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Safety Assessment and Caloric Value of Partially Hydrolyzed Guar Gum

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ABSTRACT: Guar gum and partially hydrolyzed guar gum (PHGG) are food ingredients that have been available for many years. PHGG is the partially hydrolyzed product from guar gum obtained from the Indian cluster bean (*Cyanopsis tetragonolopus*). The gum (CAS Registry No. 9000-30-0) is composed of galactomannan, a gel-forming polysaccharide with a molecular weight ranging from 200 to 300 kDa. The intact and partially hydrolyzed forms have multiple food applications. The intact material can be used to control the viscosity, stability, and texture of foods. PHGG is highly soluble and has little physical impact on foods. Both forms are indigestible but are excellent sources of fermentable dietary fiber. The caloric value of intact guar gum is accepted as 2.0, whereas the caloric value of PHGG has not been firmly established. It is the goal of this paper to review the chemistry, safety, in vivo effects, and caloric value of PHGG.

KEYWORDS: *guar gum, partially hydrolyzed guar gum, PHGG, caloric availability*

INTRODUCTION

Most people consuming a Western diet fail to consume sufficient levels of dietary fiber, particularly fermentable fiber. Addition of fermentable fiber to the diet could result in multiple health benefits. Guar gum has a long history of use in multiple food applications. Guar gum is added to beverages, sauces, dressings, and other liquid products to increase viscosity and improve mouthfeel. In addition, guar has been shown to have an influence on blood lipids, satiety, and diarrhea control. Partial hydrolysis of the guar gum results in a soluble version with little influence on viscosity but provides many of the other benefits of intact guar gum. Guar gum and partially hydrolyzed guar gum (PHGG) are not digestible by mammals but are readily fermented by bacteria in the lower gastrointestinal tract. This paper summarizes the chemistry, safety studies, and nutritional assessments that have been done on guar gum and PHGG including data that demonstrate that the caloric value of the partially hydrolyzed guar gum is between 1.6 and 1.9 kcal/g. Partially hydrolyzed guar gum is an effective approach to adding soluble fermentable fiber to beverages.

CHEMISTRY

Guar gum is derived from the Indian cluster bean (*Cyanopsis tetragonolopus*). The gum (CAS Registry No. 9000-30-0) is composed of galactomannan polymers, gel-forming polysaccharides with molecular weights ranging from 200 to 300 kDa. The chemical structure of the material is D-galacto-D-mannan ranging from 50,000 to 8,000,000 units. The mannose/galactose ratio is approximately 2:1. The polymers are linear chains of (1−4)-linked β-D-mannospyranosyl units with (1−6)-linked α-galactosyl residues as side chains (Figure 1). Clarified guar gum is devoid of cell structure and is a white to slightly yellowish odorless free-flowing powder. It is insoluble in organic solvents but dissolves easily in cold water. The gum is stable at a pH range from 4.0 to 10.5. Intact guar gum is an excellent stabilizer. Because it is nonionic, it thickens at extremes of pH and ionic strength.[1,2] Improvement of emulsifying properties using galactomannans has been suggested to require polysaccharides of molecular sizes of more than 6000−12000.[3] PHGG has the same basic molecular structure as the parent guar gum except the D-galacto-D-mannan chains are reduced to oligomers of 3−30 units (1−100 kDa) with a midrange of about 10 units.[1,2]

SAFETY

Guar gum was evaluated in 1973 by the Select Committee on GRAS (Generally Regarded as Safe) Substances (SCOGS 1973), which concluded that guar gum is toxicologically safe at current levels based on short-term animal studies and long-term consumption by humans in India, Pakistan, and the United States. The committee suggested only that long-term animal feeding studies be conducted at levels exceeding maximum daily human doses to determine whether an increase in daily human consumption would represent a health issue.[4]

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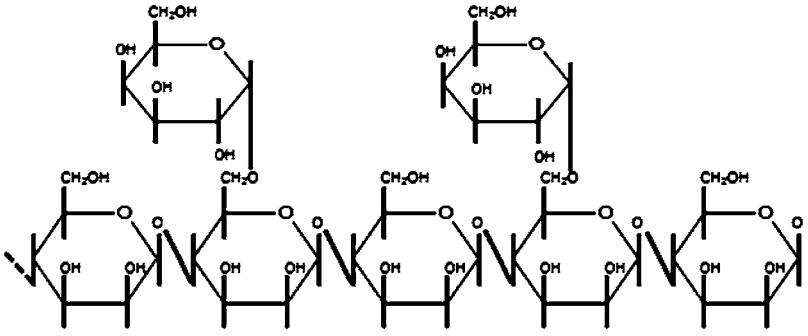

Figure 1. Structure of guar gum or PGHH. β-1,4-Mannose units with α-1,6-galactose units are randomly distributed in a 1.6:1 to 1.8:1 ratio.

On the basis of this finding, the U.S. FDA affirmed the GRAS status of guar gum and promulgated regulation 21 CFR §184.1339, which delineates GRAS conditions of use for guar gum meeting specifications in the Food Chemicals Codex. These conditions allow addition to foods at maximum levels ranging from 0.35 to 2.0% in a variety of food categories. Technical effects include use as an emulsifier, stabilizer, thickener, and firming agent. Additionally, guar gum was reviewed by the Joint FAO/WHO Expert Committee on Food Additives (JECFA 1975) and assigned an Allowable Daily Intake (ADI) of "not specified".

In 1991, PHGG was determined to be GRAS for addition to enteral feeding products.[1] The Expert Panel determined that the intended addition of PHGG to enteral products to provide up to 13 g/day PHGG (along with about 10 g/day insoluble fiber from other sources) is both safe and GRAS. Additionally, the panel noted that this concentration might prove to be inadequate for patients with special physiological conditions. The panel determined that the in the future addition of PHGG would be both safe and GRAS at higher concentrations than were then contemplated, providing up to 25 g/day PHGG.[1] The Sandoz Expert Panel regarded PHGG as GRAS when added as dietary fiber at levels up to 13 g/1000 kcal diet along with 7 g/1000 kcal of diet from an insoluble fiber from other sources; thus, a total of 20 g/1000 kcal of added fiber would be considered GRAS.

The Sandoz Expert Panel also determined that addition of PHGG to liquid oral supplements at a concentration sufficient to provide up to 19 g/day PHGG is also safe and GRAS. These supplements are intended for the treatment of geriatric patients consuming a normal diet but exhibiting diminished appetite and provide about 250 kcal/serving; up to 3 servings/day are given as determined by the patient's primary-care physician and dietitian.[1] In terms of energy content, the maximum addition of PHGG to liquid oral supplements is sufficient to provide 20 g/1000 kcal soluble fiber.

In 1993, an Expert Panel convened by the Life Sciences Research Office (LSRO) of the Federation of American Societies for Experimental Biology evaluated the safety and GRAS status of the addition of PHGG to conventional foods.[2] The panel concluded that consumption of PHGG up to 20 g/day does not represent a hazard; however, more evidence is required to determine its safety at significantly increased doses.[2]

In 2006, a different Expert Panel determined that addition of PHGG to enteral products and conventional foods as a source of functional fiber to increase the daily intake of total fiber is GRAS. This determination was based on scientific procedures as described under 21 CFR §170.30(b). In addition to information previously available, comprehensive searches of the literature through February 2006 were conducted and served as the basis for the preparation of a monograph summarizing the totality of the information available germane to determining the safety of the intended uses of PHGG.[5] The monograph included unpublished research corroborating the published information that alone was the basis for the GRAS determination.

