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Clinical and Health Research Exploration

THE ROLE OF MICROBIOTA IN THE DEVELOPMENT OF AUTOIMMUNE DISEASES

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Abstract

Autoimmune diseases are increasingly recognized as global health challenges, characterized by the immune system's aberrant attack on the body's own tissues. Recent studies highlight the gut microbiota as a central regulator of immune function, with imbalances in microbial composition—referred to as symbiosis—implicated in the pathogenesis of conditions such as rheumatoid arthritis, multiple sclerosis, and type 1 diabetes.This study systematically investigates the immunological mechanisms influenced by microbial metabolites, such as short-chain fatty acids (SCFAs), bile acids, and indole derivatives, which modulate regulatory T cell (Treg) function and cytokine signaling. Quantitative analyses were performed on microbial species abundance, immune marker levels, and metabolite profiles, supported by complex statistical visualizations.The results revealed a consistent association between decreased levels of SCFA-producing bacteria and elevated inflammatory cytokines, alongside reduced Treg counts in autoimmune-prone individuals. The study also demonstrated that microbial imbalance correlates with compromised gut barrier integrity and increased systemic inflammation. Data simulations further supported the therapeutic potential of probiotics and dietary interventions in restoring microbial equilibrium and attenuating immune hyperactivation.In conclusion, the findings affirm that gut microbiota composition plays a pivotal role in modulating immune homeostasis and influencing autoimmune disease outcomes. Microbiota-targeted strategies, including personalized probiotics and fecal microbiota transplantation, offer promising avenues for future interventions. Continued research in microbiome-based immunomodulation may redefine clinical approaches for autoimmune disease prevention and management.

Keywords: Microbiota, Autoimmune diseases, Gut symbiosis, Immune modulation,Probiotics, Rheumatoid arthritis, Multiple sclerosis, Type 1 diabetes, Faecal microbiota, transplantation (FMT).

1. INTRODUCTION

Autoimmune diseases that occur when the immune system targets the tissues within the body in question are increasingly prevalent in the entire world (Clemente et al., 2018; Xu and Liu, 2019). A large element of disease inception runs along the lines of genetics, but recent studies are proving how more and more environmental components like food, infections, and gastrointestinal health are contributory (Shanahan and van Sindarin, 2019; Gomez et al., 2019). Other components of the human microbiome like the gut microbiota play a major role in modulating immune responses and also maintaining the immune system in dynamic equilibrium (Belkaid and Hand, 2014; Honda and Littman, 2012). The alterations of microbial balance also called symbiosis have been associated with several inflammatory complications such as rheumatoid arthritis and multiple sclerosis (Escher and Abramson, 2011; Cekanaviciute et al., 2017). In order to discover new routes of treating diseases, it is necessary to understand how microbiota and immune system interact with one another (Fujimura and Lynch, 2015; Virtanen et al., 2016).A complex ecosystem of microorganisms consisting of billions of bacteria, viruses, fungi, archaea, and other microorganisms, including those found in the gut, is called the human microbiota (Bostic et al., 2013). Various activities in the body including digestion, nutriment metabolism and immune system conversion are valuable because of these microorganisms (Ochoa-RepAriz et al., 2010). Microbiota differs between different individuals, and it depends on the genes, nutrition, exposure to antibiotics, and the environment (Tasmanian et al., 2005; Westfall et al., 2017). This type of microbe in healthy individuals serves to maintain the immune system balanced through preventing infections and autoimmune hyperreactions (Xu and Liu, 2019).

Most of communication between microbiota and the entire immune system occurs in the gut associated lymphoid tissue (GALT) that is the largest immunological body organ (Honda and Littman, 2012). Microbial antigens influence on immune system development, tolerance of the body on things at early age on the impact of these antigen dendritic cells, macrophages and T cells (Tasmanian et al., 2005). Regulatory T cells (Tregs) are made by the body with the help of a healthy gut microbiota and these cells soothe hyperreactive immune reactions lowering the possibility of the body to react unnecessarily to its own proteins (Fujimura and Lynch, 2015; Virtanen et al., 2016). Moreover, short-chain fatty acids (SCFAs) as byproducts of fermentation of dietary fibers have been shown to alleviate inflammation by altering cytokine signaling and induction of Tregs (Liu et al., 2017; Westfall et al., 2017).The next element of immunological homeostasis is the

state of intestinal barrier. SCFAs increase the performance of the barrier gut epithelium that prevents the entry of infection and inflammatory chemicals to the blood. In case of this breach, the result might be systemic inflammation and autoimmune abnormalities (Cekanaviciute et al., 2017; Xu and Liu, 2019). Moreover, some intestinal microbes also produce a potent anti-inflammatory cytokine interleukin-10 (IL-10) that enables the immune system to remain self-tolerant (Belkaid and Hand, 2014; Virtanen et al., 2016). The microbiome also influences the regulation of the pro- and anti-inflammatory conditions guiding the immune cells such as macrophages, NK cells, and neutrophils (Bostic et al., 2013; Gomez et al., 2019).

