

Digestibility and bulking effect of ispaghula husks in healthy humans

P Marteau, B Flourié, C Cherbut, J-L Corréze, P Pellier, J Seylaz, J-C Rambaud

Abstract

The digestibility of ispaghula, a mucilage from *Plantago ovata* composed mainly of arabinoxylans, and its faecal bulking effect were studied in seven healthy volunteers who ingested a low fibre controlled diet plus either placebo or 18 g/day of ispaghula for two 15 day periods. Whole gut transit time and gas excretion in breath and flatus were not different during the periods of ispaghula and placebo ingestion. Faecal wet and dry weights rose significantly, however, during ispaghula ingestion. Faecal short chain fatty acid concentrations and the molar proportions of propionic and acetic acids also increased. Most of the ispaghula had reached the caecum four hours after ingestion in an intact highly polymerised form. During ispaghula ingestion, the increase in the faecal output of neutral sugars was accounted for by the faecal excretion of arabinose and xylose in an intact highly polymerised form; the apparent digestibilities of these sugars were 24 (11) and 53% (6) respectively (mean (SEM)). In conclusion, ispaghula is more resistant to fermentation than previously reported in humans, and its bulking effect largely results from intact material.

(Gut 1994; 35: 1747-1752)

The possible mechanisms by which dietary fibres increase stool weight fall into two broad categories.^{1,2} Firstly, mechanisms that act by increasing stool volume. These include the physical presence of the undigestible fibre in the colon, this fibre's water holding capacity, the stimulation of microbial growth, and the production of gas. The resulting increase in stool volume may cause colonic propulsion. Secondly, mechanisms that act primarily by stimulating colonic motility, particularly by mechanical action of the fibre on the colon, and an increase in certain colokinetic end products of fibre fermentation.

Plantago ovata husks, often referred to as ispaghula husks, are highly branched arabinoxylans widely used as a faecal bulking agent.³⁻⁸ The mechanism of the bulking effect of ispaghula is still unclear. In rats and during in vitro incubation with mixed faecal bacteria harvested from humans, ispaghula is only partially fermented,⁹⁻¹³ whereas in the only human study reported so far, Prynne and Southgate¹⁴ showed that it could be extensively fermented.

In this study, we aimed at determining the mechanism of the bulking effect of ispaghula in

humans by assessing the digestibility of ispaghula, faecal bacterial mass, and transit time. Special care was taken to assess the production of gas and changes in the proportions of short chain fatty acids. In addition, caecal contents were sampled to discover the form in which ispaghula reached the caecum.

Methods

Seven healthy volunteers (five men and two women, aged 21-35 years) gave informed written consent to the study protocol, which was approved by the local ethics committee. None of them had a history of gastrointestinal disease, the use of laxatives or recent treatment with antibiotics. Four subjects were identified as producers of methane because their breath methane concentrations were at least one $\mu\text{l/l}$ higher than the room air concentration.¹⁵

EXPERIMENTAL DESIGN

The study comprised two 15 day periods with an interval of 21 days between them. In a randomised, blind, and cross over fashion, the subjects were given 12.9 g ispaghula husk pellets twice daily - that is, 18 g ispaghula/day (Spagulax, Beecham, Nanterre, France) throughout one period, and placebo (sucrose pellets) throughout the other. Ispaghula was mainly composed of neutral sugars, 90% of which consisted of arabinose and xylose (Table I). The ispaghula and placebo pellets were packaged in tubes, which also contained five grams of the faecal recovery marker polyethylene glycol 4000 (PEG). The contents of the tubes were swallowed whole during breakfast and dinner.

During each period, the subjects consumed a basal diet that consisted of conventional low fibre foods and provided 6 g dietary fibre and 1-2 g resistant starch per day as determined from food tables.^{16,17} Great care was taken to ensure that the dietary constituents were the same throughout the two periods. The ingredients for breakfast were given to the subjects to be prepared and eaten at home; lunch and dinner were prepared and eaten in the laboratory. At the end of each period (days 13-15), subjects were admitted to the clinical centre and given standardised test meals.

TABLE I Neutral sugar composition of ispaghula husks (g/100 g dry matter)

Rhamnose	2.1	Mannose	1.1
Arabinose	17.6	Galactose	3.7
Xylose	47.4	Glucose	Traces

Neutral sugars were measured according to Hoebler *et al.*²⁴ The rest of the 100 g dry matter was not specifically assessed.

INSERM U 290
Hôpital Saint-Lazare,
Paris, France
P Marteau
B Flourié
P Pellier
J-C Rambaud

INRA, Nantes, France
C Cherbut

CNRS UA-641, Paris,
France
J-L Corréze
J Seylaz

Correspondence to:
Dr P Marteau, INSERM U
290 Hôpital Saint-Lazare,
107 bis rue de Faubourg
Saint-Denis, 75010 Paris,
France.

