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The role of gut microbiota in the development of autoimmune disease - a literature review

1. Karolina Baran [KB]
Independent Public Health Care Facility of the Ministry of Internal Affairs and Administration in Krakow, Kronikarza Galla 25 Street, 30-053 Krakow: Cracow, Malopolska,
https://orcid.org/0009-0004-1627-5065
E-mail: 99barankarolina@gmail.com

2. Marlena Jankowska [MJ]
Stefan Zeromski Specialised Hospital (SP ZOZ) in Cracow: Cracow, os. Na Skarpie 66 Street 31-913 Kraków, Małopolska, Poland, PL
https://orcid.org/0009-0005-2240-8853
E-mail: marlena.rosol@wp.pl

3. Natalia Jańczyk [NJ]
Stefan Zeromski Specialised Hospital (SP ZOZ) in Cracow: Cracow, os. Na Skarpie 66 Street 31-913 Kraków, Małopolska, Poland, PL
https://orcid.org/0009-0000-1862-9681
E-mail: nataliajanczyk34@gmail.com

4. Karolina Mędrysa [KM]
Voivodeship Hospital in Bielsko-Biała,
Armii Krajowej Street 101, 43-316 Bielsko-Biała
https://orcid.org/0009-0002-1557-0244
E-mail: karolinamedrysa@gmail.com

5. Jakub Jan Pokrzepa [JP]
5th Military Hospital with Polyclinic in Cracow
Wrocławska 1-3 Street, 30-901 Krakow; Cracow, Malopolska,
https://orcid.org/0009-0000-7907-1511
E-mail: jakub.pokrzepa@onet.pl

6. Michał Presak [MP]
5 Military Clinical Hospital SPZOZ in Cracow
Wrocławska 1-3 Street, 30-901 Krakow; Cracow, Malopolska,
https://orcid.org/0009-0006-0335-5917
E-mail: michal.presak@gmail.com

7. Gabriela Blecharz [GB]
5 Military Clinical Hospital SPZOZ in Cracow
Wrocławska 1-3 Street, 30-901 Krakow; Cracow, Malopolska,
https://orcid.org/0009-0007-9275-3913
E-mail: blecharzgabriela@gmail.com

8.Julia Szwech [JS]
5 Military Clinical Hospital SPZOZ in Cracow
Wrocławska 1-3 Street, 30-901 Krakow; Cracow, Malopolska,
https://orcid.org/0009-0006-3792-0101
E-mail: julia.szwech@op.pl

9. Mikołaj Pograniczny [MPG]
Andrzej Frycz Modrzewski Kraków University: Cracow, Gustawa Herlinga-Grudzińskiego 1 Street 30-705 Kraków, Małopolska,Poland,PL
https://orcid.org/0009-0009-8407-3605
E-mail: m.pograniczny@gmail.com

10. Adrianna Mielżyńska [AM]
Andrzej Frycz Modrzewski Kraków University: Cracow, Gustawa Herlinga-Grudzińskiego 1 Street 30-705 Kraków, Małopolska,Poland,PL
https://orcid.org/0009-0006-7359-4796
E-mail: adrianna.mielzynska.03@gmail.com

Abstract

The gut microbiota plays a crucial role in maintaining immune balance, and its dysregulation has been increasingly linked to the development of autoimmune diseases. This review summarizes evidence connecting microbial alterations to conditions such as type 1 diabetes, rheumatoid arthritis, multiple sclerosis, and inflammatory bowel disease. Changes in microbial diversity and the overgrowth of pro-inflammatory species appear to trigger immune pathways that drive disease progression. Therapeutic strategies aimed at restoring gut balance, including probiotics, prebiotics, fecal microbiota transplantation (FMT), and dietary and lifestyle interventions, show promise in modulating immune responses and improving outcomes. However, despite encouraging results, significant gaps remain regarding the causality of these associations, the long-term safety of interventions like FMT, and the need for individualized therapeutic approaches. Advances in microbiome research, including

metagenomic profiling, offer hope for developing personalized treatments. Further studies are necessary to better understand the complex interactions between the gut microbiota and the immune system in autoimmune diseases.

