Studies on the effects of polydextrose intake on physiologic functions in Chinese people[1–3]

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ABSTRACT

Background: Previous studies showed that polydextrose has physiologic effects similar to those of dietary fiber.

Objective: Ingestion of 4, 8, and 12 g polydextrose/d was studied to determine the physiologic effects in Chinese subjects.

Design: In a placebo-controlled, randomized, double-blind study, we evaluated the effects of polydextrose ingestion on clinical biochemistry indexes, glycated hemoglobin, glucose tolerance, the glycemic index, bowel function, stool weight and pH, short-chain fatty acid production, fecal microflora, and cecal mucosa cell proliferation.

Results: Polydextrose had no significant effect on blood biochemistry indexes. Ingestion of 12 g polydextrose plus 50 g glucose resulted in a glycemic index of 89% (compared with a glycemic index of 100% after ingestion of 50 g glucose). Bowel function (frequency and ease of defecation) improved significantly and there were no reports of abdominal distention, abdominal cramps, diarrhea, or hypoglycemia. Fecal weight (wet and dry) increased and fecal pH decreased proportionally to polydextrose intake. Short-chain fatty acid production—notably that of butyrate, isobutyrate, and acetate—increased with polydextrose ingestion. There were substantial changes in fecal anaerobes after polydextrose intake. *Bacteroides* species (*B. fragilis, B. vulgatus*, and *B. intermedius*) decreased, whereas *Lactobacillus* and *Bifidobacterium* species increased. The cecal mucosa whole-crypt labeling index increased, with colonocyte proliferation mainly occurring in base compartments, which provided an indirect confirmation of butyrate production in the colon.

Conclusion: Polydextrose ingestion had significant dietary fiber–like effects with no laxative problems. *Am J Clin Nutr* 2000;72:1503–9.

KEY WORDS Polydextrose, dietary fiber, blood glucose, glycemic index, colonic fermentation, short-chain fatty acids, bowel function, fecal anaerobes, *Lactobacillus*, *Bifidobacterium*, China

INTRODUCTION

Dietary fiber has attracted much attention since the 1970s because of its beneficial effects on human physiology. Dietary fiber was initially defined by Trowell and Burkitt as "remnants of plant cell walls which were not hydrolyzed and digested by human enzymes" (1). The definition recognized in China is "the sum of food components that are not digested by intestinal enzymes and absorbed into the body" (2). Dietary fiber is classified as water soluble or non–water soluble and includes cellulose, gum, pectin, polysaccharides, and food additives.

Polydextrose is a polysaccharide synthesized by random polymerization of glucose, sorbitol, and a suitable acid catalyst at a high temperature and partial vacuum. It is used widely in many countries as a bulking agent and as a lower-energy ingredient (4.2 kJ/g) in a variety of prepared foods. Polydextrose is not digested or absorbed in the small intestine, and a large portion is excreted in the feces (3). Several studies of polydextrose showed physiologic effects consistent with those of dietary fiber (4–14). Polydextrose is partially fermented in the large intestine, leading to increased fecal bulk, reduced transit time, softer stools, and lower fecal pH (4–9). Fermentation of polydextrose leads to the growth of favorable microflora, diminished putrefactive microflora, enhanced production of short-chain fatty acids (SCFAs), and suppressed production of carcinogenic metabolites (eg, indole and *p*-cresol) (3, 8, 10).

Therefore, the safety and efficacy of polydextrose as a water-soluble bulking agent and fiber has been widely and thoroughly investigated. The metabolic route has been established in animals and humans (3). The aim of the present trial was to evaluate the effects of polydextrose on various body functions and blood biochemical indexes in healthy Chinese subjects.

