Digestive and metabolic effects of potato and maize fibres in human subjects

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The physiological effects of dietary fibres in humans are due to their physico-chemical properties. However, it is difficult to predict these effects simply by measuring certain characteristics *in vitro*. Studies in human subjects are still required to assess the effectiveness of new substrates. The aim of the present study in healthy human subjects was to evaluate the effects of two novel fibres, potato (PF) and maize (MF), on fasting and postprandial blood concentrations of carbohydrate and lipid metabolites as well as on stool ouput and transit time. The chemical composition, water-binding capacity (WBC) and fermentative properties of the fibres were also characterized in order to determine their possible involvement in digestive and metabolic effects. Stools, as well as breath and blood samples, were collected after consumption for 1 month of either a basal diet (control) or a basal diet supplemented with fibre (15 g/d). MF resisted fermentation better than PF and had lower digestibility. However, both fibres increased faecal output of dry matter, neutral sugars and water. There was an inverse relationship between stool weight and orofaecal transit time, although only MF significantly reduced transit time. Orocaecal transit was lengthened by PF, probably because of its high WBC. PF ingestion also decreased postprandial plasma levels of total and esterified cholesterol but had no effect on fasting concentrations. In contrast, MF lowered fasting cholesterolaemia and increased free:esterified cholesterol. These particular physiological and fermentative properties suggest that PF and MF would be suitable ingredients in a healthy diet.

Dietary fibre: Stool output: Transit time: Plasma lipids

The mechanisms by which a particular dietary fibre acts on human physiology are mainly related to its physico-chemical properties. For instance, solubility seems to be particularly involved in the metabolic effects of fibre. Soluble fibres can apparently reduce serum cholesterol concentration, whereas insoluble ones are usually ineffective (Glore *et al*. 1994). Other properties, such as viscosity and the ability to sequester bile salts, can also influence glucose and cholesterol metabolism (Wolever, 1995). The effects of dietary fibre on digestive functions also depend on several physico-chemical properties of substrates. It is now recognized that fibre can affect stool weight and transit time through two main mechanisms. First, it may act by increasing lumen volume. This mechanism depends on the amount of indigestible residue in the colon, the water-binding capacity (WBC) of the residue, the stimulation of microbial growth, and the production of gas (Cummings, 1986). The resulting increase in stool volume may cause colonic propulsion through excitation of enteric mechanoreceptors, thus decreasing transit time. Second, fibre may act directly on colonic motility either by mechanical stimulation of submucosal neural receptors (Cherbut & Ruckebusch, 1985) or by an effect of fermentation end-products, e.g. short-chain fatty acids (SCFA), on the contractile activity of the colon (Cherbut, 1995).

Even though our understanding of the mechanisms of dietary fibre action is improving, it is still difficult to predict the observable physiological effects of a new fibre preparation in man simply by measuring its physico-chemical and fermentative properties *in vitro.* Examination of some blood variables and gastrointestinal factors is required to assess the potential action of a novel substrate. The present study in healthy human subjects constitutes the first attempt to evaluate the effects of potato fibre (PF) on blood glucose, insulin, cholesterol and triacylglycerol concentrations and to assess faecal output and composition as well as transit time. The chemical composition, water-holding properties and fermentative pattern of this new fibre are also characterized. In addition, the effects of maize fibre (MF) are studied and compared with those of PF.

MATERIALS AND METHODS

Characterization of fibres

Two different dietary fibres were prepared from maize cob and potato pulp (Roquette, Lestrem, France) by mechanical processes (grinding, air-classification, sieving and grinding) and reduced to the same average particle size (0·08 (SE 0·02) mm). The chemical composition of the fibre preparations is given in Table 1. Native substrates were prepared by amylase (*EC* 3.2.1.1; 3.2.1.3) digestion to remove starch (Prosky *et al.* 1988). The neutral and acidic sugars of the fibres were determined respectively by GLC (Hoebler *et al.* 1989) and colorimetry (Thibault, 1979) after the fibres were hydrolyzed in H_2SO_4. Each value is given as a proportion of dry matter determined at 103°.

Swelling was measured by the bed-volume technique (Kuniak & Marchessault, 1972), with slight modification. Dry fibre (100 mg) was weighed into a glass cylinder and left overnight at 25° in 0·15 M-NaCl (pH 6–6·5). Results are expressed as ml swollen sample/g dry initial sample. WBC was determined by centrifugation (McConnell *et al.* 1974). Samples (100 mg) were stirred overnight at 25° in 0·15 M-NaCl and centrifuged for 1 h at 14 000 *g* at 25°. The supernatant fractions were then discarded and the residues left for 1 h on G2 sintered glass (porosity 41–100 μm). The pellets were weighed, dried and weighed again. Results are expressed as g water/g dry residue.