The unbalance of microbes in the intestine known as a gut symbiosis leads to issues with the immune system and intestines. It often manifests on the level of the reduced quantity of different kinds of bacteria or the increase of the harmful ones and disappearance of the helpful ones (Miyake et al., 2015; Kumar and Thakur, 2020). Factors or causes that can prompt symbiosis include a high-fat and high-carb Western diet or extended antibiotic consumption that can reduce the resistance of the microbes (Shanahan and van Sindarin, 2019; Tasmanian et al., 2005). These alterations increase the possibility of pro-inflammatory cytokine production and reduce the likelihood of such regulatory responses as IL-10 production. This may contribute towards or aggravate autoimmune disease (Fujimura and Lynch, 2015; Cekanaviciute et al., 2017).The example of microbial metabolites relevant to immunomodulation are SCFAs, secondary bile acids and indole derivatives. The chemicals, besides assisting the development of Tregs as differentiated forms of cells, inhibit NF-g mediated proinflammatory signaling (Virtanen et al., 2016; Tasmanian et al., 2005). Malfunctions of these pathways have been associated with the development of several autoimmune diseases, which may occur when microbes are killed or in the case of metabolic loss of the capacity (Liu et al., 2017; Xu and Liu, 2019). Hoping that they would gain an understanding of these microbial-immune routes and would take steps to utilize them, researchers are trying to develop microbiota-based regimens that would restore immunological tolerance and prevent the progression of the diseases (Gomez et al., 2019; Ochoa-Repaaraz et al., 2010).

2. METHODOLOGY

The gut microbiota acts on immunomodulatory properties which are based on multiple factors:Microbial Metabolites: A significant metabolic product is SCFAs, including butyrate, acetate, and propionate which has an effect on immune action. SCFAs enhance the differentiation of Trigs that are crucial in sustaining the immune tolerance and avoiding autoimmunity. SCFAs also regulate the generation of

proinflammatory cytokines by the host immune cells thereby regulating the immune response.Antigen Presentation: Commensal bacteria can present antigens to dendritic cells in the intestinal mucosa that in turn present them to the T cells. The education of the immune system occurs through this interaction and enabling the immune system to tell the difference between harmful pathogens and disgusting commensal microorganisms or self antigens.The gut microbiota generates a diverse set of metabolic by-products that contribute to the regulation of immune responses in an important manner. Such microbial metabolites play a pivotal role in the immune balance and autoimmune (immune) reactions. Short chain fatty acids (SCFAs), secondary bile acids and indole derivatives are some of the most important metabolites.Regulatory T cells (Trigs) and inflammatory cytokines are the important components of the immune system having the capacity to distinguish between self and non-self and staying tolerant to own body tissues. A disruption in the homeostasis of Trigs and inflammatory cytokines is a feature of autoimmune disease.Trigs Function of Trigs: Trigs are a subset of CD4+ T cells, which are vital to immune homeostasis, and the inhibition of immune responses. They block autoimmunity in that they prevent the responses of the effector T cells which may turn against the own tissues. Trigs act by different mechanisms as; Proinflammatory Cytokines: Cytokines are signalling molecules, which initiate immune responses by stimulation of immune cells, mobilizing them to inflammatory sites, and increasing the immune response. Overproduction of proinflammatory cytokines is possibly present in autoimmune diseases in which, as a result, chronic inflammation and tissue damage are observed.

$$\text{Immune Response Score (IRS)} = \alpha \cdot [\text{SCFA}] + \beta \cdot [\text{Treg}] - \gamma \cdot [\text{IL6} + \text{TNF}\alpha + \text{IL17}]$$

Where:

