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Comparison of Impact of High versus Low FODMAPs diet on Gut Flora in Patients with Irritable Bowel Syndrome

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Abstract – FODMAPs diet abbreviated for fermentable, oligosaccharides, disaccharides, monosaccharides and polyols significantly influence the gut microbiota. Low FODMAPs diet reduces the intake of fermentable molecule and thus alleviate the symptoms of IBS. This happens because of decreasing gut dysbiosis and visceral hypersensitivity. On the other hand, high FODMAPs diet exacerbates GI symptoms by promoting dysbiosis and increasing harmful short chain fatty acids. Population of firmicutes, Bacteroidetes and Actinobacteria plays a crucial role in development of IBS. Factors such as age, sex, ethnicity and diet influence gut microbiota and symptom severity. IBS patients have a lower composition of beneficial bacteria like bifidobacterium and lactobacillus and increased population of the harmful ones like Enterobacter. Current clinical evidence supports the efficacy of low FODMAPs diet in improving IBS symptoms and altering gut microbiota composition with patients having a better quality of life because of reduced GI symptoms. However, the diet alters the gut environment, and the effect of its long-term impact needs further research. Future studies should also aim on studying personalized dietary intervention for a precise IBS management in patients. Overall, low FODMAPs diet shows promise as therapeutic approach for IBS by providing a better quality of life and symptom reduction.

Keywords – High FODMAPs, Low FODMAPs, Irritable Bowel Syndrome, Gut Microbiota, Gut Dysbiosis, Lifestyle Medicine, Gastroenterology.

I. INTRODUCTION

Considered as one of the ideal non-interventional therapeutic approaches for various bowel disorders. FODMAPs is an abbreviation for fermentable, oligosaccharides, disaccharides, monosaccharides and polyols which includes lactose, fructose, in excess of glucose, sorbitol, mannitol, fructans and galacto-oligosaccharides **[1,51]**. This diet has shown to have a significant effect on the alteration of gut microbiota, in patients with bowel diseases such as Irritable Bowel Disease (IBS), predominantly in individuals with subtypes IBS-M and IBS-D **[1,5]**.This diet method when consumed in low levels, decreases the consumption of fermentable molecules, which are poorly absorbed in the gut henceforth resulting in reduction in short chain fatty acids (SCFA's) such as butyrate which has been proved to have harmful effects on the physiological mechanisms of the human body such as the gut, sleep and mental health **[1,2]**. On the other hand, when consumed in high levels (High FODMAPs) this diet can cause gut dysbiosis which can provoke visceral hypersensitivity and worsen GI symptoms **[51]**. The recommended intake of FODMAPs in patients with IBS is 5-18g/d **[2]**. An ideal diet plan for a low-FODMAPs diet should have: not more than 0.2-0.3 g/serve of oligosaccharides, below 0.4 g/serve of total polyols, under 0.4 g/serve of fructose and not more than 1g/serve of lactose **[51,53]**. This diet excludes several categories of food such as (a) dairy products like milk, soft cheese and ice cream, (b) fruits such as

apples, watermelon, mangoes, cherries, apricots, pears and peaches, (c) legumes such as chickpeas and lentils (d) vegetables such as asparagus, beetroot, broccoli, garlic, onions, and cauliflower (e) cereals, such as bread and rye f) sweeteners **[3]**. In addition, several studies have shown that the physiological effects of consuming high levels of FODMAPs diet involves promoting increased levels of intestinal water content which may cause abdominal pain and bloating **[1,2,5]**. It has also been associated with the increased production of hydrogen, carbon dioxide and methane gases which induces the production of colonic gas and later results in luminal distension **[2,4,5]**. A still under-researched topic, this review aims to discuss the potential effects of consumption of high and low FODMAPs diet and its effect on the gut flora in patients with irritable bowel syndrome (IBS).