Key words:
gut microbiota, autoimmune disease, gut-associated lymphoid tissue (GALT), dysbiosis, fecal microbiota transplantation (FMT), probiotics, prebiotics,

1. Introduction

The human gut microbiota is a complex system consisting of bacteria, viruses, fungi and aechaea. It is essential for maintaining homeostasis in the body. Recent research increasingly focuses on the correlation between gut microbiota imbalances and autoimmune disorders. Autoimmun diseases arise as a result of a dysregulated immune response, leading to the immune system mistakenly attacking its own tissues. The microbiome is now recognized as a key factor influencing immune system function, either promoting immune tolerance or triggering autoimmune responses [1].
Given the growing interest in microbiome research, understanding how gut microbiota interacts with the immune system is essential. This study aims to explain the concepts of microbiota, microbial imbalances (dysbiosis) and autoimmune diseases, and to provide an insight into their correlations based on current evidence.

2. Gut Microbiota Composition and Function

2.1 Normal Gut Microbiota Composition

The gut microbiota is present at four major colonization sites in the human body: the oral cavity, gut, vagina and skin. Among these the main focus of recent scientific interest is the gut microbiota. Although it is composed of 150-170 bacterial species, it primarily consists four dominant bacterial phyla: Firmicutes (90 % of total population), Bacteroidetes, Actinobacteria, and Proteobacteria [1][2]. These bacteria contribute to various physiological functions, including virtually the entire nutrient metabolism, maintenance of gut barrier integrity and modulation of immune responses. Even though the microbiota contains microorganisms that can cause inflammation, it is the healthy microbiota that ensures a balanced immune environment by regulating pro-inflammatory and anti-inflammatory mechanisms [3].

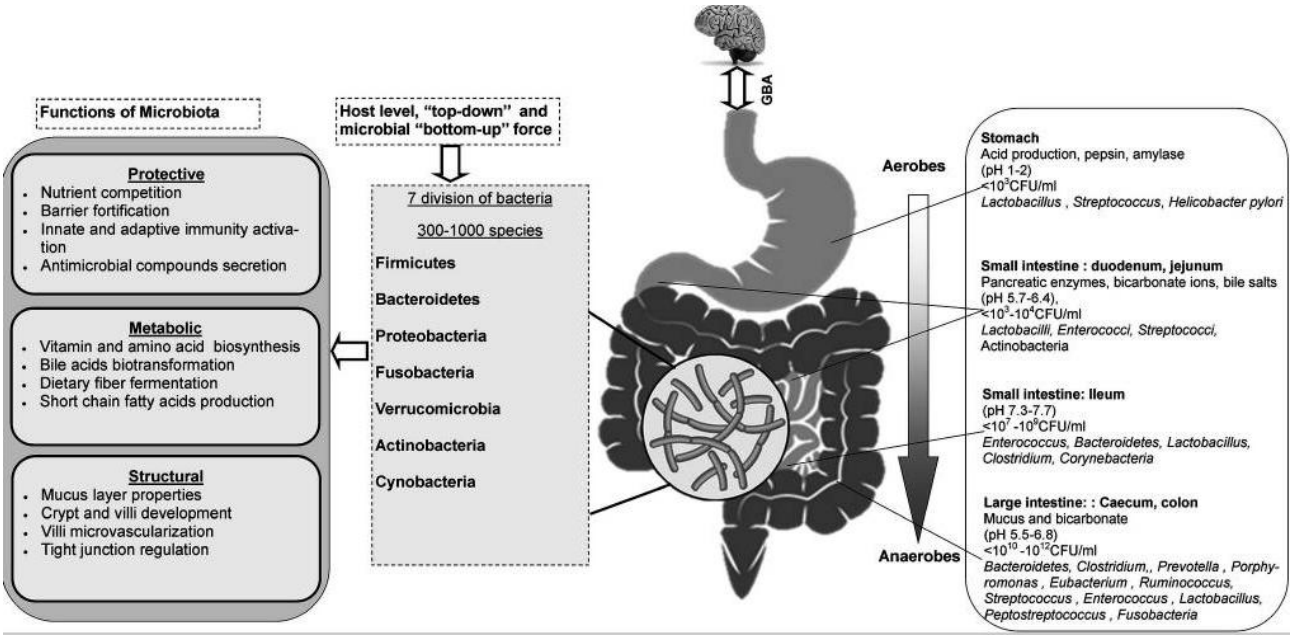

Fig. 1. Distribution of normal gut flora in different parts of intestine and its functional activities and GBA. *GBA* gut brain axis [1].