Accepted for publication
23 March 1994

TABLE II Test meal composition

Eggs*	2	Coffee	250 ml
Cheese (Gruyère)	30 g	Hydrolysed milk	10 g
White bread	100 g	Sucrose	10 g
Butter	30 g	Ispaghula or placebo	9 g
Strawberry jam	30 g		

*Boiled for 10 minutes.

During both periods, subjects were asked to record daily symptoms (bloating, borborygmos, cramp, and flatus), which they themselves graded on a scale from 0 to 3 (none to severe). After seven days of equilibration, all stools were collected from day 8 to day 13 and immediately frozen at -20°C . On days 8, 9, and 10, subjects ingested with breakfast 20 radio-opaque pellets of different shapes to permit measurement of mean oro-faecal transit time according to the method of Cummings and Wiggins.¹⁸ On day 13, a test meal was given at 8 am (composition in Table II) with either ispaghula or placebo. End expiratory breath samples were taken before the meal, and every 30 minutes thereafter to measure oro-caecal transit time and total excretion of hydrogen in breath over 12 hours. During the same period, rectal gases were collected using a flexible canula 4 mm in diameter whose tip was inserted into the rectum 12 cm from the anal verge, and which was connected to a laminated gas bag (ABS, Saint-Dié, France). Gas bags were closed with a stopcock, and changed every four hours. The effectiveness of this gas collection method has been previously validated in a water bath, and in six other subjects by instilling the non-absorbable gas sulphur-hexafluoride into the rectum and checking its recovery.¹⁹ After test meal ingestion, no food was permitted until the end of exhaled breath and rectal gas collection, and subjects had to refrain from smoking, and stay in a recumbent position. In two subjects, leaks of gas were felt, and the experiment was repeated on the following day.

On day 14 of each period, four of the subjects were intubated with a polyvinyl tube led by an inflatable mercury bag as previously described.²⁰ Progression of the tube was stopped when the sampling port had reached the caecum, as confirmed fluoroscopically. On day 15, the same test meal as on day 13 was given to these subjects at 8 am, and 0.5 ml of caecal contents was sampled within five minutes at two, four, six, and eight hours after the meal, while the subjects continued to fast. After each sampling, the tube lumen was flushed with nitrogen and the sample was immediately frozen at -20°C .

ANALYTICAL METHODS AND CALCULATION

For each stool, the pH was measured using a pH meter (Radiometer, Copenhagen, Denmark), PEG by turbidimetry,²¹ nitrogen by the Kjeldahl method, and ammonia, according to Assous *et al.*²² An aliquot from each stool was lyophilised to measure the dry weight and faecal bacterial mass according to the fractionation procedure of Stephen and Cummings.²³ Briefly, the fractionation was

achieved by repeated mixing in a stomacher of 0.5 g dried stools combined with formylsaline (0.9% vol/vol NaCl and 1% vol/vol formalin) and sodium lauryl sulphate (0.1%) followed by filtering through nylon meshes. This procedure gives a fraction of coarse particles mainly of plant cell wall material, a fraction of fine particles, which also are from the plant cell wall, and the suspension of bacteria in large volume of washings. The bacterial fraction was centrifuged at 30 000 g for 30 minutes, and, after discarding the supernatant, the pellets were dried to constant weight. For the other measurements, the five day faecal collections obtained from each subject throughout each period were pooled. Short chain fatty acids were determined by gas chromatography.²⁴ The solubility of carbohydrate molecules was determined as described by Delort-Laval and Mercier.²⁵ Briefly, carbohydrates were extracted by ethanol 80° (extraction of oligosides), ethanol 40° (extraction of carbohydrate molecules with a degree of polymerisation <65), and water. Neutral sugars were subsequently measured in the three extracts and in the residue after freeze drying, according to Hoebler *et al.*²⁶

The caecal contents of the four subjects sampled were pooled for each sampling time throughout the placebo and ispaghula periods, and analysed for carbohydrate molecule solubility and neutral sugars as described above. As the duration of sampling was limited to five minutes to reduce in vitro fermentation in the tube and to avoid interference with the normal physiology, we could not collect the caecal contents before the meal. The volume of the sample taken eight hours after ingestion of the test meal containing ispaghula was not sufficient to permit solubility analysis.

Concentrations of breath hydrogen were measured using an electrochemical cell (Exhaled Hydrogen Monitor, GMI Medical Ltd, Renfrew, Scotland). The total excess volume of hydrogen excreted in breath after the test meal was determined by integrating the area under the hydrogen concentration curve over basal values. For each subject, tidal volume was determined from the reference nomogram and data were expressed in ml per total test period (12 hours). Oro-caecal transit time was defined as the time elapsing between the beginning of the meal and the sustained increase in breath hydrogen ($>5 \mu\text{l/l}$). Methane concentrations in breath were measured by gas-liquid chromatography (IGC 121 DFL-Intersmat, Country, France). Rectal gas volume was measured manually after its evacuation from the bag into a greased glass syringe, and gas composition was analysed using a mass spectrometer (QMG 511, Balzers, Balzers, Liechtenstein).

