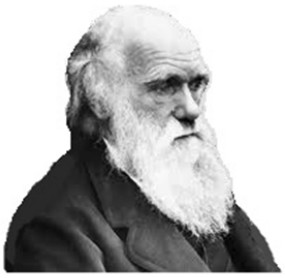
Available online at www.worldnewsnaturalsciences.com



World News of Natural Sciences

An International Scientific Journal

WNOFNS 60 (2025) 138-155 EISSN 2543-5426

Polycystic Ovarian Syndrome (PCOS) Management In Indian Women: Through Native Diet and Gut Microbiota Regulation

Thomas Sowmya[1,a] and **Gabriel Jeya Jothi**[1,b,*]

[1] Department of Plant Biology and Biotechnology, Loyola College, Chennai 600034, Tamil Nadu, India

[a,b]E-mail address: sowmyathomas19@gmail.com , gjjothiloyola@gmail.com

ABSTRACT

Polycystic ovarian syndrome (PCOS) has become a common metabolic syndrome among women globally. In recent times, Indian women are more prone to PCOS following unhealthy eating patterns that involve overconsumption of commercially available Western diets (WD), including sugary food and processed food (PF). Ingestion of WDs and PFs causes dysbiosis (DB) in the gut. Dysbiosis (DB) – an imbalance between beneficial and pathogenic gut bacteria is suspected to undermine the health of the intestine by triggering obesity, insulin resistance (IR), disrupted ovulation (DO), and hyperandrogenism (HA). DO is termed anovulation. Pregnant women who consume commercial foods are prone to intergenerational implications for PCOS risk. Following dietary patterns focusing on GM regulation, such as probiotics and prebiotics, are emerging. Consumption of such a diet can regulate GM and revert the associated metabolic disorders (MD), such as IR, inflammation, and HA, which are the leading factors of PCOS. Emphasizing the choice of traditional, home-cooked food can address nutritional deficiencies and support gut health. The systematic review renders the effect of dietary choices on GM in PCOS regulation.

***Keywords*:** Western Diet, Dysbiosis, Metabolic Disorders, Polycystic Ovarian Syndrome (PCOS), Probiotics, Prebiotics, PCOS Management

1. INTRODUCTION

Over the years, PCOS prevalence rates have varied drastically based on the diagnostic criteria focusing on the different outcomes of the individual [1]. A meta-analysis by Bharali

(Received 11 February 2025; Accepted 11 March 2025; Date of Publication 13 March 2025)

and his co-workers in 2022 reported that the pooled prevalence of PCOS in India is approximately 11.33%, according to the Rotterdam criteria [2]. Zhao and his colleagues have determined that IR is an influential contributor to PCOS by analyzing the various levels of IR and hyperinsulinemia influenced by every PCOS individual's genetic and environmental factors [3]. A connection between PCOS and hepatic disorders was identified. It depicted that these women with PCOS often exhibited MD, including IR and HA, leading to non-alcoholic fatty liver disease (NAFLD) [4]. The origin of PCOS in any patient is marked by the protracted overconsumption of unhealthy commercial food to satisfy the craving [5].

2. GUT MICROBIAL CONSORTIUM

Trillions of distinct microbial communities constitute the GM, which resides in the gastrointestinal (GI) tract and contributes significantly to homeostasis, comprising beneficial, pathogenic, or opportunistic bacteria commonly called gut bacteria that outnumber human cells [6, 7]. The common GM phyla include *Firmicutes*, *Bacteroidetes*, *Actinobacteria*, and *Proteobacteria*. The former two are classified as beneficial, while the latter are classified as pathogenic. These bacteria are distributed throughout the colon of every healthy adult [8]. According to a study by Vera-Santander and colleagues, *Bifidobacterium* and *Lactobacillus* are two kinds of beneficial bacteria (BB) belonging to the phylum Firmicutes. The significance of BBs is that they inhibit harmful bacteria, produce vitamins, boost immunity, inhibit tumor development, minimize infection, alleviate allergic reactions, and facilitate nutritional absorption [9].

A symbiotic relationship referred to as "host-microbe interaction" facilitates cross-talk between the BBs and the host in the intestinal milieu enabled by the encapsulated microRNAs (miRNAs) [10]. The symbiotic interplay is crucial for the host’s well-being, including digestion, metabolism, and the immune system. They protect the intestine against infections by colonizing the mucosal surface. Additionally, they control the proliferation and differentiation of epithelial cells [11]. The BBs affect brain-gut communication, enabling insulin secretion [12, 13]. The colonization of the GI microbiome occurs at birth, as neonates pass through their mother's GM-rich vaginal canal during the process of natural delivery. Consequently, the neonatal GM composition exhibits distinct maternal characteristics following birth; however, this composition gradually diverges to become increasingly distinct approximately one year later [14].

Perturbations in the GM equilibrium characterize DB. Such a condition occurs when opportunistic and pathogenic bacteria outnumber beneficial bacteria when a sedentary lifestyle and unfavorable dietary habits are followed [15, 16]. Liu and his co-scientists in 2017 identified a strong correlation between DB in women's GI tracts and the onset of PCOS [17]. It was later proved that it is the root cause of the blooming of opportunistic and endotoxin-secreting pathogens, marking the route to inflammation beginning from intestinal inflammation leading to obesity-associated PCOS [18].

Chronic DB results in the proliferation of pathogenic strains, predominantly *Klebsiella pneumoniae* and *Clostridium leptum,* which secrete endotoxins, leading to the risk of several illnesses, including cancer [19]. The opportunistic bacteria include *Escherichia coli, Proteus, Morganella, Serratia, Streptococcus, Shigella,* and *Klebsiella pneumoniae* [20]. According to research by He and Li published in 2020, DB may lead to IR, which is closely linked to the

emergence of PCOS [21]. Torres and associates confirmed their findings that the GM of PCOS-affected women differed from that of healthy individuals with higher pathogenic bacteria. According to their research, 73 PCOS individuals exhibited a substantially lower proportion of beneficial GM than healthy women without PCOS difficulties. It was found that the induction of hirsutism and HA in the host was directly correlated with the increased levels of pathogenic and opportunistic gut bacterial composition, such as Porphyromonas spp., *Bacteroides coprophilus*, *Blautia* spp., and *Faecalibacterium prausnitzii* [22]. Oral gavage transplantation of fecal samples from PCOS patients and healthy women into two groups of mice resulted in the appearance of PCOS-like symptoms. Balancing the microenvironment and GM is crucial because, as He and Li indicated, GM works as the second genome of the human body, influencing immune responses, metabolic pathways, and overall health [21]. Additionally, a mouse model study by Yang and his colleagues in 2021 demonstrated the predominant presence of Bacteroides in the PCOS mouse model, indicating it is a crucial microbiological marker in PCOS [23]. Rectifying faulty dietary practices by consuming more probiotics and a higher-fiber diet is the most fundamental part of the rehabilitation process for such women [24, 25]. The widespread practice of dietary transition from native home-cooked food to WDs and PFs is crucial to determining obesogenic (obesity-causing) genetic modification [26, 27].