2.2 Factors Influencing Gut Microbiota

Several factors shape the composition of gut microbiota:

- **Diet:** Dietary patterns play a major role in shaping our gut microbiota. A diet rich in fiber promotes the growth of beneficial bacteria such as *Bifidobacterium* and *Lactobacillus*, which leads to production of short-chain fatty acid and gut health. In contrast, a „Western diet" rich in saturated fats, simple sugars, and low in fiber is associated with reduced microbial diversity and the expansion of pro-inflammatory bacteria, contributing to dysbiosis and increased risk of metabolic diseases.[4].

- **Genetics:** Our genes also have a say in which bacteria live in our gut. Research shows that identical twins have more similar microbiomes than fraternal twins, suggesting that genetic makeup partly shapes the gut microbial community.[5]

- **Antibiotics:** While antibiotics are important for fighting infections, they can seriously disturb the gut microbiota. They often wipe out both harmful and beneficial bacteria, sometimes leading to long-term changes, making it harder for the gut to return to a healthy state. [6].

- **Early-Life Exposure:** The first few months of life are critical for gut microbiota development. Babies born vaginally pick up bacteria from their mother's birth canal, while those delivered by cesarean section are colonized mainly by skin microbes[7]. Breastfeeding further supports healthy microbiota growth by providing beneficial bacteria and important nutrients that help shape the developing immune system [8].

3. Immune System and Gut Microbiota Interaction

3.1 Gut-Associated Lymphoid Tissue (GALT)

To understand the association between immune system and gut microbiota it is crucial to understand GALT - gut-associated Lymphoid Tissue. It is the key factor of close communication between immune system ang gut microbiota. GALT is made up of structures like Peyer's patches, mesenteric lymph nodes, and immune cells in the lamina propria, which work together to recognize the difference between harmful intruders and helpful microbiota ingredients. [9] Its main focus is to detect dangerous bacteria in the intestine and to trigger immune response when it is needed. It happens through specialized cells called M cells which transport microbes from guts to immune system. Then there's a key moment of immune reaction - time when immune system make a decision: whether to stay quiet - tolerate microbes or to trigger defensive reaction. When it overreacts, that's when an autoimmune reaction starts. [10]

3.2 Mechanisms of Immune Regulation

There is a lot of complicated mechanisms involved in maintaining gut microbiota and immune system balance left. Below we discuss the most critical process involved in maintaining immune homeostatsis.

T-regulatory (Treg) cells

T-regulatory (Treg) cells are one of the key elements of this communication. They have the ability to suppress the excessive immune response - in other words the ability to prevent the body from attacking their own tissues. There are several species of gut bacteria, especially *Clostridia* and *Bacteroides* types that were proven to drive the expansion of Treg populations, in the same time helping to maintain immune homeostatsis. [11]

T-helper 17 (Th17) cells

Other equally important cells present in this immune response are T-helper 17 cells which are essential for defending intestinal cells against pathogens. Unfortunately, however, if they become over-activated, they can trigger an autoimmune reaction, e.g. inflammatory bowel disease (IBD) or multiple sclerosis (MS). Animal studies have shown that imbalanced microbiota promotes their over-stimulation [12].

Pattern Recognition Receptors (PRRs)

Pattern Recognition Receptors (PRRs), such as Toll-like receptors (TLRs) and NOD-like receptors (NLRs), are another critical components of the complex network. Their task is to recognize molecules known as pathogen-associated molecular patterns (PAMPs) and to

activate and initiate appropriate responses of immune system. Here it is all about balance, because too weak PRRs reaction may result in failure to recognize the pathogen and too weak defense reaction, while a reaction that is too strong will result in chronic activation, which will result in inflammation and an autoimmune response. [13]

Short-chain fatty acids (SCFAs)

Lastly, **short-chain fatty acids (SCFAs)** like acetate, propionate, and butyrate, which are produced during bacterial fermentation of dietary fibers, have a significant impact on the immune system. They supportive integrity of gut barrier, meaning also regulation of inflammatory processes and directly promote the differentiation of Treg cells. In summary, they help maintain immune tolerance and prevent their too intensive activation. Balanced intestinal microbiota is essential for the production of SCFAs.[14].
All of these mechanisms described above work together to protect the delicate structure of the intestinal microbiota

4. Dysbiosis and Autoimmune Diseases

4.1 Defining Dysbiosis

Dysbiosis refers to disruptions in the composition and function of the gut microbiota, typically characterized by an overgrowth of potentially harmful microorganisms, a reduction in beneficial bacteria, and an overall loss of microbial diversity. This imbalance is currently associated with decreased immune tolerance, the initiation of chronic inflammation, and the development of autoimmune processes. [15]

4.2 Role of Increased Intestinal Permeability ("Leaky Gut”)

The intestinal barrier, formed by the intestinal endothelium, is responsible for preventing the translocation of pathogens to the blood stream and maintaining immune homeostasis. Damage of this barrier, commonly referred to as "leaky gut," allows bacterial components such as lipopolysaccharides (LPS) as well as other normally excluded substances (e.g., proteins, gluten, microbes, and food antigens) to cross into systemic circulation, resulting in inflammation that may trigger an array of autoimmune diseases in the course of the processes described below.

