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Acacia Gum is a Bifidogenic Dietary Fibre with High Digestive Tolerance in Healthy Humans

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The objective of the present study was to determine whether acacia gum (GUM) is a prebiotic fibre and to evaluate its intestinal tolerance in healthy subjects. The effects of increasing doses of GUM were compared to those of sucrose (SUC) on stool output, concentration of the main bacterial populations in stools, and occurrence and severity of intestinal symptoms (flatulence, bloating, abdominal cramps, diarrhoea). Ingestion of GUM 10 and 15 g/day for 10 days increased total lactic acid-producing bacteria and bifidobacteria counts in stools, without affecting total anaerobe and aerobe counts. The magnitude of this selective effect was greater in subjects with a low initial faecal concentration of bifidobacteria. Faecal digestibility of GUM was around 95% and its caloric value was estimated to range between 5.5 and 7.7 kJ/g. In addition, stool weight increased 30% because of greater faecal water content. Digestive tolerance of GUM was high and not statistically different from that of SUC up to a daily dose of 30 g. Above this dose, the main complaint was excessive flatulence. However, the mean degree of severity remained mild (< 1), even at doses > 50 g/day. Other intestinal events were rarely reported. Thus GUM is a very well tolerated dietary fibre with bifidogenic properties believed to benefit intestinal health. *Key words:* acacia gum, prebiotic, soluble fibre, stool output, digestive tolerance.

INTRODUCTION

Nutritionists agree that increasing dietary fibre intake up to 25–30 g/day is part of a healthy diet. Some dietary fibres influence digestion processes and thus metabolic responses, while others improve transit and stool output (1). In addition, some of them appear to be essential for mucosal barrier integrity and intestinal ecosystem balance by selectively stimulating the growth and/or activity of bacteria, such as bifidobacteria or lactobacilli, which is considered to benefit the host (2). However, some fibres are rapidly fermented into short-chain fatty acids and associated with a pronounced increase in gas production, a condition that may induce undesirable symptoms: bloating, flatus, abdominal cramps and diarrhoea. Thus, although dietary fibres have health-promoting effects, these adverse events may limit their consumption.

Acacia gum (GUM) or gum arabic is a soluble dietary fibre obtained from the stems and branches of *Acacia senegal* and *A. seyal*. It is composed mainly of complex polysaccharides (95%) that consist of highly branched galactan polymers, with galactose and/or arabinose side chains, possibly terminated by rhamnose or glucuronic acid residues (3). While $> 80\%$ of current production is used by the food industry for various applications (emulsification,

encapsulation, coating, gum candies, etc.), GUM is traditionally consumed by African and Indian populations to improve digestive comfort and intestinal transit. In vitro, its fermentation is slow (4) and supports the growth of bifidobacteria (5). Thus, GUM may be a bifidogenic soluble dietary fibre that would not induce uncomfortable intestinal side effects in the healthy consumer. The purpose of the present study was to investigate the effects of GUM on bowel performance and the bacterial composition of stools and to evaluate the occurrence and severity of digestive symptoms associated with increasing intake of GUM in healthy subjects.

SUBJECTS AND METHODS

Substrates

GUM (Fibregum[®]; Colloides Naturels International, Rouen, France) consists of branched arabino-galactans polymers (92.8% of the dry matter) containing the following sugars (molar percentage in parentheses): galactose (38), arabinose (46), rhamnose (4), glucuronic acid (6.5), methylglucuronic acid (5.5). Its molecular weight is 882 kDa and its polydispersity is 1.74. The effects of GUM were compared with that of sucrose (SUC, placebo), and short-chain fructo-oligosaccharides (FOS; Actilight[®], Eridania-

Beghin-Say, Paris, France), a rapidly fermented soluble fibre. The three substrates were provided by CNI as odourless, white powders packed in 5-g sachets.