Nurturing more than thousands of bacteria, and other organisms triple to that of the number of cells in the human body, the GI tract is the home to a variety of bacterial populations such as bacteria, fungi and protozoa **[2,6]**. These bacteria's have both physiological as well as pathological roles in the maintenance of the human body and the prevention and development of diseases within the body **[6]**. This relationship between the human body and microbiome has been described as a mutualistic, symbiotic or parasitic relationship **[2]**. Healthy individuals have at least three main species of bacteria's that predominate the GI and these include: *Firmicutes* and *Bacteroidetes* dominating at least 90% of the population and lastly *Actinobacteria*, dominating less than 10% of the population **[2,6]**. Another lesser known species include *Proteobacteria* which dominates around 5-10% of the population **[6]**. Several host factors influence the diversity and growth of the gut microbiome as illustrated in Fig 1 **[2]**. These factors include age, sex, ethnicity and diet.

1. Age

Several studies have found that advances in chronological age have shown an increased variability in gut microbiome **[6]**. In a systematic review comprising of 27 studies, Badal et al investigated and concluded that microbiome composition is different across different age groups, showing higher levels in species of *Akkermansia* and lower levels in *Faecalibacterium*, *Bacteroidaceae* and *Lachnospiraceae* **[7]**. Similarly, another cross-sectional study performed involving 153 participants over a range of age groups, found that certain bacteria species, that of *Bifidobacterium*, *Faecalibacterium*, *Bacteroides* group and *Clostridium* has been reduced in individuals over 80 years of age **[8]**.

2. Sex

According to Human Microbiome Project, done by Ding and his colleague, it has been shown that, males were three times more likely to have lower levels of *Bacteroides* and higher levels of *Prevotella* species **[9]**.

3. Ethnicity

Although this topic requires further research, scientists have always believed that ethnicity plays a crucial role in gut microbiome due to different lifestyles and dietary habits. Moreover, there has been evidence in a study conducted between South Asian and Caucasian infants, concluding that South Asians had higher levels of lactic acid and Caucasians having higher levels of *Clostridiales* **[10]**.

4.. Diet

Certain studies have shown that intake of a plant based protein diet results in higher levels of some bacterial species like *Bifidobacterium* and *Lactobacillus* and lower levels in species like *Bacteroides fragilis* and *Clostridium perfringens* **[11]**. On the other hand, intake of animal-based protein diets causes an increase in the levels of species such as *Bacteroides*, *Alistipes* and *Bilophila* and reduced levels of *Bifidobacterium*. Fiber rich diets have also shown an increase in the number of *Bifidobacterium* and *Lactobacillus* **[11]**.

These microbiomes play several different roles within the gut both physiologically and pathologically. Some of which include breaking down of simple carbohydrates to produce short chain fatty acids (SCFA's), decomposition, biotransformation and chemical modification of natural products to produce rich metabolites, regulation of bile-acid metabolism, increasing the production of vitamins B and K and lastly working on improving the different metabolic and immune function **[2]**. Over the last couple of decades, several studies have been carried out to learn more about the microbiota, its existence, and how it relates to human physiology.

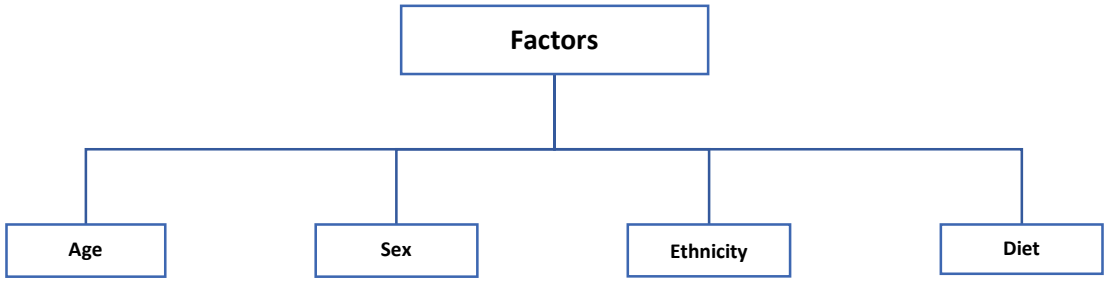

Fig 1: illustration of host factors that influence the diversity and growth of gut microbiome [1]**.**

