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Intestinal dysbiosis in the pathogenesis of systemic lupus erythematosus and rheumatoid arthritis: an analysis of the interaction between microbiota and autoimmunity.

Intestinal dysbiosis in the pathogenesis of systemic lupus erythematosus and rheumatoid arthritis: analysis of the interaction of microbiota and autoimmunity

Intestinal dysbiosis in the pathogenesis of systemic lupus erythematosus and rheumatoid arthritis: analysis of the interaction between microbiota and autoimmunity

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SUMMARY

Introduction: The gut microbiota acts as an essential functional barrier for immune homeostasis, and its imbalance, known as dysbiosis, is strongly associated with the development of systemic autoimmune diseases. Objective: Therefore, the present study is
This is an integrative literature review aimed at analyzing the interaction between the gut microbiota and the pathogenesis of Systemic Lupus Erythematosus (SLE) and Rheumatoid Arthritis (RA). Methodology: This is an integrative literature review, conducted in the PubMed, SciELO, and Google Scholar databases, encompassing publications from the last five years. Results: The study consisted of 12 articles selected from scientific databases, applying the inclusion criteria related to the theme. Discussion: It was observed that the loss of microbial diversity leads to hyperpermeability of the epithelial barrier, allowing the passage of bacteria and their metabolites into the systemic circulation. In SLE, the increase in pathobionts, such as Ruminococcus gnavus, stands out, while in RA, the exacerbated proliferation of Prevotella copri induces pro-inflammatory pathways. In both pathologies, molecular mimicry and protein citrullination cause a breakdown in self-tolerance, inducing an imbalance in the Th17/Treg axis and generating hyperactivation of autoreactive immune responses. Final Considerations: The study confirmed the hypothesis that dysbiosis acts as a trigger in the immunopathology of SLE and RA. Microbiota control is not only a supportive treatment but also a fundamental therapeutic target capable of silencing systemic inflammation, opening avenues for advanced biomedical diagnosis and tolerance-inducing therapies.

Keywords: Autoimmune Diseases, Dysbiosis, Gastrointestinal Microbiome, Systemic Lupus Erythematosus, Rheumatoid Arthritis, and Immune Tolerance.

1 INTRODUCTION

The human gut microbiota is composed of a vast and complex community of Microorganisms that perform essential functions in maintaining health. This ecosystem It acts as a physical and functional barrier, being necessary for the development of the immune system. and ensuring protection against pathogens (Chen; Zhou; Wang, 2021). However, the balance of this Its composition can be altered by genetic factors, environmental factors, and dietary habits, leading to a imbalance (Yao *et al.,* 2023).

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This imbalance is called intestinal dysbiosis and is characterized by the loss of
The diversity of beneficial microorganisms compromises the integrity of the intestinal barrier.
Altered permeability allows bacterial components to pass into the bloodstream.
triggering chronic inflammatory responses. This process becomes a trigger for rupture.
of self-tolerance, being directly associated with the pathogenesis of several autoimmune diseases (Vojdani *et al.,* 2022; Zhou *et al.,* 2021).

Immunological self-tolerance is a fundamental process for the body to recognize
and not attack its own tissues. When the interaction between the microbiota and the immune system is
If the negative occurs, these control mechanisms fail, resulting in a loss of tolerance. Thus, the
The immune system begins to identify self-antigens as threats, initiating the process.
autoimmunity (Song; Li; Wu, 2024).

Among the pathologies influenced by this scenario, systemic lupus erythematosus stands out.
(SLE), an autoimmune and multisystemic disease. Its main characteristic is...
production of autoantibodies, which can cause damage to multiple organs and tissues in the body.
In the context of the current research, it is observed that alterations in the gut microbiota in these patients act...
as an aggravating factor, stimulating inflammatory pathways that intensify the activity and clinical course.
of the disease (Zhang *et al.,* 2021).

Similarly, rheumatoid arthritis (RA) is defined as an autoimmune disease.
A systemic, chronic, and progressive condition that primarily affects synovial joints. The pathogenesis
This condition is characterized by an exacerbated immune response that culminates in inflammation of the
synovial membrane and progressive cartilage destruction, which can evolve into conditions of
Osteoporosis. In this context, intestinal dysbiosis plays a determining role in...
compromising the functionality of the epithelial barrier. This scenario favors molecular mimicry,
which intensifies the inflammatory response and accelerates the joint damage typical of RA (Yang et al., 2024).