Subjects

Twenty subjects (9 men and 11 women, 22–38 years of age) were included in the study after undergoing a preliminary medical check-up. None had a history of gastrointestinal disease or antibiotic or laxative use during the preceding 3 months. All were lactose absorbers, as tested by an H₂ breath test after ingestion of 50 g of lactose in water. They gave their informed consent to the protocol, which was approved by the local ethics committee (CCPPRB, Nantes, France). Each subject ingested GUM, SUC and FOS powders that were identical in appearance and colour. The powders were added to orange juice to mask different tastes, and ingested two to six times daily at the subject's convenience.

Experimental design

The study was divided into two independent experiments. The first was intended to assess the effects of GUM on stool output and faecal bacterial concentration in 10 volunteers (4 men and 6 women, aged 22–33 years). The second sought to determine the nature and severity of digestive symptoms during consumption of increasing doses of GUM in 10 volunteers (5 men and 5 women, aged 22–38 years). The two experiments were separated by at least a 2-month interval.

Experiment 1. Faecal output and the bacterial composition of stools were determined over four 10-day periods (no interruptions) in which supplements of SUC 10 g/day, GUM 10 g/day, SUC 15 g/day and GUM 15 g/day were given successively. Menus were established for each day of each period to keep the diet as constant as possible, especially for fibre intake (15–20 g/day). Foods containing inulin, oligosaccharides or alcohol sugars were excluded from the diet, as well as dairy products enriched with bifidobacteria or lactobacilli.

Volunteers were asked to record the times at which daily meals were consumed and sachets ingested, as well as the number of sachets used. All stools excreted on days 5–10 of each period were collected in plastic boxes and maintained at 4°C under anaerobic conditions using an AnaerocultA® (Merck, Darmstadt, Germany) for rapid transportation (within 1 h) to the laboratory. Each stool was weighed and homogenized before one 1-g aliquot was collected for immediate bacterial analysis and two others for determination of dry matter (16 h at 103°C) and total neutral sugars content. The analysis of the faecal microflora was made by V.R., who knew the order of the successive periods of supplementation. The other analyses were made blindly. Thus, only a part of the experiment was double-blinded in nature.

Experiment 2. Tolerance to increasing amounts of GUM (10–70 g/day) was compared to that of SUC and FOS ingested according to the same escalation schedule lasting 18 days: 10 g/day during days 1 and 2, then a 5 g/day increase every 2 days up to 30 g/day on days 9 and 10, followed by a further 10 g/day increase every 2 days up to 70 g/day on days 17 and 18. Intake periods for each substrate were separated by at least 2 weeks of wash-out. The experiment was designed as a double-blind, randomized cross-over study.

Throughout experiment 2, volunteers were asked to avoid foods known to promote abdominal symptoms (cabbage, artichoke, onions, beans, lentils, wheat bran, prunes and plum juice) and to record the time at which meals were consumed, and their composition, and the times at which sachets were ingested, and the number used. They also filled out a diary card concerning tolerance and gastrointestinal symptoms. The following items were recorded: number of bowel movements per day, stool consistency (1 = liquid; 2 = soft or 3 = hard) and size (1 = small, 2 = medium or 3 = large), episodes of flatulence and abdominal discomfort (bloating, borborygmi, cramps) and uneasiness (nausea, headaches, others). Subjects rated symptom severity as absent (0), mild (1), moderate (2), or severe (3). Volunteers were asked to report diarrhoea (one or more watery stools or more than three liquid stools per day) immediately, as well as any grade 3 symptoms.

At day 6 (15 g/day) and 12 (30 g/day) of each period, H₂ excretion was measured in breath. Daily intake of the tested substrate was provided by thirds in meals at breakfast, lunch and dinner. A latex balloon was used to collect samples of expired gas before breakfast and then every hour for 16 h after breakfast. Each sample was stored in a syringe before being analysed for H₂ by gas chromatography (Quintron microlyser model DP, Quintron Instruments Co., MI, USA). On the 2 days during which breath tests were performed, meals were standardized and special care was taken to ensure identical timing for meals and substrate ingestion.

