RESEARCH ARTICLE

Structural analysis of active tea polysaccharide from *Polygonatum* and their mechanism of action in regulating intestinal barrier damage

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Intestinal barrier dysfunction is closely related to various diseases. Damage to intestinal barrier function not only causes intestinal disorders but may also induce tumors in severe cases. Preventing intestinal barrier damage is an urgent issue that needs to be addressed. Active tea polysaccharides from *Polygonatum* have potential intestinal protective effects, but their structural characteristics and mechanisms of action are not yet clear. This research analyzed the fine structure of active tea polysaccharide from *Polygonatum* and explored their repair mechanism on intestinal barrier damage to address the intestinal barrier dysfunction by using anion exchange chromatography combined with infrared spectroscopy, mass spectrometry, and methylation analysis. The reparative effect of active tea polysaccharides from *Polygonatum* was analyzed by using a rat model with low oxygen induced intestinal barrier injury. The results showed that the active tea polysaccharides of *Polygonatum sibiricum* had a main chain structure consisting of alternating connections between 1→4)-β-D-xylose and 1→4)-α-D-galacturonic acid, as well as branched chains containing β-D-mannose and α-D-glucose. Functionally, it could significantly improve intestinal morphological indicators with significant statistical differences in villus length, goblet cell number, and intestinal gland depth between different groups ($P < 0.05$) with obvious statistical difference between protein occludin and claudin-1 ($P < 0.001$). Active tea polysaccharides from *Polygonatum* reduced the inflammatory factors and effectively regulated the abundance of bacterial phyla such as *Bacteroidota* and *Verrucomicrobiota* ($P < 0.05$). The research confirmed that active tea polysaccharides from *Polygonatum* could synergistically improve intestinal barrier function through multiple pathways, providing a scientific basis for intestinal diseases. Subsequent research should emphasis structural optimization and clinical translation verification.

Keywords: tea polysaccharide; polygonatum; rat; intestinal barrier injury; structure; glycosidic bond.

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Introduction

In recent years, intestinal health problems have become increasingly prominent. Intestinal barrier dysfunction is linked with various metabolic diseases, inflammatory bowel diseases, and immune disorders. The integrity of the intestinal barrier relies on the stable tight junction proteins and the balanced intestinal microbiota. Oxidative stress, low oxygen environment, or excessive activation of inflammatory factors increase intestinal epithelial permeability, causing the "intestinal leakage" [1, 2]. Therefore, the search for natural

active ingredients that can repair intestinal barrier function has become a research hotspot.

In existing research, *Polygonatum* as a traditional medicinal and edible plant is rich in polysaccharides and has activities such as antioxidant, anti-inflammatory, and immune regulation [3, 4]. However, the detailed structural characteristics of *Polygonatum* polysaccharides, especially tea polysaccharide complexes, and their specific mechanisms of protective effects through the "microbiota - intestinal barrier" axis have not been systematically elucidated. Plant polysaccharides can improve intestinal barrier damage by regulating the tight junction proteins, inhibiting the TLR4/NF-κB inflammatory pathway, or reshaping gut microbiota. However, whether active tea polysaccharide from *Polygonatum* exerts their effects through similar or unique pathways still needs further exploration.

To analyze the actual mechanism of active tea polysaccharides from *Polygonatum* in intestinal barrier damage, this study took active tea polysaccharides from *Polygonatum* to explore its potential mechanisms on intestinal barrier damage through the measurements of inflammation factors, intestinal microbiota, and intestinal protein expression in rats. The actual application structure of active tea polysaccharides from *Polygonatum* was detected by using mass spectrometry. The results of this research provided theoretical basis for *Polygonatum sibiricum* polysaccharides and opened up new ideas for natural intervention strategies for intestinal barrier injury.