Given the impact of dysbiosis on the regulation of the immune system, further investigation is justified.
This study aims to gather scientific evidence linking the gut microbiome to
autoimmunity. Analyzing this relationship is important so that healthcare professionals can...
To better understand the clinical manifestations, seeking strategies to restore balance.
to reduce intestinal damage and mitigate the effects of autoimmune diseases.

This study aimed to analyze, through a literature review, the relationship
between intestinal dysbiosis and the occurrence and development of Systemic Lupus Erythematosus and
Rheumatoid Arthritis.

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2 METHODOLOGY

This study was an integrative literature review, guided by...
Question: "What is the relationship between intestinal dysbiosis and the occurrence of autoimmune diseases, such as..."
Systemic Lupus Erythematosus (SLE) and Rheumatoid Arthritis (RA)? A literature review
It occurred between September 2025 and May 2026, using a structured search strategy.
The search was conducted in the PubMed, PubMed Central, SciELO, and Google Scholar databases.
The study was conducted without language restrictions, and articles published in the last 5 years were selected.
using the following descriptors obtained from the search in the Health Sciences Descriptors
(DeCS): *Autoimmune Diseases, Dysbiosis, Gastrointestinal Microbiome, Lupus Erythematosus Systemic, Rheumatoid Arthritis ,* and *Immune Tolerance,* as well as the connectors: AND and NOT.

The selection of articles was carried out in two stages. Initially, the articles were read.
titles and abstracts of the studies retrieved to assess their relevance to the central theme and
in line with the proposed objectives. Subsequently, only the pre-selected articles had their texts published.
Complete data retrieved for in-depth analysis. The data relevant to achieving the
Objectives were extracted and organized systematically, ensuring quality and coherence.
from the information used in the synthesis of the theoretical framework.

The Inclusion Criteria (IC) and Exclusion Criteria (EC) were defined to ensure rigor.
methodological. The CI included studies that addressed the interrelationship between dysbiosis
intestinal and pathogenesis of autoimmune diseases (SLE and RA), with text available.
Complete for analysis, without language restrictions, including literature reviews and clinical trials.
However, the CE considered the articles in the database to be repeated or duplicated, the studies carried out
with animals and items that deviated from the central theme.

3 RESULTS

Based on the structured search performed in the PubMed and PubMed Central databases,
SciELO and Google Scholar initially identified 883 studies. After removing...
143 duplicates, the sample was subjected to screening by title and abstract, resulting in the exclusion of
670 articles were rejected for involving animal studies and 52 for deviating from the topic. Of the 18 articles
Of the remaining 6, 6 were excluded after full reading because they did not meet the objectives. In the end, 12
The articles were selected to comprise the corpus of analysis for this review, as detailed in
Figure 1.