II. Understanding Irritable Bowel Syndrome and its Pathogenesis

As stated by Cumming in 1849, IBS has been described as "The bowels are at one time constipated, another lax, in the same person. How the disease has two such different symptoms I do not profess to explain" **[12]**. Recognized over 150 years ago and having a global prevalence of 11-21%, a chronic bowel disorder, Irritable Bowel Syndrome (IBS) is characterized by shifting abdominal pain and chaotic bowel habits **[12, 13,50]**. Other unspecified symptoms include flatulence, bloating, urgency, fibromyalgia in 31.6% to 63% patients, mental health issues (depression in 26-45.5% and anxiety in 30-39.1%), lethargy, migraines and headaches in 50% of the patients **[2,6,50]**. Moreover, studies and research has shown that individuals suffering from IBS have multiple triggering elements, such as regular caffeine intake, alcohol, and lastly consuming spicy and fat-rich meals **[14]**. Consisting of more than 60% of gastroenterology consultations, its usual age of onset is less than <40 years and primarily affects around 14% of females and 9% of males globally **[2]**. Clinically, there has been several different subtypes of IBS based on the Bristol Stool Form Scale (BSFS) and these are both diarrhea and constipation, mixed (IBS-M), diarrhea-predominant (IBS-D) also the most prevalent type in among 40-50% of patients, constipation-predominant (IBS-C) and unspecified (IBS-U) which has normal stools **[2,6,14,15]**. Although no biomarkers available currently, diagnosis of this syndrome is usually done according to Rome IV criteria, which was published in May 2016 and states that the individual needs to report at least 1 day/week in the last 3 months to report either two or more of the following symptoms **[1,16]**:

i. Changes related to defecation

ii. Changes associated with stool frequency

iii. Changes in stool consistency and appearance

In addition, several blood tests are also conducted along with invasive procedures like col[1] H. M. Staudacher and K. Whelan, "Altered gastrointestinal microbiota in irritable bowel syndrome and its modification by diet: probiotics, prebiotics and the low FODMAP diet," *Proceedings of the Nutrition Society*, vol. 75, no. 3, pp. 306–318, Aug. 2016, doi: 10.1017/S0029665116000021.

onoscopy, gastrointestinal symptoms severity are usually measured through a scale called the IBS Symptom Scoring System (IBS-SSS) **[6,12]**. Recent studies and evidence based research have shown that there have been various mechanisms implicated in the etiology and pathogenesis of IBS, some of these include visceral hypersensitivity, food sensitivity, carbohydrate malabsorption, immune dysregulation, gastrointestinal (GI) microbiota, intestinal inflammation, serotonin dysregulation, gut-brain axis and psychosocial factors **[2,6,13,17]**.

Currently and clinically, treatment and management of this disease has always followed a symptomatic based approach combining both lifestyle as well as medication based interventions **[2]**. Medicines such as serotonin agonists and antagonists, chloride channel agonists, antispasmodics, tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs) have been prescribed **[2,6,14].** Lifestyle interventions require doing regular exercise, avoiding foods that trigger IBS such as high levels of FODMAPs, processed foods and increasing fiber intake via consumption of fruits **[2,14]**.

III. FACTORS THAT PLAY ROLE IN PATHOGENESIS OF IBS

Although not well understood pathophysiology is highly complex, involving multiple mechanisms, it is believed that this disorder has components of both the environment and genetics. Potential factors that play crucial role in pathogenesis of IBS are tabulated in table 1.