In vitro fermentation

In vitro fermentation of GUM was compared to that of FOS, chosen as a rapidly fermented substrate, and sugar beet fibre (Fibrex®, Fibrex AB, Malmö, Sweden), chosen as a slowly fermented substrate. The study was carried out according to Barry et al. (6), with slight modifications. Briefly, two faecal slurries were prepared by homogenizing faeces (20% w/v) from two healthy volunteers in anaerobic CO₂-saturated nutritive buffer and then filtered through six layers of surgical gauze. Volunteers, who ate Western-style diets, had not taken antibiotics for at least 3 months before the study. Serum bottles (60 ml working volume) containing 10 ml of substrate solution (2% w/v in carbonate/phosphate buffer) were inoculated with 10 ml of faecal slurry. Bottles

with no additional substrates served as controls. Air was displaced by a flow of oxygen-free nitrogen. Cultures were incubated at 37°C for 24 h under anaerobic conditions. Gas production was recorded every hour for 8 h, then at 12 and 24 h, and samples were taken at 0 and 24 h for pH determination, SCFA analysis and substrate disappearance. All incubations were performed in duplicate and the study was repeated twice.

Analyses

Microbiological analysis. All bacterial analyses were performed within 2 h of defecation. Faecal samples (1 g) were serially diluted 10-fold in half-strength peptone water anaerobic broth. Triplicate plates for each dilution were then inoculated in appropriate agars and incubated at 37°C either aerobically or in an anaerobic chamber (H₂ 5%, CO₂ 10%, N₂ 85%). Total anaerobic bacteria were counted on Wilkins–Chalgren agar, facultative anaerobes on nutrient agar, *Bacteroides fragilis* group on modified Bacteroides mineral salt, lactobacilli on Rogosa agar, and total lactic acid-producing bacteria on Man–Rogosa–Sharpe agar. Bifidobacteria were counted on Beerens' medium after microscopic examination of gram-stained bacteria. Clostridia were determined on Wilkins–Chalgren medium after heat treatment at 80°C for 10 min. All media were purchased from Oxoid (Basingstoke, UK).

Short-chain fatty acids. A mercuric chloride (1% w/v) and orthophosphoric acid (5% w/v) solution was added (10%) to aliquots of fermentation media. The samples obtained were then stored at –20°C. Before analysis, samples were thawed and centrifuged. A methyl valerate solution (86.1 mM) was then added (10%) to supernates as internal standard. SCFA were measured by gas chromatographic procedures (7).

In vitro substrate disappearance. The amount of total sugars was determined in the initial substrate + inoculum solutions and their residues after fermentation for 24 h.

Faecal neutral sugars. The faecal neutral sugars were determined in each stool collected for 5 days from each subject, throughout the SUC 15 g/day and GUM 15 g/day periods of experiment 1, by gas-liquid chromatography after the stools had been hydrolysed at 35°C for 1 h in H₂SO₄ (720 g/kg) (8).

Calculations and statistical analysis

All the data are given as means ± SEM. All statistical analyses were performed with Statview 5.0® software (SAS Institute).

Experiment 1. The effect of the substrate and dose on stool output was assessed first by two-way and then by one-way ANOVA. The bulking index of GUM, expressed in grams of wet stool per gram of ingested GUM, was calculated by subtracting the wet stool weight measured on day *i* (*i* = 5 to 10) of the SUC period from that measured on the corresponding day of the gum period, and then

dividing the difference by the daily dose of GUM. Faecal digestibility of the total neutral sugars of GUM was calculated after correction for the excretion of these sugars during the control period.

The effect of the substrate and dose on faecal bacteria concentrations was assessed by two-way and then by one-way ANOVA. The influence of the basal concentration of bifidobacteria on the magnitude of the bifidogenic effect of GUM was evaluated. On the basis of their mean faecal concentration of bifidobacteria during the SUC ingestion period, volunteers were classified into two groups of five subjects: one with > 9.5 log cfu/g of stool and the other with less. The difference between the bifidobacterial concentration (expressed in cfu/g) measured on day *i* of the SUC period and that measured on the corresponding day of the GUM period was then calculated for each day and each volunteer. The means of the two groups were compared by the Mann–Whitney test for unpaired data.