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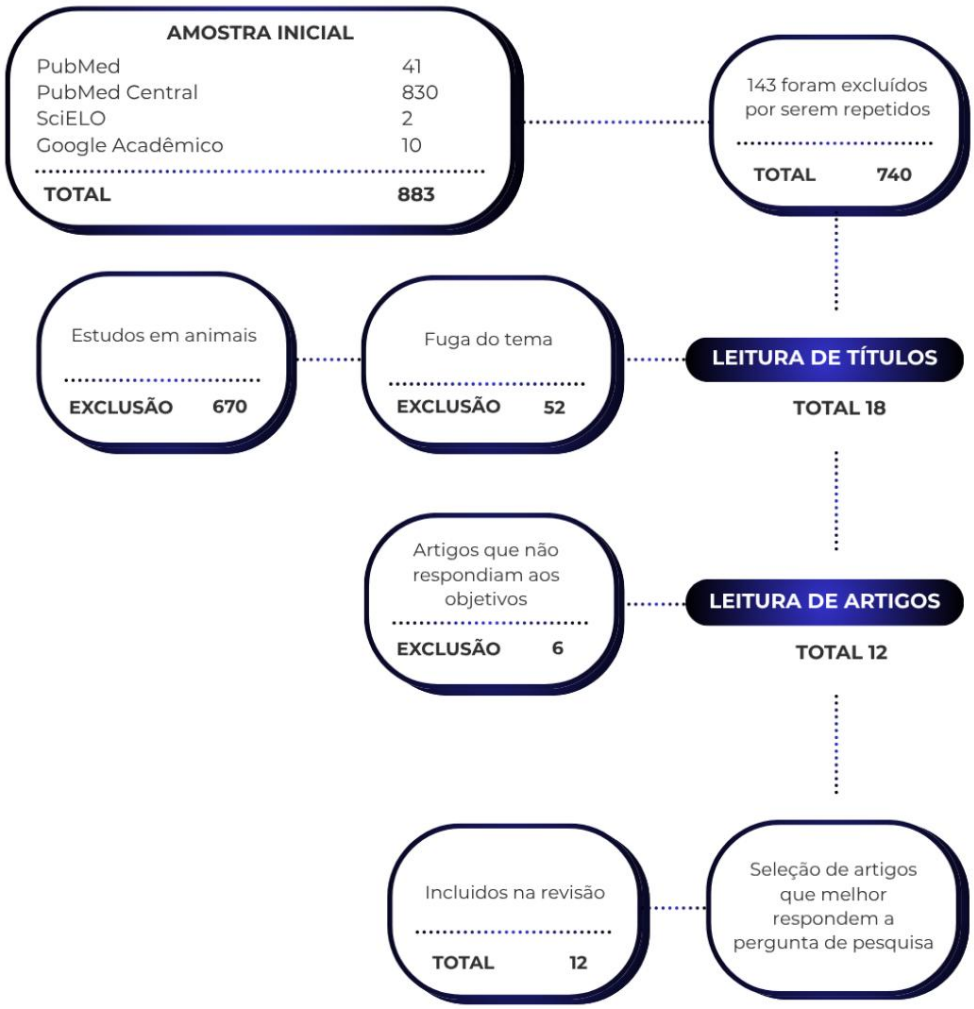
Figure 1 - Article selection flowchart, 2026.



(Source: prepared by the author, 2026)

The 12 articles selected for this integrative review are detailed in Table 1, with

The years of publication, authors, titles, and main findings related to intestinal dysbiosis in SLE.

and in AR.

Table 1 - Summary of articles included in the review.

YEAR	AUTHOR	TITLE	RESULTS
2021	Kinashi; Hase.	Partners in Leaky Gut Syndrome: Intestinal Dysbiosis and Autoimmunity.	It was shown that intestinal dysbiosis disrupts the epithelial barrier, resulting in leaky gut syndrome, which allows the passage of bacterial components and harmful antigens and contributes to the onset of autoimmune diseases, such as arthritis. rheumatoid.
2021	Zhang et al.	Gut Microbiome and Metabolites in Systemic	It was identified that the disruption of the intestinal microbiota and its alterations

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		Lupus Erythematosus: Link, Mechanisms, and Intervention	Metabolic factors contribute to the hyperactivation of the immune system and the production of autoantibodies; it highlights that metabolites derived from the microbiota interact with the host at a distance, influencing the phenotype of immune cells in SLE.
2022	Vojdani et al.	The Role of Exposomes in the Pathophysiology of Autoimmune Diseases II: Pathogens	It demonstrated that the structural similarity between pathogens and human tissues induces erroneous immune responses; it highlighted that dysbiosis and increased intestinal permeability allow the passage of components that trigger systemic inflammation and autoimmunity.
2022	Zhao et al.	Gut microbiota and rheumatoid arthritis: From pathogenesis to novel therapeutic opportunities	It was confirmed that dysregulation of the gut microbiota precedes the onset of RA and is associated with loss of immune tolerance.
2024	Balakrishnan et al.	Gut Microbiota-Immune System Interactions in Health and Neurodegenerative Diseases: Insights into Molecular Mechanisms and Therapeutics Applications	He highlighted that dysbiosis promotes the passage of metabolites and the elevated secretion of pro-inflammatory cytokines, leading to chronic systemic inflammation; he also showed that the integrity of the intestinal barrier is fundamental for regulating immunity and mitigating inflammatory processes.
2024	Macura; Kiecka; Szczepanik.	Intestinal permeability disturbances: causes, diseases, and therapy	It was confirmed that damage to the intestinal barrier allows microorganisms to pass into the tissue, activating immune cells that migrate to other organs; it was highlighted that the persistent inflammation resulting from this failure facilitates the development of systemic diseases such as arthritis. rheumatoid arthritis and lupus.
2024	Song, Li, Wu.	Involving an understanding of autoimmune mechanisms and new therapeutic strategies for autoimmune disorders	He explained that the loss of tolerance involves the aberrant activation of T and B cells; he highlighted that the induction of regulatory T cells (Tregs) and the secretion of immunosuppressive cytokines are essential to reverse autoimmunity and promote immunological anergy against autoantigens.
2024	Yang et al.	Rheumatoid arthritis and the intestinal microbiome: probiotics as a potential therapy	It was confirmed that patients with RA have an altered microbial composition, which impacts the maintenance of the immune system; it was highlighted that probiotic supplementation can restore intestinal homeostasis and modulate the systemic immune response, attenuating clinical symptoms and disease progression.
2025	Cui; Cong.	Role of Gut Microbiota in the Development of Some Autoimmune Diseases	He highlighted that mucosal barrier dysfunction allows the invasion of bacteria and metabolites that activate responses.