3. UNHEALTHY EATING HABITS AND THEIR IMPACT ON GM

The Ferriman-Gallwey (mFG) score variability for hirsutism was evident across all pooled PCOS prevalence data, primarily due to unhealthy lifestyle and dietary habits alongside a minor genetic influence. The molecular crosstalk between GM and the host contributes to the onset of PCOS by inducing low-grade inflammation and MD, such as IR and HA [28, 29]. Consuming a diet rich in trans-fat and sugar can lead to DB-associated GI disorders, instigating increased permeability of the intestines, endotoxemia, HA, and DO, expressing PCOS symptoms [30]. Currently, the coherence of PCOS is not comprehended [31]. The potential for curing metabolic problems through GM regulation has been overlooked in PCOS treatments hitherto, prioritizing obesity, IR, and HA (the primary antecedent of PCOS) [32]. However, recent investigations suggest that conserving a higher population of GM may positively influence metabolism [33].

Several studies documented that unhealthy eating patterns and overconsumption of PF, including pastries containing artificial sweeteners, preservatives, and synthetic additives, besides unhealthy lifestyle choices, cause DB [34, 35]. The adoption of WD, primarily focused on fats and sugars, has been linked to obesity, which is a precursor to various endocrine problems, particularly the dysfunction of women's reproductive systems. High sugar content, trans fat, food additives, and micronutrients are among the ingredients that are included in WD and PF (convenient) food. These non-nutritive ingredients disrupt the GM consortium, affecting metabolism and gut health, which can result in obesity and associated MD [36]. PFs, a major part of WDs, are correlated to several MDs and contain unnatural elements contrary to traditional and home-cooked foods [27]. Thus, long-term inflammation, elevated testosterone levels, excessive weight gain, and diabetes can all result from gut DB due to protracted unhealthy diet practices. The consequences lead to the synthesis of choline, bile acids, and lipopolysaccharides, increased intestinal permeability (IP), and discordant brain-gut connection. A combination of these elements can lead to the development of PCOS [37].

3. 1. Processed and convenient meals in India

The food sector in India is growing consistently, with the bakery segment at the forefront. Therefore, the demand for baked goods is increasing along with the growing desire for pastries [38]. In 2020, Dhir and Singla investigated and documented that the Indian ready-to-eat market has experienced rapid growth, reaching Rs 3500 crore in 2016 with a 40% annual growth rate. Within the same year, Dhir *et al.* 2020 examined the correlation between the frequency and health status of convenience food-consuming working and non-working women in Ludhiana. They found that working women (41.7%) spent over 30% of their total food expenditure on such foods, compared to non-working women (8.3%). Bakery goods, ready-to-eat snacks, and drinks were consumed thrice a week by working women and only infrequently or every two weeks by non-working women. Overindulgence in convenience foods has been identified as a contributing factor to women's obesity and non-communicable diseases [39, 40]. Szynal and his colleagues found that common food-graded chemicals included in refined meals exhibit detrimental effects on gut microbial populations when taken at acceptable everyday consumable rates [41]. Furthermore, prior studies have linked PF consumption to conditions such as obesity, type 2 diabetes (T2D), cardiovascular problems, and neuropsychiatric disorders. Brichacek et al. 2024), which can lead to PCOS. Consuming WD that is low in fiber and high in fat and carbs causes gut DB. Conversely, adhering to dietary patterns that oppose the WD may assist in preventing DB and reversing the ensuing metabolic syndromes [42, 43].

3. 1. 1. High-Sugar in Pastries and Sweet PFs:

Confectionery products such as hard candies are manufactured with higher sugar content to overcome spoilage due to surrounding moisture and to prolong product shelf life [44]. Consuming such products is detrimental in the long run. Sun and his colleagues in 2021 administered a high-sucrose diet to Wistar male rats for four weeks. High blood fat levels and NAFLD have developed in those rodents. Increased Bacteroidetes/Firmicutes ratios with fewer beneficial strains altered the GM population. This study investigated the role of GM in lipid metabolic issues driven by consuming excessive amounts of sucrose [45]. Earlier research on NAFLD in PCOS women published by Targher and his colleagues in 2016 was consistent with the recent findings. Such detections highlight the urgency of dietary guidelines that address the detrimental impacts of PFs on gut health and general well-being [46].

3. 1. 2. Non-nutritive artificial sweeteners (NNS) – A primary ingredient in PFs pastries and their association with DB:

Increased consumption of high-glycemic index foods fosters DB in the GM by elevating levels of pathobionts such as *Escherichia coli* and *Candida* spp., and decreasing BB, leading to heightened IP and contributing to leaky gut and systemic inflammation [47, 48]. The World Health Organisation (WHO) advises limiting daily sugar consumption below 10% for better lifestyle, yet the global preference for sweet flavors remains strong. Consequently, the food industry has substituted conventional sugar with calorie-free NNS.

Aspartame, acesulfame-K, and sucralose are among the most commonly used NNS. Furthermore, manufacturers incorporate NNS into certain food products that usually do not require added sugar to enhance their taste appeal (for instance, flavoring potato chips) [49]. Although a collection of data reviewed by researchers indicated that consumption of NNS has benefited individuals in maintaining weight loss, controversial results were observed and

published that prolonged intake of NNS and NNS-containing food results in obesity and associated MD in the later part of life. Associated experiments have demonstrated that prolonged administration of emulsifiers and NNS to rodent models have expressed obesity and observed similar outcomes. In one of the studies, the gene expression assessment revealed that the sweet taste receptor was upregulated on subjection to long-term nutritive sweeteners, non-nutritive sweeteners, and high-fat diets [50-54]. A recent review suggested that regular daily intake of NNS could be potentially associated with cardiovascular disease and related MD [55]. Given this evidence, the WHO has recommended against the protracted use of NNS as a weight loss strategy [49]. Therefore, minimizing the consumption of PFs is recommended. The GM is not directly influenced by aspartame or acesulfame-K; however, the body does not break down and assimilate sucralose. Being a disaccharide with a low absorption rate, it travels directly to the intestines, where it mutates the GM's makeup by impending BB and encouraging the proliferation of the Proteobacteria phylum, which can result in various MDs [56].

3. 1. 3. Impact of Sucralose on the Gut:

Researchers approved sucralose, marketed under the brand name Splenda, as an NNS for its exceptional quality and stability in high temperatures in the food industry as a replacement for table sugar [57]. However, a cohort of databases reviewed by Del Pozo and his colleagues suggests that long-term consumption of sucralose-containing food expressed a toxic effect on GM. Their toxic levels were higher than saccharin. It was understood from the previous analysis that NNS shifted the GM and elevated the biofilm-forming bacterial communities [58]. It is comprehended from a recent study published in Cell Press by Wu and his colleagues that the consumption of NNS causes inflammation triggered by IR and associated MDs [59]. The MDs elevate the production of the male hormone androgen in the ovaries, resulting in HA, hirsutism, acne, facial hair development, alopecia, and anovulation. Anovulation results in irregular menstruation and the development of multiple tiny ovarian cysts [60].

The non-metabolized NNS, on the other hand, directly enter the intestines, where they express the taste receptor protein (T1R3) and Alpha-gustducin protein (α-gustducin) in the extra-gustatory regions that resemble the lingual sweet receptors present in the human colon. The expression triggers DB and proinflammatory factors, contributing to LG [61-63]. The increased percentage of pathogenic gut bacteria brought on by the consumption of WDs, which are low in fiber and high in saturated adulterants, appears to cause IP [64].