In vitro *fermentation*

Fibres were incubated at 40° in a N_2 atmosphere with a human faecal inoculum provided by three healthy subjects accustomed to eating an unspecified Western diet, as previously detailed (Salvador *et al.* 1993). Three experiments were performed. For each experiment, two batches of each material were prepared, as well as two control batches containing only the faecal inoculum. Fermentation was stopped after 6, 12 and 24 h incubation. Medium pH was immediately measured in batches removed for analysis. Bottle contents were then

Table 1. *Chemical composition of fibre preparations (g/kg dry weight)*

	Maize	Potato
Starch	173	237
Protein (N × 6·25)	97·5	30
Fat	23	13
Ash	5	18·5
Total fibre	701·5	701·5

centrifuged and the supernatant fraction analysed for SCFA by GLC (Jouany, 1982). Pellets were analysed for cell-wall sugars as described previously. The fermentability of fibre sugars, i.e. the rate of sugar disappearance, was calculated as follows (Salvador *et al.* 1993):

$$\text{fermentability (\%)} = 100 \times (\text{amount of sugars digested/initial amount of sugars}).$$

Subjects

Eighteen healthy volunteers (ten women and eight men, aged 24–48 years) gave their informed written consent to the study protocol which was approved by the local Ethics Committee. None had a history of gastrointestinal disease, use of laxatives or recent treatment with antibiotics.

Experimental design

The subjects were distributed into two groups, one testing MF and the other PF. The study comprised two 1-month periods with an interval of 21 d. In randomized, blind, cross-over fashion the subjects of each group were given 22 g (DM basis) of either maize or potato substrate twice daily (providing about 15 g fibre/d) during one period, and no fibre supplement (control period during which fibre supplement was replaced by sucrose, 15 g/d) during the other period. The fibres were packaged in tubes which also contained 5 g of the faecal recovery marker, PEG 4000. The contents of the tubes were mixed with food and ingested during breakfast and dinner.

During each period the subjects consumed a basal diet consisting of conventional low-fibre foods and providing 6 g dietary fibre and 1–2 g resistant starch/d, as determined from food tables (Renaud *et al.* 1979; Englyst *et al.* 1992). Great care was taken to ensure that the dietary constituents were the same throughout the two periods. A weekly menu was established and rotated each week. The ingredients for breakfast, lunch and dinner were given to the subjects to be prepared and eaten at home. At the end of each period (day 27), subjects were admitted to the clinical centre and given standardized test meals.

During both periods, after 20 d equilibration, stools were collected from day 21 to day 27 and immediately frozen at $-20°$. On days 22, 23 and 24 the subjects ingested twenty radiopaque pellets of different shapes with their breakfast to allow measurement of mean orofaecal transit time according to the method of Cummings & Wiggins (1976). On day 27 a test meal, composed of egg (1), yoghurt (1), cheese (40 g), vegetable margarine (20 g), white bread (60 g), sucrose (10 g), orange juice (100 ml) and tea or coffee, was given with either 7·5 g fibre or placebo (7·5 g cellulose, Arbocel type B00, Durieux, France). In addition, subjects ingested 10 g lactulose (Duphalac®, Duphar, Villeurbanne, France). End-expiratory breath samples were taken before the meal and every 30 min thereafter to measure orocaecal transit time over 6 h. Blood samples were taken 15 min before and immediately before the meal was ingested, and at 30 min intervals thereafter for 6 h. After test-meal ingestion no food was permitted until the end of exhaled breath gas and blood collection, and subjects had to refrain from smoking.

Analytical methods and calculation

Each stool was weighed and a portion was lyophilized to measure the dry weight. The 5 d faecal collections obtained from each subject throughout each period were then pooled. Neutral sugars were measured in each pool as described above. PEG 4000 was measured by the turbidimetric method (Hyden, 1955).

Concentration of breath H_2 was measured using a Quintron model DP instrument (Quintron Microlyser, MI, USA). Orocaecal transit time was defined as the elapsed time between the beginning of the meal and the sustained increase in breath H_2 (> 15 ppm).

Plasma was separated from whole blood by centrifugation ($10°$, 10 min, 910 g). Plasma glucose was measured using the glucose oxidase (*EC* 1.1.3.4) method (Beckman Analyzer, Fullerton, CA, USA), and plasma insulin was determined by radioimmunoassay (Oris, Gif sur Yvette, France). Triacylglycerols were determined by an enzymic procedure (Buccolo & David, 1973) using commercial kits (BioMerieux, Marcy l'Etoile, France). Total and free cholesterol were assayed by the cholesterol oxidase (*EC* 1.1.3.6) method (Siedel *et al.* 1983) using BioMerieux kits. Phospholipids were assayed by an enzymic procedure (Takayama *et al.* 1977) using BioMerieux kits. Total serum apoprotein A1 (Sievet-Desrumeaux *et al.* 1980) and apoprotein B (Sievet-Desrumeaux *et al.* 1979) were assayed by laser-nephelometry (Behring Werke AG, Marburg, Germany).

