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Effects of Pectin on Intestinal Microbiota and Human Health

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Abstract. Gut microbes are important players in human metabolism and their genomes are abundant in genes that regulate the metabolism of SFCAs, methane, amino acids, and carbohydrates. Pectin is a water-soluble dietary fiber, an abundant heteropolysaccharide in the main and intermediate cellular lamellae of plant cell walls. Pectin is degraded by a varied group of microorganisms residing in the human gut, which depolymerize pectin by secreting both cell-bound and extracellular enzymes to produce monosaccharides. The pectin fermentation degradation pathway is the main mechanism for the degradation of carbohydrates that cannot be digested in human GIT, and the end products include SCFAs, ethanol, CO_2 and H_2. The makeup of intestinal microbiota is impacted by these products. Additionally, the effect of pectin on intestinal microbial fractions is related to the structure of pectin, the intestinal environment and the degree of pectin esterification. Moreover, pectin itself and pectin degradation products contribute to human health by influencing intestinal microorganisms. Pectin is effective in modifying allergies to sensitization, reducing body composition and has a preventive effect on type I diabetes.

Keywords: pectin, human intestinal flora, human body health

1. Introduction

Intestinal microbes refer to the large number of microbial communities that present in the host's gut. They rely on the host's gut to live, and the human gut provides a good habitat for microbes. The amount of intestinal microbes is up to 10^{14}, around 10 times the amount of human somatic cells, and the weight reaches 1.2 kg, close to the quality of the human liver. Research has indicated that gut microorganisms are important players in human metabolism, and their genomes contain several genes that the human body lacks, including a large number engaged in the metabolism of amino acids, carbohydrates, methane, and short-chain fatty acids (SCFAs) [1]. Furthermore, major functions of gut microbiota include carbohydrate metabolism, energy conversion, and cellular subdivision [2]. Intestinal microbiota generally keeps the environment both within and outside the human body in a dynamic balance. When intestinal microbiota is dysregulated, it can lead to digestive system diseases, metabolic diseases and other diseases.

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Dietary fiber is an edible component of plants or carbohydrate analogs that is fermented entirely or partially in the large intestine rather than being digested and absorbed in the small intestine. Dietary fiber is composed of polysaccharides, oligosaccharides, lignin, and other related plant substances. It is mainly derived from unprocessed grains and their products, fruits, and crude fiber.

Pectin is mostly a water-soluble dietary fiber, and it is a heterosaccharide found in the main wall and middle layers of the plant cell wall (Figure 1) [3]. Natural pectins are widely present in the roots, stems, leaves and fruits of plants in the form of protopectins, pectins and pectins. Pectin can promote gastrointestinal peristalsis, be digested by intestinal microorganisms to generate SCFAs, control the digestion of lipids, inhibit the synthesis of liver cholesterol, and promote the proliferation of probiotics. Studies have shown that pectin can prevent cancer, diabetes and other diseases.