3. 2. Emulsifiers – Negative Impact on the Gut

Among the prevalent constituents in many processed meals, emulsifiers can cause DB in their users. Their main purpose is to blend the immiscible liquids to create stable emulsions of different treats [65]. While naturally synthesized emulsifiers are employed to make jams, jellies, mayonnaise, and similar products, chemically manufactured emulsifiers are used in baked goods and sweets [66, 67].

Various emulsifiers alter the GM, disrupting intestinal morphology. Recent investigation indicates emulsifiers, specifically polysorbate-80 (P80) and carboxymethylcellulose (CMC), can directly impact GM by fostering inflammation and related MDs. The emulsifiers mutate the gene expression and disturb the GM population, leading to LPS secretion. Based on current studies, both CMC and P80 release pro-inflammatory molecules, exerting a long-lasting negative effect on the composition and function of the microbiota when compared to naturally

occurring emulsifiers that include lecithin, carrageenan, and gums. The common pathogen that proliferates after the consumption of emulsifiers-containing food is *Ruminococcus gnavus*, which primarily contributes to LG [68, 69]. Research on mice has demonstrated that modest levels of popular food emulsifiers can cause metabolic syndrome, obesity, and mild inflammation in mice that are already at risk for colitis, leading to severe consequences. Emulsifiers damage the intestinal lining, causing the colon to change the natural GM composition and triggering inflammation [70]. These outcomes imply that the application of emulsifiers in PFs generally contributes to a spike in MS, obesity, and other inflammatory disorders.

3. 3. Genistein - Endocrine Disruptor

Genistein, which is often included in plant-based meat products to fortify functional foods, appears to disrupt endocrine functions. It is incorporated into the consumables to enhance the nutrition profile and commercial purpose [71]. Despite its nutritive value and health effects, overconsumption of genistein and its associates in food products leading to potentially negative endocrinal issues, particularly obesity and obesity-associated illness, has been studied [72]. Because of the structural similarity to the hormone estradiol, genistein can bind to the estrogen receptor alpha (Erα) and estrogen receptor beta (Erβ) and elicit estrogenic effects, particularly at moderate dosages. Being a selective estrogen receptor modulator (SERM), genistein can either enhance or inhibit estrogenic action, depending on the tissue type and concentration. The disruption of normal hormonal signalling pathways caused by this dual effect may lead to breast cancer [73].

3. 4. Tight Junction Dysfunction

The intestine's first line of defense for the cellular bypass pathway is the intestinal mucosal tight junctions (TJs), which are found at the top and edge of the mucosal cell membrane and regulate the openings of intercellular channels. Although biologically complete, intracellular TJs are compromised by pathological diseases brought on by poor dietary choices, such as high-fat diet, high-sugar diet, food containing additives that are non-dietary factors, and lifestyle modifications. This increases the absorption of lipopolysaccharides (LPS). LPS is released externally by the proliferation of gram-negative bacterial spp leading to endotoxemia. Elevated LPS levels enter the bloodstream and weaken the immune system (IS). The buildup of blood LPS serves as a biomarker for several MDs, such as T2D and IR. [30, 74-77]. The IS promotes macrophage formation when infections are initiated.

The cytokines, including the proinflammatory cytokines tumor necrosis factor-alpha (TNF-alpha) and interferon-gamma (IFN-γ), are upregulated by macrophages. The synchronization of macrophages with the signaling pathways, such as Nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and Mitogen-activated protein kinase (MAPK), causes phosphorylation of TJ proteins. Consequently, it alters the structural integrity and function of the intestinal wall, thereby elevating the permeability of the intestinal epithelial barrier by downregulating Zonula occludens-1 (ZO-1), cingulin, and afadin [63, 78-81]. Immune cell-resistant LPS leaks out of the phosphorylated colon and enters the bloodstream, initiating inflammation and eventually MD [82]. According to research, TJ protein levels are aberrant in PCOS patients and are associated with blood markers of metabolic problems and inflammation [83].

Conversely, DB prevents amino acids conjugated with primary bile acids from converting to secondary bile acids. Intestinal inflammation and permeability brought on by the buildup of amino acid-conjugated primary bile acids cause a leaky gut [84, 85].

4. THE ROLE OF PROBIOTICS AND PREBIOTICS IN GM REGULATION

Maintaining a GI microbiome compatible with BBs involves lifestyle modifications, including exercise and a regulated healthy diet. Lifestyle modifications that influence the proliferation of BBs and regulate the body's metabolism are regarded as the primary therapy for PCOS [86]. Recently published manuscripts have illustrated that probiotics and prebiotics may help manage PCOS and its associated disorders. Data suggests that the probiotics improve lipid profiles, lower body mass index, and accelerate plasma glucose level [87, 88]. Talebi and colleagues reported in a 2023 study that prebiotics can effectively reduce fasting glucose levels, improve lipid profiles, and reduce waist/hip circumference, all of which replenish GM and may be correlated with PCOS treatment [87, 89, 90].

4. 1. Probiotics

Scientists are currently focusing on the advantageous qualities of probiotics and products made from probiotic fermentation in response to the drawbacks of pharmaceutical therapies. According to Parichat and Pongsak (2023), probiotics are "live microorganisms that, when delivered in suitable quantities, impose beneficial effects on the recipient" [91]. In 2021, Yao and his colleagues updated in their manuscript that in rats with NAFLD, traditional probiotic therapy, such as *Lactobacillus reuters* (GMNL 263), significantly reversed the condition and its associates, such as IR, satiety, intestinal gluconeogenesis, inflammation, and fat deposition [92]. Probiotics could thus represent a viable treatment approach for the management of PCOS, especially in obese adolescents, according to current studies. According to studies, probiotic supplements can help PCOS patients' lipid metabolism, inflammatory indicators, and hormonal profiles [1].

The resistant capacity of probiotic strains against common antibiotics is crucial. Bubnov and his associates 2018 evaluated and reported the probiotic potential and resistant properties of two bifidobacteria strains and six lactic acid bacteria strains. While strains of *L acidophilus* IMV B-7279 and *Bifidobacterium animalis* displayed strong resistance to gastric juice, *Lacticaseibacillus rhamnosus* LB-3 VK6 and *L. delbrueckii* LE VK8 showed high antibiotic resistance. *B. animalis* and *L. casei* IMV B-7280 showed the most potential effects [93]. Another randomized double-blind controlled study by Karamali and his colleagues confirmed that probiotic supplementation rich in *L acidophilus*, *L casei*, and *B bifidum* (2 × 109 CFU/g each) significantly increased serum levels of sex hormone-binding globulin (SHBG), decreased total serum testosterone levels as well as the mFG scores, and improved chronic inflammatory conditions as evidenced by reduced serum concentrations of malondialdehyde (MDA) and high-sensitivity c-reactive protein (hs-CRP) [94].

The 12 weeks administration of probiotics that included *Lactobacillus acidophilus*, *Bifidobacterium bifidum*, *Bifidobacterium langum*, and *Bifidobacterium lactis* on an everyday basis to diabetic retinopathy (DR) intending to improve insulin sensitivity. Positive outcomes were observed; however, lesser improvement was noticed on other metabolic profiles. This was consistent with the previous findings [95].

Therefore, probiotics' positive benefits in PCOS are ascribed to their capacity to regulate IR, inflammation, host metabolism, and reproductive function [96]. Furthermore, probiotics may help address the imbalance in GM communities associated with PCOS and its related symptoms, including HA [97]. These findings imply that probiotic supplementation may be a viable therapeutic approach for treating PCOS.