Materials and methods

Structural analysis of active tea polysaccharide from *Polygonatum*

(1) Extracting tea polysaccharides

The *Polygonatum sibiricum* active tea polysaccharide was purchased from Beijing Soleibao Technology Co., Ltd. (Beijing, China) with 70% purity. 300 g of *Polygonatum sibiricum* crude polysaccharides were dried and mixed with 4 times volume of water for reflux extraction in a standard glass reflux device with a heating jacket temperature controlled to maintain a slight boiling point for 30 min. The filtrate was collected, and the procedures were repeated twice before combining all filtrates together. The combined filtrates were filtered through a Buchner funnel and medium speed qualitative filter paper before concentrating to 350 mL using a rotary evaporator at 60℃ and -0.09 MPa. The concentrates were precipitated with 4 times volume of 95% ethanol at 4℃ overnight before centrifugation and freeze-dry to obtain the final sample [5, 6]. Polysaccharide was extracted by using a 1.6 × 20 cm DEAE Sepharose Fast Flow anion exchange chromatography column at 10 mg/mL and eluted using ultrapure water, 0.15 M, 0.25 M, and 0.6 M sodium chloride solutions at a flow rate of 1.0 mL/min with an elution volume of 1,000 mL. The neutral polysaccharide component obtained through ultrapure water elusion was labeled as N, while the acidic polysaccharide component eluted with 0.25 M NaCl was treated by a dialysis membrane with a molecular cut-off of 3,000 Da for 72 hours and then freeze-dried as the sample A [7, 8].

(2) Polysaccharide structure analysis

The standard curve of glucose was obtained by mixing 0, 0.2, 0.4, 0.6, 0.8, and 1.0 mL of 0.1 mg/mL glucose solution with water to 1 mL and adding 1 mL of 5% phenol solution followed by adding 5 mL of concentrated sulfuric acid. After standing for 10 min, the reaction mixture was put into a boiling water bath for 15 min followed by quick cooling down to room temperature and measuring the absorbance at 490 nm using Multiskan GO spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA) [9, 10]. The total sugar content was determined as follows.

Total sugar content = (measured sugar mass × dilution factor/sample mass) × 100

The purity of the sample was determined by measuring the polysaccharide solution in the wavelength range of 190 – 400 nm and observing

if there was an absorption peak near 200 nm. Subsequently, infrared spectroscopy analysis was performed by mixing the dried polysaccharide sample with potassium bromide and compressed into tablets followed by using Fourier transform infrared spectroscopy to scan within 4,000 – 400/cm [11, 12]. Monosaccharide component was determined by combining acid hydrolysis with high-performance liquid chromatography (HPLC). 10 mg of polysaccharide sample was mixed with 2 mL of 2M trifluoroacetic acid (TFA) and hydrolyzed in a 110℃ oven for 6 hours. The hydrolyzed product was then subjected to vacuum evaporation to remove residual acid followed by reduction with sodium borohydride and acetylation with acetic anhydride to produce sugar alcohol acetate derivatives. After drying with hydrolyzed liquid nitrogen, 1 mL of water and 10 mg of $NaBH_4$ were added and incubated at room temperature for 2 hours. The reaction was then neutralized with glacial acetic acid and then dried with nitrogen. 1 mL each of acetic anhydride and pyridine were then added and reacted at 100℃ for 1 hour for acetylation. After drying with nitrogen, the sample was dissolved in chloromethane. HPLC analysis was performed by using an amino column with acetonitrile-water (75:25) as the mobile phase and combining with an evaporative light scattering detector for separation and detection [13-15].

Construction of intestinal barrier injury animal model

60 8-week-old Sprague Dawley (SD) rat (Beijing Weitonglihua Experimental Animal Technology Co., Ltd., Beijing, China) with the body weights ranging from 250 to 300 g were divided into 5 groups with 12 rats in each group including control (group 1), hypoxia model (group 2), low-dose polysaccharide (150 mg/kg·d) (group 3), medium-dose polysaccharide (350 mg/kg·d) (group 4), and high-dose polysaccharide (550 mg/kg·d) (group 5) [16, 17]. All animals had free access to food and water. Except for group 1, all other groups were placed in a DG-2 animal experimental low pressure oxygen chamber (Nanjing Quanfeng Instrument Co., Ltd., Nanjing, Jiangsu, China) with the oxygen concentration of 10.0 ± 0.5%, which was equivalent to the low-oxygen conditions at an altitude of about 5,500 m, the temperature inside the cabin at 22 ± 2℃, and the humidity of 50 ± 10%. The animals were exposed to a low-oxygen environment for 20 hours daily for 5 weeks to induce intestinal barrier damage, while the remaining 4 hours daily were returned to a normal pressure and oxygen environment for administration, pad replacement, and other procedures. After adaptively fed for about 3 days, the animals were subjected to a one-week gastric lavage treatment using physiological saline for groups 1 and 2 and different doses of active tea polysaccharides from *Polygonatum* for groups 3 to 5 once a day in a normal environment after exiting the cabin [18]. After constructing the intestinal barrier injury model, the intervention was stopped. In the 6th week of the experiment, the feeding and weight changes of rats were recorded, and the rats in each group were fasted for 12 hours before being euthanized for serum isolation and intestinal tissue (jejunum) extraction. All experimental procedures were approved by the Animal Ethics Committee of Foshan University with the approval number of FOSU386120260203.