STATISTICAL ANALYSIS

Results are expressed as mean (SEM). Comparisons between the two periods were made using Wilcoxon's matched pair signed rank test.

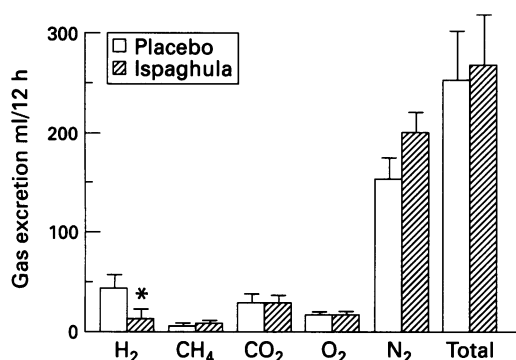


Figure 1: Rectal gas excretion in seven healthy volunteers after ingestion of test meals plus placebo or ispaghula. Results are expressed as means, bars represent standard error. * $p < 0.05$.

Results

SYMPTOMS AND TOLERANCE

Ispaghula was well tolerated; the cumulative score of each symptom was very low, and was not different for the two periods. Cumulative scores (maximal value 36) of bloating, borborygmus, cramps, and flatus were 4.4 (3.3), 1.9 (1.9), 2.0 (1.0), 9.3 (5.2) during the placebo period. They were 4.0 (2.6), 3.9 (2.2), 5.3 (3.0), 6.1 (2.8) during the ispaghula consumption.

BREATH AND RECTAL GASES

The excess volumes of hydrogen excreted in breath were 23.8 (5.9) and 38.6 (9.2) ml/12 hours after ispaghula and placebo meals respectively ($p = 0.20$). In the four hydrogen producers, breath hydrogen excretion was not different for the two periods either in the fasting state, or after the test meal. The gases excreted by the rectum during these periods were not significantly different as regards total volume and composition, except for hydrogen, whose excretion was lower during the ispaghula period (Fig 1).

TRANSIT TIMES

Breath hydrogen excretion increased after both test meals in each subject permitting determination of orocaecal transit time. Mean orocaecal transit time and orocaecal transit time were not significantly affected by ispaghula consumption. Mean orocaecal transit times

TABLE III Number of stools, faecal weight, and composition during the periods of placebo and ispaghula ingestion

	Placebo	Ispaghula	p Value
Number of stools/day	1.0 (0.10)	1.20 (0.12)	0.037
Faecal wet weight g/day	122 (11)	231 (13)	0.009
Faecal dry weight g/day	38 (6)	51 (4)	0.037
Faecal water g/day	85 (7)	180 (8)	0.009
Faecal water content (%)	69 (1)	77 (3)	0.042
Bacterial dry mass g/day	14 (2)	16 (2)	0.20
Nitrogen g/day	3.8 (0.7)	4.3 (0.5)	0.25
Ammonia mg/day	33 (3)	34 (4)	0.25
Non-ammonia nitrogen g/day	3.8 (0.7)	4.3 (0.5)	0.60

Results are expressed as mean (SEM).

TABLE IV Faecal concentrations of neutral sugars during the periods of placebo and ispaghula ingestion

Sugars mg/g		Placebo	Ispaghula	p Value
Fucose	dw	Traces	Traces	NS
	ww	Traces	Traces	NS
Ribose	dw	Traces	Traces	NS
	ww	Traces	Traces	NS
Rhamnose	dw	5.9 (0.5)	6.5 (0.6)	0.027
	ww	1.8 (0.1)	1.4 (0.1)	0.027
Arabinose	dw	7.8 (0.7)	58.1 (6.6)	0.027
	ww	2.4 (0.2)	12.3 (1.0)	0.027
Xylose	dw	11.6 (1.1)	98.7 (12.2)	0.027
	ww	3.6 (0.4)	20.7 (1.5)	0.027
Mannose	dw	5.5 (0.5)	7.6 (0.9)	0.12
	ww	1.7 (0.1)	1.5 (0.1)	0.25
Galactose	dw	13.0 (1.2)	20.5 (1.9)	0.046
	ww	4.0 (0.3)	4.3 (0.2)	0.35
Glucose	dw	49.4 (4.6)	54.1 (9.6)	0.91
	ww	15.5 (1.4)	11.4 (1.7)	0.17
Total	dw	93.2 (2.1)	254.1 (11.2)	0.027
	ww	29.0 (0.7)	54.6 (4.5)	0.027

ww=mg/g faecal wet weight; dw=mg/g faecal dry weight. Results are expressed as mean (SEM).

were 53 h (8) and 42 h (6) for placebo and ispaghula respectively ($p = 0.33$), and orocaecal transit times were 325 min (38) and 330 min (41) ($p = 0.94$).