Experiment 2. The occurrence dose was regarded as the first dose producing a symptom that the subject constantly rated higher with GUM or FOS than with SUC. If some of the symptoms were not experienced with the maximal dose (70 g/day), it was assumed that these symptoms would have occurred at the next step dose (80 g/day). Mean occurrence doses for GUM and FOS were compared for each symptom using the Wilcoxon rank test for paired data. At each dose, the percentage of subjects who experienced symptoms at a level constantly higher with GUM or FOS than with SUC was calculated. The 50% effective dose (ED50) was then plotted for each symptom.

The effect of each substrate dose on symptom severity was determined using one-way ANOVA followed by a post hoc test. The possible correlation between the severity of each symptom and the doses of ingested substrates was studied by linear regression analysis.

Hydrogen excretion (expressed in ppm × 16 h) was quantified using a trapezoidal method to compute the area under the discontinuous curve of breath H₂ concentration. The effect of the substrate and dose on mean H₂ excretion was determined first by two-way and then by one-way ANOVA.

In vitro fermentation. SCFA production (mmol/l) was calculated after subtraction of the amount of SCFA present in the initial inocula. Substrate disappearance was expressed as percentage of the initial substrate amounts: (IS – FS) × 100/IS, where IS and FS are the initial and final quantities of total sugars, respectively.

RESULTS

Stool output

The number of stools per week was slightly, although not significantly, increased by ingestion of GUM (10 and 15 g/day) as compared with SUC. Accordingly, daily stool weight was higher during GUM consumption (Table I).

Table I

Stool output measured in subjects ($n = 10$) consuming supplements of sucrose (SUC) or acacia gum (GUM) added to a controlled diet

Supplement (g/day)	Number of stools/7 days	Wet stool weight (g/day)	Stool water (g/day)	Stool dry matter content (%)
SUC 10	7.7±0.7	103.6±8.3	77.6±6.4	26.1±1.4
GUM 10	8.1±0.6	115.8±10.4	84.6±8.0	28.2±1.7
SUC 15	7.8±0.2	100.0±8.6	71.9±7.3	27.3±2.5
GUM 15	8.0±0.6	130.2±11.4 *	108.4±9.2 *	25.5±1.8 *

Data are means ± SEM.

* Different from SUC 15 g/day, $p < 0.05$.

GUM intake also increased water excretion and decreased the dry matter content of stools, although these effects were only significant at a dose of 15 g/day. The overall bulking index for GUM was 1.6 ± 0.5 g of added stool per gram of ingested GUM. Faecal output of total neutral sugars tended to be slightly higher during the ingestion of 15 g/day GUM (3.19 ± 0.21 g/day) as compared with the period of 15 g/day SUC (2.7 ± 0.21 g/day, $p = 0.107$). Faecal digestibility of the neutral sugars of GUM, as calculated after correction for the excretion of these sugars during the SUC period, was $96.5 \pm 2.0\%$ (range 87.7–100).

Faecal microflora

After substrate administration, the number of total bacteria was constant throughout the study, although differences were recorded in the counts of certain bacterial groups. A significant increase in bifidobacteria, lactobacilli and total lactic acid-producing bacteria was observed during GUM consumption (Table II). Considerable variations in faecal bifidobacteria concentrations occurred among subjects during the first period of SUC ingestion (10 g/day). Bifidobacteria counts ranged from 7.5 to 10.2 log cfu/g in this period. These inter-individual variations were reduced during the second SUC period following the period of GUM 10 g/day (Table II). A significant interaction was found between the subject and the substrate for faecal

bifidobacteria concentration ($p = 0.007$), which led us to compare the magnitude of the GUM-induced increase in bifidobacteria in subjects with low (< 9.5 log cfu/g) and high (> 9.5 log cfu/g) basal concentration. The ability of GUM to stimulate bifidobacteria growth was significantly higher in subjects whose basal faecal concentration was < 9.5 log cfu/g (Fig. 1).