Fluorescence staining of intestinal mucus

The intestinal tissue was fixed with 4% paraformaldehyde (China National Pharmaceutical Group Chemical Reagent Co., Ltd., Shanghai, China) followed by dewaxing and hydration before acidic mucus staining with Alcian Blue 8GX (Sigma Aldrich, St. Louis, MO, USA) at pH 2.5 for 30 min. After rinsing with running water, the tissue was oxidized with 1% periodic acid for 5 min to expose aldehyde groups and reacted with Schiff reagent (Beijing Solaibao Technology Co., Ltd., Beijing, China) for 15 - 20 min. After termination with sulfite water, the cell nucleus was stained with hematoxylin followed by dehydrating and sealing the slide [19]. The acidic mucus would show blue color, while the neutral mucus appeared as red, and the mixed type was purple red.

Intestinal tissue processing

After rinsing the contents of the intestinal lumen with pre-cooled physiological saline, the tissues were divided into three parts with one part being immediately fixed in 4% paraformaldehyde for 24 h for subsequent paraffin embedding using EG1150H paraffin embedding machine (Leica Microsystems, Wetzlar, Germany) and histological staining with Hematoxylin and Eosin (HE). The second part of fresh tissue was embedded with Optimal Cutting Temperature Compound (OCT) and frozen in liquid nitrogen for frozen sectioning using RM2245 paraffin sectioning machine (Leica Microsystems, Wetzlar, Germany) and immunofluorescence tests. The third part of fresh tissue was directly frozen in liquid nitrogen and stored at -80℃ for subsequent protein and RNA extraction [20].

16S rRNA gene sequencing

The study employed 16S rRNA gene sequencing to analyze gut microbiota. After extraction of microbial genomic DNA, polymerase chain reaction (PCR) was performed to amplify the highly variable V3-V4 regions of the 16S rRNA by using bacterial universal primers 338F and 806R. KAPA HiFi HotStart ReadyMix (Roche, Basel, Switzerland) and Bio-Rad T100 PCR instrument (Bio-Rad, Hercules, CA, USA) were used for PCR experiment by following the standard PCR procedures and manufacturer's instructions. The sequencing library was constructed by using the 16S V4 kit (PerkinElmer, St, Waltham, MA, USA). The PE250 double-ended sequencing was performed by Beijing Novogene Technology Co., Ltd. (Beijing, China) on the Illumina NovaSeq 6000 platform (Illumina, San Diego, CA, USA). Species annotation was then completed through quality control filtering, operational taxonomic unit (OUT) clustering, and SILVA database (https://www.arb-silva.de) comparison. α/β diversity index was determined by using QIIME 2 (https://qiime2.org), and community structure differences were analyzed. Microbial functional potential was predicted by using PICRUSt2 (https://github.com/picrust/picrust2). The entire process required setting up negative controls and strictly controlling the sequencing depth of ≥ 50,000 reads/sample) to ensure data reliability.

Indicators of intestinal barrier damage

The inflammatory factors and barrier damaging hormones were measured as indicators of intestinal barrier damage, which included TNF-α, IL-1β, and Myeloperoxidase (MPO). In addition, diamine oxidase (DAO) and gastrin (GAS) were tested because DAO mainly protected intestinal barrier function by degrading biogenic amines and its activity level reflected the integrity of intestinal mucosa, while GAS maintained digestive function by stimulating gastric acid secretion, promoting gastric mucosal growth, and regulating gastrointestinal motility. The concentrations of inflammatory factors TNF-α and IL-1β in serum and intestinal tissue homogenate were measured by using a rat specific Enzyme-linked immunosorbent assay (ELISA) kit (Wuhan Huamei Bioengineering Co., Ltd., Wuhan, Hubei, China) following the manufacturer's instructions. The colorimetric assay kit (Nanjing Jiancheng Bioengineering Institute, Nanjing, Jiangsu, China) was used to detect the activity of myeloperoxidase and diamine oxidase following supplier's instructions. Gastrin levels were measured by using a radioimmunoassay kit (Beijing Northern Institute of Biotechnology, Beijing, China) following manufacturer's instructions.