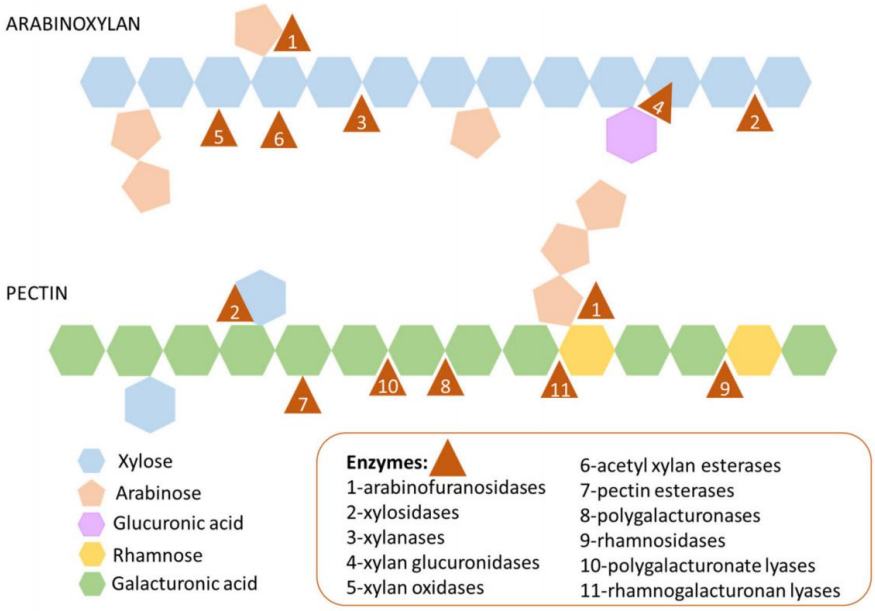


Figure 1. Diagrams for the structure of citrus pectin.

In this paper, metabolic process of pectin in intestine, the effect of pectin on intestinal microbial diversity and metabolism, and the relationship between pectin and human health were summarized, so as to provide some reference for the study of the regulation of pectin and intestinal microorganisms.

2. The effect of pectin on intestinal flora

2.1. Influence of intestinal flora on pectin metabolism

Human digestive enzymes are unable to disintegrate the bulk of glycosidic connections within the pectin molecule since it is a non-digestible plant polysaccharide [4]. Therefore, the many microbial populations that live in the human gut are responsible for pectin breakdown. Ingested pectin reaches the large intestine almost intact and is subjected to the action of a small number of microorganisms present in it, which usually have the enzymatic mechanisms required to degrade pectin. Plant polysaccharides, like pectin, have high molecular weights, making it unable to directly absorb whole molecules for intracellular breakdown. Instead, the pectin breakdown process entails the secretion of extracellular cell-free and cell-bound enzymes that depolymerize the pectin structure and remove the secondary side chains to create mono/oligosaccharides that can then be ingested and used for intracellular catabolic metabolism.

There are twelve distinct phyla found in human gut among the representatives of the phylum Bacteroides and Thick-walled Bacteroides often make up between 50 and 70 percent of the microbial population and the four phyla Bacteroides, Thick-walled Bacteroides, Aspergillus and Actinomycetes

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account for over 95% of the microflora [5]. Fermentation processes dominate in the human gut because of the hypoxic circumstances and lack of alternative electron acceptors, with 100 to 1000 times more strictly anaerobic bacteria than aerobic and parthenogenic anaerobes [6]. The fermentation degradation pathway is the main mechanism for the degradation of indigestible carbohydrates, including pectin, in human GIT, ultimately producing SCFAs, ethanol, carbon dioxide (CO_2), and H_2. (Figure 2). The fermentation products are taken up by the host cells as a source of energy and mediate a series of physiological effects that will proceed in the next stage.

In 1941, it was discovered that microbes have a role in the process of dietary pectin breakdown. Through