The Expert Panel determined that the amount of functional fiber provided by the intended use of PHGG is within recommended levels and the total exposure to PHGG resulting from this use is well within concentrations shown by extensive animal research and human studies to be safe and tolerable. The panel further determined that the safety of this level of intake was based on publicly available and accepted information and is generally recognized by experts qualified by scientific training and experience to evaluate the safety of substances added to food. The panel concluded that PHGG is safe for its use in enteral nutrition at a maximum of 30 g/100 kcal and in foods according to cGMP based on its characterization, history of consumption, and lack of evidence suggesting toxicity.[5]

EFFECTS ON SATIETY AND WEIGHT CONTROL

Slower weight gain in animal studies often has been associated with ingestion of guar gum.[2] Calvert et al.[6] fed rats, and over 4 weeks the rats on a fiber-free diet gained 138 g while the rats fed 5% guar gum gained only 106 g. Poksay and Schneeman[7] observed that rats on a fiber-free diet consumed 16 g food/day and weighed 298 g after 4 weeks compared to rats on a 10% guar gum diet that consumed 16.5 g food/day and gained 251g over the same period. Shah et al. reported significant decreases in body weight gain, food consumption, and food efficiency in rats when diets containing 5 or 10% guar gum were fed for 3 or 4 weeks.[8,9] However, Ikegami et al. found that food consumption and body weight gain were not decreased in rats fed a diet containing 5% guar gum for 2 weeks.[10]

Body weights of male Osborne Mendel rats fed diets containing 1−15% guar gum for 91 days were significantly lower than those of control males even though food consumption was at least 90% of control animals' intake.[11] After 12 weeks, male rats on diets containing 7.5 and 15% guar gum gained 8.3 and 15% less weight, respectively, than rats on the control diet. In the same period, female rats receiving 7.5% guar gum gained 10% less weight than control animals, and females consuming 15% guar gum gained 16.5% less than control. In another subchronic study, the weight gain of male Fischer 344 rats fed a diet containing 5 or 10% guar gum was depressed 7 and 16% relative to control animals.[12] Only a slight depression in weight gain was observed for female Fischer 344 rats fed the diets containing 10% guar gum. Using B6C3F1

Table 1. Clinical Trials on Influence of Guar Gum and PHGG on Satiety and Weight Gain

reference	subjects	study	results
French and Read, 1994[130]	8 healthy males consumed low-fat and high-fat soups with and without added guar gum	times to return to hunger and decline in fullness were measured along with gastric emptying	return to hunger and fullness were greater when guar gum was added to high-fat soup
Pasman et al., 1997[14]	17 free-living obese women who had lost weight	for 1 week, subjects received 20 g PHGG/day compared to 1 week with no added fiber; food diary recorded last 3 days	when on PHGG, energy intake was 5.4 MJ compared to 6.7 MJ with no intervention
	14 obese women on fixed energy intake of 4 or 6 MJ/day compared to no intervention	1 week on each diet; food diary recorded last 3 days	no differences were observed in hunger scores no differences in hunger and satiety scores, at either energy level, were observed with or without added PHGG
Heini et al., 1998[32]	25 obese, otherwise healthy females	subjects received 3.3 MJ/day diet with or without added 20 g/day added PHGG	PHGG resulted in higher postprandial CCK responses in pmol/L: time placebo PHGG 0 3.31 4.51 15 3.34 4.45 30 4.89 4.39 60 3.68 4.27 no other significant differences were observed
Lavin and Read, 1995[131]	30 healthy men consumed 250 mL of a 30% glucose drink with or without 2% guar gum	glucose and insulin were monitored for 2 h	added guar gum resulted in lower peak glucose (7.8 vs 6.9 mmol/L) and lower peak insulin (82.1 vs 47.4 mU/L) guar resulted in ~20% lower hunger rating over 3 h and doubling of fullness rating over the same time
Kovacs et al., 2001[33]	28 overweight males	1 week on baseline diet; three treatments of 2 weeks each receiving semisolid meal with or without guar gum or a solid meal with the same energy (947 kJ)	all three treatments were effective for weight loss; guar added to semisolid meal reduced increase in appetite, hunger, and desire to eat no changes were statistically significant
Kovacs et al., 2002[132]	15 overweight males	subjects consumed low-energy diet 3 times a day and had one free-choice dinner; lowenergy meals were semisolid, semisolid plus 2.5% guar gum, or a solid meal	peak glucose was lower in guar group (115 mg/dL) compared to the solid group (120 mg/dL) or the semisolid control (125 mg/dL); peak time appeared to be delayed about 30 min appetite, energy intake, and diet patterns were not different

mice in the same experimental protocol, weight gains of female mice fed diets containing 5 and 10% guar gum were depressed by 15 and 16% relative to control animals. Weight gains of male mice were decreased to a lesser extent, 6 and 9%, for the 5 and 10% guar gum diets, respectively. Food consumption of the guar-fed mice was equal to or greater than that of the control animals.[12]

In a 28 day subchronic toxicity study of PHGG cited in LSRO,[2] Sprague–Dawley rats were given 500 or 2500 mg/kg body weight/day PHGG by gavage. A slight but not significant growth depression was reported in male rats given the higher dose of PHGG; however, these animals also consumed somewhat less food. No significant differences were reported in body weight gain or food consumption in male weanling Wistar rats fed diets containing 0, 5, or 10% guar gum or PHGG for 3 weeks.[13] In the 3 week study, animals receiving 0, 5, or 10% PHGG or 5% guar gum all had similar body weights at the end of the study (177.7–186 g).

The slower weight gain observed when animals were fed guar gum was not observed in human clinical trials in which guar gum was added to the diet.[14] Other clinical trials that show little influence on weight gain and satiety are found in Table 1. The majority of clinical studies reported no weight change in human adults fed guar gum.[15−29] These studies included investigations in healthy adult subjects and persons with diabetes and/or hypercholesterolemia who consumed from about 9 to 32 g of guar gum daily for periods ranging from 3 weeks to 12 months. In a study with hypercholesterolemic women, subjects received 15 g guar gum per day for 4 months. The guar group lost 2.3 kg but showed no changes in serum lipids.[30] When guar gum was fed to non-insulin-dependent diabetics with poor metabolic control small (<4 kg), no statistically significant reductions in body weight were observed. In a short study over 3 weeks with hyperlipidemic patients, no changes were seen in body weight.[31] The LSRO[2] panel observed that the protocols of these studies may have been designed for promotion of weight maintenance rather than weight loss and that weight loss may be regarded as a beneficial effect in some instances, whereas it may be an undesirable side effect in other situations.

Assessing the effect of PHGG on weight control in obesity, Heini et al.[32] placed 25 pre- and postmenopausal obese women (average age 46; average body mass index = 35) on a controlled weight-loss diet for 2 weeks, then gave them either 20 g/day of PHGG or a placebo for 1 week, followed by a 1 week washout and a 1 week crossover. On days 1, 3, and 7 of the intervention

Table 2. Studies with Animals and Clinical Trials Assessing Influence of Guar Gum and PHGG on Serum Lipids

reference	subjects	markers	results
Takeno et al., 1990[42]	rats	compared intact guar gum and PHGG at 5% of diet, compared to nondietary fiber diets	both intact guar gum and PHGG suppressed total triglyceride and cholesterol levels (abstract)
Ide et al., 1991[43]	rats fed high saturated fat diets (lard or palm oil)	guar gum, PHGG and FOS were fed at 8% of diet at expense of sucrose	in lard diet, cholesterol was lowered from 114 to 73.6 and 84.6 mg/dL by intact guar gum and PHGG, respectively in palm oil diet, cholesterol was reduced from 140 to 85.3 mg/dL triglycerides were reduced by >60% by both intact and PHGG on lard diets; in palm oil diets reductions were 37% for intact guar gum and 31% for PHGG
Evans et al., 1992[41]	rats	3-galctomannans with differing galactose/mannose ratios	serum cholesterol from guar reduced from 3.90 mmol in control to 3.26 mmol when 80 g/kg in diet
Yamada et al., 1999[36]	rats	fed diets with water-insoluble cellulose or intact guar gum, PHGG, glucomannan, or highly methoxylated pectin	all four soluble fibers reduced total cholesterol from 127 mg/dL (cellulose control) to ranges from 77 to 89 mg/dL control cellulose triglycerides was 57 mg/dL; guar and PHGG were 27−30 mg/dL
Favier et al., 1997[44]	rats	fed intact guar or PHGG at the expense of starch in diet that contained 0.4% cholesterol	total cholesterol was 2.18 mmol/L for control and 1.37 and 1.97 mmol/L for intact and PHGG, respectively triglycerides were reduced from 1.41 to 1.22 and 1.07, respectively
Suzuki and Hara, 2004[56]	rats fed fructose	glucose tolerance tests on rats fed fructose, glucose, and fructose plus PHGG	on day 28, rats on glucose AUC was 10.6 mmol/L/h compared to 9.5 mmol/L/h for rats receiving fructose and PHGG insulin in fructose group at 28 days was 0.54 nmol/h/L compared to 0.29−0.36 nmol/h/L for other groups

weeks, measures were made of fasting serum glucose and insulin, plasma leptin and cholecystokinin (CCK), respiratory quotient, and hunger/satiety ratings. These same measures were taken 0, 15, 30, 60, and 120 min after a test meal providing 320 kcal with or without 8 g of PHGG. No significant effect was found on fasting values of satiety, glucose, insulin, CCK, leptin, or respiratory quotient or on 2 h postprandial insulin, glucose, respiratory quotient, or satiety. However, PHGG significantly increased postprandial CCK increases by 0.57 pmol/L in the placebo group compared to 2.5 pmol/L in the group receiving PHGG. There was no change in postprandial hunger ratings between the two groups. The authors concluded that soluble fiber is unlikely to play a critical role in weight control.