FAECAL COMPOSITION

The percentages of ingested PEG recovered in stools were 100 (7) and 92 (6) for the placebo and ispaghula periods respectively. The number of stools, faecal wet and dry weights, and faecal water were significantly greater during the ispaghula period (Table III). Faecal bacterial mass, and faecal excretion of nitrogen, ammonia, and non-ammonia nitrogen were not different for the two periods (Table III).

Table IV gives the concentrations of neutral sugars (expressed per g dry and wet weight) in the pooled faecal contents during both the ispaghula and placebo periods. The total concentrations of neutral sugars were significantly higher during the ispaghula than the placebo ingestion period because of the higher faecal concentrations of arabinose and xylose. Faecal outputs of neutral sugars also increased significantly during ispaghula ingestion, and again, this was accounted for by the increased outputs of arabinose and xylose (Table V). The digestibilities of arabinose and xylose, calculated after correction for the excretion of these sugars measured during the placebo period, were respectively 24% (11) (range 12–36) and 53% (6) (range 46–60). Most of the arabinose and xylose excreted in stools was present in the insoluble fraction – that is, in the highly polymerised fraction – compared with the ingested ispaghula (Fig 2).

TABLE V Faecal outputs of neutral sugars during the placebo and ispaghula ingestion periods

Sugars g/day	Placebo	Ispaghula	p Value
Fucose	Traces	Traces	NS
Ribose	Traces	Traces	NS
Rhamnose	0.21 (0.02)	0.31 (0.02)	0.027
Arabinose	0.28 (0.03)	2.70 (0.16)	0.027
Xylose	0.42 (0.04)	4.57 (0.30)	0.027
Mannose	0.20 (0.02)	0.35 (0.04)	0.027
Galactose	0.47 (0.05)	0.97 (0.07)	0.027
Glucose	1.78 (0.19)	2.64 (0.55)	0.34
Total	3.37 (0.35)	11.54 (0.87)	0.027

Results are expressed as mean (SEM).

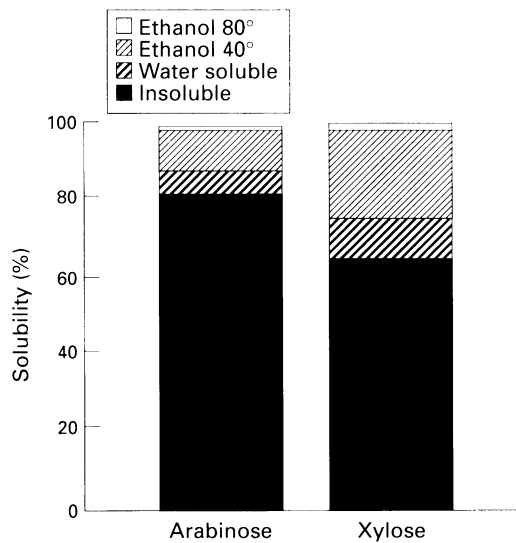


Figure 2: Solubility (mean %) of arabinose and xylose excreted in the stools of seven healthy volunteers. Arabinoxylans in the ingested ispaghula were totally insoluble; most of the arabinoxylans recovered in faeces remained insoluble – that is, in the highly polymerised form.

TABLE VI Concentrations of short chain fatty acids in faecal water during the placebo and ispaghula ingestion periods

Concentration (mmol/l)	Placebo	Ispaghula	p Value
C ₂	25.7 (2.3)	60.1 (6.3)	0.027
C ₃	7.5 (1.3)	24.9 (5.4)	0.039
IC ₄	1.1 (0.3)	1.2 (0.2)	0.46
C ₄	7.3 (1.0)	11.1 (2.0)	0.30
IC ₅	1.8 (0.4)	1.5 (0.3)	0.62
C ₅	1.2 (0.2)	2.2 (0.5)	0.05
Total	44.7 (4.4)	100.9 (12.6)	0.027

Results are expressed as mean (SEM). C₂=acetic acid; C₃=propionic acid; IC₄=isobutyric acid; C₄=butyric acid; IC₅=isovaleric acid; C₅=valeric acid.

The faecal concentrations and 24 hour outputs of acetate, propionate, and total short chain fatty acids were significantly higher during the ispaghula than placebo period (Table VI). Outputs of butyrate and valerate were also higher but not their faecal concentrations. Faecal pH was slightly but significantly lower during ispaghula ingestion at 6.5 (0.1) v 6.7 (0.1) (p=0.03).