Statistical analysis

SPSS 20.0 (IBM, Armonk, NY, USA) was employed for statistical analysis of this research. The experimental data were expressed as mean ± standard error. GraphPad Prism 8.0 (https://www.graphpad.com/) were used for data processing and analysis. Mann-Whitney U test was used for the intergroup comparison, and Kruskal-Wallis test was applied for multi-group difference comparison. *P* value less than 0.05 was defined as statistically significant difference.

Results

Structural analysis of active tea polysaccharide from *Polygonatum*

The glycosidic bonds in the active polysaccharide structure of *Polygonatum sibiricum* were

Table 1. Analysis results of polysaccharide structure of *Polygonatum sibiricum*.

Mutual connection	Chemical shift (ppm)
H1 peak of Unit B ↔ C2 peak of Unit A	4.79 (H1) / 72.4 (C2)
H1 signal of Unit A ↔ C4 signal of Unit C	4.79 (H1) / 768 (C4)
H1 peak of Component E ↔ C2 peak of Component C	4.92 (H1) / 81.9 (C2)
H4 peak of Structure C ↔ C1 peak of Structure A	4.42 (H4) / 100.4 (C1)
H3 signal of Molecule B ↔ C1 signal of Molecule D	4.29 (H3) / 100.2 (C1)

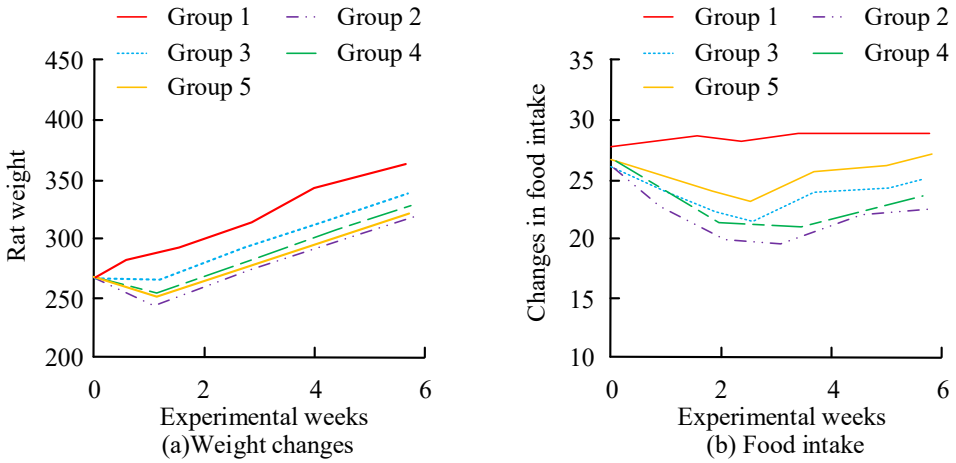

Figure 1. Changes in food intake and body weight of rats.

determined using methylation. The results showed that the active polysaccharide structures of *Polygonatum sibiricum* included five glycosides of 1-O-acetyl-2,3,4,5-tetra-O-methylxylitol, 1,2,4-tri-O-acetyl-5-O-methylxylitol, 1,3-di-O-acetyl-2,46-tri-O-methylmannitol, 1-O-acetyl-2,3,4,6-tetra-O-methylglucitol, and 1,2,4-tri-O-acetyl-3,6-di-O-methylgalactol, while 1,2,4-tri-O-acetyl-3,6-di-O-methylgalactol had the longest retention time and the highest molar ratio of 0.8. The mass spectrometry analysis demonstrated five significant cross-peak signals in the H2/C2 region with chemical shifts (δ) of 4.61/72.1 ppm, 3.75/74.8 ppm, 4.13/82.0 ppm, 3.70/71.6 ppm, and 3.72/67.8 ppm, respectively. The results indicated that the C2 position of the sugar residue had been replaced by a carbon atom connected to an oxygen atom, which provided key information for inferring the connection mode and branching structure of the polysaccharide chain, confirming that the polysaccharide had a complex molecular configuration. The polysaccharide spectrum showed that the β-D-xylose in the polysaccharide structure was connected by a 1→2 bond. The main chain involved →4)-β-D-xylose, and →4)-α-D-galacturonic acid alternately connected by 1→4 bonds. The branched chain contained β-D-mannose connected by a 2→1 bond, while α-D-glucose connected by a 3→1 bond (Table 1). The entire structure reflected the β/α configuration of sugar residues in polysaccharides, the branching sites of the main and branch chains, and the involvement of uronic acids.