Statistical analysis of data

Data are expressed as mean and standard error of the mean (SE). Comparisons between the two treatments were made using Wilcoxon's non-parametric test for paired data; $P < 0.05$ was considered as the significance level. A two-way ANOVA was performed to test the interaction between treatment (control *v.* fibre) and period (order of distribution). The P value for treatment × period was above 0.3 for all the considered variables (stool weights, transit time, blood glucose, insulin and lipids) indicating that the wash-out period was effective. All calculations were performed using StatView^{TM} SE + Graphics (Abacus Concepts, Inc., CA, USA) run on a Macintosh computer.

RESULTS

Fibre characteristics

The chemical, physico-chemical and fermentative properties of PF and MF were very different. PF contained 140 g soluble fibre/kg and was composed of both neutral (glucose and galactose) and acidic sugars (Table 2), whereas MF was completely insoluble and composed mainly of neutral sugars (arabinose, xylose and glucose). The difference between the total fibre content and soluble plus insoluble fibres of the preparation was about 20 g/kg, which represents the normal variability between the two methods (Prosky *et al.* 1988). A totally different method (non-gravimetric, hydrolysis then chromatography or colorimetry) was used to analyse the sugar content of the fibre preparation. This may explain the slight discrepancy between the sum of the neutral and acidic sugars and the total fibre content (Tables 1 and 2). Moreover, it is likely that the analysis of sugars did not take into account all the components of the fibre preparation.

In vitro fermentation of the fibres led to extensive disappearance of PF and low degradation of MF (Table 3). The resulting SCFA profiles also differed, acetate–propionate–butyrate being 66:23:11 and 52:31:17 respectively for PF and MF (Table 3). Although native PF had higher swelling and WBC than MF, it lost this property after *in vitro* fermentation (Fig. 1).

Faecal outputs

The percentages of ingested PEG 4000 recovered in stools were 100 (SE 9), 90 (SE 1) and 96 (SE 8) for the control, PF and MF periods respectively. The number of stools was

Table 2. *Chemical composition of maize and potato fibres (g/kg dry weight), determined after starch was removed from the native fibre preparations by amylase digestion*

	Maize	Potato
Fibre content		
Insoluble	684	588
Soluble	0	96
Neutral sugars		
Rhamnose	4	8
Arabinose	144	32
Xylose	229	14
Mannose	11	8
Galactose	37	145
Glucose	202	206
Acidic sugars	31	197

*For details of procedures, see p. 34.

Table 3. *Percentage disappearance (fermentability) and concentration of individual short-chain fatty acids (SCFA) during* in vitro *incubation of maize and potato fibres* with a human faecal inoculum†*

(Mean values with their standard errors of three measurements)

| | | Fermentability (%) | | Concentration of SCFA (mmol/l) | | | | | |
| | | | | Acetate | | Propionate | | Butyrate | |
Fibre	Incubation time (h)	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Maize	6	12·0	0·7	15·9	5·3	6·0	0·6	2·7	1·1
	12	19·8	0·6	25·5	7·3	11·2	0·4	6·1	0·5
	24	31·0	1·8	31·6	7·8	18·8	2·2	10·6	2·4
Potato	6	43·5	2·5	50·1	4·8	15·4	3·9	5·6	0·5
	12	65·8	0·5	79·5	0·4	25·6	3·9	13·2	0·6
	24	82·3	0·2	87·4	3·2	30·4	5·6	15·1	4·0

*Before incubation, maize and potato fibre materials were prepared by amylase digestion to remove starch.
†For details of procedures, see pp. 34–35.

slightly increased by ingestion of the fibre preparations (Table 4). Faecal wet and dry weights, as well as faecal water, were significantly greater during the PF and MF periods (Table 4). Accordingly, faecal outputs of neutral sugars increased during MF and PF ingestion (Table 5). However, PF fibre was more degradable than MF throughout the gut: faecal digestibilities of the total neutral sugars of PF and MF, as calculated after correction for the excretion of these sugars during the control period, were respectively 67·9 (SE 12·5; range 19–96)% and 48·4 (SE 4; range 33–66)%.