SUBJECTS AND METHODS

Subjects and polydextrose intake

One hundred twenty healthy volunteers participated in the study, of whom 66 were men and 54 were women with average ages of 32.9 and 29.4 y, respectively (**Table 1**). None of the

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TABLE 1
General characteristics of the subjects and polydextrose intakes

	Group A (control) (n = 30)	Group B (n = 30)	Group C (n = 30)	Group D (n = 30)
Men/women	16/14	17/13	16/14	17/13
Average age (y)	30.2	32.7	31.7	29.6
Polydextrose intake (g/d)	0	4	8	12
Morning	0	4	4	6
Evening	0	0	4	6

subjects reported fever or diarrhea during the trial period and all subjects gave written consent. Subjects had no history of heart, lung, kidney, liver, or metabolic disease. The subjects were randomly assigned to 4 groups: A (0 g polydextrose/d; control), B (4 g polydextrose/d), C (8 g polydextrose/d), and D (12 g polydextrose/d). Polydextrose was provided in a double-blind manner as a powder dissolved in warm water (100 mL) and was drunk within 10 min of preparation. Polydextrose (Litesse) samples were provided by Pfizer (now Danisco Cultor), Ardsley, NY. The study was conducted according to the ethical guidelines of the Rui Jin Hospital Ethics Committee (Shanghai, China).

Study design

Before the feeding phase (days −4 to −7) began, subjects underwent a screening that included a physical examination, a dietary history, a family medical history, questions about bowel habits, and measurements of clinical chemistry indexes. An oral-glucose-tolerance test was conducted at baseline. On days −10 and 29, colonoscopy biopsy samples were taken from a cohort group (n = 3/group) for pulse labeling of colonocytes with [^{3}H]thymidine to study colonic crypt cell proliferation. A dietary control was conducted on days −4 to −1, ie, all meals were provided at the clinic (also on days 26–28). Before the feeding phase began on day −1, baseline measurements of clinical chemistry indexes and hemoglobin were made and feces were collected for determination of pH, microflora, sterols, and moisture (3-d pooled sample). The feeding phase, including polydextrose intake, began on day 0 and continued for 28 d. Body weight was measured on day 0. Clinical chemistry indexes, hemoglobin, body weights, and dietary records were conducted and a lifestyle questionnaire was completed on days 7, 14, 21, and 28. Modified glucose tolerance (ie, response to 50 g glucose + polydextrose) and the glycemic index were determined on days –7, 0, and 29. At the end of the feeding phase (day 28), feces were collected for determination of pH, microflora, sterols, and moisture (3-d pooled sample). An exit interview was conducted on day 29.

Feeding

All subjects were required to eat meals provided by the clinic during the dietary control phase (days −4 to −1 and 26–28). The meals were prepared as typical Chinese food and provided 9450–10710 kJ/d (2250–2550 kcal/d), 60–70 g protein/d, 50–60 g lipid/d, and 15–18 g fiber/d. Fruit consumption was limited to one piece per day. During the feeding phase, each subject was asked to record the foods consumed at each meal, to approximate the amount of food consumed at each meal, to report his or her daily activities, and to report any adverse effects experienced after polydextrose intake. Weekly visits to the clinical center were made to ensure compliance and for the conduct of biochemistry tests.

Serum analysis

Plasma electrolytes, indexes of liver and renal function, fasting blood sugar, lipids, and cholesterol were measured by using a SMAC-II automatic biochemistry analyzer (Beckman, Palo Alto, CA) in the clinical laboratories of Rui Jin Hospital. Glycated hemoglobin (Hb A_{1c}) was analyzed at the Shanghai Endocrinology Institute with a DCA 2000 Analyzer (Bayer, Tarrytown, NY). Glucose tolerance and the glycemic index were determined after ingestion of 50 g glucose. Blood samples were taken to determine fasting glucose concentrations at baseline and 0, 30, 60, 90, 120, and 150 min after glucose consumption. The blood glucose response to the modified oral-glucose-tolerance test versus time was plotted for each subject. The glycemic index of the test samples was calculated from the incremental area under the curve (IAUC) of the blood glucose response divided by the IAUC of the baseline response and expressed as a percentage (15). Any area beneath the fasting concentration curve was ignored. To determine the statistical significance of these differences, the glycemic index for each subject was calculated and averaged within each group [groups A (control), B, C, and D] and for each day (−7, 0, and 29).