4. 2. Prebiotics

Prebiotics are non-digestible saccharides that confer health benefits by selectively promoting changes in the composition and activity of the GI microflora that confer benefits upon the host's well-being in the GM [98]. They have been associated with numerous health benefits, including improved gut health, enhanced mineral absorption, reduced blood cholesterol levels, and potential cancer risk reduction [99]. Prebiotics are often incorporated into functional foods or nutraceuticals, sometimes in combination with probiotics as symbiotics. These compounds resist digestion in the small intestine and are fermented by GM in the large intestine, producing short-chain fatty acids (SFAs) and other beneficial compounds [100]. SCFAs are byproducts that are essential for the significant return of NAFLD to normalcy and the prevention of obesity [101]. According to Zeng et al. (2024), these gut metabolites can increase the release of glucagon-like peptide-1 (GLP-1) [102]. A 2024 study by Bu and associates indicates that improved insulin sensitivity in obese people corresponds to increased expression of the GLP-1 receptor (GLP-1R) in adipose tissue. This implies that by encouraging lipolysis in adipose tissue via receptor-dependent pathways, GLP-1 may help reverse IR [103].

Double-blinded placebo-controlled trials in medical settings have evaluated food items with prebiotic properties to improve the clinical results and overall health of individuals with relevant conditions. The findings displayed a significant enhancement of fecal IgA levels compared to the placebo group. This study illustrated that prebiotics could effectively modulate GM composition, particularly an upsurge in bifidobacteria levels [104]. Common plant-based dietary prebiotics are predominantly constituted of inulin and inositol, which are found naturally in various fruits and vegetables [99, 105, 106]. India's varied ethnic communities and tribes contribute distinct culinary practices based on prebiotic-rich local grains and vegetables, including mandua, jhangora, makka, and bhang seeds. These natural products provide various health advantages, such as promoting weight loss, regulating blood sugar levels, protecting kidney function, and supporting heart health due to their abundance of micronutrients, fiber, antioxidants, and polyphenols.

Moreover, locally made fermented drinks improve gut health by offering probiotics like Lactobacillus and Bacillus species. In addition to their health benefits, these foods reflect the rich cultural diversity of India, highlighting the importance of preserving and documenting them for future generations [107]. The rich source of dietary fiber in various Indian staples, cole vegetables, and frequent intake of those functional foods have been documented recently [108]. Inulin-type fiber (ITF), a prebiotic and a potential modulator of GM, is widely present in vegetables, offering the best replacement for junk foods [109]. Research findings by Hiel et al. (2019) indicated that consuming inulin-type fructans increased beneficial Bifidobacterium levels by 3.8-fold while reducing less favorable bacteria, with microbiota shifts normalizing after the intervention, although minimal GI discomfort was reported. Li and colleagues further demonstrated that dietary inulin might help manage obesity-related PCOS by influencing the GM-inflammation-hormone axis, suggesting that inulin-rich foods could be a cost-effective strategy for managing this condition [110, 111].

Foods containing isoforms of inositol, such as myoinositol (MI) and diciroinositol are effective in reversing PCOS [106]. Consumed inositol-depleted PF causes metabolic issues and endocrine dysfunction corresponding to PCOS. Such conditions commonly emerge from inadequate consumption of an inositol-rich diet, impaired absorption of inositol from the consumed food, or alterations in the GM [112]. Unprocessed foods with low molecular weight carbohydrates, such as saccharides, polyalcohols, sugar acids, and glycosides, serve as significant reservoirs of inositol [106]. MI acts as a second messenger for follicular stimulating hormone (FSH) in the ovary and contributes to hypoglycemic effects through a PPAR-gamma-based mechanism, expanding research on its potential in treating PCOS [113]. Previous research has indicated that the dietary inclusion of MI conjugated with Lactobacillus strain (α-LA) is essential. A notable rise in the amount of MI in the plasma has been recorded due to the α-LA in the GM promoting MI absorption. Across a study, MI caused a 62% ovulation rate among 37 anovulatory women. Following treatment with MI + α-LA, 86% of the remaining non-ovulatory women ovulated, successfully enhancing ovulation [114, 115].

5. CONCLUSION

Overall, this review article provides insight into how the DB affects the metabolic pathways in women, leading to PCOS on choosing WDs and PFs as their dietary choice. The cohort of findings validates the decision to adhere to traditional and home-cooked food to regulate the GM, the associated MDs, and PCOS management. The findings have contributed to the detection and analysis of the bacterial profiles related to the occurrence and development of PCOS.

References

[1] Calcaterra, V., Rossi, V., Massini, G., Casini, F., Zuccotti, G., & Fabiano, V. (2023). Probiotics and polycystic ovary syndrome: a perspective for management in adolescents with obesity. *Nutrients*, *15*(14), 3144.

[2] Bharali, M. D., Rajendran, R., Goswami, J., Singal, K., & Rajendran, V. (2022). Prevalence of polycystic ovarian syndrome in India: a systematic review and meta-analysis. *Cureus*, *14*(12).

[3] Zhao, H., Zhang, J., Cheng, X., Nie, X., & He, B. (2023). Insulin resistance in polycystic ovary syndrome across various tissues: an updated review of pathogenesis, evaluation, and treatment. *Journal of Ovarian Research*, *16*(1), 9.

[4] Alhermi, A., Perks, H., Nigi, V., Altahoo, N., Atkin, S. L., & Butler, A. E. (2025). The Role of the Liver in the Pathophysiology of PCOS: A Literature Review. *Biomolecules*, *15*(1), 51.

[5] Begum, R. F., Singh, A., & Mohan, S. (2023). Impact of junk food on obesity and polycystic ovarian syndrome: Mechanisms and management strategies. *Obesity Medicine*, *40*, 100495.

[6] Afzaal, M., Saeed, F., Shah, Y. A., Hussain, M., Rabail, R., Socol, C. T., ... & Aadil, R. M. (2022). Human gut microbiota in health and disease: Unveiling the relationship. *Frontiers in Microbiology*, *13*, 999001.

[7] Khalil, M., Di Ciaula, A., Mahdi, L., Jaber, N., Di Palo, D. M., Graziani, A., ... & Portincasa, P. (2024). Unraveling the role of the human gut Microbiome in Health and diseases. *Microorganisms*, *12*(11), 2333.

[8] Rizzatti, G., Lopetuso, L. R., Gibiino, G., Binda, C., & Gasbarrini, A. (2017). Proteobacteria: a common factor in human diseases. *BioMed Research International*, 2017, 1, 9351507. doi: 10.1155/2017/9351507

[9] Vera-Santander, V. E., Hernández-Figueroa, R. H., Jiménez-Munguía, M. T., Mani-López, E., & López-Malo, A. (2023). Health benefits of consuming foods with bacterial probiotics, postbiotics, and their metabolites: a review. *Molecules*, *28*(3), 1230.

[10] Zeng, Y., Yin, Y., & Zhou, X. (2024). Insights into Microbiota–Host Crosstalk in the Intestinal Diseases Mediated by Extracellular Vesicles and Their Encapsulated MicroRNAs. *International Journal of Molecular Sciences*, *25*(23), 13001.

[11] Zhou, A., Yuan, Y., Yang, M., Huang, Y., Li, X., Li, S., ... & Tang, B. (2022). Crosstalk between the gut microbiota and epithelial cells under physiological and infectious conditions. *Frontiers in Cellular and Infection Microbiology*, *12*, 832672.