Transit times

The increase in breath H_2 excretion in each subject after both test meals enabled us to determine orocaecal transit time. Mean orocaecal transit time was not significantly affected by MF consumption but was increased by PF ($P = 0.045$, Fig. 2). Mean orofaecal transit
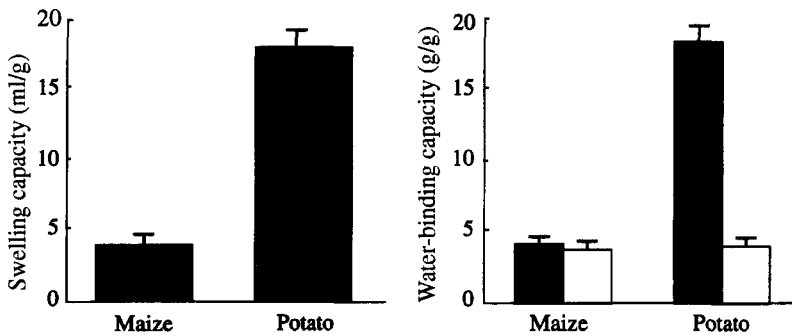
Fig. 1. Swelling and water-binding capacities of maize and potato fibres before (■, ▨) and after 24 h fermentation with a human faecal inoculum *in vitro* (□). For details of procedures see pp. 34–35. Values are means with their standard errors of three measurements.

Table 4. *Number of stools and faecal weights recorded in nine subjects for 7 d after 20 d of equilibration to either the control diet (no fibre supplement) or the fibre diet (15 g maize or potato fibre/d)**

(Mean values with their standard errors; ranges are given in parentheses)

		Number of stools/d		Faecal wet weight (g/d)		Faecal dry weight (g/d)		Faecal water (g/d)		Faecal water content (g/kg)	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Maize	Control	0·85	0·03	72·7	7·4	19·3	1·9	53·2	5·5	728	11
	(range)	(0·7–1·0)		(23–93)		(7–25)		(16–70)		(670–770)	
	Fibre	0·96	0·06	108·4	5·4	32·4	1·5	76·9	4·2	708	7
	(range)	(0·7–1·2)		(89–142)		(28–39)		(61–102)		(680–750)	
P value		0·118		0·007		0·007		0·007		0·086	
Potato	Control	0·9	0·06	79·3	9·7	18·7	2·4	60·6	7·7	760	14
	(range)	(0·7–1·1)		(44–115)		(11–31)		(33–92)		(710–820)	
	Fibre	1·1	0·14	115·7	9·1	28·3	2·3	86·9	7·5	748	13
	(range)	(0·6–1·5)		(59–141)		(17–38)		(42–107)		(700–790)	
P value		0·088		0·011		0·011		0·011		0·262	

*The experimental periods (control and fibre) were separated by an interval of 21 d. For details of diets, see p. 35.

time was not changed during the PF period but was significantly reduced during the MF period ($P = 0\cdot011$, Fig. 2). By combining the data for each subject during all periods (n 36) we calculated a significant inverse relationship between orofaecal transit time and faecal wet weight ($y = -0\cdot42x + 114$, r 0·530, $P = 0\cdot001$).

Blood variables

Fasting plasma concentrations of glucose, insulin and triacylglycerols were not affected by consumption for 1 month of either MF or PF (Table 6). However, MF ingestion

Table 5. *Faecal outputs of neutral sugars (g/d) recorded in nine subjects after 20 d of equilibration to either the control diet (no fibre supplement) or the fibre diet (15 g maize or potato fibre/d)**

(Mean values with their standard errors; ranges are given in parentheses)

| | Maize | | | | | Potato | | | | |
| | Control | | Fibre | | | Control | | Fibre | | |
Sugars	Mean	SE	Mean	SE	P value	Mean	SE	Mean	SE	P value
Rhamnose	0·13	0·01	0·28	0·09	0·015	0·14	0·03	0·20	0·03	0·017
(range)	(0·05–0·19)		(0·14–1·0)			(0·06–0·29)		(0·10–0·38)		
Arabinose	0·22	0·08	1·61	0·13	0·007	0·17	0·03	0·30	0·06	0·012
(range)	(0–0·82)		(1·11–2·40)			(0·05–0·32)		(0·09–0·65)		
Xylose	0·25	0·07	2·51	0·20	0·007	0·25	0·06	0·47	0·11	0·017
(range)	(0–0·78)		(1·60–3·53)			(0·04–0·52)		(0·15–0·92)		
Mannose	0·11	0·03	0·17	0·02	0·109	0·14	0·03	0·34	0·14	0·093
(range)	(0–0·028)		(0·06–0·26)			(0·06–0·27)		(0·06–1·23)		
Galactose	0·25	0·02	0·75	0·05	0·007	0·27	0·04	0·50	0·11	0·025
(range)	(0·11–0·34)		(0·54–1·04)			(0·13–0·40)		(0·15–1·02)		
Glucose	0·88	0·17	3·17	0·26	0·007	1·53	0·61	3·56	1·39	0·012
(range)	(0·25–1·81)		(2·02–4·61)			(0·37–4·95)		(0·72–10·22)		
Total	1·84	0·34	8·54	0·62	0·007	2·49	0·77	5·38	1·77	0·012
(range)	(0·64–4·10)		(5·90–11·97)			(0·73–6·50)		(1·29–13·38)		

*The experimental periods (control and fibre) were separated by an interval of 21 d. For details of diets, see p. 35.

significantly reduced the basal concentrations of total and esterified cholesterol ($P = 0.045$ and 0·038 respectively), and increased the free:esterified cholesterol ratio ($P = 0.065$). PF had no significant effect (Table 6).