4.3 Molecular Mimicry, Bystander Activation, and Cross-Reactivity

One of the most important processes in the etiology of autoimmune diseases is molecular mimicry. In this process, certain microbial antigens closely resemble the body's own proteins.

As a result, the immune system may mistake self-tissues for foreign invaders and attack them. It turns out that this mechanism is one of the basic ones involved in the formation of conditions like type 1 diabetes and multiple sclerosis.

Another path that infection contributes to autoimmunity is bystander activation. During an infection, the immune system responds aggressively, releasing a range of cytokines and other signaling molecules. When this mechanism is constantly activated, it can cause nearby, auto-reactive immune cells to become inadvertently activated, thereby damaging healthy tissue.

Lastly, cross-reactivity between microbial molecules and host tissues highlights how crucial a stable relationship with the gut microbiota is for immune regulation. Disruptions in the microbial balance, often referred to as dysbiosis, may make the immune system more prone to these kinds of errors, increasing the risk of autoimmune reactions. [17][18]

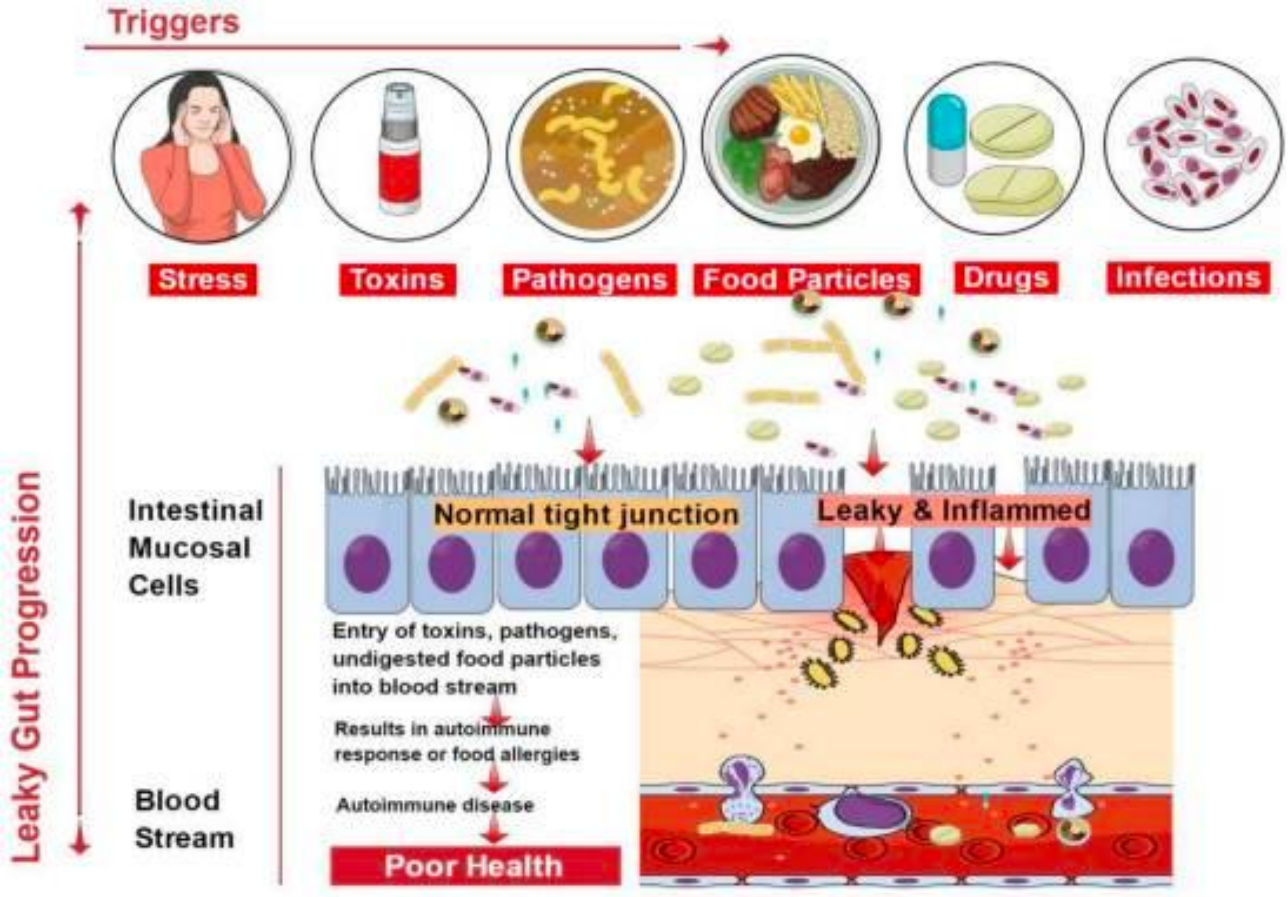

Fig. 2. Factors contributing to the development of leaky gut and its relationship to autoimmune diseases.[16]

5. Microbiota in Specific Autoimmune Diseases

5.1 Type 1 Diabetes (T1D)

Recent studies suggest that changes in the gut microbiota may occur even before the clinical symptoms of type 1 diabetes become evident.Several studies have shown gut dysbiosis, or rather an overrepresentation of pro-inflammatory bacterial species, in people at risk for developing type 1 diabetes. It is suspected that it is this dysregulation in the microbiota that contributes to the activation of immune pathways that attack the beta cells of the pancreas. Over time, this autoimmune attack leads to the progressive destruction of the cells responsible for insulin production, which ultimately leads to the onset of the disease. [19]

5.2 Rheumatoid Arthritis (RA)

The connection between gut health and joint inflammation is increasingly recognized through the concept of the gut-joint axis. Evidence suggests dominance of *Prevotella copri* in the microbiota of patients with early-stage rheumatoid arthritis. It is thought that the presence of this particular bacteria may point immune responses toward a pro-inflammatory state, thereby facilitating the development or exacerbation of arthritis symptoms. While the exact mechanisms are still being unknown, changes in gut flora appear to play a key role in modulating the immune system's attack on joint tissues.[20]

5.3 Multiple Sclerosis (MS)