The effect of active tea polysaccharides from *Polygonatum* on dietary changes in rats

The effects of active tea polysaccharides from *Polygonatum* on the feeding behavior of rats showed that the animal weight changes in all groups significantly increased with the increase of the experimental period, and group 1 demonstrated the fastest weight changes (Figure 1a). There was basically no change in the food

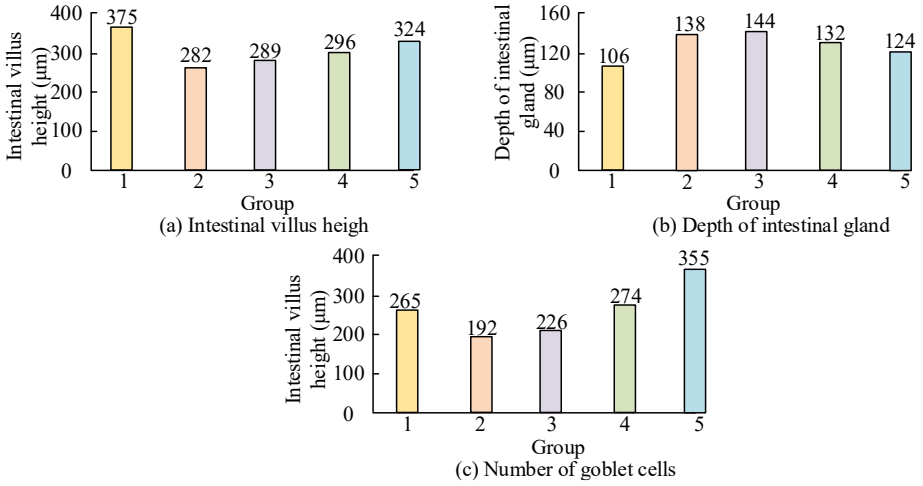

Figure 2. Monitoring results of intestinal mucosal injury.

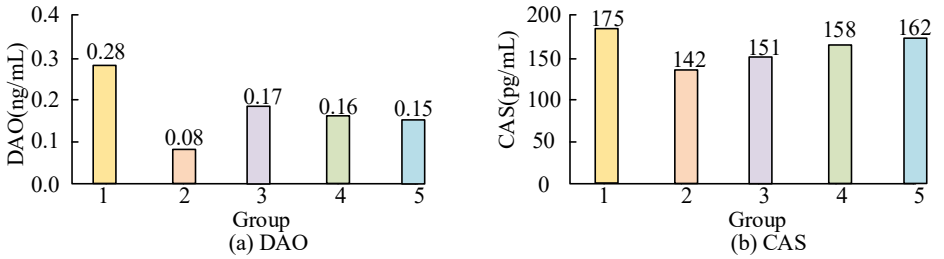

Figure 3. Comparison of changes in DAO and GAS hormone levels.

intake of group 1, while the food intake of other groups showed a significant downward trend followed by a slow upward trend with statistically significant differences in food intake and weight changes between group 1 and other groups ($P < 0.01$) (Figure 1b). The results indicated that active tea polysaccharides from *Polygonatum* had no significant effects on the food intake and weight changes in intestinal barrier damaged rats.

The effect of active tea polysaccharides from *Polygonatum* on intestinal barrier injury in rats

The monitoring results of intestinal mucosal damage demonstrated that the control group had longer intestinal villi than that in other intestinal mucosal damaged groups, while group 2 had the lowest intestinal villi of only 282 μm. After intervention with different doses of active tea polysaccharides from *Polygonatum*, the length of intestinal villi gradually increased. The intestinal gland depth and goblet cell number was also increased after using different doses of active tea polysaccharides from *Polygonatum* with the high dose showing good effect on resisting intestinal mucosal damage (Figure 2).