TABLE VII Caecal concentrations of neutral sugars after ingestion of the test meal with placebo and the test meal with ispaghula

Time after ingestion	Two hours		Four hours		Six hours		Eight hours	
	Placebo	Ispaghula	Placebo	Ispaghula	Placebo	Ispaghula	Placebo	Ispaghula
% Water	96.8	96.5	95.5	94.4	94.2	94.9	92.9	92.8
Rhamnose	dw 1.9	3.3	7.0	1.5	4.1	5.3	2.5	5.1
	ww 6.1	14.8	31.5	8.4	23.8	27.0	17.7	35.7
Fucose	dw 7.4	5.5	1.0	3.1	4.9	4.4	8.8	0
	ww 23.7	19.2	4.5	17.4	28.4	22.4	62.5	0
Ribose	dw 3.4	3.0	5.8	1.7	3.8	5.2	3.5	3.5
	ww 10.9	10.5	29.0	9.5	22.0	26.5	24.8	24.5
Arabinose	dw 4.1	11.4	2.8	13.8	31.2	45.1	13.6	39.2
	ww 13.1	40.0	12.6	77.3	181	230	96.6	274.4
Xylose	dw 8.6	21.0	5.2	25.8	43.1	95.3	95.8	92.7
	ww 27.5	73.5	23.4	144	250	486	183	649
Mannose	dw 5.9	11.1	3.2	8.2	11.4	12.1	13.4	6.6
	ww 18.8	38.8	14.4	45.9	66.1	61.7	95.1	46.2
Galactose	dw 16.6	17.2	10.9	30.9	19.6	22.0	19.9	15.0
	ww 53.1	60.2	49.0	173	114	112	141	105
Glucose	dw 96.3	180	44.8	301	93.7	82.0	119	75.2
	ww 308	631	201	1687	543	418	843	526
Total	dw 144	253	81	387	212	271	206	237
	ww 462	885	363	2165	1228	1383	1464	1661

dw = mg/g dry weight, ww = mg/100 g wet weight.

COMPOSITION OF CAECAL CONTENT

The percentages of water in pooled caecal contents at any sampling performed were similar after ingestion of the meal with ispaghula and placebo (Table VII). Glucose was the main neutral sugar found in the caecum at all the times sampled after ingestion of the test meal with placebo. Caecal concentrations of neutral sugars, expressed per g dry and wet weight, were higher after ingestion of the test meal with ispaghula. Within the first four hours this was accounted for by the high concentration of glucose, and as from six hours by the high concentrations of arabinose and xylose, which became the predominant sugars (Table VII). Figure 3 shows the distribution of glucose, arabinose, and xylose in the fractions with different solubilities. Forty six per cent of the arabinose reaching the caecum two hours after the meal and 39% of the xylose were extracted in the 80° ethanol soluble fraction – that is, in the form of oligosides. As from four hours after the meal, however, these sugars were mainly present as insoluble highly polymerised molecules (Fig 3).

Discussion

This study confirms that ispaghula is a potent stool bulker in humans,^{3, 8} and shows that it is fermented to a limited extent throughout the human colon.

The digestibility of arabinose and xylose was calculated after correction for basal excretion, assuming that ispaghula did not increase faecal excretion of the arabinose and xylose from the basal diet. Theoretically, some of the saccharides excreted may have been bacterial polysaccharides or endogenous materials, or both, the excretion of which could have been increased by fibre intake leading therefore to an underestimation of the ispaghula digestibility. Faecal bacterial mass and non-ammonia nitrogen did not rise significantly during ispaghula consumption, however, and arabinose and xylose are very rarely present in bacterial saccharides.²⁷ Intestinal glycoproteins do not contain arabinose or xylose,²⁸ but do contain fucose, which was not detected here in stools.

The apparent digestibility of xylose was greater than that of arabinose. This suggests that the ispaghula backbone containing xylose might be more easily accessible to fermentative enzymes than the highly branched chains containing arabinose.²⁹ Overall, the apparent digestibility of total neutral sugars contained in ispaghula was 54%. This is in agreement with the digestibility found in vitro and in rats,^{9, 13, 30} and in two of four volunteers studied by Prynne and Southgate¹⁴ during the consumption of 25 g ispaghula in the form of Isogel. In this last study, however, in which the mechanism of faecal bulking was not assessed and neutral sugars were not specifically measured in faeces, the apparent digestibility of ispaghula was higher in the two other volunteers (as much as 100%). Differences in the adaptation period (one week instead of two) do not seem to play an important part in digestibility of