Studies with PHGG on appetite ratings from 7 to 11 days of supplementation[14,32−34] showed limited effects on satiety and long-term weight control (see Table 1). Van de Ven et al.[35] observed reduced food intake in 15 healthy women following consumption of a fructose/PHGG drink before a test meal. They reported that PHGG-supplemented meals reduced hunger and the desire to eat, but there was little influence on weight loss.

MAINTENANCE OF SERUM TRIGLYCERIDES AND ATTENUATION OF BLOOD CHOLESTEROL CONCENTRATIONS

Tables 2 and 3 summarize the numerous animal and clinical trials that have been conducted with intact guar and PHGG. Four-week-old male Sprague−Dawley rats were given diets with water-insoluble cellulose or a water-soluble fiber (intact guar gum, PHGG, glucomannan, or highly methoxylated pectin) to compare the effects of the fibers on serum lipids.[36] All four soluble fibers reduced total cholesterol from 127 mg/dL (cellulose control) to ranges from 77 to 89 mg/dL with no significant difference among the soluble fibers. Glucomannan, guar gum, and PHGG lowered triglycerides to 27−30 mg/dL compared to 57 mg/dL for the cellulose-fed animals.[36] Most other studies fail to show large changes in serum triglycerides.[37−39] (See Table 2 for animal studies and Table 3 for human clinical trials.)

The LSRO[2] report cited Gee et al.[40] as attributing the effect of guar gum on blood lipids mainly to its viscosity. On the other hand, a study in rats by Evans et al.[41] suggests that viscosity is not the complete answer to the question of mechanism of action in reducing blood cholesterol. Male adult Wistar rats weighing 200−230 g were fed for 2 weeks on a fiber-free diet or a diet containing 80 g/kg galactomannans with different galactose/mannose ratios (G:M); all diets contained 10 g/kg of cholesterol. The fibers were fenugreek gum (G:M = 1:1), guar gum (G:M = 1:2), and locust-bean gum (G:M = 1:4), which also differed substantially in their viscosity. In comparison with the fiber-free diet, all three galactomannans lowered the concentrations of cholesterol in both liver and blood plasma, as well as the rate of hepatic synthesis of cholesterol from 300 to 113 mg/liver for locust bean, 75 mg/liver for fenugreek, and 76 mg/liver for guar. Interestingly, guar gum, which caused the greatest increase in viscosity, was the least effective in lowering blood cholesterol, whereas fenugreek gum, with a slightly lower viscosity than that of locust-bean gum, was the most effective. Serum cholesterol from guar reduced total serum cholesterol in the rats from 3.90 mmol in control to 3.26 mmol when 80 g/kg guar gum was in the diet. In another study, the effect of PHGG (average molecular weight 24 kDa) was compared with that of intact guar gum (average molecular weight 300 kDa), the intact material having much greater viscosity.[42] Rats were fed hypercholesterolemic diets containing either 5% PHGG or 5% intact guar gum for 3 weeks. Intact guar gum suppressed the elevation of total cholesterol and triacylglycerides in both the plasma and the liver, whereas PHGG suppressed only plasma concentrations. Thus, PHGG retained some ability to lower plasma cholesterol despite its lower viscosity.[42]

Table 3. Clinical Trials of Guar Gum and PHGG Effects on Serum Lipids and Glycemic Markers

reference	subjects	markers	results
Jenkins et al., 1977[64]	hyperlipidemic humans	glucose changes after liquid test meal	liquid test meal with 14.5 g guar gum; 30 min glucose level reduced from 3.33 to 4.77 mmol/L; insulin reduced 50%
Kahn et al., 1981	24 subjects received 9 g guar gum/day or glucose for 4 weeks	blood lipid profiles were measured before and after 2 weeks and full 4 week treatments	after 4 weeks, total cholesterol in test group was reduced from 179.2 to 150.1 mg/dL; LDL cholesterol reduced from 118.9 to 86.5 mg/dL; triglycerides were not significantly different; there were no changes in placebo group
Aro et al., 1984[15]	14 hypercholesterolemic males received 15 g of guar for 12 weeks in a crossover design; subjects consumed 5 g guar or placebo in a beverage before each meal	fasting glucose, total and LDL serum cholesterol; serum triglycerides; serum phosphate, magnesium, iron, calcium, and urinary calcium	no significant change in glucose or serum phosphate, magnesium, iron, calcium, and urinary calcium; LDL cholesterol was reduced trom 5.32 to 4.70 mmol/L and total cholesterol was reduced from 8.23 to 7.27 mmol/L when guar gum was fed
Bosello et al., 1984[16]	12 patients with familial hyperlipoproteinemia received 16 g guar gum/day for 15 days	lipoproteins and apolipoprotein (apoC-II) and apolipoprotein C-III (apoC-III) and isoforms of apo C-III were measured	total serum cholesterol was reduced from 7.83 to 6.75 mmol/L after 15 days and was 6.94 mmol/L after 60 days of treatment; apoC-III decreased from 24.5 to 15.5% and persisted at reduced levels throughout the guar gum treatment; apoC-III returned to basal levels after guar gum was discontinued
Jarjis et al., 1984[63]	normal and diabetic humans	2.5 and 14.5 g guar gum with a 50 g glucose load	at 30 min, 2.5 g guar gum caused 40% reduction in serum glucose; 14.5 g reduced by 60%; both doses resulted in 60% reduction in insulin at 30 and 60 min
Ebeling et al., 1988[17]	9 insulin-dependent type 1 diabetics were given placebo or 5 g of guar gum four times a day for 4 weeks in a crossover study design	glucose response was tested initially and after the 4 week intervention	mean blood glucose was reduced from 8.7 to 7.7 mmol/L when guar gum was administered; no differences were observed in the HbA values; LDL cholesterol values fell from 3.58 to 2.63 mmol/L during the guar treatment; total serum cholesterol fell 21% in the same period
Uusitupa et al., 1989[28]	10 month open trial with 39 non-insulin-dependent diabetics test group received 5 g guar gum three times a day in a beverage control group received a placebo	glucose, glycosylated hemoglobin, and total cholesterol	no difference in glucose, glycosylated hemoglobin no change in control group cholesterol test group total cholesterol dropped from 6.55 to 5.69 mmol/L no change in HDL cholesterol or serum triglycerides
Ellis et al., 1991[62]	healthy humans	guar in bread high, medium, low MW	no postprandial effect on serum glucose up to 120 min 25−45% reduction in postprandial insulin at 60 and 90 min
Spiller et al., 1991[24]	11 g/day fiber from guar gum or oat fiber was given to 13 free-living men and women in water at meal time for 21 days	blood lipids were analyzed at baseline and on days 14 and 21	guar gum resulted in greater reduction in LDL cholesterol and total cholesterol than oat fiber; total cholesterol reduction from guar gum was from 244 to 2 mg/dL (average days 14 and 21); LDL cholesterol reduction was from 152 to 125 mg/dL (average days 14 and 21)
Vuorinen-Markkola et al., 1992[29]	17 hypercholesterolemic insulin-dependent patients (8 test; 9 control) consumed 5 g of guar gum or placebo four times a day for 6 weeks	hemoglobin A_{1c}, blood lipids, and fasting glucose	fasting glucose dropped from 8.7 to 7.0 mmol/L; hemoglobin A_{1c} dropped from 8.3 to 7.7%; total serum cholesterol dropped from 4.92 to 4.12 mmol/L
Landin et al., 1992[19]	25 healthy nonobese middle-aged males in a placebo-controlled crossover study	insulin sensitivity, total cholesterol, serum triglycerides; insulin, glucose, and blood pressure	in guar gum leg of study, serum glucose was 4.5 vs 4.8 mmol/L during control phase; insulin was not changed; glucose disposal measured with euglycemic clamp increased 1.2 mg/kg lean body mass/min; total cholesterol was 5.1 vs 5.5 mmol/L in guar gum vs placebo phases systolic and diastolic blood pressure dropped 3 and 3 mmHg, respectively, during guar gum intervention