[12] Kelly, C. J., Zheng, L., Campbell, E. L., Saeedi, B., Scholz, C. C., Bayless, A. J., ... & Colgan, S. P. (2015). Crosstalk between microbiota-derived short-chain fatty acids and intestinal epithelial HIF augments tissue barrier function. *Cell Host & Microbe*, *17*(5), 662-671.

[13] Zheng, P., Zeng, B., Liu, M., Chen, J., Pan, J., Han, Y., ... & Xie, P. (2019). The gut microbiome from patients with schizophrenia modulates the glutamate-glutamine-GABA cycle and schizophrenia-relevant behaviors in mice. *Science Advances*, *5*(2), eaau8317

[14] Russell, A.L., McAdams, Z.L., Donovan, E. *et al.* The contribution of maternal oral, vaginal, and gut microbiota to the developing offspring gut. *Sci Rep* 13, 13660 (2023). https://doi.org/10.1038/s41598-023-40703-7

[15] Malard, F., Dore, J., Gaugler, B., & Mohty, M. (2021). Introduction to host microbiome symbiosis in health and disease. *Mucosal Immunology*, *14*(3), 547-554.

[16] Redondo-Useros, N., Nova, E., González-Zancada, N., Díaz, L. E., Gómez-Martínez, S., & Marcos, A. (2020). Microbiota and lifestyle: a special focus on diet. *Nutrients*, *12*(6), 1776.

[17] Liu, R., Zhang, C., Shi, Y., Zhang, F., Li, L., Wang, X., Ling, Y., Fu, H., Dong, W., Shen, J., Reeves, A., Greenberg, A. S., Zhao, L., Peng, Y., & Ding, X. (2017). Dysbiosis of Gut Microbiota Associated with Clinical Parameters in Polycystic Ovary Syndrome. *Frontiers in Microbiology*, *8*, 234522. https://doi.org/10.3389/fmicb.2017.00324

[18] Wang, K., Lai, W., Min, T., Wei, J., Bai, Y., Cao, H., ... & Su, Z. (2024). The Effect of Enteric-Derived Lipopolysaccharides on Obesity. *International Journal of Molecular Sciences*, *25*(8), 4305.

[19] Dempsey, E., & Corr, S. C. (2022). Lactobacillus spp. for gastrointestinal health: current and future perspectives. *Frontiers in Immunology*, *13*, 840245.

[20] Guillemard E., Poirel M., Schafer F., Quinquis L., Rossoni C., Keicher C., et al.. (2021). A randomised, controlled trial: Effect of a multi-strain fermented milk on the gut microbiota recovery after helicobacter pylori therapy. *Nutrients* 13, 3171. doi: 10.3390/nu13093171

[21] He FF, Li YM. Role of gut microbiota in the development of insulin resistance and the mechanism underlying polycystic ovary syndrome: a review. *J Ovarian Res.* 2020 Jun 17; 13(1): 73. doi: 10.1186/s13048-020-00670-3

[22] Torres, P. J., Siakowska, M., Banaszewska, B., Pawelczyk, L., Duleba, A. J., Kelley, S. T., & Thackray, V. G. (2018). Gut microbial diversity in women with polycystic ovary syndrome correlates with hyperandrogenism. *The Journal of Clinical Endocrinology & Metabolism*, *103*(4), 1502-1511.

[23] Yang, Y. L., Zhou, W. W., Wu, S., Tang, W. L., Wang, Z. W., Zhou, Z. Y., ... & Zhou, H. W. (2021). Intestinal flora is a key factor in insulin resistance and contributes to the development of polycystic ovary syndrome. *Endocrinology*, *162*(10), bqab118.

[24] Leung, W. T., Tang, Z., Feng, Y., Guan, H., Huang, Z., & Zhang, W. (2022). Lower Fiber consumption in women with polycystic ovary syndrome: a meta-analysis of observational studies. *Nutrients*, *14*(24), 5285.

[25] Wang, X., Xu, T., Liu, R., Wu, G., Gu, L., Zhang, Y., ... & Ding, X. (2022). High-fiber diet or combined with acarbose alleviates heterogeneous phenotypes of polycystic ovary syndrome by regulating gut microbiota. *Frontiers in Endocrinology*, *12*, 806331.

[26] Rasaei, N., Fatemi, S. F., Gholami, F., Samadi, M., Mohammadian, M. K., Daneshzad, E., & Mirzaei, K. (2025). Interaction of genetics risk score and fatty acids quality indices on healthy and unhealthy obesity phenotype. *BMC Medical Genomics*, *18*(1), 16

[27] Clemente-Suárez, V. J., Beltrán-Velasco, A. I., Redondo-Flórez, L., Martín-Rodríguez, A., & Tornero-Aguilera, J. F. (2023). Global impacts of western diet and its effects on metabolism and health: A narrative review. *Nutrients*, *15*(12), 2749.

[28] Bharali, M. D., Rajendran, R., Goswami, J., Singal, K., & Rajendran, V. (2022). Prevalence of polycystic ovarian syndrome in India: a systematic review and meta-analysis. *Cureus*, *14*(12).

[29] Zhou, P., Feng, P., Liao, B. *et al.* Role of polyphenols in remodeling the host gut microbiota in polycystic ovary syndrome. *J Ovarian Res* 17, 69 (2024). https://doi.org/10.1186/s13048-024-01354-y

[30] Netto Candido, T. L., Bressan, J., & Alfenas, R. C. G. (2018). Dysbiosis and metabolic endotoxemia induced by high-fat diet. Disbiosis y endotoxemia metabólica inducidas por la dieta rica en grasa. *Nutricion Hospitalaria*, *35*(6), 1432–1440. https://doi.org/10.20960/nh.1792

[31] Louwers, Y. V., & Laven, J. S. (2020). The polycystic ovary syndrome (PCOS). *Female reproductive dysfunction*, 1-23.

[32] Dong, Y., Yang, S., Zhang, S., Zhao, Y., Li, X., Han, M., ... & Zou, K. (2025). Modulatory impact of Bifidobacterium longum subsp. longum BL21 on the gut–brain–ovary axis in polycystic ovary syndrome: insights into metabolic regulation, inflammation mitigation, and neuroprotection. *mSphere*, e00887-24.

[33] Cowan, S., Lim, S., Alycia, C., Pirotta, S., Thomson, R., Gibson-Helm, M., ... & Moran, L. (2023). Lifestyle management in polycystic ovary syndrome–beyond diet and physical activity. *BMC Endocrine Disorders*, *23*(1), 14.

[34] Redondo-Useros, N., Nova, E., González-Zancada, N., Díaz, L. E., Gómez-Martínez, S., & Marcos, A. (2020). Microbiota and lifestyle: a special focus on diet. *Nutrients*, *12*(6), 1776.

[35] Brichacek, A. L., Florkowski, M., Abiona, E., & Frank, K. M. (2024). Ultra-processed foods: a narrative review of the impact on the human gut microbiome and variations in classification methods. *Nutrients*, *16*(11), 1738.

[36] Miclotte, L., & Van de Wiele, T. (2020). Food processing, gut microbiota and the globesity problem. *Critical Reviews in Food Science and Nutrition*, *60*(11), 1769-1782.