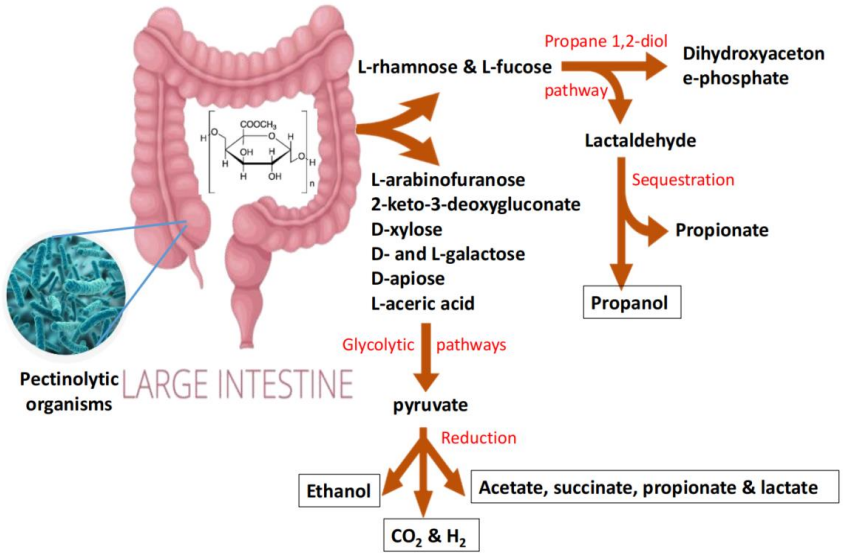


Figure 2. Outline of the human intestine microbiota's methods for degrading pectin.

enrichment and separation on growing media with pectin as the only or primary carbon source, follow-up research has isolated pure cultures of pectin-degrading bacteria from human feces and tested the pectin-degrading potential of previously isolated common media [7, 8]. Genomic analysis has recently been used to pinpoint the genes for carbohydrate-active enzymes (CAZymes) in a number of GIT isolates and predict their pectin-degrading potential [9]. Thick-walled Bacteria and members of the phylum Bacillariophyta have both been shown to be capable of decomposing pectin thus far.

The extracellular cell-free and cell attached enzymes used in the pectinolytic microbial process depolymerize the pectin backbone and eliminate the secondary side chains. Glycolic acids and sugars generated, such as 2-Keto-3-deoxygluconate, L-arabinose, D-xylose, L-rhamnose, L-focus, D- and L-galactose are all available to a wider range of secondary degraders present in the gut microbiota in addition to serving as carbon and energy sources for the pectin-degrading members of the community.

2.2. Pectin affects intestinal microbial diversity

Pectin and pectin-like substances have a significant regulatory effect on intestinal microorganisms. Martinov et al studied the increase of E. coli and S. aureus in the presence of pectin-derived oligosaccharides and hydroxyl radicals and found that pectin oligosaccharides reacted with hydroxyl radicals to produce carbon dioxide radical anions and that apple pectin oligosaccharides significantly inhibited the increase of E. coli and S. aureus [10]. The low-molecular-mass oligosaccharide products after pectin depolymerization were able to promote more significantly the multiplication of Bifidobacterium and Lactobacillus and to produce large amounts of lactic acid, propionic acid and acetic acid [11]. The growth-promoting properties of citrus pectin oligosaccharides for bifidobacteria were also evaluated by fecal fermentation experiments. Although many studies have demonstrated that oligosaccharides, including pectin oligosaccharides, have bifidobacterial growth promoting properties, the relationship and the mechanism of activity are still unclear and need further in-depth study [12].

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The fine structural features of pectin specifically activate the polysaccharide utilization site genes (PUL) of specific intestinal microorganisms, which induce the secretion of related glycosidases, and multiple specific glycosidases synergistically degrade the corresponding pectin structural units, and the resulting energy enables the rapid proliferation of the specific genus, which leads to population dominance. In contrast to mice exposed to the same fiber following depolymerization treatment, RG-I enriched pectin animals had different microbiota profiles, suggesting a possible connection between pectin structure and gut microbiota composition. Compared to caracal berries, which have a largely side chain structure, the pectin derived from spinach has a predominantly linear chain structure that facilitates the growth of Bacillus mimicus [13].

The intestinal microorganisms generate metabolites such as SCFAs and carbon dioxide while degrading pectin fragments, which in turn inhibit the proliferation of non-dominant or harmful microorganisms by altering the pH of the intestinal environment, thus exerting a probiotic effect. Bacillus spp. and Prevotella spp. are the main pectin degraders. These bacteria use lytic enzymes, methylestrolases and acetylases to break down pectin mole fractions and the desubstrates can be used as growth substrates by surrounding bacteria, thus affecting the composition of the microbial community. Cultivation of the genus of bacteria that dominates the gut flora in a special polysaccharide mixture illustrated the dominance of Ovomycetes in the utilization of pectin and production of major propionic acid SCFAs; while Prauschnitzia faecalis had a significant advantage over Polymorphomycetes and Eumycetes in co-culture utilizing apple pectin as the main carbon source, while producing large amounts of butyrate as the major metabolite [14].