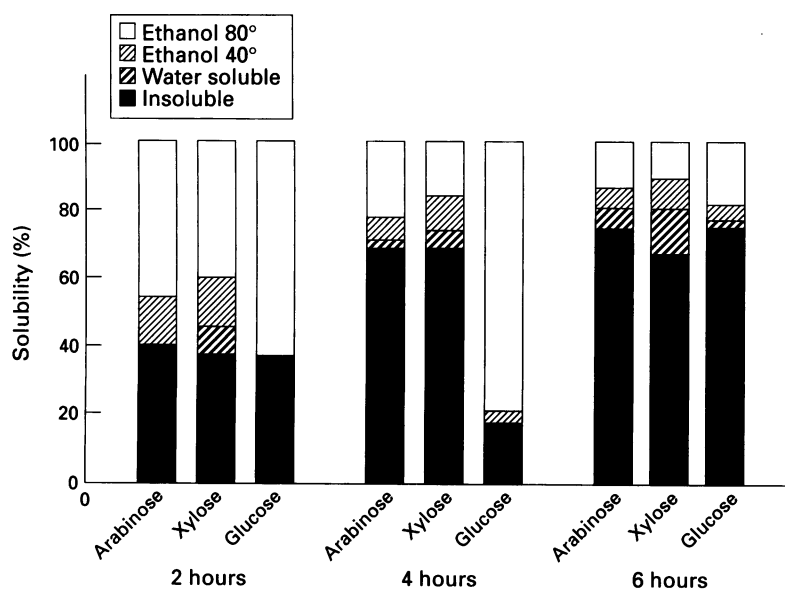


Figure 3: Solubility (%) of arabinose, xylose, and glucose in the pooled caecal contents of four subjects sampled two, four, and six hours after ingestion of the test meal with ispaghula. A large part of the molecules recovered two hours after the meal were soluble in ethanol 80° – that is, in the form of oligosides; arabinosylans recovered four hours after the meal were insoluble (highly polymerised form).

ispaghula as we found no significant modifications of digestibility in four other volunteers who ingested ispaghula for 31 days (data not shown). Individual differences in colonic flora might be responsible for individual variations in ispaghula digestibility; in our study, however, these variations were small. The nature of the ispaghula used might also influence its digestibility, as shown with other varieties of the same fibre.³¹ An interesting feature of our ispaghula preparation was its insolubility in both water and ethanol.

The partial fermentation of ispaghula led to increased faecal concentrations and excretion of short chain fatty acids but did not significantly affect the faecal bacterial mass. Ispaghula was well tolerated in our study as in others,^{5 32} and did not increase either rectal expulsion of gas or the excretion of methane and hydrogen in breath.^{7 10 33} For unknown reasons, rectal hydrogen was even slightly lower after ingestion of the meal with ispaghula. The rise in the faecal concentration of short chain fatty acids was mainly accounted for by the increased concentrations of propionic acid (+230%) and acetic acid (+130%). An increase in the concentration of propionic acid was previously reported during the incubation of ispaghula with human faeces,¹⁰ and in the colonic and faecal contents of rats consuming ispaghula.^{11 12} Propionic acid may reduce contractile activity in rat isolated colon mounted in vitro.³⁴ In our study, ispaghula did not affect transit time. This has also been reported by other workers in healthy volunteers^{3 4}; in constipated patients, however, ispaghula has been shown to reduce transit time.^{5 35}

The incomplete fermentation of ispaghula in humans is probably caused by its slow metabolism by colonic bacteria, as shown in rats.¹² It has been suggested that the distal fermentation of fibres in the rectosigmoid could have a beneficial effect on colonic carcinogenesis due to

the beneficial actions of butyrate³⁶; in our volunteers, however, the increase in faecal concentration of butyrate during ispaghula consumption did not reach statistical significance.

The rise in faecal dry weight (about 13 g/day) was mainly accounted for by the increased excretion of neutral sugars (8 g/day), especially arabinose and xylose. As in the ingested ispaghula, arabinose and xylose in stools were mainly present in the insoluble highly polymerised fraction, and only a small proportion consisted of osmotically active oligosides – that is, in the 80° ethanol soluble fraction. Compared with the placebo period, a slight non-significant increase in bacterial mass occurred during ispaghula ingestion. The large increase in faecal water may be consistent with previous in vitro determinations of the water holding capacity of ispaghula.³⁷ In rats consuming ispaghula, Nyman and Asp⁹ reported also an increase in faecal water holding capacity, which they determined by a centrifugation method. Such an increase was not found, however, by Edwards *et al.*,¹² who used a dialysis method, and the water holding capacity determined by dialysis on a residue fermented in vitro was only 4 g H₂O/g ispaghula.¹³ These last findings could suggest that a proportion of the additional water in faeces is not bound to undegraded fibre or bacteria. Short chain fatty acids resulting from the distal fermentation and to a lesser extent undigested oligosides could maintain water in the lumen, and reduce the amount of free water absorbed.