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Table 3. continued

reference	subjects	markers	results
Yamatoya et al., 1993[52]	6 healthy volunteers	15 g of guar gum and 75 g of glucose in glucose tolerance test; lipid tolerance test	cholesterol reduced 6 mg/dL at 4 h VLDL lowered 35 mg/dL at 6 h and 30 mg/dL at 6 h glucose levels lowered from 60 to 20 mg/dL insulin reduced from 35 to 25 μUmL at 60 min
Golay et al., 1995[54]	non-insulin-dependent diabetics	fed formula with sucrose, fructose, or fructose plus PHGG	glucose area under curve for sucrose reduced from 16 to 9 mol/Lh when sucrose was replaced by fructose and guar added
Blake et al., 1997[30]	11 hypercholesterolemic subjects consumed 4 rolls with and without 4 g PHGG	total and LDL cholesterol were measured at the beginning and end of each 3 week exposure period	LDL cholesterol dropped from 4.28 to 3.81 mmol/L; total cholesterol dropped from 6.52 to 5.89 mmol/L; serum triglycerides were unchanged
Yamatoya et al., 1997[38]	15 healthy young subjects with total serum cholesterol ≥190 mg/dL ingested 5 mg (n = 9) or 15 mg (n = 6) PHGG for 2 weeks	cholesterol, triglycerides, free fatty acids, and glucose were measured before and after 2 weeks of subjects consuming PHGG in a fruit juice mixture	in the group receiving 5 g/day free fatty acids were reduced from 0.82 to 0.48 mequiv/L/In the 15 g/day group; cholesterol was reduced from 5.05 to 4.88 mmol/L; free fatty acids were reduced from 0.82 to 0.53 mequiv/L; glucose was reduced from 88.3 to 84.5 mg/dL
Wolf et al., 2003[34]	30 healthy nondiabetics with BMI of 24.4 ± 0.4mkg/m^2	tested glycemic response to high-glycemic meal with added fructose, added guar gum, or a combination on postprandial glucose response; guar gum and guar gum + fructose treatments contained 3.67 and 3.69 g guar gum/240 g serving	baseline peak glucose response was similar for fructose and control groups; peak glucose response was reduced nearly 50% in the two guar gum-containing groups
Kondo et al., 2004[45]	11 healthy adult males	6 g PHGG in a fat tolerance test	reduced AUC for triglyceride from 297.2 to 251.5 mg h/dL serum RLP cholesterol reduced from 312 to 238 mg/dL
Trinidad et al., 2004[133]	glycemic index of control and test foods in 11 normal and 9 diabetic subjects	white bread supplemented with 3−15 g PHGG; japonica rice was prepared with 3 or 5 g added PHGG; a drink with PHGG was provided in combination with white bread	white bread GI was assumed to be 100 in diabetics and was 93.3 in normal; 3 g of PHGG resulted in GI of 63.4 and 75.1 in normal and diabetic subjects, respectively; 15 g resulted in GI of 56.4 and 55.4 in the same groups; 10 g of PHGG in beverage with white bread reduced GI to 56.9 and 58 in normal and diabetic subjects, respectively, compared to values from 66.3 to 68.5 for inulin and maltodextrin; GI of rice was reduced from 88 to 72 (average of both groups)
Williams et al., 2004[134]	48 subjects received bar with or without guar gum or alginate in a double-blind crossover design	postprandial glucose excursions were measured for 3 h; test bars (2/serving) delivered 5.5 g guar and 1.6 g alginate prior to glucose tolerance test	incremental peak glucose was 1.78 vs 1.24 mmol/L for control vs test bar, respectively; incremental AUC (mmol/L/min was reduced from 134 in control to 90 after consumption of test bar compared to control

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Table 4. Summary of Studies on Influence of Guar Gum and PHGG on Serum Glucose

reference	species	study type	results
			Guar Gum
Aro et al., 1984[15]	hypercholesterolemic males	15 g guar gum/day for 12 weeks	no significant change in glucose
Ellis et al., 1991[62]	healthy human	guar in bread	no postprandial effect on serum glucose up to 120 min
		high, medium, low MW	25−45% reduction in postprandial insulin at 60 and 90 min
Jarjis et al., 1984[63]	normal and diabetic humans	2.5 and 14.5 g guar gum with a 50 glucose load	at 30 min, 2.5 g guar gum caused 40% reduction in serum glucose; 14.5 g reduced response by 60%; both doses resulted in 60% reduction in insulin at 30 and 60 min
Jenkins et al., 1977[64]	hyperlipidemic humans	glucose changes after liquid test meal	liquid test meal with 14.5 g guar gum fed for 30 min reduced glucose level from 3.33 to 4.77 mmol/L; insulin reduced by 50%
			PHGG
Golay et al., 1995[54]	non-insulin-dependent diabetics	fed formula with sucrose, fructose, or fructose plus PHGG	glucose area under curve for sucrose reduced from 16 to 9 mol/L/h when sucrose was replaced by fructose and guar added
Suzuki and Hara, 2004[56]	rats fed fructose	glucose tolerance tests on rats fed fructose, glucose, and fructose plus PHGG	on day 28, for rats on glucose AUC was 10.6 mmol/L/h compared to 9.5 mmol/L/h for rats receiving fructose and PHGG
			insulin in fructose group at 28 days was 0.54 nmol/h/L compared to 0.29−0.36 nmol/h/L for other groups
Yamatoya et al., 1993[52]	healthy humans	15 g of guar gum and 75 g of glucose in glucose tolerance test	glucose levels lowered from 60 to 20 mg/dL
			insulin from 35 to 25 μU/mL at 60 min

Another possible mechanism suggested by LSRO[2] is that, having passed through the small intestine, fibers such as guar gum are readily available for colonic fermentation, with the production of short-chain fatty acids (SCFA), particularly propionate, which has been shown in animal and human studies to lower blood cholesterol through a suppression of hepatic cholesterol synthesis. This hypothesis is supported by the rat study by Evans et al.[41] and the study by Ide et al.,[43] which also evaluated the effect of intact versus PHGG using an enzyme hydrolysate prepared in the laboratory. Four-week-old male Sprague−Dawley rats were fed a basal diet or the basal diet supplemented with either 8% intact guar gum, 8% PHGG, or 8% fructooligosaccharide (FOS) for 3.5 weeks. After sacrifice, the bile ducts were cannulated and bile drained for 2 h. The rats were then bled and the liver and small intestine excised. Guar gum, but not PHGG or FOS, reduced feed intake and growth. Both guar gum and PHGG increased cecum weights to the same degree and increased cecum contents of SCFA. In lard-based diets, total cholesterol was lowered from 114 to 73.6 and 84.6 mg/dL by intact guar gum and PHGG, respectively. In palm oil diets, cholesterol reductions were from 140 to 85.3 and 111.8 mg/dL, respectively. Triglycerides were reduced by >60% by both intact and PHGG in lard-based diets; in palm oil diets, reductions were 37% for intact guar gum and 31% by PHGG.

In a later study, Favier et al.[44] fed diets containing 0.4% cholesterol to male Wistar rats weighing about 150 g for 21 days. The diets were supplemented with either 8% intact guar gum (average molecular weight 2000 kDa, average viscosity 2800−3400 cps at 1% concentration) or 8% PHGG (Fiberon; average molecular weight 15 kDa, average viscosity <10 cps at 10% concentration). After 21 days, the total cholesterol was 2.18 mmol/L for control and 1.37 and 1.97 mmol/L, respectively, for intact and PHGG, respectively. The intact guar gum effectively lowered blood cholesterol concentrations, chiefly in the triacylglyceride-rich lipoprotein fraction. The researchers concluded that, even if PHGG alters some characteristics of the enterohepatic cycle of cholesterol and bile acids, its effects are not sufficient to elicit a significant cholesterol-lowering effect.

Limited clinical evidence suggests that PHGG may reduce serum triglycerides. Kondo et al.[45] conducted a randomized, single-blind, placebo-controlled crossover design in 11 healthy adult males consuming yogurt with or without 6 g/day of PHGG for 1 week, with no washout period between administrations of the test and control yogurts. In the fat tolerance test, when PHGG was added to the yogurt, the area under incremental curve (AUIC) was reduced from 11.1 to 8.5 mg/h/dL for serum triglycerides and from 297.2 to 251.5 mg/h/dL for cholesterol. There were no complaints of diarrhea or gastrointestinal discomfort and no change in body weight.[45] The authors suggested that PHGG may have affected the rate of fat absorption.