Depending on the pectin methoxyl content, pectins can be divided into high ester pectins (esterification above 50%) with 7.0%-16.3% methoxyl content and low ester pectins (esterification below 50%) corresponding to ≤7% methoxyl content (Figure 3); Fak et al found that compared to low ester pectins (DM 20%), high ester pectins (DM 70%) more significantly increased the short chain [15]. This phenomenon may be attributed to the fact that high ester pectin allows more complete fermentation and produces more SCFAs, while low ester pectin has more carboxyl groups and forms more molecular cross-links at multiple sites, thus hindering the microbial enzymatic process. When feeding experimental rats, the addition of highly esterified pectin to their diets reduced Oscillospira and Ruminococca- ceae in the colon and promoted acetic and propionic acid production, while the addition of 3% of low esterified pectin to the diet increased the colonic concentrations of Prevotella, Dialister, Ruminococcaceae sp. in healthy piglets as well as butyric and propionic acid concentrations [16].

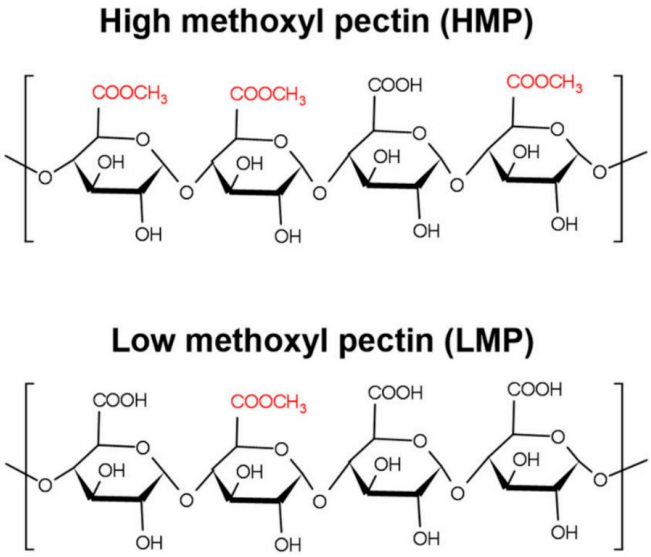


Figure 3. Schematic of the heteropolysaccharide pectin's chemical structure showing variations in esterification levels.

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3. Pectin promotes human health

3.1. Immunity

Prebiotics are considered an immunoactive agent with a beneficial function to human health. Factors such as environmental factors, certain drugs, low-fiber/ high-fat diets, and antimicrobial agents can cause gut microbiota dysbiosis that can lead to allergies [17]. However, ingestion of prebiotics such as dietary fiber maintains the homeostasis of gut microbiota. Prebiotics can promote health of host in two ways: by increasing the amount of microbial metabolites entering the gut, and then by injecting some molecular and cellular processes, or by promoting immune responses in cells such as intestinal epithelial cells and immune cells [18]. In addition, intestinal microbes can produce SCFAs for dietary fiber fermentation, which have immunomodulatory properties. SCFAs, on the other hand, can regulate human immunological reaction through three ways: (I) direct activation of g-coupled receptors, (II) induced epigenetic modification through inhibition of histone deacetylase, and (III) serves as an energy substrate for immune cells and non-immune cells [19]. Pectin, as one kind of dietary fiber, has the above properties. Additional evidence indicates that SCFAs can influence the epigenome by metabolically regulatory receptors, with the potential to reduce allergy [20]. Moreover, the skeletons of pectin macromolecules also have immunosuppressive activity [21]. Pectin can form a new product through the enzymatic reaction of certain enzymes, namely, pectin-derived oligosaccharides (POS), and one of the main effects of this component is to resist the invasion of various pathogens [22].