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			autoimmune diseases; showed that strategies for modulating the gut microbiota represent promising avenues for restoring homeostasis and immune regulation in patients with SLE and RA.
2025	Feliz, G.; Grasselli, C.	Gut microbiota and autoimmune diseases: an integrative review	The study concluded that dysbiosis is directly related to the progression of autoimmunity, possibly through molecular mimicry; it identified that strains of Lactobacillus and Bifidobacterium have therapeutic potential, while an increase in *Bacteroides* and *Prevotella* may be associated with the risk of autoimmune diseases.
2025	Mohamed et al.	The Role of probiotics in promoting systemic immune tolerance in systemic lupus erythematosus	It showed that dysbiosis in SLE induces autoimmunity through molecular mimicry and the passage of components into the body; it demonstrated that the use of probiotics stimulates the production of Treg cells and anti-inflammatory cytokines, restoring self-tolerance.
2026	Hirano *et al.*	Association between autoimmune diseases and the gut microbiome	It confirmed specific microbial signatures associated with SLE and RA; it highlighted that advances in precision therapeutic technologies will allow for a deeper understanding of the field beyond mere correlation, enabling personalized strategies for predicting, preventing, and treating immune dysregulation.

(Source: prepared by the author, 2026)

4. DISCUSSION

Studies confirm that the integrity of the intestinal barrier is central to... maintenance of immunological homeostasis. As evidenced by Kinashi and Hase (2021), the Intestinal dysbiosis can break down this epithelial protection, resulting in gut syndrome. Leaky gut. This condition occurs when an imbalance in the gut microbiota leads to a rupture. of the structures that seal the epithelium, increasing the permeability of the mucosa. This allows the Continuous displacement of antigens, toxins, and bacterial components from the lumen into the circulation. systemic. This phenomenon is corroborated by Macura et al. (2024), who describe how damage to The barrier allows the migration of harmful microorganisms and antigens into internal tissues. activating immune cells that can subsequently migrate to other organs and systems of the host.

Once permeability is compromised, the flow of substances into the circulation becomes... continuous and pathological. Balakrishnan et al. (2024) highlight that the passage of components

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Bacterial enzymes and metabolites, such as lipopolysaccharides (LPS), increase the secretion of proactive cytokines. Inflammatory, establishing a chronic state of systemic inflammation. Convergingly, Cui and Cong (2025) reinforce that this bacterial invasion is the starting point for the triggering autoimmune responses, since the presence of these elements in the bloodstream Blood pressure directly interferes with the regulation of the immune system.

Disruptions in the gut microbiota precede the clinical manifestations of diseases. autoimmune diseases and are associated with loss of immune tolerance, as argued by Zhao et al. (2022). The impact of this persistent inflammation is reflected in the failure of self-tolerance mechanisms. The process is detailed by Song, Li, and Wu (2024), who clarified how the breakdown of this tolerance... It involves the activation of T and B lymphocytes, transforming a protective response into an attack against... the host itself.