Reduction of serum cholesterol and other serum lipids has been reported with guar ingestion in numerous short-term clinical studies in humans (about 2 weeks−3 months), but reductions were not consistently sustained over the long term (up to 12 months).[15−19,22−29,39,46−51] For example, Yamatoya et al.[52] fed 75 g of glucose dissolved in 200 mL of water to six healthy volunteers, either with or without 15 g of PHGG (source unspecified) dissolved in 150 mL of water. Blood total cholesterol, triacylglycerides, LDL, very low density lipoprotein (VLDL), and phospholipid concentrations were measured after 1, 2, 4, 6, and 8 h. The addition of PHGG reduced the concentrations of all of these lipids at nearly all time points; the differences were statistically significant only for total cholesterol at 4 h (−6 mg/dL), VLDL at 6 (−34 mg/dL) and 8 (−29 mg/dL) h, and phospholipids at 4 h.[52] In a follow-up study, Yamatoya et al.[38] prepared PHGG with a peak molecular weight of ∼20 kDa by means of enzymatic hydrolysis. Healthy young females with serum cholesterol concentrations of 190 mg/dL or higher ingested either 5 or 15 g/day of PHGG for

2 weeks. In the 5 g/day group, serum cholesterol was slightly reduced (−0.18 mmol/L) and free fatty acids decreased (−0.34 mequiv/L); in the 15 g/day group, both cholesterol (−0.17 mmol/L) and free fatty acids (−0.29 mequiv/L) were significantly reduced. However, no changes were noted in triacylglycerides or phospholipids.[38]

The cholesterol-lowering effect of PHGG has also been confirmed when used in a food matrix. In a randomized, double-blind crossover study of 20 individuals with moderately elevated plasma cholesterol,[30] participants received either control wheat bread or wheat bread supplemented with a PHGG having an average molecular weight of 1070 kDa, approximately midway between the average size of intact guar and PHGG in GRAS approval. Study participants received each diet for 3 weeks with a 4-week washout; while on the test diet, they consumed about 13 g/day of guar gum. There were no changes in body weight or dietary intake on either diet, but the guar gum-supplemented diet resulted in a significant reduction in plasma total cholesterol (−0.63 mmol/L), due primarily to a reduction in LDL (−0.47 mmol/L). There were no significant differences in HDL or triacylglycerides. Although there were some reports of mild flatulence with the guar gum-supplemented diet, there were no palatability issues and no serious side effects. Increased viscosity and binding of flavors by guar gum, however, may reduce the release of highly volatile flavor compounds in the food matrix to 40−60%; thus, this must be considered in the formulation of foods containing guar gum or PHGG.[53]

■ POSTPRANDIAL GLYCEMIC RESPONSE

A limited body of evidence, based on studies in animals, in small studies with healthy humans, and in NIDDM patients, suggests that administration of PHGG may be effective in attenuating the postprandial rise in both plasma insulin and plasma glucose; however, the data are insufficient to support a strong claim (Table 4).

Golay[54] administered 20 g of PHGG in 500 mL of a fructose-containing enteral formula and observed significant reductions in blood glucose and insulin concentrations compared to fiber-free formulas containing fructose or sucrose in six patients with insulin-dependent diabetes. In a glucose tolerance test, the area under the curve was reduced from 16 to 9 mmol/h/L in the fructose plus PHGG group. Alam[55] performed an oral glucose tolerance test with administration of 75 g of glucose in 10 healthy men consuming enteral formulas with or without 21 g/day PHGG in which plasma concentrations of glucose and insulin were measured. No significant differences were found in plasma concentrations of these compounds.

Suzuki and Hara[56] fed 5-week-old male Sprague−Dawley rats high-fructose diets and dextrin diets with or without 7.5% PHGG (source unspecified) for 30 days. Glucose tolerance tests were given on days 0, 14, and 28. Initially the glucose area under curve (0−60 min) was the same for all groups, ranging from 9.13 to 9.32. On day 28, the glucose tolerance test area under the curve for the fructose without PHGG was 10.6, which was significantly higher than those for all other groups, which ranged from 9.46 to 9.66. They concluded that the AUC 60 insulin level for the fructose without PHGG was 0.54 mmol/h/L, which was significantly higher than those for the other groups, which ranged from 0.29 to 0.37 mmol/h/L. The animals receiving fructose without PHGG also had higher triglycerides (3.3 vs 1.8 to 2.4 mmol/L for the other groups). Similar trends existed throughout the study. The free fatty acids exhibited a similar pattern. They concluded that PHGG improved glucose tolerance and reduced hyperinsulinemia on day 28 and improved hyperlipidemia caused by the high-fructose diet.

In a double-blind crossover study, 12 patients with NIDDM were fed enterally on each of three formulas, two containing PHGG and one control.[31] After an overnight fast, patients were brought to steady state with a blood glucose level of 8.4 mmol/L; at time 0 and every 15 min thereafter for 4 h, the patients ingested 30 mL of formula for a total of 480 mL. Blood glucose was measured every 30 min. The two formulas containing PHGG (concentration not specified) were not effective in attenuating the postprandial glucose excursion.

Yamatoya et al.[52] gave 75 g of glucose dissolved in 200 mL of water to five healthy volunteers, either with or without 15 g of PHGG (source unspecified) dissolved in 150 mL of water. Blood glucose and insulin concentrations were measured after 30, 60, 90, 120, and 180 min. The addition of PHGG did not affect the glucose peak time, but reduced the concentrations of blood glucose and insulin at all time points; the differences were statistically significant only at 60 min for glucose (glucose increase reduced from 58 to 30 mg/dL and at 90 min the insulin increase was reduced from 25 to 16 μU/mL).

Similar results were observed in a study by Wood et al.[57] in which guar gum containing 82% galactomannan significantly reduced postprandial blood glucose similarly to oat gum as indicated by the glycemic index (48 and 59% reductions, respectively). In addition, a correlation was observed between the reduction in postprandial glucose and the kinematic viscosity of the gums at 1%; viscosity is not expected to be altered by stomach pH given the neutral and linear nature of the galactomannan polysaccharides. In a later study, Ou et al.[58] identified three mechanisms by which dietary fibers including guar gum may reduce postprandial glucose response, including a viscosity increase of the small intestine juice that reduces glucose diffusion, direct glucose binding that reduces the glucose availability in the small intestine, and encapsulation of starch and α-amylase, which retards the action of the enzyme and might even inhibit the enzyme itself. Reduction of glucose release was confirmed by Tudorică et al.[59] when guar gum was used in a pasta formulation at 3−10%. The study showed that enrichment of pasta with guar gum at low levels (3%) preserved the cooking qualities while forming a network that encapsulated the starch granules and prevented excessive swelling and diffusion, resulting in a significant decrease in glucose release after an in vitro digestibility test.

The Institute of Medicine Panel on Macronutrients concluded that most studies indicate that guar gum had a beneficial impact on postprandial blood glucose.[60] LSRO[2] also reported that postprandial blood glucose and insulin concentrations were reduced in human subjects following ingestion of guar gum, citing the following published papers: Aro et al., 1981; Ellis et al., 1991; Jarjis et al., 1984; Jenkins et al., 1977; Morgan et al., 1985; and Peterson et al., 1987.[21,61−65] Additional research cited by LSRO at other points that bears on the effect of guar gum on blood glucose concentrations includes Jenkins et al. (1980), Smith and Holm (1982), Blackburn et al. (1984), Ebeling et al. (1988), Uusitupa et al. (1989), Landin et al. (1992), and Vuorinen-Markella et al. (1992).[17,19,23,28,29,66,67]

PREBIOTIC EFFECTS

Maintaining populations of bifidobacteria and lactobacilli is increasingly regarded as beneficial.[68] One means of encouraging the proliferation of these lactic acid bacteria is by ingesting fermentable carbohydrates that provide a substrate upon which these bacteria can grow. Several in vitro studies have explored the ability of PHGG to encourage proliferation of lactic acid bacteria, either by direct measurement of microbial prevalence or by evaluation of bacterial production of SCFA. Thirteen bacterial species were screened for growth on several oligosaccharide preparations and PHGG (Benefiber) by Miller-Fosmore et al.[69] Little growth was shown of any species exposed to PHGG, although bifidobacteria grew on the oligosaccharides.

The proliferation of lactic acid bacteria results in a reduction in the prevalence of putrefactive bacteria, possibly due to competitive exclusion.[68] Ishihara et al.[70] supplemented the feed of 9-week-old White Leghorn pullets or 72-week-old ISA Brown laying hens with 0.025, 0.05, or 0.1% PHGG. The treatments (most effective was the lowest concentration, 0.025%) increased *Salmonella enteriditis* secretion in the feces in both groups of hens. Laying hens also showed lower *S. enteriditis* counts on eggshells (34.5% incidence reduced to 12.5%) and in egg whites (1.7% incidence to 0) and yolks (6.9% to 0). Pullets showed lower *S. enteriditis* in organs (63.3 vs 16.7%).