Pectin has various immunomodulatory properties that can directly affect immune cells, and the products of pectin being metabolized by gut microorganisms also have immunomodulatory properties. [23]. Before dietary fiber is metabolically degraded by microbes in large intestine, they will interact with immune barrier cells in small intestine. There is a thin, loose layer of mucus in small intestine. And the mucus layer can allow molecules like dietary fiber to interact with immune cells and intestinal epithelial [24]. When pectin interacts with the intestinal immune barrier cells, it can enhance mucus layer, strengthen barrier function of epithelial cells, and modulate the intestinal immune response. Dietary fiber can improve human health and prevent disease because of its interaction with the intestinal immune system [25].

Pectin can influence growth of certain gut microbial communities, and can also be used as a substrate for the enzymatic digestion of gut microorganisms. Furthermore, the stimulation of the commensal microflora growth through dietary fiber may help to limit or reduce the growth of pathogens adhering to intestinal wall [26].

Pectins can enhance mucus layer by certain mechanisms. And the strengthening of the mucus layer will restrict the entry of harmful substances into the underlying tissues, and then preventing the inflammatory response [27]. Dietary fiber is beneficial in promoting the integrity of the gastrointestinal epithelial cell layers. Dietary fiber can stimulate gut microbial metabolism to produce SCFAs, and it can directly interact with pattern recognition receptors (PRRs) to enhance epithelial cell integrity. Furthermore, pectin can also directly interact with innate immune cells, like dendritic cells or macrophages, and then affect their response. There are in vitro studies showing that pectin will interact directly with immune receptors to activate or inhibit responses in innate immune cells like macrophages or dendritic cells. [28].

3.2. Obesity

In recent years, obesity has increased rapidly worldwide, posing a serious threat to human health and is an independent risk factor affecting the clinical prognosis of surgical patients. A recent report by the World Health Organization noted that more than 1.9 billion adults worldwide were overweight in 2016, with more than 650 million of them suffering from obesity [29]. The prevalence, high mortality and morbidity of obesity have direct and indirect negative effects on quality of life, which has not only triggered widespread concern among medical and scientific workers, but has also become a major public health problem that threatens human life and health. Recent epidemiological studies have shown that obesity increases the stress on weight-bearing joints and eventually wears down cartilage, leading to

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osteoarthritis in many obese individuals, one of the most disabling diseases [30]. Obesity can also lead to an increased risk of several metabolic diseases and problems such as non-alcoholic fatty liver disease, diabetes, and cardiovascular disease, hypertension and dyslipidemia, which in turn shorten human life expectancy and greatly increase the health and socioeconomic burden.

Since there is a close relationship between intestinal flora and blood lipids and liver function, regulation of intestinal flora can play a role in regulating obesity-related indicators. Recent studies have confirmed that disorders of the intestinal flora, especially the dysbiosis of the thick-walled phylum and the bacteriophage phylum, are intimately connected to the emergence of obesity; the composition of the thick-walled and bacteriophage phylum bacteria plays a significant role in intestinal health. The ratio of abundance of thick-walled and bacteriophage bacteria affects the development of obesity in mice; many studies have demonstrated that probiotics and prebiotics can regulate the intestinal flora and that this regulation can prevent obesity, for example, Lactobacillus and Bifidobacterium can reduce metabolic syndrome by regulating intestinal flora in mice [31].

Several animal experiments and clinical research has been conducted in recent years to confirm the effect of dietary fiber in improving blood lipids, blood glucose and reducing body weight. Pectin is the predominant soluble dietary fiber, which can be catalyzed by gut flora to generate metabolites in the colon with local and systemic functions. In addition, pectin can inhibit the accumulation of lipids in adipocytes. In animal experiments, pectin was found to significantly increase the relative abundance of Bacillus and Paramecium, Eubacterium and Bifidobacterium, and Lactobacillus while inhibiting weight gain and fat deposition in obese rats on a high-fat diet, reducing blood lipids, blood glucose, and insulin levels, improving liver fat metabolism, enhancing intestinal tight junction protein expression, and protecting intestinal barrier function [32].