Hormonal effects of active tea polysaccharides from *Polygonatum*

The changes of two barrier damaging hormones, DAO and GAS, were compared in this study. The results showed that the levels of DAO and GAS were significantly increased with the increase of *Polygonatum* dose. The significant statistical differences were observed between groups 3, 4, 5 and group 1 ($P < 0.05$). The changes of GAS levels between groups 3, 4, 5 and group 1 were also significantly different ($P < 0.05$) (Figure 3). The changes in the dosage of *Polygonatum sibiricum* significantly affected the secretion of intestinal mucosal hormones with the positive effects.

Table 2. Comparison of changes in gut microbiotas.

ID (Phylum Level)	Group 1 (%)	Group 2 (%)	Group 5 (%)
Bacillota	83.87 ± 5.08	84.92 ± 6.41*	83.12 ± 4.73
Bacteroidota	12.15 ± 4.03	11.32 ± 6.38*	12.04 ± 4.25
Patescibacteria	1.18 ± 1.27	0.91 ± 0.72	0.93 ± 0.65
Pseudomonadota	2.24 ± 1.49	1.42 ± 0.88*	2.35 ± 1.77
Unclassified	0.31 ± 0.15	0.85 ± 0.42*	0.63 ± 0.34#
Actinomycetota	0.33 ± 0.21	0.48 ± 0.51*	0.25 ± 0.11
Spirochaetota	7.12 ± 12.63	2.91 ± 3.41*	4.21 ± 6.87#
Verrucomicrobiota	27.6 ± 50.28	102.15 ± 217.30**	218.43 ± 565.91##
Mollicutes	2.41 ± 2.87	6.38 ± 7.15**	1.41 ± 1.39##
Synergistota	0.00 ± 0.00	2.03 ± 3.42*	0.00 ± 0.00

Note: The data were expressed as mean ± standard deviation. *: $P < 0.05$. **: $P < 0.01$ compared to group 1. #: $P < 0.05$. ##: $P < 0.01$ compared to group 1.

Changes in inflammatory factor hormone levels

The results of inflammatory factor tests showed that, compared with the control group, the indicators in the jejunum and serum of the hypoxia model group and intervention groups demonstrated significant changes. There was a significant difference in TNF-α levels between the intervention groups and the control group in the jejunal tissue ($P < 0.05$). The decrease in IL-1β levels was more significant, while the intervention groups showed extremely significant differences ($P < 0.01$) compared to the control group in both the jejunum and serum. In addition, the activity of MPO representing the degree of neutrophil infiltration showed a highly significant decrease in both the jejunum and serum of the intervention groups compared to the control group ($P < 0.001$). These results indicated that high-dose active tea polysaccharides from *Polygonatum* significantly affected the inflammatory factor in the intestinal barrier.

The impact of protein expression on intestinal barrier

The protein levels of occludin and claudin-1 were measured to analyze the effect of intervention on the expression of intestinal tight junction proteins. Compared with the control group, the hypoxia model group showed the differences at the protein levels. After polysaccharide intervention, the expression levels of occludin in the jejunal tissue and serum of rats were significantly upregulated compared to the control group ($P < 0.001$), while claudin-1 of the polysaccharide intervention groups also showed significant statistical differences ($P < 0.001$) in the jejunum and serum compared to the control group. The results indicated that high-activity active tea polysaccharides from *Polygonatum* could effectively regulate the expression of intestinal tight junction proteins.

The impact of changes in gut microbiota

The results showed statistically significant difference between groups 1 and 2 in the number of different gut microbiotas ($P < 0.05$) (Table 2). The active tea polysaccharides from *Polygonatum* affected the changes in the gut microbiota. The gut microbiota in the exposed group also showed significant statistical differences in some microbiota, indicating obvious changes in the exposed group.