Fermentation of food fibers with fecal inocula has demonstrated that dietary fiber fractions from various foods are effective in the differential production of SCFA including acetate, butyrate, and propionate.[71] Velazquez et al.[72] and Pylkas et al.[73] used fresh fecal inocula to compare the effects of glucose, soy oligosaccharide, fructooligosaccharide, inulin, hydrolyzed inulin, cellulose, powdered cellulose, methylcellulose, psyllium husk, indigestible dextrin, arabinogalactan, Polydextrose, and PHGG on the production of SCFA. Inoculation with each of these carbohydrates resulted in the production of SCFA, with PHGG producing high concentrations of propionate (19.8 mg/mL) and butyrate (15.5 mg/mL) after 24 h.[72] Psyllium was slightly higher in butyrate at 24 h (16.8 mg/mL) but, at 12 h, the propionate and butyrate concentrations were 2−5 and 6−8 mg/mL higher than for other fibers, respectively.[72] In the study by Pylkas et al. PHGG resulted in the highest production of SCFA at all time points (2, 4, 8, 12, and 24 h).[73] Additionally, the SCFA produced in the presence of PHGG were high in acetate (0.17−0.21 mmol/g organic matter) and butyrate (0.4−0.5 mmol/g organic matter) but low in propionate (0.0075 mmol/g organic matter).[73] The acetate concentrations were 50−100% greater and the butyrate concentrations were 4−5 times higher than the levels of other fibers tested.[73]

In a study of the effects of PHGG on the colonic microbiota of rats, Takahashi et al.[74] assigned male Wistar rats to one of five groups, standard chow, liquid low-residue diet, liquid elemental diet, low-residue diet with 1.5% PHGG, or an elemental diet with 1.5% PHGG, for 2 weeks. The liquid diets led to atrophy of the terminal ileum villi, but PHGG lessened the effect. Rats on the liquid diets showed reduced activity of diamine oxidase (0.59 U/mg protein in low-residue diet, 0.97 U/mg protein with low-residue diet + PHGG, 0.39 U/mg protein for elemental diet, and 0.96 for elemental diet + PHGG) and alkaline phosphatase (4.82 U/mg protein for low-residue diet and 4.95 U/mg protein for low-residue diet + PHGG, 4.79 U/mg protein for elemental diet, and 4.8 U/mg protein for elemental diet + PHGG) in serum.

Effects of PHGG on the population and diversity of gut microbiota have also been observed. Okubo et al.[75] gave 21 g/day (three 7-g doses) of PHGG to nine healthy 22−39-year-old males for 14 days as a 7% (w/v) solution in water, and feces were collected on days 12 and 14. A significant increase was seen in the cell counts and percentage of bifidobacteria (10.0−10.32 $\log_{10}$ bacteria/g wet feces) and in the frequency of occurrence of lactobacilli (4.41−4.95 $\log_{10}$ bacteria/g wet feces). As a result, fecal pH was decreased (from 6.16 to 5.77), probably due to the production of SCFA. The SCFA were not increased in the feces, however, possibly due to absorption in the colon.[75] These results may not be exclusive of PHGG, as observed in a double-blind randomized placebo-controlled crossover study that investigated the prebiotic effects of PHGG and fructooligosaccharide.[76] In the study, 31 healthy males and females consumed either placebo biscuits or biscuits with 6.6 g/day of fructooligosaccharide and 3.4 g/day PHGG for 21 days, and then they were crossed over (no washout). No significant differences were found in the number of total bacteria, *Bacteroides* spp., *Clostridium* spp., or *Lactobacillus* spp. There was a significant increase in *Bifidobacterium* spp. (from 9.10 to 9.59 $\log_{10}$ bacteria/g wet feces), but it did not persist after treatment. There were no effects on fecal pH, mean daily stool frequency, abdominal pain, intestinal bloating, or flatulence.[76] Similarly, Beards et al.[77] compared fatty acid production and distribution of bacterial species with a range of food ingredients fermented with human colonic microbiota. Samples compared in this study were oligofructose, short-chain inulin, and Taiyo galactomannan. The results demonstrated clear similarity in the production of SCFA and in the distribution of bacteria after fermentation.[77]

Partial hydrolysis of guar gum results in a product that can be used as a soluble fiber. It appears that PHGG may have a prebiotic effect, reflected in increased proliferation of lactic acid bacteria and production of SCFA, but this effect is not as clearly elucidated as are effects of other well-studied prebiotic carbohydrates such as fructooligosaccharides and galactooligosaccharides. The physiological effects of the PHGG are similar to those of other soluble dietary fibers (fructooligosaccharides and Fibersol-2). Salyers et al.[78] demonstrated that guar gum was fermented by human fecal microbiota and that the effects were bifidogenic. Flickinger et al.[79] conducted a comparative study of fermentable fibers, including FOS, PHGG, and Fibersol-2, in a model system using human fecal microflora. In the study, they monitored the change in pH and accumulation of SCFA. Figure 2 illustrates that there were few differences in change in pH over time among the three soluble fibers. Guar gum was added as a comparison showing similarity to PHGG. Figure 3, using data from the same paper, illustrates SCFA accumulation over time. There appear to be small differences with Fibersol-2, showing a slightly different pattern of fatty acid accumulation, suggesting that the fermentation of PHGG was more like that of FOS than that of Fibersol-2, although all three soluble fibers illustrated clear prebiotic activity. The total fatty acid accumulation is further demonstrated in Figure 3, which illustrates the total SCFA accumulation over time. The overall conclusion that one can draw from the data presented is that although the fermentations differ slightly, all three soluble fibers are similar in the conversion of fiber to SCFA. As a result, the energy

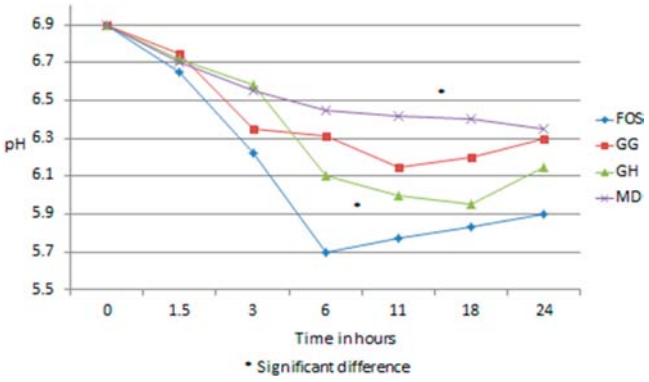

Figure 2. Fecal pH values of fermentable fibers at various times. Adapted from ref 79.

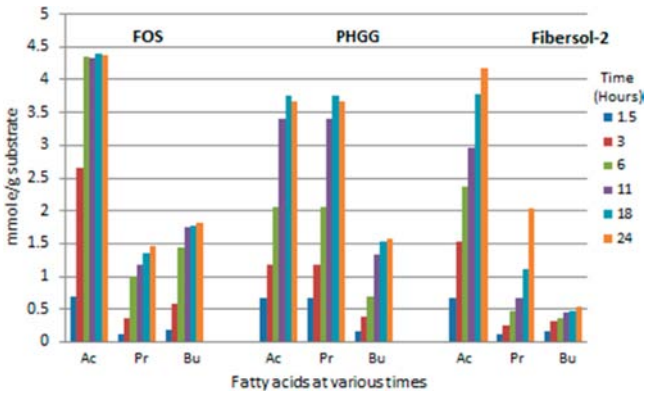

Figure 3. In vitro short-chain fatty acid accumulation over time. Adapted from ref 79.

recovery from the three sources should be considered to be similar.

Acacia gum, another food gum, is an arabinogalactan. Like guar gum, acacia gum is completely nondigestible in routine tests such as described by Southgate et al.[80] Phillips[81] established that the gum was essentially completely fermentable and assigned it a maximum caloric value of 2.0 kcal/g.

Soluble fermentable fibers also share the common characteristic of enhancing colonic absorption of calcium, magnesium, and iron. Both long- and short-chain inulin enhanced calcium uptake and bone mineralization.[82−86] Miyazato[87] reported that resistant polydextrin improved absorption of calcium (25%), magnesium (27%), iron (100%), and zinc (50%). The PHGG improved the absorption of minerals such as calcium[88,89] and iron.[90,91] Hara et al.[89] reported a doubling of the calcium pool in gastrectomized rats fed PHGG compared to control animals. The effect was strongly correlated with higher concentrations of propionic acid in the fecal material. Takahashi et al.[90] demonstrated that iron absorption improved from 29.1% in iron-deficient rats to 32.3 and 36.7% in 25 and 50% iron-deficient rats fed PHGG. DeCassia Freitas et al.[91] observed a 37% improvement in iron absorption in anemic rats fed PHGG. This further confirms the similarity among the three soluble fiber sources.