In addition, SCFAs, the enzymatic products of pectin, are not only absorbed as nutrients and provide additional energy directly to the body, but also are important signaling molecules involved in the regulation of the body's energy metabolism. As signalling molecules, SCFA can bind to G-protein-coupled receptors for inducing intestinal peptide production are found on the surface of intestinal cells such as casein, glucagon-like peptide-1 (GLP-1), and 5-hydroxytryptamine (5-HT) [33]. 5-HT, an important neurotransmitter in the body, is implicated in the control of a number of physiological processes and can contribute to weight loss by controlling appetite, reducing food intake, and increasing energy expenditure [34]. A hormone produced in the gut called GLP-1 acts with the GLP-1 receptor to increase satiety and reduce food intake. Tyrosine slows down intestinal motility, prolongs the time of food passage through the intestine, and increases intestinal substance absorption. In addition, SCFAs can also promote leptin secretion from adipocytes by activating G protein-coupled receptors, directly regulating the body's glucolipid and energy metabolism, while transmitting anorexia signals to the hypothalamic appetite regulation center, suppressing appetite and reducing food intake. It can be seen that SCFAs, as important energy-regulating signal molecules, are closely related to the body's glucolipid metabolism and influence the development of obesity.

Finally, pectin oligosaccharides as degradation products of pectin can lower serum lipid levels and reduce hepatic lipid accumulation. The intervention of pectin oligosaccharides improved the phenomenon of intestinal flora disorders, raised the SCFA content level and reduced inflammatory levels, while alleviating dyslipidemia, which explains part of its alleviating effect on obesity symptoms. Thus, pectin oligosaccharides also alleviate weight gain in mice, and the earlier the intervention the better the weight reduction effect.

3.3. Type I diabetes mellitus

An immune condition known as type 1 diabetes (T1D) is characterized by deficient insulin synthesis, which is a result of partial or complete breakdown of pancreatic β-cells mediated by auto-reactive T-cells. about 5 out of 100 diabetic patients suffer from type 1 diabetes, and they mostly start in childhood or adolescence, but also have adult onset. About 80% to 90% of adolescents and young people with diabetes have type 1 diabetes.

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The prevalence of type 1 diabetes is on a significant global rise [6]. Only dietary fiber with a certain chemical make-up has these advantageous benefits; other fibers have none. Therefore, dietary fibers that are effective at preventing ecological dysregulation and gut barrier breakdown may be effective in inhibiting T1D development. Gut homeostasis underlies the development of T1D, and current studies on the efficacy of low-methoxy-pectin (LMP) novel dietary fibre for preventing the development of T1D have shown that by selectively selecting for particular microbial species that produce SCFAs, LMP can lower the prevalence of diabetes in NOD mice [35].

Cecum homeostasis mediates the preventive impact of dietary fiber on the onset of T1D. LMP supplementation lowered the prevalence of T1D in NDS mice. The effect is the result of a favorable impact on the cecum microbiota, increased generation of the SCFA immunomodulator and enhanced cecum intestinal stability. Regulation of the gut-pancreatic autoimmune reaction is brought about by LMP-induced intestinal stability, which reduces the risk of T1D onset (Figure 4).