1. Immune Dysfunction and Infections

Scientists have found that patients with IBS, particularly IBS-D has higher levels of pro-inflammatory cytokines such as IL-β, IL-6, IL-8, IL-12 and TNF-α which may lead to mucosal and systemic inflammation **[18,19]**. This increased levels of cytokines can be triggered due to multiple factors such as any form of stress which could be an illness or psychological **[20]**. Data has also shown that these patients also have elevated levels of eosinophils and mast cells when compared to healthier individuals and this may also be a cause of increased colonic permeability, abdominal pain and diarrhea **[18,19,20]**. It has also been proven that post-infectious IBS (PI-IBS), especially after gastrointestinal illness acts as a predisposing factor to the development of IBS as this might later cause alterations in gut microbiota which in turn, increases intestinal permeability, which later may cause visceral hypersensitivity, thus causing increased abdominal pain **[2,20].** Several factors can induce PI-IBS and these include bacterial infections by *Salmonella, Shigella, Campylobacter jejuni, Escherichia coli, O157:H7,* protozoan infections by *Trichinella,* parasitic infections by *Giardia intestinalis,* various viral infections, patients with a history of gastroesophageal reflux disease, anxiety, depression and female sex **[18,20]**. In a retrospective study performed by Iacob et al involving 873 participants with positive stool test and another group of 200 control participants, whom of all were evaluated using the Rome III criteria questionnaire and Bristol Stool Form Scale found that the risk of developing PI-IBS is 4.16 times higher compared to any other systemic infection **[21]**.

2. Gut Dysbiosis

Recent research shows that imbalance of gut microbiome might be a causative agent for the development of IBS, decreases and increases in the various bacterial populations has given rise to the different symptoms of IBS. Some studies have found a decreased prevalence of *Lactobacillus*, *Bifidobacterium, Fecalibacterium* species , and higher levels of *Enterobacter* species in IBS-D patients whereas higher levels of *Veillonella* in patients with IBS-C **[18,22,23]**. These changes in gut environment can later provoke changes in nervous systems, inflammatory and pain responses as well as affecting colonic motility and sensitivity **[18]**. In a case-control study on 14 participants with IBS-C and 12 healthy participants, examination of fecal microbiota revealed reduced numbers of lactate producing and metabolizing bacteria and hydrogen consuming bacteria in participants with IBS-C **[19,49]**. Further analysis showed that flora of patients with IBS-C generated more sulphides and hydrogen and lower levels of butyrate **[19]**.

3. Genetics

Evidences show that there has been over 60 genes implicated in the pathogenesis of IBS, of these *TNFSF15* gene which encodes for TY1A protein have been found to cause activation of the immune-cell-mediated inflammatory response in the gut epithelia **[6,24]**. Moreover, in a genome-wide association study, comprising of 584 individuals with IBS and 1380 healthy individuals found that a gene *SCN5A* which encodes for voltage gated sodium channel is responsible for congenital prolonged QT syndrome, and often associated with abdominal pain has been found to have a missense mutation in more than 2% IBS patients in a genome wide association study that was performed **[19,25]**. In addition to this, IBS is also proposed to have familial aggregation, and twin studies performed showed higher levels of concordance in monozygotic twins than dizygotic twins **[26,27]**. A case control study involving 477 participants with IBS with 1492 first-degree relatives and 297 controls with 936 of their first-degree relatives demonstrated increased proportion of IBS relatives in participants with IBS compared with the controls **[20,28]**. Furthermore, epigenetic modifications such as DNA methylation and expression of non-coding microRNAs have been found in patients with IBS. These alterations can be triggered due to multiple reasons such as early stressful and traumatic life **[18,19,20]**.

4. Diet

Consumption of certain foods along with high FODMAPs diet have been constantly associated with the development of IBS **[6]**. Several animal studies performed concluded that consumption of high FODMAPs diet can cause an increase in gram-negative bacteria and luminal lipopolysaccharide which can later activate mast cells and trigger GI symptoms **[51]**. The presence of nutrients often affect gastrointestinal motility, barrier functions, sensitivity and gut microbiota **[18]**. Several different hypothesis

has been provided in order to describe the connection between food and IBS. Theories such as hypersensitivity to certain foods can trigger mucosal inflammation of the gut, alterations in the gut flora, as well as cause increased permeability of the gut have been reported in several studies **[18,22,29]**. It has also been proposed that certain chemicals such as salicylates, amines and glutamates can provoke symptoms like abdominal pain and bloating and in long term can lead to visceral hypersensitivity as seen in IBS **[18,30]**. Furthermore, studies have found that fiber deficiency in a worsening component in patients with IBS-C and consumption of soluble fiber has shown relief in these kinds of patients **[18,22,31]**.