Digestive symptoms

None of the tested substrates induced major symptoms of digestive intolerance. Diarrhoea was observed in 2 of the 20 subjects with FOS 50 g/day and in one subject with 60 g/day. The mean degree of severity of the different symptoms did not reach a score of 1 (mild), regardless of the substrate involved (Table III and Fig. 2). For the overall study, symptoms were more frequent with FOS than GUM (19.5 ± 5.0 and 9.1 ± 2.5 symptoms per subject, respectively), and both of these substrates induced more frequent symptoms than SUC (3.3 ± 1.2 symptoms per subject). The degree of symptom severity reported during consumption of substrates was higher with FOS than GUM, and higher with both fermentable substrates than with SUC. However, digestive problems were not significantly different with GUM and SUC below a dose of 30 g/day (Fig. 2). The mean occurrence doses for flatus, bloating and borborygmi were lower with GUM than FOS (Table IV). Accordingly,

Table II

Mean viable counts and pH of five faecal samples collected in subjects ($n = 10$) consuming supplements of sucrose (SUC) or acacia gum (GUM) added to a controlled diet

Bacteria log cfu/g wet stool	SUC 10 g/day	GUM 10 g/day	SUC 15 g/day	GUM 15 g/day	Substrate	Dose	Substrate × dose
					p	p	p
Total aerobes	7.94±0.11	7.81±0.09	7.72±0.13	7.55±0.13	0.19	0.04	0.89
Total anaerobes	10.45±0.05	10.42±0.04	10.49±0.05	10.50±0.05	0.89	0.42	0.72
Bifidobacteria	9.13±0.16	9.75±0.14 *	9.58±0.06	10.02±0.07 †	< 0.0001	0.002	0.44
Bacteroides	8.97±0.07	8.86±0.16	9.10±0.07	9.24±0.13	0.85	0.03	0.29
Clostridia	5.43±0.09	5.43±0.11	5.46±0.11	5.23±0.10	0.29	0.42	0.26
Lactobacilli	8.65±0.18	9.13±0.18 *	8.75±0.14	9.07±0.17	0.02	0.91	0.65
Total lactic acid-producing bacteria	9.34±0.16	10.11±0.06 *	9.75±0.06	10.22±0.05 †	< 0.0001	0.006	0.11
Faecal pH	6.74±0.07	6.65±0.07	6.64±0.07	6.61±0.08	0.42	0.34	0.72

Data are means ± SEM.

* Statistically different from SUC 10 g/day, $p < 0.05$.

† Statistically different from SUC 15 g/day, $p < 0.05$.

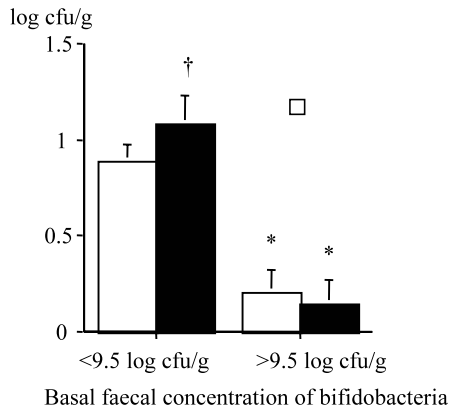


Fig. 1. Increase induced by acacia gum consumption (\square 10 g/day and \blacksquare 15 g/day) in faecal bifidobacteria in subjects with low (< 9.5 log cfu/g) and high (> 9.5 log cfu/g) basal concentration. *High vs low basal concentration were statistically different, $p < 0.02$. †Gum 15 g/day vs 10 g/day were statistically different, $p = 0.05$.

the 50% effective dose was higher with GUM than FOS for flatus and bloating. Thus, a daily dose of 17 g of FOS induced flatus in half of the subjects, whereas 46 g/day of GUM were needed to induce the same symptoms (Table IV).

Breath tests

Consumption of GUM and FOS, as compared with SUC, raised breath hydrogen concentration ($p < 0.001$). The quantity of H_2 excreted in breath, as calculated by the area under the 16-h excretion curve, was higher with FOS than with GUM (Fig. 3), and the effect depended on the ingested dose ($p = 0.06$).