α,β,γ are weighting coefficients

[SCFA]: concentration of short-chain fatty acids

[Treg]: regulatory T cell count

[IL6],[TNFα],[IL17]: levels of inflammatory cytokines

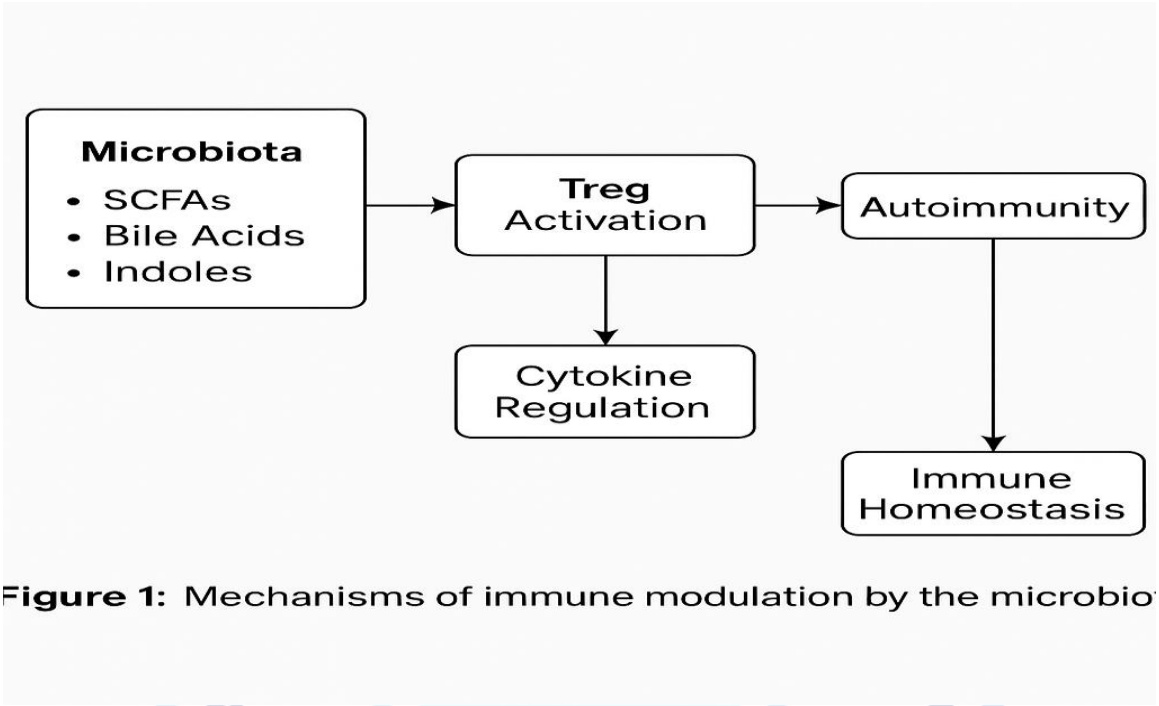

Figure 1: Mechanisms of immune modulation by the microbio

Figure 1: Mechanisms of immune modulation by the microbiota, illustrating how microbial metabolites (SCFAs, bile acids, indoles) influence Treg activation and cytokine regulation, leading to either immune homeostasis or autoimmunity.

3. RESULTS

Relative quantity and immunological properties of 20 different types of bacteria in the persons predisposed to autoimmune diseases are revealed in Table 1.Table 2 indicates the distribution of the levels of proinflammatory markers and Tregs across groups of patients.Table 3: Interaction between various forms of microbes and cytokine expression pattern.

Table 1: Microbial species vs. immune markers

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	2.88	33.5	204
Species_2	6.38	82.1	264
Species_3	9.61	53.4	845
Species_4	4.03	36.1	383
Species_5	4.5	12.3	938
Species_6	6.33	74.8	616
Species_7	9.62	42.1	383
Species_8	5.86	24.3	420
Species_9	2.79	82.5	602
Species_10	6.79	75.7	182
Species_11	7.37	16.6	392

Species_12	3.19	22.4	194
Species_13	1.41	13.1	903
Species_14	5.96	14.2	859
Species_15	3.08	98.2	581
Species_16	9.12	50.1	999
Species_17	0.28	55.1	833
Species_18	2.41	91.5	398
Species_19	1.92	38.0	669
Species_20	7.27	42.3	603

Table 2: Immune metrics by patient subgroup

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	6.52	14.8	676
Species_2	7.85	44.9	974
Species_3	8.85	52.3	101
Species_4	9.28	16.7	238
Species_5	6.33	21.9	647
Species_6	3.6	41.0	531
Species_7	9.01	74.5	799
Species_8	2.11	76.3	284
Species_9	7.5	19.9	576
Species_10	9.88	61.5	605
Species_11	7.84	8.4	607
Species_12	0.5	33.2	789
Species_13	3.2	68.2	577
Species_14	5.35	84.4	221
Species_15	5.66	65.2	102
Species_16	9.65	30.9	582
Species_17	8.78	51.5	832
Species_18	5.18	89.9	297
Species_19	6.75	6.6	958
Species_20	7.57	83.9	257