5. Serotonin Dysregulation

Serotonin or 5-HT is an important neurotransmitter of the gut and brain **[20]**. It is mostly stored in the enterochromaffin cells and in enteric serotonergic neurons. Its release is usually triggered by a number of stimuli **[18,19,20]**. It plays its role by binding to the receptors of the enteric neurons as well as vagal and spinal afferent neurons, which are primarily responsible for gut motility, secretion and sensation **[20,32,33]**. The method of reuptake of serotonin into cells is performed by 5-HT reuptake transporter, SERT **[19,20]**. In addition, several studies performed have showed that lower levels of plasma 5-HT concentrations are found in patients with IBS-C and higher levels in patients with IBS-D **[19,32]**. These higher levels of 5-HT levels causes increased hypersensitivity in patients with IBS **[52].**

Table 1: Potential factors that play role in pathogenesis of IBS

Factor	Description
Immune Dysfunction and Infections	IBS-D patients have higher levels of pro-inflammatory cytokines, leading to inflammation. Elevated eosinophils and mast cells increase colonic permeability and symptoms. Post-infectious IBS (PI-IBS) can develop after gastrointestinal illness, altering gut microbiota and increasing intestinal permeability and pain. Factors inducing PI-IBS include infections, GERD, anxiety, depression, and female sex.
Gut Dysbiosis	Imbalance in gut microbiome causes IBS. IBS-D patients have decreased *Lactobacillus, Bifidobacterium, Fecalibacterium*, but increased *Enterobacter* species. IBS-C patients have higher *Veillonella*. These imbalances affect inflammation, pain, motility, and sensitivity.
Genetics	Over 60 genes are linked to IBS. TNFSF15 gene causes immune response in the gut. *SCN5A* gene mutations found in 2% of IBS patients cause pain. IBS shows familial aggregation, higher concordance in monozygotic twins. Epigenetic modifications are also involved.
Diet	Certain foods and high FODMAP diets are linked to IBS. High FODMAP diets increase bacteria and lipopolysaccharides, triggering symptoms. Nutrients affect motility, barrier function, and gut flora. Hypersensitivity to certain foods causes inflammation and increased permeability. Fiber deficiency worsens IBS-C; soluble fiber helps.
Serotonin Dysregulation	Serotonin (5-HT) in the gut and brain affects motility, secretion, and sensation. IBS-C patients have lower 5-HT levels, while IBS-D patients have higher levels, increasing hypersensitivity.

IV. Changes in Gut Microbiota Composition in Patients with IBS

Several studies have shown that gut dysbiosis, which is primarily caused due to an imbalance of the gut environment, may be involved in the development of irritable bowel syndrome (IBS) **[2,6,34]**. These organisms play a key role in maintaining gut homeostasis and protecting the mucosal epithelium **[6]**. Gut dysbiosis can be triggered by factors such as inflammatory responses that can later provoke oxidative stress and increased intestinal permeability **[6,35]**. A summary of potential changes is illustrated in fig 2.