In addition to the direct inflammation caused by permeability, the structural similarity between the Bacterial antigens and host tissues worsen the clinical picture through mimicry. molecular. Vojdani et al. (2022) demonstrate that this similarity induces the immune system to commit Recognition errors activate autoreactive T cells that then attack human proteins. as if they were external pathogens. In the context of SLE and RA, this process is intensified. due to dysbiosis, which allows prolonged exposure to bacterial antigens in the bloodstream. According to detailed by Mohamed et al. (2025), this failure in self-tolerance is accompanied by a imbalance in the Th17/Treg axis, with hyperactivation of Th17 inflammatory lymphocytes and reduction severe deficiency of regulatory T cells (Tregs), which consolidates the initial trigger for autoimmunity. Systemic symptoms observed in both pathologies.

Given this specific scenario of SLE, intestinal dysbiosis acts as a factor that sustains the hyperactivation of the immune system. Hirano et al. (2026) point out that patients with lupus present specific microbial profiles, with an increase in bacteria such as Ruminococcus gnavus, This imbalance in the microbiota is directly associated with the severity of inflammation and kidney damage. It promotes the loss of self-tolerance and stimulates the production of autoantibodies against single-stranded DNA. double DNA (dsDNA), a hallmark of the disease.

Similarly, cytokine-mediated inflammatory signaling forms a link. fundamental to the chronicity of the condition. Mohamed et al. (2025) explain that high levels of Interferon type 1 (IFN-1) and interleukins, such as IL-6, are frequently observed. IL-6, Specifically, it inhibits the function of regulatory cells (Tregs), preventing the body from braking the autoimmune response. As reinforced by Zhang et al. (2021), this interaction between the Intestinal imbalance and the immune system create a persistent inflammatory environment that worsens the Clinical manifestations of SLE.

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Given this failure in self-tolerance, the modulation of the microbiota through probiotics
This presents itself as a promising therapeutic perspective. Yang et al. (2024) emphasize that the use
Lactobacillus and *Bifidobacterium* strains may help restore the balance of the axis.
Th17/Treg. Adequate supplementation stimulates the production of anti-inflammatory cytokines, such as
IL-10, and restores the population of Treg cells, essential for suppressing abnormal immune responses.
and to protect the host tissues from attack by the organism itself.

As with SLE, alterations in the gut microbiota act as a potent trigger in
Pathogenesis of rheumatoid arthritis (RA). Hirano et al. (2026) demonstrate that patients with RA
They exhibit a distinct dysbiotic microbial pattern, characterized by a significant increase in
bacteria of the genus *Prevotella,* especially *Prevotella copri.* This colonization
Disproportionate growth is often identified in the early stages of the disease and is
intrinsically associated with the activation of pro-inflammatory immune responses. The exacerbated presence
P. *copri* stimulates Th17 lymphocyte-mediated pathways, resulting in the overproduction of IL-17.
and related cytokines, which directly potentiates inflammation and progressive destruction.
of the articular cartilage that characterizes RA.

In addition to direct inflammation, impairment of the intestinal barrier acts as a facilitator.
of central processes in the immunopathology of the disease. The main events triggered by this
The failures are molecular mimicry and citrullination. As described by Macura et al. (2024), the
Loss of epithelial integrity allows opportunistic bacteria to reach the systemic circulation.
and reach distant tissues.

The point of pathogenic association lies in the ability of these pathogens to secrete the
enzyme peptidylarginine deiminase (PAD), as explained by Vojdani et al. (2022). This enzyme
The bacterium catalyzes the citrullination of the host's own proteins, modifying their structures.
originals. This structural alteration induces the immune system to make recognition errors, leading it to treat human
proteins as self-antigens. This is precisely the immune response.
sustained reaction against these citrullinated proteins, mediated by autoreactive T and B cells, which defines
The progression of RA culminates in the production of autoantibodies (ACPA) that perpetuate the destruction.
articulate.

Understanding the close relationship between intestinal permeability and rheumatoid arthritis opens doors to...
perspectives focused on the restoration of the intestinal epithelium. According to Macura et al. (2024), the reduction
of mucosal permeability and the stimulation of the production of protective metabolites, such as butyrate,
They limit the passage of opportunistic bacteria into the systemic circulation. Consequently, the
Restoring this physical barrier attenuates the systemic release of the PAD enzyme, interrupting
It inhibits the citrullination cascade and slows the cycle of joint destruction.