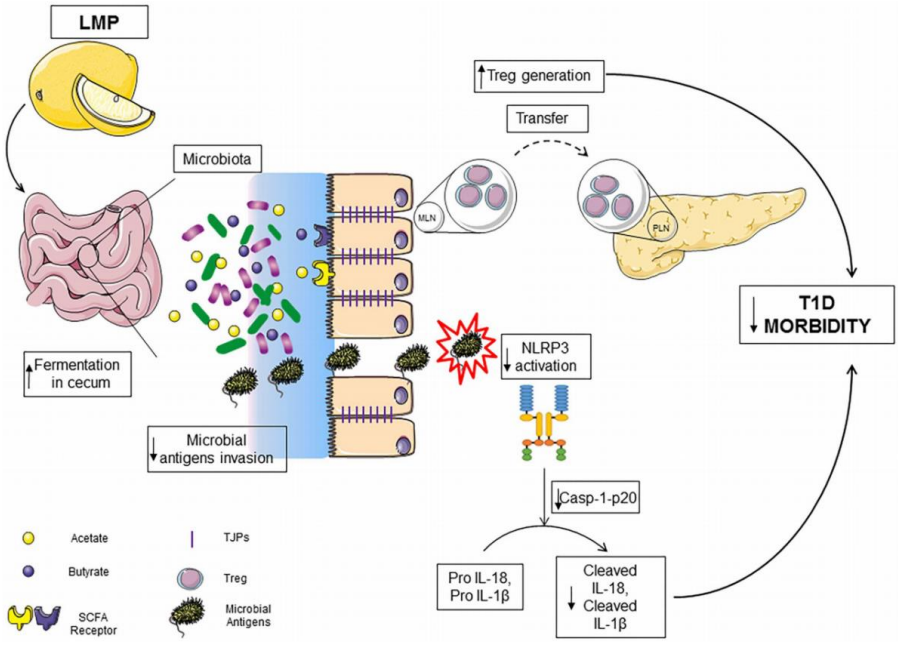


Figure 4. In NOD mice, LMP supplementation reduces T1D through modifying intestinal homeostasis.

Hypoesterified pectin, may improve T1D through selectively enhancing for particular types of bacteria that make SCFAs. LMP supplementation significantly increased the proportional abundance of microorganisms from the thick-walled phylum, TM7, Aspergillus and Clostridium species, and the ratio of thick-walled phylum to anaplasma in NOD + LMP mice. Compatible with increased microbial abundances and Clostridium perfringens producing butyrate, more SCFAs were found in the cecum of LMP-fed mice. lMP administration prevented T1D-associated intestinal ecological dysregulation in NOD mice.

The extent to which the pectin component has been esterified influences the location of fermentation, the products and the health-promoting effects conferred in the intestine, where pectins, particularly LMP, reduces intestinal permeability and has direct anti-inflammatory properties [36]. Thus, it may contribute to the prevention of T1D. LMP reduces the morbidity of NOD mice by improving intestinal flora and maintaining intestinal homeostasis. The main manifestations are that LMP dietary intervention reduces fasting blood glucose, inflammation in the intestinal pancreatic region, strengthens the intestinal epithelial barrier and regulates intestinal flora in mice.

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4. Conclusion

Pectin and pectin-like substances have significant regulatory effects on intestinal microorganisms. Among them, pectin oligosaccharides have pro-bifidobacterial growth properties, but the mechanism of activity is not clear. There is also a potential relationship between the structure of pectin and the composition of intestinal microbiota, and pectin with a predominantly linear chain structure is more favorable to the growth of Bifidobacterium. The effect of methoxy content in pectin on the intestinal microbial fraction may also vary, with high ester pectin (DM 70%) more significantly increasing the abundance of Ekmania spp. in the cecum. Pectin has a direct effect on immune cells, and bacterial metabolites have an indirect effect on pectin fermentation in the intestine. By modifying gut flora, pectin reduces allergic sensitivity. Pectin oligosaccharides lower serum lipid levels and reduce hepatic lipid accumulation. SCFAs are intimately connected to the body's glycolipid metabolism and influence the development of obesity, which in turn modulates the intestinal flora to play a role in regulating obesity-related indicators. Oligomerized pectin exerts direct anti-inflammatory effects and reduces intestinal permeability, which may contribute to the prevention of type I diabetes.

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