[37] Ferreira, R. D. S., Mendonça, L. A. B. M., Ribeiro, C. F. A., Calças, N. C., Guimarães, R. D. C. A., Nascimento, V. A. D., ... & Franco, O. L. (2022). Relationship between intestinal microbiota, diet and biological systems: an integrated view. *Critical Reviews in Food Science and Nutrition*, *62*(5), 1166-1186.

[38] Kumar, S., & Singh, C. (2022). Productivity growth in India's bakery manufacturing industry. *Journal of Agribusiness in Developing and Emerging Economies*, *12*(1), 94-103.

[39] Dhir and Singla. 2020. Consumption Pattern and Health Implications of Convenience Foods: A Practical Review. *Current Journal of Applied Science and Technology* 38 (6): 1-9. DOI: 10.9734/CJAST/2019/v38i630455

[40] Dhir, Bhavya, Neerja Singla, and Rohini Jain. 2020. Relationship Between Consumption of Convenience Foods and Health Status of the Working Women. *Current Journal of Applied Science and Technology* 39 (3): 87-94. https://doi.org/10.9734/cjast/2020/v39i330518

[41] Szynal, K., Polaniak, R., Górski, M., Grajek, M., Ciechowska, K., & Grochowska-Niedworok, E. (2021). Processed Food And Food Additives In The Context Of Dysbiosis And Its Health Consequences. *Advancements of Microbiology*, *60*(3), 223-230.

[42] Brichacek, A. L., Florkowski, M., Abiona, E., & Frank, K. M. (2024). Ultra-processed foods: a narrative review of the impact on the human gut microbiome and variations in classification methods. *Nutrients*, *16*(11), 1738.

[43] Jawhara, S. (2023). Healthy diet and lifestyle improve the gut microbiota and help combat fungal infection. *Microorganisms*, *11*(6), 1556

[44] Macuglia Spanemberg, F. E., Sellitto, M. A., Mailan Porto, L., Cruz dos Santos, A., & Canez Lemos Souza, Á. (2022). Shelf life estimation of glassy confections using moisture sorption isotherms. *Journal of Food Process Engineering*, *45*(5), e14024.

[45] Sun, S., Araki, Y., Hanzawa, F., Umeki, M., Kojima, T., Nishimura, N., ... & Oda, H. (2021). High sucrose diet-induced dysbiosis of gut microbiota promotes fatty liver and hyperlipidemia in rats. *The Journal of Nutritional Biochemistry*, *93*, 108621.

[46] Targher, G., Rossini, M., & Lonardo, A. (2016). Evidence that non-alcoholic fatty liver disease and polycystic ovary syndrome are associated by necessity rather than chance: a novel hepato-ovarian axis? *Endocrine*, *51*, 211-221.

[47] Thaiss, C. A., Levy, M., Grosheva, I., Zheng, D., Soffer, E., Blacher, E., ... & Elinav, E. (2018). Hyperglycemia drives intestinal barrier dysfunction and risk for enteric infection. *Science*, *359*(6382), 1376-1383.

[48] Fajstova, A., Galanova, N., Coufal, S., Malkova, J., Kostovcik, M., Cermakova, M., ... & Kostovcikova, K. (2020). Diet rich in simple sugars promotes pro-inflammatory response via gut microbiota alteration and TLR4 signaling. *Cells*, *9*(12), 2701.

[49] Khan, T. A., Lee, J. J., Ayoub-Charette, S., Noronha, J. C., McGlynn, N., Chiavaroli, L., & Sievenpiper, J. L. (2023). WHO guideline on the use of non-sugar sweeteners: a need for reconsideration. *European Journal of Clinical Nutrition*, *77*(11), 1009-1013.

[50] Nadolsky, K. Z. (2021). COUNTERPOINT: artificial sweeteners for obesity—better than sugary alternatives; potentially a solution. *Endocrine Practice*, *27*(10), 1056-1061.

[51] Wilk, K., Korytek, W., Pelczyńska, M., Moszak, M., & Bogdański, P. (2022). The Effect of Artificial Sweeteners Use on Sweet Taste Perception and Weight Loss Efficacy: A Review. *Nutrients*, *14*(6), 1261. https://doi.org/10.3390/nu14061261

[52] Rogers, P. J. (2024). Non-nutritive sweeteners and body weight management: another brick in the wall of evidence. *International Journal of Obesity*, *48*(1), 1-2.

[53] Laster, J., Bonnes, S. L., & Rocha, J. (2019). Increased use of emulsifiers in processed foods and the links to obesity. *Current gastroenterology reports*, *21*, 1-6

[54] Zhang, Y., Chen, L., Gao, J., Cheng, Y., Luo, F., Bai, X., & Ding, H. (2023). Nutritive/non-nutritive sweeteners and high fat diet contribute to dysregulation of sweet taste receptors and metabolic derangements in oral, intestinal and central nervous tissues. *European Journal of Nutrition*, *62*(8), 3149-3159.

[55] Debras, C., Chazelas, E., Srour, B., Druesne-Pecollo, N., Esseddik, Y., de Edelenyi, F. S., ... & Touvier, M. (2022). Artificial sweeteners and cancer risk: Results from the NutriNet-Santé population-based cohort study. *PLoS Medicine*, *19*(3), e1003950.

[56] Conz, A., Salmona, M., & Diomede, L. (2023). Effect of non-nutritive sweeteners on the gut microbiota. *Nutrients*, *15*(8), 1869.

[57] Binns, N. M. (2003). Sucralose–all sweetness and light. *Nutrition Bulletin*, *28*(1).

[58] Del Pozo, S., Gómez-Martínez, S., Díaz, L. E., Nova, E., Urrialde, R., & Marcos, A. (2022). Potential effects of sucralose and saccharin on gut microbiota: a review. *Nutrients*, *14*(8), 1682.

[59] Wu, W., Sui, W., Chen, S., Guo, Z., Jing, X., Wang, X., ... & Cao, Y. (2025). Sweetener aspartame aggravates atherosclerosis through insulin-triggered inflammation. *Cell Metabolism*. https://doi.org/10.1016/j.cmet.2025.01.006

[60] Jin, Q., Xu, G., Ying, Y., Liu, L., Zheng, H., Xu, S., ... & Chen, Y. (2025). Effects of non-pharmacological interventions on biochemical hyperandrogenism in women with polycystic ovary syndrome: a systematic review and network meta-analysis. *Journal of Ovarian Research*, *18*(1), 8.

[61] Turner, A., Veysey, M., Keely, S., Scarlett, C. J., Lucock, M., & Beckett, E. L. (2020). Intense sweeteners, taste receptors and the gut microbiome: a metabolic health perspective. *International Journal of Environmental Research and Public Health*, *17*(11), 4094

[62] Posta, E., Fekete, I., Gyarmati, E., Stündl, L., Zold, E., & Barta, Z. (2023). The Effects of Artificial Sweeteners on Intestinal Nutrient-Sensing Receptors: Dr. Jekyll or Mr. Hyde? *Life*, *14*(1), 10

[63] Moonwiriyakit, A., Pathomthongtaweechai, N., Steinhagen, P. R., Chantawichitwong, P., Satianrapapong, W., & Pongkorpsakol, P. (2023). Tight junctions: from molecules to gastrointestinal diseases. *Tissue Barriers*, *11*(2), 2077620.