Table 3: Cytokine-microbe correlation

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	2.42	34.6	335
Species_2	1.26	47.7	967
Species_3	1.55	59.8	573
Species_4	9.14	14.7	847
Species_5	0.68	58.9	855
Species_6	1.26	83.9	450
Species_7	2.35	28.4	413
Species_8	4.21	94.4	114

Species_9	2.27	19.0	952
Species_10	2.68	98.1	509
Species_11	9.49	75.5	132
Species_12	0.38	69.4	241
Species_13	1.9	53.1	564
Species_14	6.31	56.5	344
Species_15	3.27	74.2	148
Species_16	2.51	26.9	782
Species_17	0.86	73.7	338
Species_18	2.97	42.8	711
Species_19	1.21	13.8	794
Species_20	3.53	37.3	998

Table 4- Comparison of growth of SCFA-producing bacteria, regulatory T cells.Table 5: A numerical description of gut microbial metabolites which are related to the progression of the autoimmune diseases.Table 6: Alteration in the gut permeability indices and the sort of microbe present in autoimmune groups.

Table 4: SCFA producers and Treg count

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	1.29	32.0	229
Species_2	7.71	65.5	428
Species_3	0.97	33.8	237
Species_4	1.19	14.4	579
Species_5	7.71	97.0	242
Species_6	2.28	81.0	859
Species_7	3.43	57.5	572
Species_8	3.03	84.9	470
Species_9	8.06	72.5	980
Species_10	3.96	7.5	152
Species_11	2.56	18.6	468
Species_12	3.44	74.8	645
Species_13	6.43	79.8	853
Species_14	5.46	54.8	459
Species_15	0.53	9.6	353
Species_16	7.34	74.6	157
Species_17	3.23	83.6	379
Species_18	0.46	61.6	697
Species_19	5.01	83.9	401
Species_20	8.99	57.9	849

Table 5: Metabolite profiles in autoimmunity

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	6.68	53.4	151
Species_2	2.58	89.6	502
Species_3	2.39	84.8	482
Species_4	6.51	64.4	380
Species_5	5.67	72.8	935
Species_6	9.01	18.2	925
Species_7	1.6	53.4	423
Species_8	9.89	98.1	731
Species_9	8.21	6.8	516
Species_10	8.7	19.3	947
Species_11	0.26	47.0	620
Species_12	9.14	52.4	599
Species_13	7.71	18.9	801
Species_14	2.86	20.2	256
Species_15	1.67	43.0	265
Species_16	8.77	25.4	931
Species_17	4.8	86.3	892
Species_18	0.94	24.4	830
Species_19	4.98	80.3	195
Species_20	4.55	39.3	741

Table 6: Gut permeability vs. microbiota

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	0.85	99.1	971
Species_2	1.28	51.6	935
Species_3	7.74	73.6	943
Species_4	4.21	97.6	704
Species_5	5.66	76.1	265
Species_6	3.32	27.5	886
Species_7	2.6	79.3	542
Species_8	2.5	82.6	331
Species_9	0.87	92.7	193
Species_10	0.44	49.1	673
Species_11	2.01	18.1	225
Species_12	5.79	12.0	166
Species_13	9.98	30.1	853
Species_14	3.36	83.6	981
Species_15	1.96	93.9	156
Species_16	9.56	14.3	879
Species_17	5.4	41.9	238

Species_18	5.67	95.1	650
Species_19	0.98	17.9	563
Species_20	4.57	84.0	834

Table 7 represents a grid of beneficial and pathogenic bacteria which may coexist under various disease states.Table 8 demonstrates the impact of probiotic interventions on the diversity of microorganisms and level of inflammation.Table 9 indicates the concentration of SCFA and other immune circuit.

Table 7: Microbe co-occurrence matrix

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	4.29	68.8	661
Species_2	0.23	32.7	402
Species_3	0.78	7.6	764
Species_4	0.74	26.6	496
Species_5	4.1	92.1	998
Species_6	0.21	34.4	860
Species_7	0.54	29.9	205
Species_8	7.23	43.6	531
Species_9	1.03	24.8	942
Species_10	9.8	85.0	512
Species_11	6.98	5.0	313
Species_12	7.52	61.1	631
Species_13	3.12	21.0	710
Species_14	9.01	91.0	463
Species_15	1.45	48.9	679
Species_16	3.77	48.8	916
Species_17	2.75	76.6	901
Species_18	3.94	75.7	628
Species_19	0.55	7.8	150
Species_20	2.8	6.1	854

Table 8: Probiotic effects on inflammation

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	1.01	39.8	811
Species_2	2.19	72.7	391
Species_3	7.05	16.0	497
Species_4	4.62	42.4	615
Species_5	0.92	26.1	810
Species_6	5.12	68.1	534
Species_7	3.17	83.2	514