Stool sampling

The stool was collected over 3 consecutive days during the dietary control period to determine fecal wet and dry weights and pH. The fresh stool collected on days −1 and 28 was sent to the Clinical Bacteriological Laboratory (Rui Jin Hospital) within 1 h after defecation for culture of microflora and determination of SCFAs.

Fecal culture

Bacteria

The fresh stool samples were analyzed for the presence of *Bacteroides fragilis*, *B. vulgatus*, *B. intermedius*, and *Bifidobacterium* and *Lactobacillus* species. Stool samples (0.5 g) were diluted with water (2 mL) and drop-seeded onto anaerobic blood agar plates. Plates were incubated for 48 h at 35°C and then counted, smeared, and Gram stained. An oxygen resistance test was then performed, followed by incubation for 48 h at 30°C in selective culture media (*Lactobacillus* selection agar for *Lactobacillus*, blood liver agar for *Bifidobacterium*). An evaluation followed.

Short-chain fatty acids

Fecal SCFA determinations were made at the Medical Testing Center of the Shanghai Second Medicine University. Samples of ≈0.5 g fresh feces were diluted with 2 mL normal saline solution followed by acidification with 1 mL of 50% H_2SO_4 solution.

TABLE 2
Glycemic indexes before and after polydextrose intake[1]

Day	Group A (control)	Group B	Group C	Group D
−7 (baseline)	100 ± 12	101 ± 12	100 ± 12	100 ± 11
0	101 ± 13	101 ± 12	95 ± 9	88 ± 12[2,3]
29	100 ± 12	102 ± 12	95 ± 10	88 ± 10[2,3]

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively.
[2] Significantly different from baseline within the same group, $P < 0.01$.
[3] Significantly different from the control group (baseline), $P < 0.01$.

This solution was then extracted with 2 mL ether and 1 μL extract was injected into the gas chromatograph. A GC-9A gas chromatograph with a flame ionization detector (Shimadzu Corp, Kyoto, Japan) was used. A column stationary phase of 10% fatty acids, a column temperature of 70°C, and a detector temperature of 230°C were used.

Measurement of colonocyte proliferation

During the pancolonoscopy, 3 biopsies of normal cecal mucosa were taken from each subject. The samples were immersed in Eagle's medium and incubated for 3 h in an equimolar sodium chloride solution. Next, the proliferating cells were pulse labeled by incubating them with [^{3}H]thymidine for 1 h. The samples were fixed in formalin, embedded in paraplast, section-cut into 4-μm slices, and stained with Schiff's acid reagent (Feulgen reaction). Sections were soaked in Ilford K_2 emulsion (Ilford Ltd, Knutsford, United Kingdom) for 15 d by using standard autoradiographic methods. The labeling frequency of colonocytes was estimated by light microscopy in 15 longitudinally sectioned crypts of each run. The number of labeled and unlabeled cells per crypt column was determined and the whole-crypt labeling index (LI) (labeled cells per crypt column/labeled cells per crypt column + unlabeled cells per crypt column) was calculated. Each crypt was equally divided into 5 compartments, with compartment 1 representing the crypt base and compartment 5 representing the crypt surface. Thus, the compartment labeling index (CLI) (labeled cells in the compartment/LI + unlabeled cells in the compartment) was calculated. The mean LI or CLI values of 15 crypts were determined (16).

Observation of physiologic effects

Physiologic reactions after polydextrose intake were recorded, including frequency of defecation, ease of defecation, abdominal distention, abdominal cramps, diarrhea, hypoglycemic symptoms (eg, sweating, pale skin, palpitation, and abdominal colic). Most symptoms were rated on a scale of 1 to 10; ease of defecation was rated on a scale of −3 to 3 and frequency of defecation was reported as the number of times per day.