[64] Severino, A., Tohumcu, E., Tamai, L., Dargenio, P., Porcari, S., Rondinella, D., ... & Ianiro, G. (2024). The microbiome-driven impact of western diet in the development of noncommunicable chronic disorders. *Best Practice & Research Clinical Gastroenterology*, 101923.

[65] Daniel, N., Gewirtz, A. T., & Chassaing, B. (2023). Akkermansia muciniphila counteracts the deleterious effects of dietary emulsifiers on microbiota and host metabolism. *Gut*, *72*(5), 906-917.

[66] Zhang, X., Wang, Q., Liu, Z., Zhi, L., Jiao, B., Hu, H., ... & Shi, A. (2023). Plant protein-based emulsifiers: Mechanisms, techniques for emulsification enhancement and applications. *Food Hydrocolloids*, 144, 109008. https://doi.org/10.1016/j.foodhyd.2023.109008

[67] Ogulur, I., Yazici, D., Pat, Y., Bingöl, E. N., Babayev, H., Ardicli, S., ... & Akdis, C. A. (2023). Mechanisms of gut epithelial barrier impairment caused by food emulsifiers polysorbate 20 and polysorbate 80. *Allergy*, *78*(9), 2441-2455

[68] De Siena, M., Raoul, P., Costantini, L., Scarpellini, E., Cintoni, M., Gasbarrini, A., ... & Mele, M. C. (2022). Food emulsifiers and metabolic syndrome: the role of the gut microbiota. *Foods*, *11*(15), 2205.

[69] Naimi, S., Viennois, E., Gewirtz, A. T., & Chassaing, B. (2021). Direct impact of commonly used dietary emulsifiers on human gut microbiota. *Microbiome*, *9*, 1-19.

[70] Panyod, S., Wu, W. K., Chang, C. T., Wada, N., Ho, H. C., Lo, Y. L., ... & Sheen, L. Y. (2024). Common dietary emulsifiers promote metabolic disorders and intestinal microbiota dysbiosis in mice. *Communications Biology*, *7*(1), 749

[71] Garbiec, E., Cielecka-Piontek, J., Kowalówka, M., Hołubiec, M., & Zalewski, P. (2022). Genistein—opportunities related to an interesting molecule of natural origin. *Molecules*, *27*(3), 815.

[72] Xiang, J., Mlambo, R., Dube, P., Machona, O., Shaw, I., Seid, Y., ... & Tan, S. (2023). The obesogenic side of Genistein. *Frontiers in Endocrinology*, *14*, 1308341.

[73] Malik, P., Singh, R., Kumar, M., Malik, A., & Mukherjee, T. K. (2023). Understanding the phytoestrogen genistein actions on breast cancer: Insights on estrogen receptor equivalence, pleiotropic essence and emerging paradigms in bioavailability modulation. *Current Topics in Medicinal Chemistry*, *23*(15), 1395-1413

[74] Mohammad, S., & Thiemermann, C. (2021). Role of metabolic endotoxemia in systemic inflammation and potential interventions. *Frontiers in Immunology*, *11*, 594150.

[75] Dai, Y. J., Liu, W. B., Abasubong, K. P., Zhang, D. D., Li, X. F., Xiao, K., ... & Jiang, G. Z. (2022). The mechanism of lipopolysaccharide escaping the intestinal barrier in Megalobrama amblycephala fed a high-fat diet. *Frontiers in Nutrition*, *9*, 853409.

[76] Tomassen, M. M., Govers, C., Vos, A. P., & De Wit, N. J. (2023). Dietary fat induced chylomicron-mediated LPS translocation in a bicameral Caco-2cell model. *Lipids in Health and Disease*, *22*(1), 4.

[77] Park, J. E., Park, H. Y., Kim, Y. S., & Park, M. (2024). The Role of Diet, Additives, and Antibiotics in Metabolic Endotoxemia and Chronic Diseases. *Metabolites*, *14*(12), 704.

[78] Ruder, B., Atreya, R., & Becker, C. (2019). Tumour necrosis factor alpha in intestinal homeostasis and gut-related diseases. *International Journal of Molecular Sciences*, *20*(8), 1887.

[79] Webster, J. D., & Vucic, D. (2020). The balance of TNF mediated pathways regulates inflammatory cell death signaling in healthy and diseased tissues. *Frontiers in Cell and Developmental Biology*, *8*, 365.

[80] Paradis, T., Bègue, H., Basmaciyan, L., Dalle, F., & Bon, F. (2021). Tight junctions as a key for pathogens invasion in intestinal epithelial cells. *International Journal of Molecular Sciences*, *22*(5), 2506.

[81] Alizadeh, A., Akbari, P., Garssen, J., Fink-Gremmels, J., & Braber, S. (2022). Epithelial integrity, junctional complexes, and biomarkers associated with intestinal functions. *Tissue Barriers*, *10*(3), 1996830.

[82] Ghosh, S. S., Wang, J., Yannie, P. J., & Ghosh, S. (2020). Intestinal barrier dysfunction, LPS translocation, and disease development. *Journal of the Endocrine Society*, *4*(2), bvz039..

[83] Lingaiah, S., Arffman, R. K., Morin-Papunen, L., Tapanainen, J. S., & Piltonen, T. (2021). Markers of gastrointestinal permeability and dysbiosis in premenopausal women with PCOS: a case–control study. *BMJ Open*, *11*(7), e045324.

[84] Yang, M., Gu, Y., Li, L., Liu, T., Song, X., Sun, Y., ... & Cao, H. (2021). Bile acid–gut microbiota axis in inflammatory bowel disease: from bench to bedside. *Nutrients*, *13*(9), 3143.

[85] Zhu, F., Zheng, S., Zhao, M., Shi, F., Zheng, L., & Wang, H. (2023). The regulatory role of bile acid microbiota in the progression of liver cirrhosis. *Frontiers in Pharmacology*, *14*, 1214685.

[86] Sivasankari, R., & Usha, B. (2022). Reshaping the gut microbiota through lifestyle interventions in women with PCOS: a review. *Indian Journal of Microbiology*, *62*(3), 351-363.

[87] Angoorani, P., Ejtahed, H. S., Ettehad Marvasti, F., Taghavi, M., Mohammadpour Ahranjani, B., Hasani-Ranjbar, S., & Larijani, B. (2023). The effects of probiotics, prebiotics, and synbiotics on polycystic ovarian syndrome: an overview of systematic reviews. *Frontiers in Medicine*, *10*, 1141355.

[88] Li, Y., Tan, Y., Xia, G., & Shuai, J. (2023). Effects of probiotics, prebiotics, and synbiotics on polycystic ovary syndrome: a systematic review and meta-analysis. *Critical Reviews in Food Science and Nutrition*, *63*(4), 522-538.

[89] Talebi, S., Zeraattalab-Motlagh, S., Jalilpiran, Y., Payandeh, N., Ansari, S., Mohammadi, H., ... & Shab-Bidar, S. (2023). The effects of pro-, pre-, and synbiotics supplementation on polycystic ovary syndrome: an umbrella review of meta-analyses of randomized controlled trials. *Frontiers in Nutrition*, *10*, 1178842.

[90] Yurtdaş, G., & Akdevelioğlu, Y. (2020). A new approach to polycystic ovary syndrome: the gut microbiota. *Journal of the American College of Nutrition*, *39*(4), 371-382.