■ NONDIGESTIBILITY OF GUAR GUM

Both intact guar gum and PHGG consist of repeating units of galactomannan as is shown in Figure 1. Humans and animals do not produce an α-galactosidase capable of removing the galactose side chains of guar gum galactomannans, nor do they have α-mannase capable of splitting the mannose polymer. Several studies have demonstrated limited or no α-mannosidase activity in mammals, and no reports of α-mannosidase activity exist in humans.[92,93] In earlier studies, the nondigestibility of guar gum was considered to be a negative, particularly in poultry diets. This was primarily associated with high water-holding capacity.[94] Rainbird et al.[95] investigated the effects of guar in cannulated pigs as a model to assess the effect on glucose uptake. The results demonstrated that the galactomannans survived the digestive tract and inhibited glucose and water uptake. Ray et al.[96] isolated mannase from commercial hemicellulose preparations and were able to overcome the "negative" effects of guar, offering evidence of the lack of mannase activity in animals.

The digestion, absorption, and metabolism of PHGG are analogous to those of intact guar gum in that neither are digested in the small intestine (although a small amount of fermentation may occur in the distal small intestine). The two products differ substantially in molecular weight (200+ kDa for intact guar gum; about 20 kDa for PHGG) and in viscosity (average of about 2700 cps for a 1% solution of intact guar gum; <10 cps for a 10% solution of PHGG). Effects influenced by these properties during transit prior to fermentation may differ between intact guar gum and PHGG. However, the initial cleavage of the mannose backbone accomplished by the endo-β-D-mannase used in the production of PHGG duplicates the action of microbial enzymes in the human gut; therefore, it would be expected that the physiological effects in the colon of intact (at the point of ingestion) and PHGG are similar. Both the Sandoz[1] panel and the LSRO[2] panel found that the digestion, absorption, and metabolism of PHGG are analogous to those of intact guar gum. The LSRO[2] reviewed two studies indicating that guar gum is not digested in the small intestine but is almost completely fermented in the colon of the rat.[97,98] In one study cited by LSRO,[2] ingestion of intact guar gum was shown also to increase the pool of SCFA in the cecum of rats.[99] In another cited study, however, feeding 10% guar gum diets increased cecal concentrations of butyrate, but not of acetate or propionate, had no effect on fecal SCFA concentrations, and did not influence the pH of either the cecal contents or feces.[100]

Tomlin et al.[101] demonstrated that incubation of a human fecal homogenate with guar gum resulted in the production of hydrogen and decreased the viscosity and pH. After incubation for 21 h, the pH of the control feces went from 8.13 to 9.91 without added substrate and to 6.18 with added guar gum, demonstrating acid production with the addition of guar gum. At the end of fermentation, the viscosity of the untreated feces was 3.0 mPas compared to 2.2 mPas when guar gum was added during the fermentation. Bayliss and Houston[102] measured fecal output in one individual for 21 days and then added 13.9 g/day guar gum to the diet for 21 days. The cumulative wet weight fecal output was doubled when guar gum was added to the diet. They concluded that when guar gum was added to the diet, 80% of the guar gum was converted to biomass in the colon.

In vitro studies with human intestinal microbiota have shown that intact guar gum is fermented by microorganisms indigenous to the colon. The α-1,4-mannosidic linkages in the mannose backbone of guar gum are hydrolyzed by mannases present in the intestinal microbiota, in particular

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those produced by *Bacillus subtilis* (Emi et al.[103] cited in ref 2) and *Bacteroides ovatus*.[104] The resulting shorter chains of galactomannans are hydrolyzed to galactose and mannose units by bacterial α-galactosidases and mannases,[105−107] and these monosaccharides are assimilated by the gut microbiota and metabolized.[108] The SCFA resulting from this fermentation may be metabolized to CO_2 or used to synthesize other compounds within the cells of the intestinal mucosa, liver, or peripheral tissues; utilized as an energy source by colonic bacteria; or excreted in the feces.[109] It has been shown in vitro that rat and human colonocytes utilize SCFA for energy, suppressing glucose metabolism.[110,111] The LSRO[2] panel concluded that, whereas failure to find increased concentrations of fermentation products such as SCFA in intestinal contents following ingestion of guar gum (see, e.g., ref 112) has been interpreted as evidence against fermentation of guar gum, the absence of SCFA in feces may be accounted for by the ability of the gut to absorb SCFA readily.[113]

With the exception of the initial cleavage of the mannose backbone, which is accomplished by the endo-β-D-mannase used in the production of PHGG, the digestion, absorption, and metabolism of PHGG follow the same steps as outlined above for intact guar gum.[1,2] The LSRO[2] panel considered that products of the endo-β-D-mannase hydrolysis are of sufficiently high molecular weight that they are not digested or absorbed.

ENERGY VALUE OF NONDIGESTED CARBOHYDRATES

Carbohydrates traditionally were divided into available and unavailable on the basis of conceptual and analytical definitions.[114] Unavailable carbohydrates were defined as celluloses and hemicelluloses, whereas available carbohydrates were sugars and starch. Improved understanding of carbohydrate metabolism has resulted in the need for more rigorous definitions. For example, lactose is not absorbed by some populations,[115] and some starches are not completely digested and are considered to be resistant.[116,117] A major consideration is the oligosaccharides, which are nondigestible. Oligosaccharides include raffinose and stachyose in beans and fructooligosaccharides from onions, leeks, and artichokes.[118−120] Partially hydrolyzed guar gum and Fibersol-2 (FS-2) (a digestion-resistant maltodextrin) have DPs of >10 but share many physical attributes such as solubility and biological attributes such as nondigestibility with oligosaccharides.

Natural oligofructans and FS-2 have been established as reduced calorie carbohydrates. FS-2 is produced by a combination of heat and enzyme treatments of cornstarch to produce a low-viscosity, low-digestible dextrin with an average molecular weight of 2000 Da. FS-2 contains glucopyranosyl units with 1,6-anhydro-β-D-glucose (levoglucosan) at some of the reducing terminals. Linkages in the molecule are randomly distributed among units consisting of α- and β-(1→4), -(1→6), -(1→2), and -(1→3) glycosidic bonds. In vitro digestion of FS-2 with successive treatments of salivary α-amylase, a gastric juice preparation, pancreatic α-amylase, and intestinal mucosal enzymes gave 89.8% recovery of the starting material. Thus, it was concluded that the FS-2 was not digested but is fermented in the colon.[121] Rats gavaged with FS-2 were found to have only a 5% increase in plasma glucose concentrations over 120 min compared to rats administered an equal amount of glucose. Approximately 38% of FS-2 administered to rats by gavage is recovered in feces, which may indicate that it is not completely fermented. Growth rates of rats fed FS-2 indicate <10% of the dextrin is contributing net metabolizable energy. FS-2 has an energy value of 2.2 kcal/g.[122]

Acacia gum, another food gum, is an arabinogalactan. Like guar gum, acacia gum is completely nondigestible in routine tests such as described by Southgate et al.[80] Phillips[81] established that the gum was essentially completely fermentable and assigned it a maximum caloric value of 2.0 kcal/g.

CALORIC VALUE OF PHGG

Nutrition labeling regulations, at 21 CFR §101.9(c)(1)(i), provide that the caloric content of food may be calculated by any of the following methods: (A) using specific Atwater factors; (B) using the general factors of 4, 4, and 9 calories per gram for protein, total carbohydrate, and total fat, respectively; (C) using the general factors of 4, 4, and 9 calories per gram for protein, total carbohydrate less the amount of insoluble dietary fiber and total fat, respectively; (D) using data for specific food factors for particular foods or ingredients approved by the FDA; or (E) using bomb calorimetry data and subtracting 1.25 calories per gram of protein to correct for incomplete digestibility.

The method proposed for determining and labeling the caloric content of PHGG is method D, above (use of data indicating the appropriate caloric factor to apply to this substance).

The regulations for the caloric calculation method (C), above, regard the caloric content of insoluble dietary fiber as zero but, unfortunately, regard soluble dietary fiber as contributing the same amount of energy as other carbohydrates.

At the time the regulation was formulated, the situation regarding labeling and defining dietary fiber in the United States and many other countries was arbitrary due to its reliance on analytical methods as opposed to an accurate definition that includes its role in health.[123] For this reason, the Institute of Medicine's (IOM) Food and Nutrition Board assembled a Panel on the Definition of Dietary Fiber. The panel recommended defining "dietary fiber" as "non-digestible carbohydrates and lignin that are intrinsic and intact in plants" (p 2).[123] The panel then suggested defining "functional fiber" as "isolated, non-digestible carbohydrates that have beneficial physiological effects in humans" (p 2).[123] Finally, the panel defined "total fiber" as the sum of dietary fiber and functional fiber.[123]

With regard to the traditional segregation of dietary fibers into soluble and insoluble varieties, the panel recommended gradually replacing the terms "soluble" and "insoluble" fiber with their corresponding characterized physicochemical property.[123]

It is noteworthy that the IOM panel emphasized consideration of physicochemical properties and strongly urged the inclusion of a requirement that a substance must provide a beneficial physiological effect in humans to be regarded as a functional fiber. This condition would appear to exclude isolated nondigestible carbohydrates with no demonstrated benefits from being defined as fibers. The IOM's Panel on Macronutrients used the term "functional fiber" in place of the earlier panel's term "added fiber"[124] in discussing recommended and safe levels of intake of total fiber to avoid confusion with the term "added" fiber that already was in use.