Statistics

The results are presented as means ± SDs. Analysis of variance was used to compare groups (before compared with after polydextrose intake) and Dunnett's multiple (pairwise) comparison procedure was used to determine differences between groups A, B, C, and D. SAS (version 6.12; SAS Institute Inc, Cary, NC) was used for the analyses.

RESULTS

Clinical biochemistry indexes (eg, measures of liver and renal function, blood electrolytes, fasting blood sugar, triacylglycerol, cholesterol, and serum Hb A_{1c}) did not change significantly after polydextrose intake for 28 d (data not shown). The IAUCs for glucose were flattened in groups C and D after polydextrose ingestion. The glycemic index decreased significantly from baseline on days 0 and 28 in group D (**Table 2**).

The subjects who consumed polydextrose had marked changes in bowel function (**Table 3**). Groups B, C, and D showed significant improvements in bowel function (increased frequency and ease of defecation) and reported no laxation problems (abdominal distention, cramps, and diarrhea). There were no significant differences between groups in ratings of abdominal distension and no reports of abdominal cramps, diarrhea, hypoglycemic symptoms, or other discomforts (data not shown). Fecal weights (wet and dry) increased and fecal pH decreased after polydextrose intake (**Table 4**). This change was most significant in groups C and D. The constitution and contents of SCFAs in feces changed greatly after polydextrose intake (**Table 5**). Particularly interesting was the significant increase in butyrate, isobutyrate, and acetate in groups C and D. Significant changes in fecal microflora were noted after polydextrose intake (**Table 6**). *Bacteroides* species decreased, whereas *Lactobacillus* and

TABLE 3
Bowel function before and after polydextrose intake[1]

	Group A (control)	Group B	Group C	Group D
Before polydextrose intake				
Frequency of defecation (times/d)	1.04 ± 0.07	1.05 ± 0.10	1.11 ± 0.16	1.05 ± 0.15
Ease of defecation	−0.21 ± 0.10	−0.18 ± 0.12	0.20 ± 0.14	−0.14 ± 0.13
After polydextrose intake				
Frequency of defecation (times/d)	1.10 ± 0.21	1.47 ± 0.28[2,3]	1.74 ± 0.37[2,3]	1.89 ± 0.26[2,3]
Ease of defecation	0.41 ± 0.15	1.36 ± 0.22[2,3]	1.88 ± 0.26[2,3]	2.35 ± 0.30[2,3]

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively. Ease of defecation was rated on a scale of –3 to 3.
[2] Significantly different from value before polydextrose intake within the same group, $P < 0.01$.
[3] Significantly different from the control group (baseline), $P < 0.01$.

TABLE 4
Stool weight and pH before and after polydextrose intake[1]

	Group A (control)	Group B	Group C	Group D
Before polydextrose intake				
Fecal wet weight (g/d)	103 ± 12.3	106 ± 16.4	101 ± 13.6	98 ± 12.5
Fecal dry weight (g/d)	32.2 ± 8.3	34.0 ± 6.8	31.5 ± 7.4	29.6 ± 8.9
Fecal pH	7.06 ± 0.18	7.05 ± 0.21	7.00 ± 0.25	6.93 ± 0.18
After polydextrose intake				
Fecal wet weight (g/d)	106 ± 15.9	115 ± 16.7	128 ± 27.4[2,3]	142 ± 18.3[3,4]
Fecal dry weight (g/d)	34.5 ± 8.8	38.3 ± 13.1	41.8 ± 16.3[2]	47.8 ± 18.2[3,4]
Fecal pH	7.04 ± 0.18	6.89 ± 0.16[5]	6.71 ± 0.23[3]	6.37 ± 0.27[3,4]

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively.
[2,4] Significantly different from value before polydextrose intake within the same group: [2] $P < 0.05$, [4] $P < 0.01$.
[3,5] Significantly different from the control group (baseline): [3] $P < 0.01$, [5] $P < 0.05$.