In vitro fermentation

The volume of gas produced during *in vitro* incubation of faecal bacteria with carbohydrates reflects the extent of fermentation, and the kinetics of gas production is indicative of the fermentation rate. Gas volume increased steadily during the first 12 h of GUM incubation then remained stable, but rose very rapidly (within 2 h) to its maximal

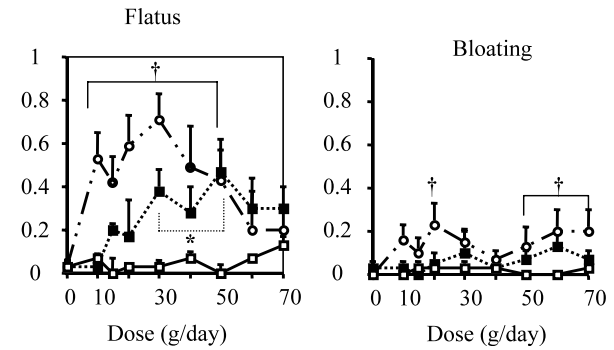


Fig. 2. Flatus and bloating severity reported by healthy subjects consuming sucrose (\square) or acacia gum (\blacksquare) or fructo-oligosaccharides (\circ) at doses increasing from 10 to 70 g/day. Symptom severity was rated as absent (0), mild (1), moderate (2), or severe (3). *GUM is statistically different from sucrose, $p < 0.05$. †FOS is statistically different from sucrose.

value during FOS incubation and then decreased slightly before stabilizing after 8 h. Gas volume increased more progressively during sugar beet fibre fermentation (Fig. 4). The final pH of the *in vitro* incubation medium was lower with FOS (4.9 ± 0.1) than with GUM (5.7 ± 0.3) and sugar beet fibre (6.3 ± 0.2). GUM led to a total SCFA concentration (47.8 ± 9.4 mmol/l) between those induced by FOS (61.6 ± 2.4 mmol/l) and sugar beet fibre fermentation (39.5 ± 5.1 mmol/l). Accordingly, the disappearance of total sugars was higher after the 24-h fermentation of FOS ($76.8 \pm 15.7\%$) than after fermentation of GUM ($68.4 \pm 24.2\%$) and sugar beet fibre ($56.6 \pm 18.9\%$).

DISCUSSION

The present study shows that acacia gum (GUM) did not induce adverse gastrointestinal effects even when consumed at high doses, and demonstrates that the gum is a bifidogenic factor. Dietary fibre may cause several symptoms of digestive discomfort during the first days of intake and progressive adaptation is generally recommended. Our study showed that the digestive tolerance of GUM was high. Flatus, bloating and borborygmi occurred at lower

Table III

Mean degree of symptom severity and correlation with the dose during regular consumption of sucrose (SUC), acacia gum (GUM) or fructo-oligosaccharides (FOS)

Symptom	SUC			GUM			FOS		
	Degree	<i>r</i>	<i>p</i>	Degree	<i>r</i>	<i>p</i>	Degree	<i>r</i>	<i>p</i>
Flatus	0.15 ± 0.04	0.536	0.170	0.61 ± 0.09 *	0.842	0.009	0.92 ± 0.09 *†	0.587	0.126
Bloating	0.06 ± 0.03	0.118	0.780	0.15 ± 0.04 *	0.749	0.032	0.40 ± 0.07 *†	0.391	0.338
Borborygmi	0.01 ± 0.01	0.312	0.451	0.10 ± 0.04 *	0.711	0.048	0.41 ± 0.08 *†	0.733	0.038
Cramps	0.01 ± 0.01	0.612	0.107	0.22 ± 0.07 *	0.134	0.751	0.29 ± 0.08 *	0.079	0.853

Data are means \pm SEM.

* Statistically different from SUC, $p < 0.05$.

† Statistically different from GUM, $p < 0.05$.