Table 5. Fraction of Each Saccharide and Total

saccharide	% dry wt	adjusted to 94.2% dry wt	fraction (g/g PHGG)	total fraction (g/g PHGG)	assumption
mono	5.2	4.9	0.0488	0.083	completely absorbed and metabolized in the small intestine
di	3.6	3.4	0.0342		
tri	2.4	2.2	0.0224	0.859	not absorbed and completely fermented in the large intestine
tetra	2.1	2.0	0.0196		
penta	1.5	1.5	0.0145		
hexa	1.8	1.7	0.0170		
hepta	1.5	1.4	0.0143		
octa	1.8	1.6	0.0165		
nona	1.5	1.4	0.0141		
deca	1.7	1.6	0.0161		
>deca	76.9	72.5	0.7246		

Energy from Other Fractions of PHGG

fraction	fraction (g/g PHGG)	energy (kcal/g PHGG)	energy (kJ/g PHGG)
protein	0.006	0.024	0.10
ash	0.011	0	0
moisture	0.041	0	0
sum	0.052	0.024	0.10

Whereas the 2001 panel did not attempt to restrict the range of physiological benefits that might be demonstrated as being provided by putative fiber substances, it specifically described three "established physiological effects" that are frequently characteristic of added fibers.[123] These are attenuation of the postprandial blood glucose response, attenuation of blood cholesterol concentrations, and improved laxation.

These effects are the same as those identified in 1988 by Health Canada.[125] The panel did not suggest that a substance needs to provide all three of these benefits to be regarded as added functional fiber,[123] nor did it exclude defining a substance as a functional fiber based on an ability to confer benefits other than these; the intention was to require that a substance could not be defined as a functional fiber unless it was shown to provide a benefit.

The Panel on Macronutrients suggested that guar and other gums might be regarded as either dietary fibers or functional fibers.[124] It is clear, however, that the partially hydrolyzed material, if it is to be considered a fiber at all, must be regarded as a functional fiber rather than a dietary fiber, because part of the definition of dietary fiber is "intact." Following the IOM panel's recommendation, in supporting the intended use of PHGG as a functional (added) fiber, it is appropriate to consider the extent to which it has been shown to provide physiological benefits.

Roberfroid[126] calculated the caloric values of inulin and oligofructans using the equation

$$\text{kcal (kJ)} = (A - B) \times (1 - C) \times (1 - D) \times E$$

where A is the kcal (kJ) entering the colon as fermentable substrate, B is the kcal (kJ) excreted in the feces, C is the proportion of C atoms and energy due to fermentation, D is the loss of C atoms and energy due to fermentation, and E is the efficiency of utilization of SFCA in the host compared to glucose.

According to the report of the FASEB/LSRO[127] and on the basis of what was known for inulin and oligofructose, it was assumed that A = 3.9 kcal (16.3 kJ), B = 0, C = 0.15−0.21, D = 0.25−0.30, and E = ∼0.70. On the basis of these assumptions and applying Roberfroid's[126] equation based on the similarities between PHGG and oligofructans, the model for PHGG would be as follows: A = 3.9; B = 0; C = 0.15−0.21; D = not determined for PHGG so we use the same value as oligofructans; and E = not determined for PHGGn so we use the same value as oligofructans:

$$\begin{aligned}\text{kcal (kJ)} &= 3.9\ (16.3\ \text{kJ}) \times [1 - (0.15 \text{ or } 0.21)] \\ &\quad \times [1 - 0.25 \text{ or } 0.30] \times 0.70 \\ &= 1.5{-}1.7\ \text{kcal/g or } 6.3{-}7.3\text{kJ/g}\end{aligned}$$

Using this approach, they accounted for biochemical and fermentation effects and metabolic balance.

Available energy was estimated assuming that mono- and disaccharides present in PHGG are completely available, delivering 4 kcal/g. It was further assumed that the remainder of the saccharide content in PHGG is completely fermented. The typical saccharide composition of PHGG is illustrated in Table 5. On the basis of these assumptions, after adjustment for moisture, the saccharide portion delivers 1.83 kcal/g and the protein provides an additional 0.024 kcal/g. Thus, the estimated caloric value using these assumptions would be 1.9 kcal/g.

Livesey[128] applied a slightly different calculation and, using his model, we get 2.1 kcal/g. Cummings et al.[129] used 1.5 kcal/g for completely fermentable fiber. Applying this assumption, the saccharide fraction of PHGG would be 1.62 kcal/g. Thus, in total, PHGG would deliver 1.65 kcal/g.

According to the LSRO,[127] energy estimates for the saccharide portion of PHGG are calculated as follows:

$$\text{ME} = \text{IE} \times [1 - ([A \times (1 - B)] + [1 - A] \times [1 - C])]$$

IE is ingested energy of a substance as measured by bomb calorimetry (4.0 kcal/g or 16.75 kJ/g), A is the fraction of ingested substance absorbed by the small intestine, B is the fraction of A metabolized after absorption in the small intestine, and C is the fraction of energy from substance fermented in small intestine.

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Therefore, energy from absorbed portion = 4.0 kcal/g or 16.75 kJ/g, the proportion of energy from fermented portion = 0.5 kcal/g or 2.095 kJ/g, A = 0.083, B = 0.083, and C = 0.429 (0.5 × 0.859).

$$\text{ME in kcal/g} = 4 \times [1-[[0.083 \times (1 - 0.083)] + [(1 - 0.083) \times (1 - 0.429)]]] = 1.6 \text{ kcal/g}$$

$$\text{ME in kJ/g} = 16.75 \times [1-[[0.083 \times (1 - 0.083)] + [(1 - 0.083) \times (1 - 0.429)]]] = 6.70 \text{ kJ/g}$$

fraction	kcal/g	kJ/g
saccharide	1.9	6.70
nonsaccharide	0.024	0.10
total	**1.624**	**6.80**

It is important to consider lot-to-lot variation. Table 6 illustrates the lot-to-lot variation and the calculations of energy availability based on the analysis of eight lots of PHGG.

Table 6. PHGG Typical Analytical Data on Main Nutrients and Ash

lot	water loss on drying (%)	protein (%)	ash (%)	fat (%)	total dietary fiber by AOAC 2009.01 (%)	mono- and disaccharides (%)
A	4.80	0.54	0.98	0	93.13	0.6
B	4.76	0.55	0.98	0	86.18	7.5
C	4.62	0.54	0.97	0	88.74	5.1
D	4.71	0.53	0.97	0	86.62	7.2
E	4.66	0.55	0.97	0	87.21	6.6
F	4.75	0.54	0.98	0	90.23	3.5
G	4.72	0.53	0.98	0	91.24	2.5
H	4.73	0.54	0.98	0	92.03	1.7
mean	**4.72**	**0.54**	**0.98**	**0.0**	**89.42**	**4.35**
SD	**0.06**	**0.01**	**0.01**	**0.0**	**2.62**	**2.65**

PHGG is a water-soluble partially hydrolyzed galactomannan chemically similar to intact guar gum, but reduced to shorter chain lengths. The PHGG is soluble and appears not to offer any health risk. There is some evidence of improved blood lipid profiles with both intact guar gum and PHGG. Neither form is digestible but readily fermented by gut microbiota. On the basis of the data reviewed, we conclude that the energy of PHGG is the same as that of guar gum, which is approximately 1.9 kcal/g (7.9 kJ/g). Cummings et al.[129] reviewed dietary carbohydrate chemistry, physiology, and health and concluded that for labeling purposes, all carbohydrates that are more or less completely fermented in the human colon should be given a caloric value of 1.5 kcal/g (6.3 kJ/g). If this system were adopted, it would reduce the proposed caloric value of PHGG to 1.5 kcal/g (6.4 kJ/g) (Table 7).

It, therefore, is rational to conclude that the caloric value of PHGG is between 1.6 and 1.9 kcal/g (6.7 and 7.9 kJ/g).

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Notes

The authors declare no competing financial interest.

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Table 7. Caloric Contribution of Various Components to Total Value Based on Mean Values of Eight Lots of PHGG

	water	protein	ash	fat	fiber	mono- and disaccharides	total kcal/g	total kJ/g
LSRO 2.0 kcal/g for fiber	0	0.02	0	0	1.79	0.17	1.98	8.31
Cummings 1.5 kcal/g for fiber	0	0.02	0	0	1.34	0.17	1.54	6.43

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