Bifidobacterium species increased in all groups after polydextrose intake. The whole-crypt CLI increased after polydextrose ingestion (**Table 7**). Changes in the CLI occurred mainly in groups C and D and in compartments 1, 2, and 3 (**Table 8**).

DISCUSSION

Polydextrose intake by the Chinese subjects led to many physiologic effects associated with dietary fiber. The Chinese diet is relatively low in fat (≈20% of energy from fat); therefore, we did not expect to measure an effect of polydextrose on blood lipids. Polydextrose had no influence on measured blood chemistry indexes. Fasting blood glucose and Hb A_{1c}, indicators of long-term stability of blood glucose concentrations, remained unchanged. A polydextrose intake of 12 g (plus 50 g glucose) flattened the postprandial glucose response significantly compared with a 50-g glucose control. The glycemic indexes were significantly lower in group D on day 0 (88 ± 12%) and day 29 (88 ± 10%) than at baseline (day −7). Note that the results on day 0 indicate an immediate benefit from polydextrose, confirming that polydextrose is nonglycemic. This suggests that polydextrose results in a reduction in glucose absorption from the intestine, possibly related to delayed gastric emptying due to polydextrose bulking and increased viscosity in the bowel (17–19). Similar results were obtained by others (15, 20), who found that the glycemic and insulin responses were markedly flattened in healthy and diabetic volunteers after they consumed fiber-enriched meals. Long-term consumption of foods high in dietary fiber could reduce urinary glucose losses and improve the control of diabetes.

Because of its excellent water-holding capacity, intake of undigested polydextrose resulted in an increase in bowel peristalsis and feces output. Most subjects reported a softening of feces and improved ease of defecation after ≈2 d of polydextrose ingestion. There was a dose-response increase in the frequency and ease of defecation and in both the wet and dry weights of feces in groups B, C, and D after polydextrose intake. The fecal wet weight increased by ≈25% and 40% in groups C and D, respectively. There was a dose-response decrease in fecal pH, due mainly to the production of SCFAs (21, 22). A high fecal output and a low bowel pH can suppress the production of enteric toxins, such as indole and *p*-cresol (8, 23, 24). This plays an important role in the prevention of constipation and diverticulosis and thereby reduces the risk of bowel cancer (25–27). Abdominal distention, diarrhea, cramps, and hypoglycemic symptoms were not reported by any of the subjects.

Dietary fiber is available for fermentation by anaerobes in the colon. It can increase stool weight and change the constitu-

TABLE 5
Fecal short-chain fatty acid contents before and after polydextrose intake[1]

	Group A (control)	Group B	Group C	Group D
			mg/g stool	
Before polydextrose intake				
Acetate	4.04 ± 0.16	4.12 ± 0.24	4.13 ± 0.21	4.09 ± 0.22
Propionate	1.48 ± 0.17	1.48 ± 0.23	1.52 ± 0.25	1.52 ± 0.27
Butyrate	0.92 ± 0.16	0.95 ± 0.21	0.98 ± 0.17	0.96 ± 0.20
Isobutyrate	0.17 ± 0.06	0.18 ± 0.08	0.20 ± 0.10	0.19 ± 0.09
Isovalerate	0.41 ± 0.13	0.39 ± 0.12	0.43 ± 0.18	0.42 ± 0.15
After polydextrose intake				
Acetate	4.12 ± 0.19	4.18 ± 0.26	4.70 ± 0.33[2,3]	5.12 ± 0.31[3,4]
Propionate	1.50 ± 0.24	1.52 ± 0.22	1.55 ± 0.36	1.48 ± 0.35
Butyrate	0.94 ± 0.23	1.10 ± 0.17	1.31 ± 0.24[2,3]	1.41 ± 0.34[3,4]
Isobutyrate	0.20 ± 0.15	0.23 ± 0.12	0.26 ± 0.18[2]	0.29 ± 0.16[4,5]
Isovalerate	0.38 ± 0.15	0.36 ± 0.13	0.40 ± 0.26	0.41 ± 0.28

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively.
[2,4] Significantly different from value before polydextrose intake within the same group: [2] $P < 0.05$, [4] $P < 0.01$.
[3,5] Significantly different from the control group (baseline): [3] $P < 0.01$, [5] $P < 0.05$.