In multiple sclerosis, the gut microbiota seems to have a significant influence on neuroinflammatory processes. Studies have shown that dysbiosis can disrupt the delicate balance between regulatory and inflammatory immune cells, cousing a shift toward chronic inflammation which results in pathogenic autodestructive process of myelin. Certain bacterial strains have been implicated in amplifying immune responses that target the central nervous system, potentially worsening disease progression. On the other side, the presence of beneficial microbes may help suppress inflammation, which allow us to believe that the gut-brain connection could offer new therapeutic targets for MS. [21]

5.4 Inflammatory Bowel Disease (IBD)

Both Crohn's disease and ulcerative colitis, the two main forms of inflammatory bowel disease, show a strong link to disturbances in gut microbial composition. Typically, patients with IBD exhibit a marked decrease in beneficial bacteria such as *Faecalibacterium prausnitzii*, which is known for its anti-inflammatory properties, alongside an increase in potentially harmful species. This microbial imbalance disrupts intestinal homeostasis,

weakens the gut barrier, and perpetuates cycles of inflammation that drive disease progression and symptom severity. [22]

5.5 Other Autoimmune Diseases

A growing body of research suggests that gut dysbiosis may also contribute to the pathogenesis of various other autoimmune conditions. In systemic lupus erythematosus (SLE), alterations in the gut microbiome are associated with heightened systemic inflammation and immune dysregulation. In Hashimoto's thyroiditis, imbalances in gut bacteria may influence the immune system's attack on thyroid tissue, exacerbating disease severity. Similarly, in celiac disease, a disrupted microbiota can intensify gluten sensitivity, leading to more severe intestinal damage and a heightened autoimmune response. [23]

6. Therapeutic Implications

6.1 Probiotics and Prebiotics

Probiotics, which are beneficial strains of bacteria, and prebiotics, non-digestible fibers that serve as nourishment for gut microbes, have attracted considerable attention as potential tools for managing autoimmune diseases. Numerous studies suggest that supplementation with specific probiotic strains may help rebalance gut microbiota composition, enhance intestinal barrier integrity, and regulate immune responses. For instance, some *Lactobacillus* and *Bifidobacterium* species have been shown to reduce systemic inflammation and support immune tolerance. Similarly, prebiotic compounds such as inulin or fructooligosaccharides promote the growth of beneficial commensals, indirectly modulating immune system activity. Although clinical outcomes vary depending on the individual and disease context, the general consensus highlights the importance of gut microbial health in maintaining immune homeostasis. [24] [25]