Postprandial plasma levels of glucose, insulin and triacylglycerols were not changed by MF and PF (Fig. 3). Although MF ingestion did not modify the postprandial areas under the curve (AUC) for total, esterified and free cholesterol, PF consumption caused a significant reduction in AUC for total and esterified cholesterol ($P = 0.038$ and 0·019 respectively, Table 7).

As sucrose (15 g/d) was used as a placebo, we cannot exclude the possibility that it may have affected plasma glucose, insulin and lipid concentrations. However, 15 g sucrose/d provides only 2·7 % energy on the basis of a 10·46 MJ daily intake. Thus, it is unlikely that this low consumption of sucrose as a percentage of energy would have caused major changes in blood variables. In addition, in normal men and women, 50–107 g sucrose/d, for 7–14 d, did not affect fasting glucose and insulin levels in the serum (Bossetti *et al.* 1984) and significant increases in plasma triacylglycerols and cholesterol concentrations were reported with much higher daily sucrose intakes (Macdonald, 1964; Bagley *et al.* 1976).
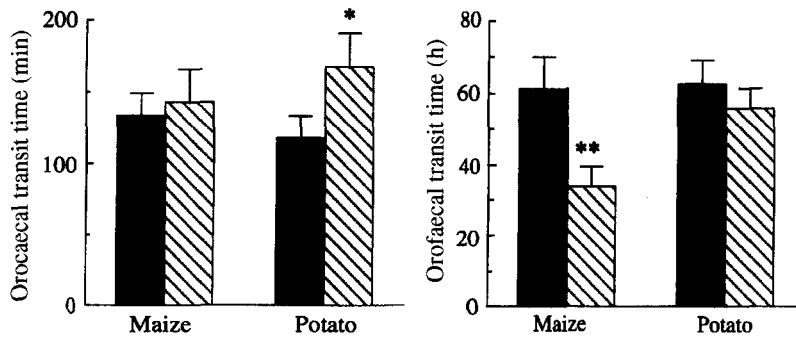
Fig. 2. Orocaecal and orofaecal transit times measured in subjects after 20–27 d of equilibration to either the control diet (no fibre supplement, ■) or the fibre diet (15 g maize or potato fibre/d, ▨). Values are means with their standard errors for nine subjects. Mean values were significantly different from control, $*P < 0.05$, $**P < 0.01$.

Table 6. *Faecal plasma concentrations of glucose, insulin, lipids and apolipoproteins measured in nine subjects after 4 weeks ingestion of either the control diet (no fibre supplement) or the fibre diet (15 g maize or potato fibre/d)**

(Mean values with their standard errors; ranges are given in parentheses)

| | Maize | | | | | Potato | | | | |
| | Control | | Fibre | | | Control | | Fibre | | |
	Mean	SE	Mean	SE	*P* value	Mean	SE	Mean	SE	*P* value
Glucose (mmol/l)	4·7	1·2	4·6	1·2	0·767	4·0	1·4	3·9	1·2	0·523
(range)	(3·7–5·5)		(3·5–5·4)			(3·1–5·2)		(3·2–5·0)		
Insulin (mU/l)	12·1	1·0	11·2	1·3	0·514	9·7	0·9	8·2	1·1	0·310
(range)	(6·4–17·5)		(5·0–17·5)			(6·9–13·6)		(0–11·0)		
Triacylglycerols (mmol/l)	0·9	0·1	0·9	0·08	0·767	0·9	0·04	0·9	0·1	0·515
(range)	(0·5–1·7)		(0·5–1·3)			(0·7–1·4)		(0·4–1·7)		
Cholesterol										
total (mmol/l)	5·7	0·2	5·0	0·3	0·045	5·8	0·5	5·1	0·3	0·138
(range)	(4·2–6·6)		(3·6–6·2)			(4·0–7·0)		(3·8–6·5)		
esterified (mmol/l)	4·6	0·4	3·6	0·3	0·038	4·4	0·3	3·9	0·3	0·260
(range)	(2·6–7·4)		(2·4–4·7)			(2·9–5·5)		(2·8–5·2)		
free (mmol/l)	1·1	0·1	1·4	0·07	0·314	1·3	0·09	1·2	0·08	0·085
(range)	(0·3–1·6)		(1·0–1·7)			(1·1–1·8)		(0·9–1·6)		
free:esterified	0·27	0·05	0·38	0·02	0·065	0·31	0·01	0·32	0·02	0·138
(range)	(0·11–0·29)		(0·29–0·4)			(0·29–0·33)		(0·28–0·4)		
Phospholipids (mmol/l)	4·1	0·2	2·8	0·6	0·109	2·5	0·3	2·0	0·1	0·085
(range)	(2·7–5·1)		(1·7–7·0)			(1·7–4·4)		(1·4–2·5)		
Apolipoprotein A1 (mg/l)	1·8	0·18	1·41	0·11	0·093	1·30	0·68	1·26	0·68	0·593
(range)	(1·3–2·76)		(0·98–2·0)			(1·0–1·58)		(0·94–1·5)		
Apolipoprotein B (mg/l)	0·93	0·73	0·9	0·6	0·674	0·90	0·54	0·87	0·39	0·767
(range)	(0·62–1·28)		(0·58–1·17)			(0·75–1·24)		(0·72–1·08)		