Multiple studies have emphasized on the number of alterations in gut microbiome in IBS patients, although these studies need further investigation and testing, evidence has found certain common features **[2,6]**. In an original study conducted with 80 subjects diagnosed with IBS, and 65 control individuals (healthy) without IBS, Jeffery et al found higher levels of *Ruminococcus gnavus* and *Lachnospiraceae* and lower levels of *Barnesiella intestinihominis* and *Coprococcus catus* **[36]**. Another meta-analysis conducted has reported depletions in the amount of *Bifidobacterium*, *Lactobacillus* and *Faecalibacterium prausnitzii* in IBS patients **[37]**. Another meta-analysis comprising of 16 articles and 777 subjects with diagnosed IBS performed suggested an increase in the *Firmicutes* and depleted levels of *Bacteroidetes* and thus higher to *Firmicutes* to *Bacteroidetes* ratio. They also found increased levels of *Clostridia* and *Clostridiales* and decreased concentrations of *Bacteroidia* and *Bacteroidales* **[2,38]**. Likewise, another meta-analysis comprising of 23 articles and involving 1340 participants which tested gut microbiome, found reduced levels of *Lactobacillus* and *Bifidobacterium,* as well as increased levels of *Escherichia coli* and *Enterobacter* in individuals who had IBS when compared to healthy patients. Increased levels of *Bifidobacterium* in IBS is often associated with abdominal pain, decreased levels of *Faecalibacterium* is associated with inflammation, *Agathobacter* is associated with body pain and lastly, *Paraprevotella* is associated with unsatisfied defecation **[39]**. Although these studies have shown the changes in gut microbiome in an IBS patient as a whole, no studies have found any significant difference in IBS subtypes **[6]**.

Moreover, data has shown that scientists have found an association between IBS-D and depression patients, these patients have lower amounts but increased prevalence of *Bacteroides, Prevotella or nondominant microbiota* **[40]**. Although further research is needed in this topic, this correlation has led through a breakthrough development.

V. Low FODMAPs and its Effect on Gut Microbiome in Patients with IBS

Evidence based on clinical trials and randomized controlled trials over the last decade has found that consumption of low FODMAPs diet had drastic changes on IBS symptomatically as well as in the growth and diversity of gut microbiome and can be considered as a primary intervention for the treatment of IBS. A concise overview of low FODMAPs and its effect on gut microbiome these patients are tabulated in table 2. One such randomized crossover trial performed in the recent years involving 29 subjects (aged 18-75) and investigating the effects of low versus moderate FODMAPs diet found that the seriousness of the gastrointestinal symptoms reduced, in addition to improved stool consistency and quality of life in patients who consumed low FODMAPs diet and worsened in patients who consumed moderate levels of FODMAPs diet **[41]**. Similarly, an original study conducted in the years 2018-2019 involving 70 patients, found that intake of low FODMAPs diet had a drastic improvement in symptoms within 1 week of consumption of the diet, this study has also proven that the diet has effects on psychological health of the patients within 3 weeks of consumption **[42]**. Another meta-analysis comprising of 22 studies (6 RCTs and 16 non-randomized trials) concluded that participants who strictly followed a low FODMAPs diet in both RCTs and non-randomized trials had a lower IB-SSS scores in addition to improved quality of life and reduced abdominal pain **[48]**. Due to the challenges often faced with adhering to a low FODMAPs diet, studies and trials have also compared the low FODMAPs diet with other diet plans **[6]**. A randomized controlled trial involving 38 participants, investigating the effects of FODMAPs versus typical Australian Diet in patients with IBS, established that patients who adhered to the low FODMAPs, had an overall relief of GI symptoms like bloating, abdominal pain and improved stool consistency **[43]**. Likewise, another randomized controlled trial conducted, involving 84 participants who had diagnosed IBS-D subtype, analyzing the comparative effects of consumption of two different diets, low FODMAPs versus modified NICE diet (mNice) concluded that although both diet plans provided similar results, low FODMAPs provided more adequate symptomatic relief for IBS-D (52%) when compared to mNICE diet (41%) **[44]**. A meta-analysis carried out comprising of 10 studies, comparing low FODMAPs diet versus traditional IBS diet (high fiber, low fat) concluded that, IBS-SSS scores were significantly lower in the group of participants who consumed low FODMAPs diet **[45]**.