Small fractions of arabinose and xylose reached the caecum two hours after ispaghula ingestion and were chiefly recovered as oligosides – that is, in the fractions extracted by 80° ethanol; these fractions probably resulted from the slight gastric and intestinal degradation of ispaghula previously described in humans by Anderson *et al.*³⁸ As shown by the rise of caecal arabinose and xylose measured six hours after ispaghula ingestion (Table VII) most of the ispaghula only reached the caecum after four hours, and most of the caecal arabinose and xylose were present in the form of highly polymerised molecules as in the ingested ispaghula (Fig 3). This result was in keeping with the oro-caecal transit time assessed by the hydrogen breath test. Interpretation of these results need to assume that caecal fluid sampled was homogeneous, as only concentrations can be measured. Aspiration of the viscous caecal contents through a narrow tube proved to be more difficult than in our previous study using a lactulose meal.³⁹

Glucose was the main sugar in caecal contents during the placebo period, and was chiefly present in the insoluble fraction at all sampling times (data not shown). Glucose concentrations were higher two and four hours after ingestion of the meal with ispaghula than after placebo, and glucose was present in the oligoside fraction extracted by 80° ethanol. The origin of this increased delivery of glucose when ispaghula was consumed is not clear. It might result from overnight residues in the

terminal ileum, which moved on in response to the test meal or to the trapping of dietary oligosides containing glucose by the small fraction of ispaghula reaching the caecum at four hours in highly polymerised form. The amount of glucose delivered to the colon during the first four hours after the meal with ispaghula was small, however, and did not lead to increased excretion of hydrogen in breath. In addition, caecal glucose concentrations, expressed per g wet weight, were small, and a real increase in caecal contents was only assumed to occur four hours after meal ingestion with the delivery of ispaghula in its highly polymerised form.

P Marteau, MD, was awarded a grant by the Fonds de Recherche du Corps des Médecins des Hôpitaux de Paris. The authors thank M C Morin, A David, C Franchisseur, M Maurel, and M Rival for skillful technical assistance, and D Ehrlich and N Baranton for preparation of the manuscript.