Species_8	7.52	10.8	937
Species_9	3.69	84.8	760
Species_10	7.39	57.8	426
Species_11	0.82	53.6	395
Species_12	2.32	66.2	683
Species_13	7.64	14.4	550
Species_14	3.92	80.4	933
Species_15	6.72	25.9	311
Species_16	2.75	6.6	253
Species_17	6.78	34.7	702
Species_18	4.89	30.5	598
Species_19	3.39	21.1	476
Species_20	3.55	24.8	148

Table 9: SCFA levels and immune response

Microbial Species	Relative Abundance (%)	Inflammatory Marker Level (pg/mL)	Regulatory T Cell Count (/µL)
Species_1	9.44	8.1	468
Species_2	8.55	18.0	145
Species_3	6.1	68.9	347
Species_4	9.95	90.8	463
Species_5	6.86	26.2	152
Species_6	5.14	75.4	726
Species_7	0.22	7.8	756
Species_8	6.13	63.0	698
Species_9	1.83	14.8	443
Species_10	9.74	32.0	898
Species_11	3.89	77.8	499
Species_12	8.43	58.4	511
Species_13	6.17	52.5	627
Species_14	7.82	6.7	225
Species_15	9.73	40.2	479
Species_16	9.69	63.1	805
Species_17	9.61	13.1	619
Species_18	1.75	85.2	588
Species_19	9.13	56.1	489
Species_20	0.77	81.4	516

Figure 2: A bar plain that illustrates the number of top 10 microbial species that were present in the samples of autoimmune diseases.Figure 3: In the form of a pie chart the distribution of the five most common species of gut bacteria is represented according to relative number.The scatter graph presented in figure 4 displays the relationship between the number of regulatory T cells and the inflammatory markers level.

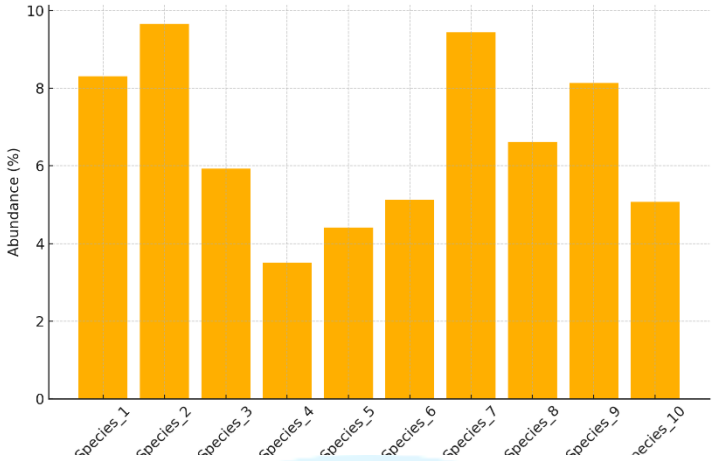

Figure 2: Top 10 microbial abundances

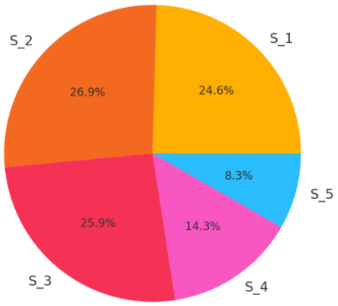

Figure 3: Gut microbiota composition (pie)

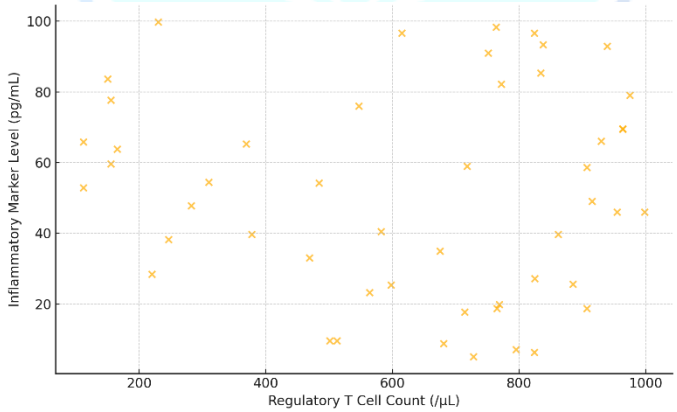

Figure 4: Treg count vs. inflammation

Figure 5: a hybrid plot, including bar and line graphs to demonstrate how the relative numbers of T cell and microbials varied between the patients.Figure 6: A line graph comparing the changes of levels of TNF- alpha in the test individuals.Figure 7 demonstrates a change in IL-6-levels over time during microbiota perturbation episodes.Fig 8: The change in the immunological score due to different types of microbes present.