Table IV

Mean occurrence doses and 50% effective doses (ED50) for each symptom during the regular consumption of acacia gum (GUM) and fructo-oligosaccharides (FOS)

Symptom	Occurrence dose (g/day)		ED50 (g/day)	
	GUM	FOS	GUM	FOS
Flatus	53.5 ± 2.5	26.5 ± 2.1 *	46	17.5
Bloating	67 ± 2.1	50 ± 3.1 *	65.7	41.7
Borborygmi	70.5 ± 2.2	51.5 ± 3.3 *	60.7	60
Cramps	71 ± 1.9	67.8 ± 2.1	55	57.5

doses with FOS than with GUM. The effect of FOS was very similar to that described previously (9, 10). Both of these studies found that tolerance did not improve during 12 or 15 days of regular intake. The main complaint related to all doses of FOS was excessive flatulence, but daily doses < 20 g/day resulted in only minor complaints, while doses > 50 g/day resulted in abdominal cramps and diarrhoea (9). In the present study, GUM did not induce flatulence below the dose of 30 g/day and daily doses higher than 50 g/day

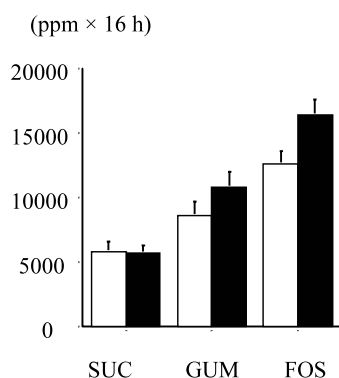


Fig. 3. Cumulated excretion of breath hydrogen for 16 h after ingestion of sucrose (SUC), acacia gum (GUM) or fructo-oligosaccharide (FOS) supplements (□ 15 g or ■ 30 g), added to standardized meals.

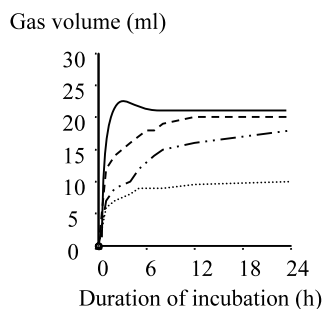


Fig. 4. Gas volume collected during *in vitro* incubation of a human faecal inoculum alone (.....), and added with sugar beet fiber (---), fructo-oligosaccharides (—) or acacia gum (-----).

did not provoke any abdominal cramps or diarrhoea. The recommended daily intake for dietary fibre worldwide ranges between 25 and 40 g/day, and it is expected that GUM intake may account for 0–50% of these doses (i.e. 0–20 g/day), which is largely below the 50% effective doses inducing abdominal symptoms. The better tolerance of GUM as compared with that of FOS may be due to several factors. First, probably owing to its high molecular weight, GUM was fermented at a slower rate than FOS and led to higher pH after incubation for 24 h. The lower pH recorded after fermentation of FOS in comparison with GUM may result from the higher production of SCFA as well as an accumulation of lactate. We did not monitor it in the present study; however, we previously observed that lactate accumulated only during the *in vitro* fermentation of FOS and not during that of GUM (5). We assume that this effect may arise because bacterial glycolysis of FOS to lactate is more rapid than the further metabolization of this intermediate to SCFA. Acid pH or lactic acid may excite some visceral sensory nervous fibres and lower nociceptive threshold for intestinal distension (11). Secondly, breath hydrogen excretion was lower during GUM than FOS intake, possibly because of lower gas production. Even if intestinal gas tolerance is normally high, some apparently normal subjects can retain gas and develop abdominal distension and symptoms (12). The origin of the increase in gas production after the FOS intake is not obvious, as this sugar is preferentially fermented by bifidobacteria and lactic acid-producing bacteria, which do not yield any gas. One possible explanation is that the lactate produced by these bacteria can be further utilized by other bacterial groups which are gas producers (13). Another explanation could be that, despite their selectivity towards lactic acid bacteria, FOS might also be metabolized to a lesser degree by gas-forming organisms such as clostridia (14).