TABLE 6
Fecal microflora contents before and after polydextrose intake[1]

	Group A (control)	Group B	Group C	Group D
		$\times 10^9$/g stool		
Before polydextrose intake				
Bacteroides fragilis	1.35 ± 0.46	1.54 ± 0.37	1.37 ± 0.40	1.36 ± 0.39
Bacteroides vulgatus	0.78 ± 0.26	0.84 ± 0.28	0.66 ± 0.22	0.80 ± 0.31
Bacteroides intermedius	0.34 ± 0.12	0.46 ± 0.11[5]	0.37 ± 0.24	0.33 ± 0.16
Lactobacillus	0.24 ± 0.15	0.26 ± 0.08	0.32 ± 0.14	0.28 ± 0.12
Bifidobacterium	0.57 ± 0.23	0.46 ± 0.21	0.40 ± 0.16[3]	0.52 ± 0.19
After polydextrose intake				
Bacteroides fragilis	1.53 ± 0.52	0.58 ± 0.32[2,3]	0.24 ± 0.07[2,3]	0.27 ± 0.08[2,3]
Bacteroides vulgatus	0.83 ± 0.26	0.45 ± 0.19[3,4]	0.13 ± 0.05[2,3]	0.16 ± 0.04[2,3]
Bacteroides intermedius	0.35 ± 0.18	0.26 ± 0.17[4,5]	0.09 ± 0.03[2,3]	Not detected[2,3]
Lactobacillus	0.27 ± 0.10	1.10 ± 0. 28[2,3]	1.36 ± 0.53[2,3]	1.92 ± 0.48[2,3]
Bifidobacterium	0.48 ± 0.17	1.54 ± 0.37[2,3]	3.05 ± 1.13[2,3]	5.29 ± 1.74[2,3]

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively.
[2,4] Significantly different from value before polydextrose intake within the same group: [2] $P < 0.01$, [4] $P < 0.05$.
[3,5] Significantly different from the control group (baseline): [3] $P < 0.05$, [5] $P < 0.01$.

tion of microflora, eg, increase the *Lactobacillus* content (28). Lactic acid, produced by *Lactobacillus*, can reduce the intestinal pH. *Bifidobacterium* has a strong inhibiting effect on *Escherichia coli* and *Bacteroides* in an acidic environment. The mechanism is probably related to the production of antibiotic-like substances during the proliferation of some specific strains (28). In the present study, *Lactobacillus* and *Bifidobacterium* concentrations were significantly higher and *Bacteroides* species were significantly lower in fresh stool after all polydextrose intakes. The relation between the proliferation of *Bifidobacterium* species with an acidic environment, dietary fiber sources, and amounts of *Bifidobacterium* in the bowel require further investigation.

The main products of fermentation are hydrogen and carbon dioxide gases and SCFAs—primarily acetate, propionate, and butyrate (26, 29). The relative amount of each product depends

TABLE 7
Whole-crypt labeling index before and after polydextrose intake[1]

	Group A (control)	Group B	Group C	Group D
Before	0.0664 ± 0.006	0.0673 ± 0.008	0.0675 ± 0.005	0.0680 ± 0.008
After	0.0671 ± 0.007	0.0816 ± 0.016[2,3]	0.1021 ± 0.017[3,4]	0.1313 ± 0.015[3,4]

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively.
[2,4] Significantly different from value before polydextrose intake within the same group: [2] $P < 0.05$, [4] $P < 0.01$.
[3] Significantly different from the control group (baseline), $P < 0.01$.