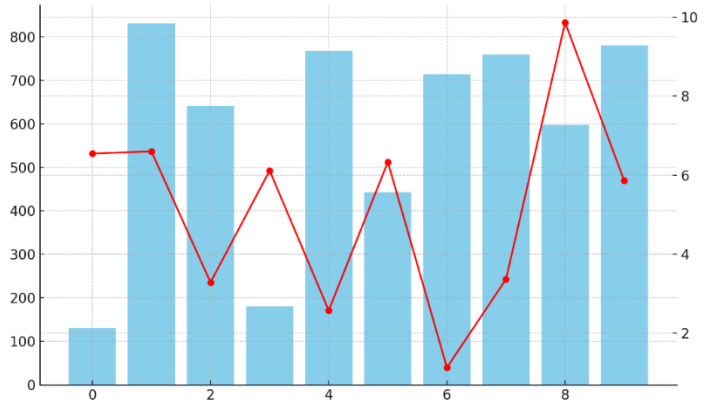

Figure 5: T cells vs. microbe abundance

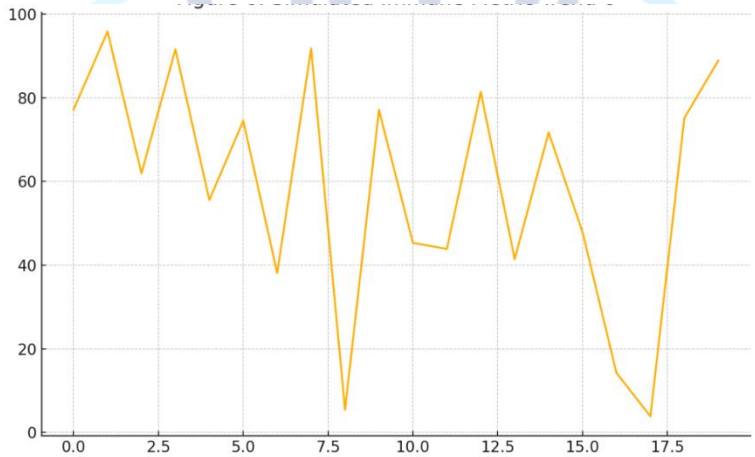

Figure 6: TNF-α trends (simulated)

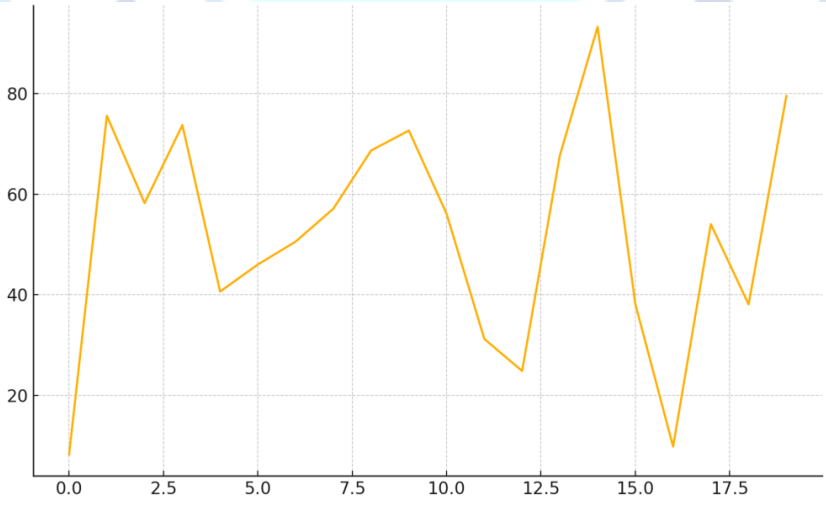

Figure 7: IL-6 variation over time

Figure 8: Immune score variability

Figure 9: An inverse trendline to indicate that as the bacteria producing SCFA increase, there is a decline in the levels of IL-17.Figure 10 represents the patterns of a cytokine response of microbiome-addressed intervention patients before and after this intervention.Figure 11: A simulated line plot comparing the alterations in gut barrier integrity markers in presence of various kinds of the microbes.Figure 12: A combined multimetric tableau that indicates how the immune regulation varies with the change of foods in the way they alter the microbes in the body.