6.2 Fecal Microbiota Transplantation (FMT)

Fecal Microbiota Transplantation (FMT), the process of transferring stool-derived microbiota from a healthy donor to a recipient, has emerged as a promising therapeutic option, particularly in conditions associated with severe dysbiosis. Originally developed as a treatment for recurrent *Clostridioides difficile* infections, FMT is now being explored for its potential to recalibrate the gut ecosystem in autoimmune diseases. Early clinical trials and animal studies have indicated improvements in disease markers and symptom severity in disorders such as ulcerative colitis and multiple sclerosis following microbiota transfer. Nevertheless, important questions regarding the durability of these effects, optimal donor selection, and long-term safety remain unresolved. Consequently, FMT is considered an

experimental but intriguing avenue for restoring immune balance through the modulation of gut microbiota. [26]

6.3 Diet and Lifestyle Interventions

Dietary and lifestyle factors exert a profound influence on gut microbial composition and function, with significant downstream effects on immune regulation. Diets rich in dietary fibers, polyphenols, and fermented foods are associated with increased microbial diversity and an enhanced abundance of anti-inflammatory species. Conversely, high-fat, high-sugar Western diets tend to foster dysbiosis and promote chronic low-grade inflammation. Incorporating foods like vegetables, legumes, whole grains, and fermented products such as yogurt and sauerkraut can encourage the growth of beneficial microbes, strengthen the intestinal barrier, and modulate immune responses. Beyond diet, regular physical activity, stress management, and sufficient sleep have all been shown to support a healthy microbiome, further underscoring the importance of holistic lifestyle strategies in autoimmune disease management. [27]

6.4 Future Directions

The rapid advancement of microbiome research paves the way for highly personalized therapeutic approaches. Emerging evidence suggests that tailoring interventions based on an individual's unique microbial profile may maximize treatment efficacy while minimizing adverse effects. Techniques such as metagenomic sequencing allow for detailed characterization of microbial communities, offering insights into specific dysbiotic patterns associated with particular autoimmune conditions. Future therapies may involve custom-designed probiotics, precision dietary recommendations, or even targeted microbiota modifications through bacteriophage therapy. While these strategies remain largely in the experimental stage, they represent a promising frontier in the quest for more effective and individualized autoimmune disease treatments.

7. Conclusion

7.1 Summary of Key Findings

This review highlights the significant role of gut microbiota in the development and progression of autoimmune diseases. Alterations in microbial diversity and composition — often characterized by a reduction in beneficial species and an increase in pro-inflammatory bacteria — are closely linked to conditions such as type 1 diabetes, rheumatoid arthritis, multiple sclerosis, inflammatory bowel disease, and other autoimmune disorders. Therapeutic strategies aimed at restoring microbial balance, including probiotics, prebiotics, fecal microbiota transplantation, and lifestyle interventions, show promising potential in modulating immune responses and

improving disease outcomes. Advances in personalized microbiome-based treatments also present new opportunities for the targeted management of autoimmunity.

7.2 Gaps in Current Research

Despite substantial progress, several gaps remain. The causal relationships between specific microbial shifts and autoimmune disease onset are not yet fully understood. Many clinical trials exploring microbiota-targeted therapies are still in early stages, with variability in outcomes and a lack of standardized protocols. Additionally, questions regarding the long-term safety and efficacy of interventions like FMT persist. More research is needed to identify precise microbial markers for disease prediction and to develop personalized, reliable therapeutic approaches tailored to individual microbiome profiles.

7.3 Final Thoughts

Growing evidence emphasizes the pivotal influence of gut microbiota on immune regulation and autoimmune disease pathogenesis. Although therapeutic manipulation of the microbiome offers a promising avenue, further research is essential to translate these insights into effective, standardized clinical practices. A deeper understanding of host-microbe interactions, combined with technological advances in microbiome analysis, holds the potential to revolutionize the prevention, diagnosis, and treatment of autoimmune diseases in the near future.

Authors' Contributions Statement:

Conceptualization: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Data Curation: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Formal Analysis: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Investigation: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Methodology: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Project Administration: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Resources: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Software: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Supervision: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Validation: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Visualization: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
Writing -original Draft: [KB][MJ][NJ][KM][JP][MP][GB][JS][MPG][AM]
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