*The experimental periods (control and fibre) were separated by an interval of 21 d. For details of diets, see p. 35.
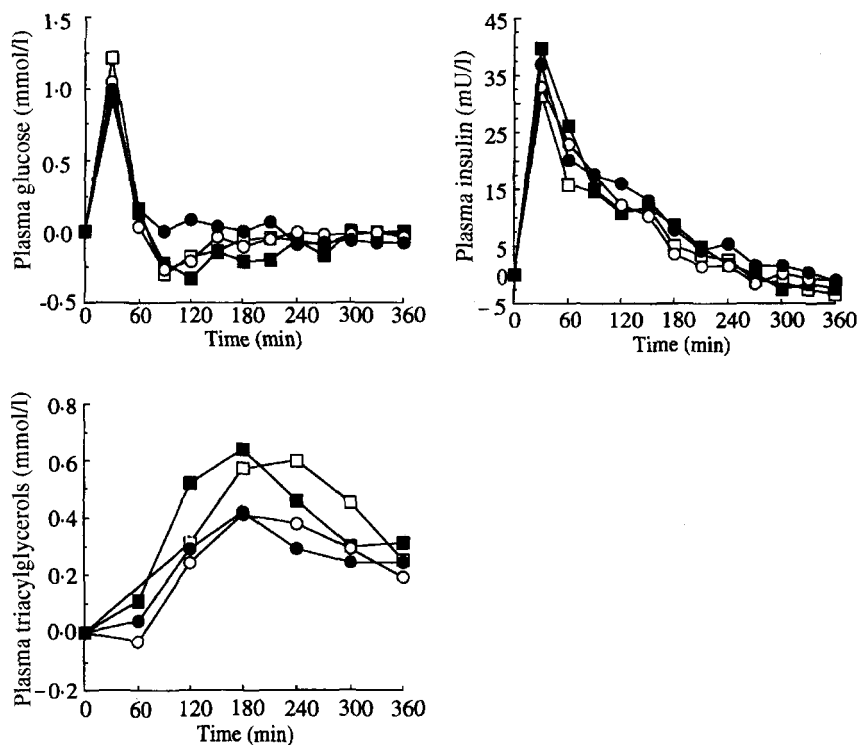
Fig. 3. Incremental plasma glucose, insulin and triacylglycerol concentrations measured after ingestion of a test meal containing either placebo (7·5 g cellulose, □ and ○) or maize (■) or potato (●) fibre (7·5 g). The response to the test meal was determined after 4 weeks of equilibration to either the control diet (no fibre supplement) or the fibre diet (15 g maize or potato fibre/d). Values are means for nine subjects. Plasma concentrations were not significantly different after placebo and fibres.

DISCUSSION

PF, a novel substrate rich in soluble fibre, and MF proved to be effective stool bulkers in humans. However, only MF, which was poorly degraded throughout the gut, shortened orofaecal transit time. In addition to providing a bulking effect, PF modified orocaecal transit time and decreased the postprandial plasma concentrations of total and esterified cholesterol. Although MF had no effect on the postprandial level of measured metabolic variables, its consumption for 1 month caused a reduction in the fasting concentrations of cholesterol and phospholipids.