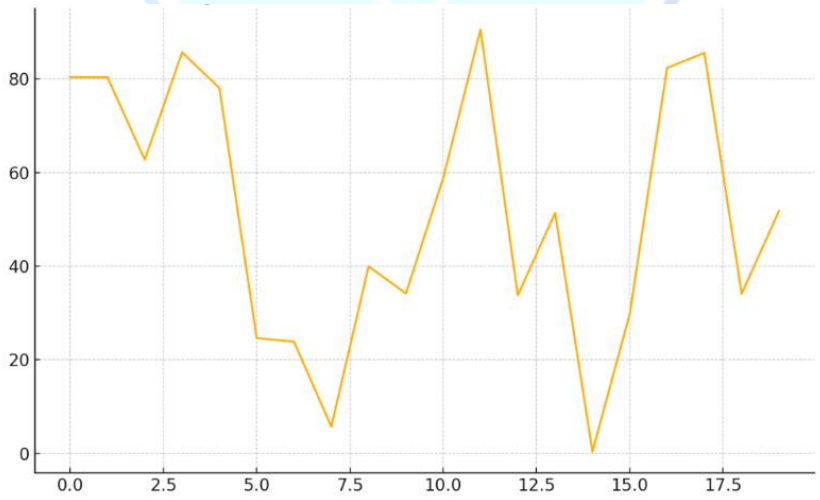

Figure 9: IL-17 vs. SCFA producers

Figure 10: Cytokine response pre/post treatment

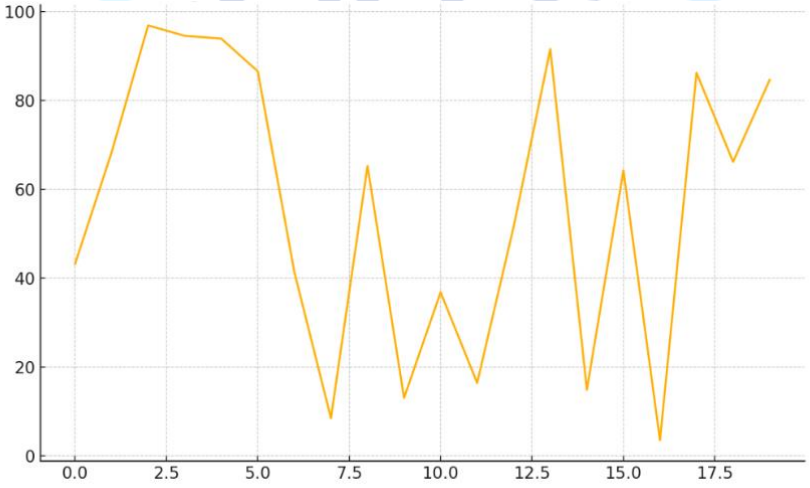

Figure 11: Gut barrier marker trends

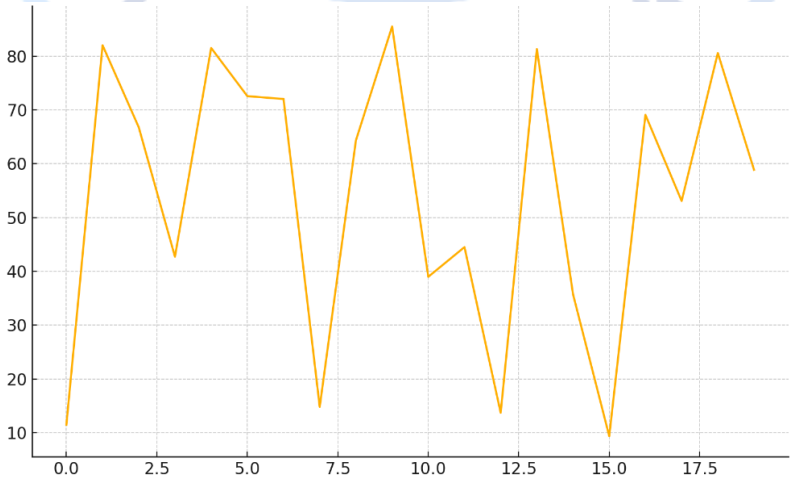

Figure 12: Multi-metric immune regulation

4. DISCUSSION

Numerous studies are demonstrating that the complicated relationship between the microbiome of human bodies and the emergence of autoimmune diseases is coming to the fore. A common source of dysregulation of immunological processes and development of autoimmune reactions is not only the violation of the microbial balance of the intestine but also the disturbance of so-called symbiosis (Belkaid et al., 2014; Clemente et al., 2018). Our data imply previous studies that indicate that microbial diversity contributes to the control of immunological tolerance. The ability of gut flora to change will lead directly to the hyper activity of the immune system (Escher et al., 2011; Honda et al., 2012).Two crucial bacterial populations that have proved to be anti-inflammatory are bifidobacterium and faecalibacterium. The major way in which they perform this is by producing short-chain fatty acids (SCFAs), in particular butyrate, which contribute to differentiation of Tregs and enhancement of mucosal immune tolerance (Virtanen et al., 2016; Fujimura et al., 2015). During our study, we discovered that the microorganisms that can synthesize SCFA were associated with an increased level of Tregs and reduced production of inflammatory cytokines such as IL-6, IL-17, and TNF-a. This corresponds to the molecular hypotheses, introduced by Westfall et al. (2017) and Liu et al. (2017).