- 1 Stephen AM, Cummings JH. Mechanism of action of dietary fibre in the human colon. *Nature* 1980; **284**: 283-4.
- 2 Edwards CA. The mechanisms of action of dietary fibre in promoting colonic propulsion. *Scand J Gastroenterol* 1987; **22** (suppl 129): 97-9.
- 3 Eastwood MA, Smith AN, Brydon WG, Pritchard J. Comparison of bran, ispaghula, and lactulose on colon function in diverticular disease. *Gut* 1978; **19**: 1144-7.
- 4 Kumar A, Kumar N, Vij JC, Sarin SK, Anand BS. Optimum dosage of ispaghula husk in patients with irritable bowel syndrome: correlation of symptom relief with whole gut transit time and stool weight. *Gut* 1987; **28**: 150-5.
- 5 Prior A, Whorwell PJ. Double blind study of ispaghula in irritable bowel syndrome. *Gut* 1987; **28**: 1510-3.
- 6 Rasmussen HS, Holtug K, Andersen JR, Krag E, Mortensen PB. The influence of ispaghula husk and lactulose on the in vivo and the in vitro production capacity of short-chain fatty acids in humans. *Scand J Gastroenterol* 1987; **22**: 406-10.
- 7 Tomlin J, Read NW. The relationship between bacterial degradation of viscous polysaccharides and stool output in human beings. *Br J Nutr* 1988; **60**: 467-75.
- 8 Stevens J, VanSoest PJ, Robertson JB, Levitsky DA. Comparison of the effects of psyllium and wheat bran on gastrointestinal transit time and stool characteristics. *J Am Diet Assoc* 1988; **88**: 323-6.
- 9 Nyman M, Asp NG. Bulk-laxatives: their dietary fibre composition, degradation, and fecal bulking capacity in the rat. *Scand J Gastroenterol* 1985; **20**: 887-95.
- 10 Tomlin J. Which fibre is best for the colon? *Scand J Gastroenterol* 1987; **22** (suppl 129): 100-4.
- 11 Edwards CA, Eastwood MA. Comparison of the effects of ispaghula and wheat bran on rat caecal and colonic fermentation. *Gut* 1992; **33**: 1229-33.
- 12 Edwards CA, Bowen J, Brydon WG, Eastwood MA. The effects of ispaghula on rat caecal fermentation and stool output. *Br J Nutr* 1992; **68**: 473-82.
- 13 Bourquin LD, Titgemeyer EC, Fahey GC, Garleb KA. Fermentation of dietary fibre by human colonic bacteria: disappearance of, short-chain fatty acid production from, and potential water-holding capacity of various substrates. *Scand J Gastroenterol* 1993; **28**: 249-55.
- 14 Prynne C, Southgate DAT. The effects of a supplement of dietary fibre on faecal excretion by human subjects. *Br J Nutr* 1979; **41**: 495-503.
- 15 Bond JH, Engel RR, Levitt MD. Factors influencing pulmonary methane excretion in man. *J Exp Med* 1971; **133**: 572-88.
- 16 Renaud S, Godsey F, Orchanian E, Baudier F. *Table de composition des aliments*. INSERM U 63 Bron, France et Département de nutrition de l'Université de Montreal, Canada. Asnières: Astra Calvé, 1979.
- 17 Englyst HN, Kingman SM, Cummings JH. Classification and measurement of nutritionally important starch fractions. *Eur J Clin Nutr* 1992; **46**: S33-50.
- 18 Cummings JH, Wiggins HS. Transit through the gut measured by analysis of a single stool. *Gut* 1976; **17**: 219-23.
- 19 Bouhnik Y, Flourié B, Marteau P, Corrèze JL, Franchisseur C, Maurel M, et al. Comparison of breath and rectal gas excreted in man [Abstract]. *Gastroenterology* 1991; **100**: 199.
- 20 Flourié B, Florent C, Jouany JP, Thivend P, Etachaud F, Rambaud JC. Colonic metabolism of wheat starch in health humans. Effects on fecal outputs and clinical symptoms. *Gastroenterology* 1986; **90**: 111-9.
- 21 Hydén S. A turbidimetric method for the determination of higher polyethylene glycols in biological materials. *Ann R Agric Coll Swed* 1955; **22**: 139-45.
- 22 Assous E, Dreux C, Girard M. Application nouvelle de la dialyse à la détermination de l'ammoniémie. *Ann Biol Clin* 1960; **18**: 319-30.
- 23 Stephen AM, Cummings JH. The microbial contribution to human faecal mass. *J Med Microbiol* 1980; **13**: 45-56.
- 24 Jouany JP. Volatile fatty acid and alcohol determination in digestive contents, silage juices, bacterial cultures and anaerobic fermentor contents. *Science des Aliments* 1982; **2**: 131-44.
- 25 Delort-Laval J, Mercier C. Evaluation de divers traitements technologiques des céréales. *Annales de Zootechnologie* 1976; **25**: 3-12.
- 26 Hoebler C, Barry JL, David A, Delort-Laval J. Rapid acid hydrolysis of plant cell wall polysaccharides and simplified quantitative determination of their neutral monosaccharides by gas-liquid chromatography. *J Agric Food Chem* 1989; **37**: 360-7.
- 27 Nyman M, Asp NG. Dietary fibre fermentation in the rat intestinal tract: effect of adaptation period, protein and fibre levels, and particle size. *Br J Nutr* 1985; **54**: 635-43.
- 28 Allen A. Structure of gastrointestinal mucus glycoproteins and the viscous and gel-forming properties of mucus. *Br Med Bull* 1979; **34**: 28-33.
- 29 Kennedy JF, Sandhu JS, Southgate DAT. Structural data for the carbohydrate of ispaghula husk. *Carbohydr Res* 1979; **75**: 265-74.
- 30 Salyers AA, Harris CJ, Wilkins TD. Breakdown of psyllium hydrocolloid by strains of *Bacteroides ovatus* from the human gastrointestinal tract. *Can J Microbiol* 1978; **24**: 336-8.
- 31 Hsu JC, Penner MH. Influence of cellulose structure on its digestibility in the rat. *J Nutr* 1989; **119**: 872-8.
- 32 Hallert C, Kaldma M, Petersson BG. Ispaghula husk may relieve gastrointestinal symptoms in ulcerative colitis in remission. *Scand J Gastroenterol* 1991; **26**: 747-50.
- 33 Wolever TMS, ter Wal P, Spadafora P, Robb P. Guar, but not psyllium, increases breath methane and serum acetate concentrations in human subjects. *Am J Clin Nutr* 1992; **55**: 719-22.
- 34 Squires PE, Rumsey RDE, Edwards CA, Read NW. Effect of short-chain fatty acids on contractile activity and fluid flow in rat colon in vitro. *Am J Physiol* 1992; **262**: G813-7.
- 35 Thorburn HA, Carter KB, Goldberg JA, Finlay IG. Does ispaghula husk stimulate the entire colon in diverticular disease? *Gut* 1992; **33**: 352-6.
- 36 MacFarlane GT, Cummings JH. The colonic flora, fermentation and large bowel digestive function. In: Phillips SF, Pemberton JH, Shorter RG, eds. *The large intestine. physiology, pathophysiology, and disease*. New York: Raven Press, 1991: 51-92.
- 37 Stephen AM, Cummings JH. Water-holding by dietary fibre in vitro and its relationship to faecal output in man. *Gut* 1979; **20**: 722-9.
- 38 Andersen JR, Bukhave K, Hojgaard L, Rasmussen HS, Hermansen N, Worning H, et al. Decomposition of wheat bran and ispaghula husk in the stomach and the small intestine of healthy men. *J Nutr* 1988; **118**: 326-31.
- 39 Florent C, Flourié B, Leblond A, Rautureau M, Bernier JJ, Rambaud JC. Influence of chronic lactulose ingestion on the colonic metabolism of lactulose in man (an in vivo study). *J Clin Invest* 1985; **75**: 608-13.