TABLE 8
Compartment labeling index before and after polydextrose intake

	Compartment				
	1	2	3	4	5
Group A (control)					
Before	0.102 ± 0.012	0.136 ± 0.025	0.068 ± 0.014	0.040 ± 0.006	0.007 ± 0.002
After	0.105 ± 0.011	0.133 ± 0.022	0.072 ± 0.016	0.038 ± 0.003	0.008 ± 0.003
Group B					
Before	0.106 ± 0.020	0.137 ± 0.030	0.071 ± 0.016	0.042 ± 0.008	0.008 ± 0.002
After	0.118 ± 0.034	0.148 ± 0.035	0.087 ± 0.027	0.051 ± 0.016[2]	0.007 ± 0.004
Group C					
Before	0.105 ± 0.011	0.134 ± 0.029	0.070 ± 0.015	0.039 ± 0.005	0.007 ± 0.003
After	0.138 ± 0.032[3,4]	0.228 ± 0.049[4,5]	0.146 ± 0.031[4,5]	0.058 ± 0.017[3,4]	0.009 ± 0.005
Group D					
Before	0.107 ± 0.016	0.135 ± 0.034	0.073 ± 0.020	0.035 ± 0.008	0.006 ± 0.002
After	0.177 ± 0.028[3,4]	0.283 ± 0.054[4,5]	0.161 ± 0.034[4,5]	0.057 ± 0.024[4,5]	0.010 ± 0.003

[1] $\bar{x}$ ± SD. 0, 4, 8, and 12 g polydextrose/d for 28 d in groups A, B, C, and D, respectively.
[2,4] Significantly different from the control group (baseline): [2] $P < 0.05$, [4] $P < 0.01$.
[3,5] Significantly different from value before polydextrose intake within the same group: [3] $P < 0.05$, [5] $P < 0.01$.

mainly on the substrates entering the colon and the types of microflora that proliferate (30). Acetate is produced in the largest amounts from fermentation of dietary fiber. It is absorbed into the bloodstream together with propionate and is metabolized in the liver and peripheral tissues. Butyrate, arguably the most important fermentation product, is generally regarded as an energy resource for colonocytes. Butyrate has been shown to have desirable effects on colonic epithelial cells, including stabilization of DNA and down-regulation of oncogenes (31–33). The production of acetate and butyrate in the feces of subjects who ingested 8 or 12 g polydextrose (groups C and D, respectively) increased significantly. Thus, a polydextrose intake ≥8 g/d can result in substantial production of butyrate and consequent desirable effects on the human colon.

Measurement of colonic crypt cell proliferation provides an indirect measure of SFCA production in the colon, particularly butyrate (16, 34–37). The present study showed that consumption of polydextrose promoted the growth of normal cecal epithelial cells. The whole-crypt labeling index increased after all polydextrose intakes, especially in groups C and D. Growth occurred mainly in the base compartments of the crypt, ie, compartments 1–3. Scheppach et al (16) found similar results after incubating normal human cecal colonocytes directly with SCFAs; the effect was most pronounced for butyrate and propionate. These investigators also found growth to be significant only in compartments 1–3. Thus, the results of Scheppach et al agree with those of the present study (Table 5). The effects of butyrate on regulation of cell phase and on morphologic changes remain unknown.

In conclusion, polydextrose is a dietary fiber that has many physiologic benefits. Consumption of polydextrose significantly improved bowel function, softened the feces, and improved the ease of defecation, with no adverse effects. Polydextrose intake inhibited excessive glucose absorption from the small intestine and was fermented in the lower gut to produce SCFAs, including butyrate. Polydextrose promoted the proliferation of favorable intestinal microflora and decreased the pH of the bowel. Therefore, daily intake of ≥4–12 g polydextrose improves physiologic function without adverse effects.

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