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Although the pathophysiology of SLE and RA involves complex inflammatory cascades, the Recognition of the gut microbiota as a therapeutic target has driven new approaches. clinical. Zhao et al. (2022) highlight the impact of conventional pharmacological management of RA, such as the use of disease-modifying antirheumatic drugs (DMARDs), like methotrexate, highlighting a strong relationship with the microbiome. The authors explain that gut bacteria not only do they undergo changes in their composition due to the action of these drugs, but also They influence the bioavailability of compounds and can act as predictors of clinical response. From patient to treatment.

To directly address this imbalance, the use of probiotics has become established as... a promising biomedical approach. Feliz (2025) highlights the importance of colonization of mucosa by strains of the genera *Lactobacillus* and *Bifidobacterium,* which demonstrate efficacy in Rebalancing of the mucosa and immune response. Further exploring this mechanism, Yang et al. (2024) They explain that these beneficial microorganisms compete for space with pathobionts and modulate actively suppresses the immune system, attenuating the differentiation of inflammatory Th17 lymphocytes and promoting the function of regulatory T cells (Tregs).

In addition to supplementation, more in-depth interventions, such as microbiota transplantation, are also possible. Fecal microtransactions (FMT) demonstrate the direct impact of the gut on autoimmunity. According to Cui According to Cong (2025), FMT has proven to be an intervention capable of remodeling the intestinal ecology. both in cases of SLE and RA. By replacing a dysbiotic flora with a healthy one, An increase in short-chain fatty acid (SCFA)-producing bacteria is observed, along with repair. of the epithelial barrier and mitigation of systemic inflammation, which proves that the control of The microbiota alters the course of the disease.

The ultimate goal of all these intestinal interventions is to correct the primary error of the diseases. autoimmune diseases: the loss of self-tolerance. Song, Li, and Wu (2024) reinforce that the great challenge of New therapies aim to induce antigen-specific tolerance, that is, to "teach" the immune system to stop to attack the body's own tissues without causing widespread immunosuppression in the patient. In this way, It is evident that ensuring microbiota homeostasis is not just a supportive treatment, but also a fundamental way to restore immune regulation and silence the self-reactive cells.

FINAL CONSIDERATIONS

This study showed that gut dysbiosis plays a determining role. and acts as an essential trigger in the pathogenesis of systemic autoimmune diseases, such as lupus.

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Systemic erythematosus (SLE) and rheumatoid arthritis (RA). The loss of microbial diversity and the
The resulting impairment of the epithelial barrier leads to intestinal hyperpermeability.
allowing the continuous passage of bacterial antigens and metabolites into the systemic circulation.
This event triggers a chronic inflammatory state which, associated with mechanisms such as
Molecular mimicry and citrullination culminate in the breakdown of immunological self-tolerance and in
hyperactivation of autoreactive T and B cells.

In this way, it was demonstrated that both pathologies present microbial signatures.
specific factors that directly influence clinical severity. Increased colonization by
Pathobionts in the intestinal environment correlate with exacerbated inflammation and act in induction.
of pro-inflammatory pathways, especially those mediated by Th17 lymphocytes. In both scenarios, the
An imbalance in the Th17/Treg axis throws the immune system off its homeostatic state and
It perpetuates the attack on the host's own tissues.

The importance of studying these interactions lies in the need to understand how the
The gut can influence the treatment of these diseases. How is the diagnosis and management of SLE and...
The challenges associated with rheumatoid arthritis (RA) remain in clinical practice; investigating the microbiota emerges as a way forward.
promising for biomedicine. Understanding these mechanisms is fundamental for development.
new forms of diagnosis and treatment in the field of clinical analysis.

Given this evidence, it can be concluded that the gut microbiome is not just a marker.
passive, but also a highly promising therapeutic target in biomedicine. Strategies of
Direct modulation of the microbiota, such as probiotic supplementation and transplantation.
Fecal Microbiota (FMT) are shown to be fundamental not only for symptomatic control, but also
also for restoring the intestinal barrier and retraining the immune system.

Therefore, advancing our understanding of the gut-immune axis is essential for the transition.
from a generalized immunosuppression model to precision therapies, aiming at the induction of
Antigen-specific tolerance. It is suggested that future research explore laboratory applications.
of biomarkers derived from the microbiota for early diagnosis, as well as the performance of
robust clinical trials that consolidate the use of nanotherapies and gut modulators such as
Standardized and safe practices in the management of autoimmune patients.

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