[91] Parichat, P., & Pongsak, R. (2023). Probiotics: Sources, selection and health benefits. *Research Journal of Biotechnology 18*, 5.

[92] Yao, M., Qv, L., Lu, Y., Wang, B., Berglund, B., & Li, L. (2021). An update on the efficacy and functionality of probiotics for the treatment of non-alcoholic fatty liver disease. *Engineering*, *7*(5), 679-686.

[93] Bubnov, R. V., Babenko, L. P., Lazarenko, L. M., Mokrozub, V. V., & Spivak, M. Y. (2018). Specific properties of probiotic strains: relevance and benefits for the host. *EPMA Journal*, *9*, 205-223.

[94] Karamali, M., Eghbalpour, S., Rajabi, S., Jamilian, M., Bahmani, F., TajabadiEbrahimi, M., ... & Asemi, Z. (2018). Effects of probiotic supplementation on hormonal profiles, biomarkers of inflammation and oxidative stress in women with polycystic ovary syndrome: a randomized, doubleblind, placebo-controlled trial. *Archives of Iranian Medicine*, *21*(1), 1-7.

[95] Farajipour, H., Matin, H. R., Asemi, Z., Sadr, S., Tajabadi-Ebrahimi, M., Sharifi, N., ... & Mirzaei, H. (2024). The effects of probiotics supplements on metabolic indices and clinical signs in patients with diabetic retinopathy, a randomized double blind clinical trial. *Journal of Diabetes & Metabolic Disorders*, *23*(1), 1133-1140.

[96] Martinez Guevara, D., Vidal Cañas, S., Palacios, I., Gómez, A., Estrada, M., Gallego, J., & Liscano, Y. (2024). Effectiveness of Probiotics, Prebiotics, and Synbiotics in Managing Insulin Resistance and Hormonal Imbalance in Women with Polycystic

Ovary Syndrome (PCOS): A Systematic Review of Randomized Clinical Trials. *Nutrients*, *16*(22), 3916.

[97] Schiattarella, A., Riemma, G., Verde, M. L., Franci, G., Chianese, A., Fasulo, D., ... & De Franciscis, P. (2021). Polycystic ovary syndrome and probiotics: a natural approach to an inflammatory disease. *Current Women's Health Reviews*, *17*(1), 14-20.

[98] Basavaiah, R., & Gurudutt, P. S. (2021). Prebiotic Carbohydrates for Therapeutics. *Endocrine, Metabolic & Immune Disorders-Drug Targets (Formerly Current Drug Targets-Immune, Endocrine & Metabolic Disorders)*, *21*(2), 230-245.

[99] Rahim, M. A., Saeed, F., Khalid, W., Hussain, M., & Anjum, F. M. (2021). Functional and nutraceutical properties of fructo-oligosaccharides derivatives: a review. *International Journal of Food Properties*, *24*(1), 1588-1602.

[100] Pedrosa, L. D. F., de Vos, P., & Fabi, J. P. (2024). From Structure to Function: How Prebiotic Diversity Shapes Gut Integrity and Immune Balance. *Nutrients*, *16*(24), 4286.

[101] Zhang, S., Zhao, J., Xie, F., He, H., Johnston, L. J., Dai, X., ... & Ma, X. (2021). Dietary fiber-derived short-chain fatty acids: A potential therapeutic target to alleviate obesity-related nonalcoholic fatty liver disease. *Obesity Reviews*, *22*(11), e13316.

[102] Zeng, Y., Wu, Y., Zhang, Q., & Xiao, X. (2024). Crosstalk between glucagon-like peptide 1 and gut microbiota in metabolic diseases. *Mbio*, *15*(1), e02032-23.

[103] Bu, T., Sun, Z., Pan, Y., Deng, X., & Yuan, G. (2024). Glucagon-like peptide-1: new regulator in lipid metabolism. *Diabetes & Metabolism Journal*, *48*(3), 354-372.

[104] Kaushal, D., & Kalsi, G. (2024). Prebiotics and vitamins enhance gut barrier in a randomized double-blind placebo-controlled trial. *Nutrition & Food Science*. https://doi.org/10.1108/NFS-08-2024-0272

[105] Xie, A., Dong, Y., Liu, Z., Li, Z., Shao, J., Li, M., & Yue, X. (2023). A review of plant-based drinks addressing nutrients, flavor, and processing technologies. *Foods*, *12*(21), 3952.

[106] Joardar, S., Duarah, P., & Purkait, M. K. (2023). Recent advances in myo-inositol recovery and purification from agricultural sources as potential dietary supplements: A review. *Sustainable Chemistry and Pharmacy*, *36*, 101331.

[107] Dwivedi, S., Singh, V., Mahra, K., Sharma, K., Baunthiyal, M., & Shin, J. H. (2024). Functional foods in the northwestern Himalayan Region of India and their significance: a healthy dietary tradition of Uttarakhand and Himachal Pradesh. *Journal of Ethnic Foods*, *11*(1), 20.

[108] Kalia, P., & Singh, S. (2023). Nutritional enhancement of vegetable crops (with major emphasis on Broccoli: a new cole crop in India). In *Vegetables for Nutrition and Entrepreneurship* (pp. 1-29). Singapore: Springer Nature Singapore.

[109] Strieder, M. M., Arruda, H. S., Pastore, G. M., & Silva, E. K. (2023). Inulin-type dietary fiber stability after combined thermal, mechanical, and chemical stresses related to ultrasound processing of prebiotic apple beverage. *Food Hydrocolloids*, *139*, 108489.

[110] Hiel, S., Bindels, L. B., Pachikian, B. D., Kalala, G., Broers, V., Zamariola, G., ... & Delzenne, N. M. (2019). Effects of a diet based on inulin-rich vegetables on gut health and nutritional behavior in healthy humans. *The American Journal of Clinical Nutrition*, *109*(6), 1683-1695.

[111] Li, X., Jiang, B., Gao, T., Nian, Y., Bai, X., Zhong, J., ... & Ma, X. (2024). Effects of inulin on intestinal flora and metabolism-related indicators in obese polycystic ovary syndrome patients. *European Journal of Medical Research*, *29*(1), 443.

[112] Caputo, M., Bona, E., Leone, I., Samà, M. T., Nuzzo, A., Ferrero, A., ... & Prodam, F. (2020). Inositols and metabolic disorders: From farm to bedside. *Journal of Traditional and Complementary Medicine*, *10*(3), 252-259

[113] Shokrpour, M., Foroozanfard, F., Afshar Ebrahimi, F., Vahedpoor, Z., Aghadavod, E., Ghaderi, A., & Asemi, Z. (2019). Comparison of myo-inositol and metformin on glycemic control, lipid profiles, and gene expression related to insulin and lipid metabolism in women with polycystic ovary syndrome: a randomized controlled clinical trial. *Gynecological Endocrinology*, *35*(5), 406-411.

[114] Kamenov, Z., Gateva, A., Dinicola, S., & Unfer, V. (2023). Comparing the efficacy of myo-inositol plus α-lactalbumin vs. Myo-inositol alone on reproductive and metabolic disturbances of polycystic ovary syndrome. *Metabolites*, *13*(6), 717.

[115] Montanino Oliva, M., Buonomo, G., Calcagno, M., & Unfer, V. (2018). Effects of myo-inositol plus alpha-lactalbumin in myo-inositol-resistant PCOS women. *Journal of Ovarian Research*, *11*, 1-7