In addition to its high level of intestinal tolerance, GUM increased stool weight. Its bulking capacity is similar to that of pectins and guar gum (15), as well as inulin and FOS (16). This effect was associated with a slight rise in dry matter excretion and with a major rise in water excretion. As faecal digestibility of GUM was high, the increase in stool dry matter and water content was probably due to faecal bacteria, as previously shown in chronic renal failure patients (17). It has already been reported that gum arabic completely disappears during its passage through the gastrointestinal tract both in animals (18, 19) and in humans (20, 21). Our results confirm that the neutral sugars from GUM are extensively degraded in the human intestine and indicate that the overall digestibility of GUM could be around 95% in healthy humans consuming the GUM for at least 5 days. A lower percentage of disappearance was found after *in vitro* fermentation for 24 h. As there are no enzymes that are able to hydrolyse GUM polysaccharides in the human stomach and small intestine,

it is unlikely that the discrepancy comes from the digestion of GUM in the upper digestive system. Furthermore, many results from animal models showed that gum arabic resists digestion in the small intestine and enters into the caecum (18, 19, 22). A more plausible explanation is that the difference between the fermentability *in vitro* and *in vivo* was due to the prior adaptation of colonic microflora in the human trial. The need for such an adaptation for a complete degradation of gum arabic has been demonstrated in rats (19, 22) and strongly suggested in humans (21). Because of this necessary adaptation of the flora to thoroughly metabolize GUM, the digestible energy value of GUM may vary. Nevertheless, because energy is lost to faeces, predominantly as unfermented carbohydrates and as bacterial biomass, the digestible energy value of GUM is less than the 17.5 kJ/g value for available starch. According to the correlations between fermentability and digestible energy value of non-starch polysaccharides, or between the metabolizable energy of fibre and production of SCFA *in vitro*, found in two European inter-laboratory studies (6, 23), the caloric value of GUM could range between 5.5 and 7.7 kJ/g, thus <2 kcal/g and close to the 1.5 kcal/g value proposed for nutritional labelling (24).

For the first time, this study demonstrated that GUM increased the counts and the proportion of bifidobacteria in human stools and is thus a strong candidate prebiotic. *In vitro* studies showed that GUM supported the growth of pure cultures of bifidobacteria strains (25) and increased total lactic acid-producing bacteria counts in continuous cultures of human faecal microflora (5). In the stools of one volunteer, Wyatt et al. (21) measured an increase in the proportion of bacteria able to ferment the gum, among which species of *Bacteroides* and *Bifidobacterium* were found. These preliminary findings suggested that GUM could be prebiotic; however, its effect in healthy humans was not established. Our results show that, compared with SUC, GUM intake (10 and 15 g/day) raised faecal concentrations of total lactic acid-producing bacteria and bifidobacteria, without changing total anaerobe and aerobe counts. Moreover, bacteroides and clostridial groups were not affected by GUM, indicating that the effect was specific. The increase in the proportion of lactic acid-producing bacteria and bifidobacteria may be related to the acidification of colonic contents during GUM fermentation. A decrease in pH could have promoted the growth of acid-tolerant bacteria, such as lactobacilli and bifidobacteria. However, the pH of colonic contents is decreased by many fermentable fibres, most of which do not appear to be bifidogenic. Therefore, even though acidification may favour the bifidogenic effect of GUM, it is likely that in our study this effect was mainly due to the ability of the gum to support the growth of *Bifidobacterium* spp., as reported for *B. longum* and *B. adolescentis* (25). The fact that GUM contains high amounts of galactose could account for this property.

Several works comparing different carbohydrates have reported that galactose-containing oligosaccharides were the most effective in stimulating growth of bifidobacteria (26–28). In addition, we observed that bifidobacteria concentration was particularly increased in subjects whose initial concentration was low. Such an inverse relationship has been observed previously for other prebiotics (28, 29). Therefore, GUM, like other prebiotics, may be particularly useful for targeting certain groups of individuals, such as the elderly who have diminished bifidobacteria populations.

In conclusion, this study suggests that GUM is a prebiotic fibre, as it was able to selectively raise the proportions of lactic acid bacteria and bifidobacteria in healthy subjects. It was fermented slowly, produced short-chain fatty acids, and its faecal digestibility was around 95%. As in the case of other soluble dietary fibres, it increased stool output by augmenting the water content of stools. In addition, its intestinal tolerance appeared to be excellent and high daily doses could be consumed without inducing any adverse intestinal events. Thanks to all these properties, GUM may improve intestinal well-being and health, although this potential remains to be confirmed in further nutritional trials including a large number of volunteers.

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