Discussion

The intestine is an important organ for digesting and absorbing nutrients, which is also the largest immune organ and microbial habitat in the body. The intestinal barrier involves mechanical barriers, chemical barriers, immune barriers, and microbial barriers. Its core function is to selectively allow nutrient absorption, while

preventing pathogens and toxins from entering the body. Damage to the intestinal barrier may cause local or systemic diseases such as inflammatory bowel disease, allergies, autoimmune diseases, *etc*. The integrity of its barrier function relies on the stable expression of tight junction proteins between intestinal epithelial cells, the chemical barrier of the intestinal mucus layer, the regulatory role of immune cells, and the dynamic balance of intestinal microbiota. However, factors such as oxidative stress, low-oxygen levels, high-fat diet, or chronic inflammation can disrupt this delicate regulatory system, leading to increased intestinal epithelial permeability and "leaky gut" phenomenon, which can cause metabolic diseases, autoimmune diseases, and even neurological disorders. *Polygonatum sibiricum* is a traditional medicinal and edible plant, which has been proven to contain polysaccharides with antioxidant, antifatigue, and immune regulatory activities. However, the fine structural characteristics of its tea polysaccharide complex and its protective mechanism against intestinal barrier damage have not been systematically elucidated. Multiple studies have shown the impact of various natural factors on the intestinal barrier. Cai *et al*. analyzed the role of the polysaccharide GP90 from Longxucai seeds by testing factor expression and analyzing gut bacteria. The results showed that the polysaccharide had anti-tumor effects, which could inhibit nitric oxide, interleukin-6 (IL-6), IL-1β, and TNF-α [21]. Different natural polysaccharide factors have positive effects on the damage of intestinal barrier. Gong *et al*. analyzed the application of *Polygonatum sibiricum* polysaccharides in different fields through clinical testing methods and found that *Polygonatum sibiricum* polysaccharides could significantly affect the microbial community and have active effects on hormone expression in different intestines, which could be used to treat intestinal barrier damage and enteritis [22]. Wu *et al*. tested the improvement effect of natural polysaccharides on gut microbiota using *Hericium erinaceus* polysaccharides and showed that *Hericium erinaceus* polysaccharides improved liver damage and metabolic disorders by optimizing gut barrier function and shaping gut microbiota. Improving intestinal barrier could significantly inhibit the liver LPS/TLR4/MyD88/NF-κB pathway and reduce liver inflammation response and cell apoptosis [23]. Those previous studies all confirmed that natural polysaccharides had significant effects on intestinal barrier protection and microbial activation. This study explored the *Polygonatum sibiricum* polysaccharides by analyzing the actual structural changes and testing the impact of polysaccharide structure on intestinal barrier. The results indicated that the active tea polysaccharides of *Polygonatum sibiricum* were mainly composed of monosaccharides of β-D-xylose, α-D-galacturonic acid, β-D-mannose, and α-D-glucose. The main chain was alternately connected by 1→4 bonds, while the branch chains were connected by 2→1 and 3→1 bonds. The structure was complex and had significant biological activity. Experimental animal results showed that active tea polysaccharides from *Polygonatum* significantly improved the intestinal barrier damage induced by low-oxygen environment, increased the intestinal villus length, intestinal gland depth, and the goblet cell number, enhanced DAO and GAS, and reduced inflammatory factors TNF-α, IL-1β, and MPO. In addition, polysaccharide intervention significantly upregulated the tight junction proteins occludin and claudin-1, enhancing the mechanical barrier function of intestinal mucosa. Occludin maintains the mechanical stability of tight junctions by connecting intracellular ZO proteins and can dynamically regulate barrier function by sensing external signals, while claudin-1 forms selective channels through its extracellular loop, precisely regulating the directional transport of water molecules and ions. Further, active tea polysaccharides from *Polygonatum* significantly altered the abundance of bacterial phyla such as *Bacteroidota* and *Verrucomicrobiota* by regulating the gut microbiota structure, thereby balancing gut microbiota. These results indicated that active tea polysaccharides from *Polygonatum* exerted protective effects on the intestinal barrier

through multiple targets and pathways including directly repairing intestinal mucosal structure, inhibiting inflammatory response, regulating intestinal hormone secretion, and reshaping intestinal microbiota. The future studies may need to additionally explore the fine structure and the structure-activity relationship between key active groups such as uronic acid and acetyl groups and clarify their biological functions. Further, the molecular mechanism of *Polygonatum* polysaccharides needs to be explored with a focus on how polysaccharides regulate specific gut microbiota and signaling pathways such as TLR4/NF-κB and PI3K-Akt through the "microbiota gut - barrier" axis to reveal their specific targets for anti-inflammatory and barrier repair.

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