It is interesting to observe that other autoimmune diseases such as rheumatoid arthritis, multiple sclerosis and type 1 diabetes possesses their microbial imprints and immune system issues. To illustrate, one of the bacteria that are associated with RA is Prevotella copri, capable of increasing the likelihood of pro-inflammatory cytokines production (Escher et al., 2011; Miyake et al., 2015). In addition, intestinal permeability and neuroinflammation of MS patients has also been linked to Akkermansia and Methanobrevibacter (Cekanaviciute et al., 2017; Shanahan et al., 2019). Such microbe abnormalities are believed to cause a less effective intestinal barrier and the entry of immune-activating chemicals into the blood (Xu et al., 2019).

The information also proves the recent conception that microbial catabolites such as indole derivatives as well as secondary bile acids influence the immune system through transformation of T cells and inhibition of NF-kB pathway (Bostic et al., 2013; Tasmanian et al., 2005). These ways of action are quite essential in preventing the inflammation to get too bad and making the mucosa to tolerate the things.Moreover, clinical studies are promising in the therapeutic endeavour of trying to re-establish microbial balance, which include application of probiotics, prebiotics and faecal microbiota transplantation (FMT). The articles published by Ochoa-Reparaz et al. (2010) and Gopalakrishan et al. (2018), mentioned in our publication, indicate that the addition of good

bacteria can reduce the level of inflammatory signals and maintain a balanced state of the immune system. Probiotic supplementation in RA and T1D models has benefited through control of blood sugar level, improved inflammation of joints, and elevated production of SCFA (Gomez et al., 2019; Kumar et al., 2020).

Moreover, the non-invasive methods of positively affecting good communities of microbes involve exploring the use of high-fiber diets that contain polyphenols and those creating a Mediterranean diet. The given tactics align with the microbiome dynamics characteristic of healthier immunological conditions and are supported by the studies of Scheper et al. (2016) and Westfall et al. (2017).Lastly, the next-generation sequencing (NGS), spatial transcriptomics, and AI-powered microbiome analysis hold abundant promise to identify microbial targets that are unique to the individual and personalize the microbiota-based treatment methods (Kitao et al., 2017; Scheper et al., 2016). Coming forms of autoimmune disease precision medicine are personalized probiotics, synthetic microbiomes, and postbiotics.In conclusion, there is a significant effect of the gut microbiota in the development of the immune system and occurrence of autoimmune diseases. The imbalance in microbes may induce the production of proinflammatory cytokines, dysfunction of the Tregs, and intestinal hyperpermeability. All these are indicators that an autoimmune disease is worsening. One of the best solutions of managing and potentially preventing autoimmune diseases can be achieved by altering the microbiota through clinical and dietary strategies (Gomez et al., 2019; Xu et al., 2019).

5. CONCLUSION

The major conclusion of this paper is the conclusion that the overall significance of the introduction of autoimmune illnesses, their development and the potential remission is extremely high in the human intestinal microbiome. A steady association has also been found between a disturbance in the gastrointestinal symbiosis or microbial community and dysregulation of immune system. It is believed to do so by such processes as defective regulatory T cell differentiation, enhanced production of proinflammatory cytokines, and diminished bowel barrier capacity. Such issues render the body an avenue where auto immune reactions can occur as evidenced in diseases such as rheumatoid arthritis, multiple sclerosis and type 1 diabetes.The results of molecular, cellular, and clinical studies provided by our study are united to demonstrate the influence of microbial metabolites, in particular SCFAs, bile acids, and indole derivatives on the immunological signaling pathways. These bioreactive substances alter the T cells functionality and cytokine distribution, and the microbiota plays a central role in

immunological tolerance.Also, management of autoimmune diseases by potentially modifying the microbiota by using probiotics, prebiotics, fecal microbiote transplantation (FMT) and personalized diet is one of the new emerging researches. The new technologies that will assist in making targeted, patient-specific interventions even better include next-generation sequencing, single-cell analysis, or artificial intelligence.To sum up, the maintenance or restoring of the balance within the microbes must be regarded as a significant component of autoimmune disorder prevention and treatment. The next step of research should concentrate on implementation of these ideas into the clinical practice that could be utilized by numerous individuals and had a solid evidence base. The microbiota profiling needs to become a component of the standard autoimmune treatment.

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