Several mechanisms may be involved in the increase in stool weight caused by MF and PF. Dietary fibre actually affects stool output by increasing unfermented residues, faecal water and microbial mass (Cummings, 1986; Edwards, 1995). In the present study, consumption of MF and PF increased faecal output of neutral sugars, accounting in part for the increase in the dry weight of stools. MF appeared to be more resistant to degradation than PF. Indeed, both *in vitro* and *in vivo*, the percentage disappearance of MF was significantly lower than that of PF. This behaviour could be due to the chemical structure of MF as well as to its low WBC. Insoluble fibres are known to be less degraded by colonic flora than soluble ones (Mortensen & Norgaard-Andersen, 1993). Furthermore, MF is composed mainly of xylose, arabinose and cellulosic glucose. In this respect, MF is close to

Table 7. *Area under the 0–6 h curves of postprandial incremental concentrations of plasma lipids measured in nine subjects who ingested a test meal together with either 7·5 g cellulosed (placebo) or 7·5 g maize or potato fibre**

(Mean values with their standard errors; ranges are given in parentheses)

	Maize					Potato				
	Control		Fibre			Control		Fibre		
	Mean	SE	Mean	SE	*P* value	Mean	SE	Mean	SE	*P* value
Triacylglycerols (mmol.h/l)	2·65	0·27	2·70	0·77	0·765	1·89	0·39	1·88	0·22	0·767
(range)	(2·05, 3·21)		(2·10, 3·49)			(1·54, 2·86)		(1·50, 2·27)		
Total cholesterol (mmol.h/l)	−5·97	1·89	−2·96	5·09	0·374	−0·42	1·04	−2·32	1·12	0·038
(range)	(−7·09, −2·84)		(−8·87, 1·53)			(−2·73, 0·96)		(−3·95, −1·34)		
Esterified cholesterol (mmol.h/l)	−5·46	0·80	−2·34	1·05	0·213	0·85	1·22	−2·25	1·84	0·019
(range)	(−6·72, −3·47)		(−5·19, −0·68)			(−1·23, 2·09)		(−3·95, 0·57)		
Free cholesterol (mmol.h/l)	−0·50	0·80	−0·71	0·24	0·314	−3·81	2·84	0·03	0·34	0·953
(range)	(−2·14, 1·39)		(−1·65, 0·83)			(−4·23, −0·58)		(−0·89, 0·42)		
Phospholipids (mmol.h/l)	5·24	2·63	1·93	2·81	0·441	−0·78	0·69	−0·68	0·46	0·374
(range)	(2·45, 7·11)		(0·98, 5·07)			(−2·38, 0·95)		(−1·87, 0·48)		

*The postprandial response to the test meal was determined after 4 weeks of equilibration to either the control diet (no fibre supplement) or to the fibre diet (15 g maize or potato fibre/d). For details of procedures, see pp. 35–36.

wheat-bran fibre which is composed largely of arabinoxylans and cellulose. Numerous studies have shown that wheat bran is poorly hydrolysed by colonic bacteria, in part because the highly branched structure of arabinoxylans limits accessibility to bacteria (Brillouet & Mercier, 1981; Stevens & Selvendran, 1988). Conversely, PF is rich in extensively fermented uronic acids (Van Soest *et al.* 1983; Stevens *et al.* 1988; Salvador *et al.* 1993) and galactose, a readily degraded sugar (Guillon *et al.* 1995). In addition, PF had a high WBC, which should make it more metabolizable since a positive relationship has been found between WBC and fibre fermentability (McBurney *et al.* 1985; Auffret *et al.* 1993).

About 6 g/d of the stool dry weight cannot be accounted for by unfermented neutral sugars. We measured neither N nor lignin content in the stools, nevertheless we may hypothesize that this extra faecal DM could come from endogenous materials (mucins, sloughed epithelial cells, etc.) and bacteria, and possibly from other undegraded fibre components, such as proteins or polyphenols.

The two fibre preparations also increased faecal water output, which may have been due to several causes. First, the increase could have been related to excreted residues. Although native PF had a high WBC, it lost this property after being degraded by colonic flora. Its WBC (less than 5 g/g) was then quite similar to that of MF. Although this WBC can account for all the extra faecal water with MF, it cannot explain the 30 % increase in water output induced by PF. Indeed, about 15 g stool water/d was not bound to the unfermented PF. This extra water might have come from faecal microbes since bacteria contain about 80 % water (Macfarlane & Cummings, 1991). As PF is extensively metabolized by colonic flora, its ingestion may favour microbial growth, thus increasing the bacterial mass in stools. Finally, the excreted water could have been free water not absorbed by the colon because of a short transit time or changes in colonic motility. It has been shown in dogs (Cherbut & Ruckebusch, 1985) that ingestion of plastic particles

inhibits the colonic contractions responsible for the mixing and retention of contents and thus increases stool water output.

