Regulation effects of resistant starch on gut microbiota.

Tao Li, Lei Chen*

College of Food Science, Fujian Agriculture and Forestry University, Fuzhou, Fujian 350002, China

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Introduction

Resistant starch (RS), a product of starch digestion, is poorly absorbed by the small intestine but makes its way to the large intestine where it acts as a substrate for fermentation by resident microbiota [1]. RS can be classified into four types on the basis of its botanical source and processing methods including RS1 (physically inaccessible starch); RS2 (native starch granule); RS3 (retrograded starch) and RS4 (chemically modified starch) [2]. RS, especially RS3, passes through the upper digestive tract to the colon producing short-chain fatty acids (mainly acetic, propionic and butyric acid) during bacterial fermentation which prevents colon cancer, inhibites fat accumulation with highlights on its regulatory effect on gut microbiota [3,4]. There are 10 trillion bacteria in the human intestine, which can affect body weight and digestive capacity, resist the risk of infection and autoimmune diseases, and control the body's response to cancer treatment. A balanced microbiota composition and associated metabolic activities are essential to human health. However, so far, information on the mechanism of RS regulating gut microbiota is limited. Therefore, the purpose of this paper is to discuss briefly regulation effects of RS on the gut microbiota and the mechanism.

Under the intervention of RS, *Coprococus*, *Ruminococcus, Bacteroides*, *Allobaculum, Roseburia,* and *Prevotella* increase significantly, especially *Bifidobacteria* and *Lactobacillus,* while potentially pathogenic members of *Enterococcus*, *Streptococcus* and *Escherichia coli* decrease in relative abundances in the hindgut of animal models [5,6]. Certain dominant species, notably among the *Bifidobacteria,* are known to possess very large numbers of genes that ferment RS to produce acetic acid and lactic acid adjusting the balance of intestinal flora. There are some published reports on the bifidobacteria growth and short-chain fatty acids (SCFAs) production of RS3 fermentation *in vitro* and *in vivo* [7,8]. RS not only alters the composition of the gut microbial community but also modulates the metabolic pathway of microbial metabolism [9]. Not surprisingly, RS can promote the level of tricarboxylic acid cycle rising, strengthen the organization of aerobic metabolism, and provide adequate energy security for intestinal metabolic bacteria. RS regulates and improves fat, amino acid and glucose metabolism associated with changes in intestinal flora, thereby improving overall metabolic status. Collectively, these studies demonstrate the potential of RS to modify the composition of the intestinal microbiota.

Generally, RS which prepared by physical methods possessed flaky and gully shapes and compact crystal state. The rough surface of RS provided the best adhesive conditions and binding capacity of the starch (number of bacteria per gram) relying on the granule surface area, therefore might play a role in efficient utilization of starch for some strains. Adhesion to the starch was considered a possible mechanism for increased bacterial survival [10]. A specific cell surface protein of probiotics would adhere to RS granules. For example, cell surface proteins of *Bifidobacteria,* which specifically bind to α-1,4-linked glucose saccharides, are involved in adhesion of the bacteria to the starch [11]. Intriguingly, the compact crystal state and easily adherent environment could provide protection towards bacteria entrapping in starch granules. Furthermore, the ordered structure could inhibit the action of gastrointestinal juices, which might contribute to enhance tolerance ability of probiotics. On the whole, the rougher surface and more perfect crystalline structure providing the better adhesive conditions and growth substrate, which contributed to the facilitation effect on the proliferation ability of probiotics. On the other hand, intestinal anaerobic bacteria, most commonly *Bifidobacteria* and *Lactobacillus*, would ferment non-digestible carbohydrates to produce SCFAs which could reduce intestinal pH value, thereby inhibiting the growth of harmful bacteria to prevent intestinal dysfunction. From cell signaling pathways analysis, consumption of RS could sitmulate the expression of Glucagon-like peptide- 1 (GLP-1) and Peptide YY (PYY) and decrease the level of Interleukin-6 (IL-6) and FFA (free fatty acids) via either IL-10 inflammatory or GLP-1R secreted hormone [12]. Fermentation of RS by anaerobic bacteria is mostly likely the primary mechanism for increased endogenous secretions of total GLP-1 and PYY in rodents, which are positively associated with fermentation and liberation of SCFAs. As previously mentioned, increasing production of SCFAs would promote the proliferation of probiotics and inhibit the growth of harmful bacteria maintaining the balance of intestinal flora.

Short chain fatty acids (SCFAs), as a bridge between RS and intestinal flora, can be closely linked with intestinal metabolism and human health. More importantly, the unique self-structure and positive promotion of genes expression which is conducive to intestinal health of RS revealed preferably the mechanism of maintaining the health of intestinal flora.

References

1. Kim J, Zhang C, Shin M. Forming rice starch gels by adding retrograded and cross-linked resistant starch prepared from rice starch. Food Sci Biotechnol. 2015;24:835-41.
2. Sajilata MG, Singhal RS, Kulkarni PR. Resistant starch-A review. Comprehensive Reviews in Food Sci Food Saf. 2006;5:1-17.

3. Lopez HW, Levrat-Verny MA, Coudray C, et al. Class 2 resistant starches lower plasma and liver lipids and improve mineral retention in rats, J Nutr. 2001;131:1283-89.
4. Sharma A, Yadav BS, Ritika. Resistant starch: physiological roles and food applications. J Nutr. 2008;24:193-34.
5. Umu OC, Frank JA, Fangel JU, et al. Resistant starch diet induces change in the swine microbiome and a predominance of beneficial bacterial populations. Microbiome. 2015;3:16.
6. Herrmann E, Young W, Rosendale D, et al. Determination of resistant starch assimilating bacteria in fecal samples of mice by in vitro RNA-based stable isotope probing. Front Microbiol. 2017;8:1331.
7. Paturi G, Nyanhanda T, Butts CA, et al. Effects of potato fiber and potato-resistant starch on biomarkers of colonic health in rats fed diets containing red meat. J Food Sci. 2012;77:216-23.
8. Silvi S, Rumney CJ, Cresci A, et al. Resistant starch modifies gut microflora and microbial metabolism in human flora-associated rats inoculated with faeces from Italian and UK donors. J Appl Microbiol. 1999;86:521-30.
9. Sun Y, Su Y, Zhu WY. Microbiome-metabolome responses in the cecum and colon of pig to a high resistant starch diet. Front Microbiol. 2016;7:779.
10. Wang X, Brown, IL, Evans AJ, et al. The protective effects of high amylose maize (amylomaize) starch granules on the survival of *Bifidobacterium spp.* in the mouse intestinal tract. J Appl Microbiol. 1999;87:631-39.
11. Crittenden R, Laitila A, Forssell P, et al. Adhesion of bifidobacteria to granular starch and its implications in probiotic technologies. Appl Environ Microbiol. 2001;67:3469-75.
12. Yang XY, Darko KO, Huang YJ, et al. Resistant starch regulates gut microbiota: structure, biochemistry and cell signalling. Cell Physiol Biochem. 2017;42:306-18.

***Correspondence to:**

Lei Chen

School of Food Science

Fujian Agriculture and Forestry University

Fuzhou, 350002

China

Tel: 86-591-83756316

Fax: 86-591-83756316

E-mail: chenlei841114@hotmail.com