Due to the complexity in its pathogenesis, it has been recently suggested that gut microbiome could be a therapeutic target for IBS **[6]**. Furthermore, in a randomized controlled trial conducted, involving 104 subjects (aged 18-65) diagnosed with IBS, according to Rome III criteria, Staudacher et al found that intake on low FODMAPs diet, in addition to providing symptomatic relief, also reduced the levels of certain bacteria species such as *Bifidobacterium* and *Faecalibacterium prausnitzii* **[46]**. On the other hand, a randomized trial conducted in 2018-2019, involving 103 patients (aged 18-70), Nordin et al found that ingestion of low FODMAPs diet produces higher levels of fecal saccharolytic bacteria such as *Anaerostipes, Bifidobacterium, Faecalibacterium, Fusicatenibacter, Agathobacter, Paraprevotella, and Oxalobacter,* these increased levels have effects on different symptoms of IBS, additionally high levels of plasma phenolic derived metabolites, 3-indolepropionate, and lower levels of isobutyrate and bile acids **[47]**.

Table 2: Overview of low FODMAPs diet and its effect on gut microbiome and patients with IBS

Aspect	Details
Effectiveness of low FODMAPs diet6tgt	Reduces GI symptoms Improves stool consistency Improves quality of life Effective as a primary intervention for IBS
Clinical trials and studies	Symptomatic improvement within 1 week Psychological health benefits within 3 weeks
Meta analysis	Lower IB-SSS score Improved quality of life Reduced abdominal pain with low FODMAP diet
Comparison with other diets like typical Australian diet, traditional IBS diet and mNICE diet	Relief of GI symptoms Improved stool consistency Lower IB-SSS score Overall better quality of life
Low FODMAPs diet and Gut microbiome	Reduced levels of *Bifidobacterium* and *Faecalibacterium prausnitzii* Increased fecal saccharolytic bacteria (*Anaerostipes, Bifidobacterium, Faecalibacterium, Fusicatenibacter, Agathobacter, Paraprevotella, Oxalobacter)*, higher plasma phenolic derived metabolites, lower isobutyrate and bile acids

VI. Conclusion

This review underscores the profound relationship between FODMAPs diet and the composition and function of gut microbiome in context of IBS. The gut microbiome plays a crucial role in GI homeostasis. Because of which the dysbiosis holds a strong implication in the pathogenesis and progression of IBS. Several factors including age, sex, ethnicity, genetics influence this diversity. Several studies have shown that higher levels of FODMAPs can exacerbate the symptoms of IBS increasing water content in intestines, increasing bloating and luminal distension. On the other hand, this review emphasizes on the benefits of low FODMAPs diet by significantly reducing IBS symptoms, improving stool consistency and enhance quality of life. Strong evidence from various clinical trials and meta-analyses suggested that a low FODMAPs diet is not only beneficial in alleviating GI symptoms but also induces positive changes in gut microbiome like reduction in *Bifidobacterium* and *Faecalibacterium prausnitzii* which are associated with symptomatic relief. This review also addresses the challenges in adhering a low FODMAPs diet and its long term impact on gut health requires further research for a better understanding. To conclude, Low FODMAPs diet presents as a promising non-pharmacological intervention in management of IBS, offering symptomatic relief and potential modulation of gut microbiota. Future studies exploring gut microbiome as a therapeutic target for IBS may lead to much more personalized and precise management model with minimal side effects and improvements in overall outlook of this condition. It would be strongly associated with better quality of life for IBS patients.

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VIII. CONFLICTS OF INTEREST

The authors declare no conflict of interests.

IX. AUTHOR CONTRIBUTIONS

AWI: Conceptualization, Writing- Orignal draft, Writing- review & editing, HSW: Writing- original draft, Writing – review & editing, Supervision & Mentorship.

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ABBREVIATIONS

FODMAPs: Fermentable, Oligosaccharides, Disaccharides, Monosaccharides And Polyols,

GERD: Gastroesophageal reflux disease

IBS: Irritable Bowel Syndrome,

IBSSS: IBS Symptom Scoring System,

PI-IBS: Post-Infectious Irritable Bowel Syndrome,

SCFAs: Short Chain Fatty Acids,

SSRIs: Selective Serotonin Reuptake Inhibitors,

TCAs: Tricyclic Antidepressants