As previously reported by Spiller (1986), stool weight was inversely related to orofaecal transit time. However, only MF had a significant effect on total transit time. It is recognized that poorly fermented fibres can reduce colonic retention time through a physical mechanism (Bardon & Fioramonti, 1983; Cherbut et al. 1991). This effect is due either to an increase in the volume of digesta, inducing distension of the colon which stimulates propulsive motility (Narducci et al. 1987), or to excitation of submucosal mechanoreceptors (Grider & Jin, 1994) by the edges of fibre particles. Although PF did not alter orofaecal transit time, it lengthened orocaecal transit, an effect possibly related to its high swelling and WBC. It has already been observed that fibres exhibiting a high WBC, such as those of peas or sugarbeet, slow the rate of orocaecal transit (Shinnick et al. 1989; Cherbut et al. 1991). Furthermore, sugarbeet and ispaghula, which have WBC similar to that of PF, modify the motility of the human small intestine (Cherbut et al. 1994).

The mechanisms by which dietary fibre influences cholesterol metabolism are still incompletely elucidated. Nevertheless, there is evidence that fibre may affect cholesterolaemia in at least five ways: altered absorption of fat and cholesterol (Borel et al. 1989), modified postprandial lipaemia and lipoproteins (Cara et al. 1992; Dubois et al. 1993), reduced insulin stimulus to cholesterol synthesis (Wolever, 1995), increased faecal bile acid excretion (Anderson et al. 1990; Jenkins et al. 1993), and systemic effects of the SCFA produced during colonic fibre fermentation (Anderson et al. 1991). The different metabolic effects observed after either PF or MF supplementation suggest that these fibre preparations are not governed by the same mechanisms. PF had no influence on fasting plasma lipid variables after 4 weeks intake but reduced postprandial plasma cholesterol concentration. It has already been reported that some fractions rich in insoluble fibres, such as concentrated wheat bran (Cara et al. 1992) or pea fibre (Dubois et al. 1993), can lower total and esterified cholesterol in plasma postprandially. This effect might be related to an alteration in lipid digestion and absorption in the upper gut (Borel et al. 1989). Actually, PF, which has notable swelling and water-holding capacities, may have modified the physico-chemistry of the intestinal contents, thereby impeding the mixing of food with digestive secretions as well as the convection of nutrients toward the absorptive area. The increase in orocaecal transit time induced by PF may account for this mechanism. Moreover, as previously suggested (Cara et al. 1992; Morgan et al. 1993), a postprandial decrease in plasma cholesterol might originate from an increased demand from the liver for de novo bile salt synthesis during the digestive period.

In contrast to PF, MF did not alter postprandial cholesterolaemia but elicited significant reductions in the total and esterified cholesterol of fasting plasma after 1 month of ingestion. Chronic consumption of MF decreased plasma total cholesterol ($-12 \cdot 2 \%$) as well as the free:esterified value. Comparable changes have been reported with soluble fibres such as guar gum, psyllium or oat bran (Jenkins et al. 1979; Anderson et al. 1984; Wolever et al. 1994). However, it is quite unusual for an insoluble fibre to produce such an effect (Krestin et al. 1990; Lampe et al. 1991). It is unlikely that SCFA, especially propionate (Stephen, 1994), could be involved in this effect. Low fibre fermentability was measured in vitro and in vivo, and the production of propionate was relatively low. Concerning the bile acid mechanism, MF may bind bile acids in the intestine and increase their excretion in stools, as has been observed for other cereal fibres (Story & Kritchevsky, 1976). This effect interrupts enterohepatic circulation, increases hepatic conversion of cholesterol into bile acids and results in lowered blood LDL and total cholesterol (Wolever, 1995). However, we did not measure either the in vitro bile acid-binding capacity of the

fibre or the faecal bile acid output. The fact that fasting plasma phospholipids and apolipoprotein (apo) A1 decreased simultaneously (-31.7 and -21.9% respectively), but apo B did not, suggests that a drop in HDL particles was the likely cause of the cholesterol changes observed. Further dedicated studies will be necessary for a fuller understanding of the mechanisms by which sources of insoluble fibres such as MF or wheat bran (Kashtan *et al.* 1992) can alter cholesterol metabolism.

Thus, daily consumption of PF and MF, even in relatively small quantities (15 g/d, representing about half of the recommended intake of dietary fibre), produced changes in digestive and metabolic physiology which may be considered beneficial for consumer health. Both fibres increased stool output, and MF also shortened orofaecal transit time. Moreover, both were fermented in the colon and produced SCFA. It has been suggested that all these effects can potentially reduce the risk of colon cancer (Gurr & Asp, 1994). In addition to their digestive action, PF and MF influenced cholesterol metabolism by reducing postprandial or fasting cholesterolaemia respectively. Furthermore, MF was able to decrease total and esterified cholesterol, an effect which could play a role in reducing the risk of coronary heart disease.

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