The digestion of pectin in the human gut and its effect on calcium absorption and large bowel function

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1. The effect of dietary fibre digestion in the human gut on its ability to alter bowel habit and impair mineral absorption has been investigated using the technique of metabolic balance.

2. Five healthy male students were studied for 9 weeks under controlled dietary conditions and during the last 6 weeks they took 36 g pectin/d. Bowel habit, transit through the gut, faecal fibre excretion, calcium balance and faecal composition were measured.

3. During the control period only 15 % of the dietary fibre ingested was excreted in the stools and when pectin was added to the diet there was no increase in fibre excretion. Stool frequency and mean transit time were unchanged by pectin but stool wet weight increased by 33 % and faecal excretion increased (%) for fatty acids 80, nitrogen 47, total dry matter 28 and bile acids 35. Ca balance remained unchanged.

4. It may be concluded from these results that dietary fibre is largely metabolized in the human gut and dietary pectin completely so. This could explain its lack of effect on bowel habit and Ca balance. Other changes in the faeces may be related to an increase in bacterial mass.

Pectin, the generic term for a family of galacturonic acid polymers which occur naturally in all plant cell walls, is one of several components of dietary fibre. Physiological studies in man have shown it to have pronounced hypocholesterolaemic properties (Keys *et al.* 1961; Palmer & Dixon, 1966; Jenkins *et al.* 1975; Kay & Truswell, 1977) but little effect on bowel habit (Durrington *et al.* 1976; Kay & Truswell, 1977). Along with other gel-forming polysaccharides it is able to modify the blood glucose and insulin response to carbohydrate containing meals (Jenkins, Leeds *et al.* 1977). It is potentially therefore a valuable component of the diet. Therapeutically pectin has been used to prevent the dumping syndrome (Jenkins, Gassul *et al.* 1977), a syndrome associated with abnormal carbohydrate digestion and absorption seen in patients after gastric surgery, whilst along with similar types of fibre it may be useful in the treatment of diabetes (Jenkins, Wolever *et al.* 1977). However pectin has a high uronic acid content and is known to complex readily with calcium and other cations (Rees, 1975; Tanaka & Skoryna, 1970). It has also been shown (James *et al.* 1978) that dietary fibre binds Ca ions in direct proportion to its uronic acid content. The potential the absorption of minerals human diet might therefore be restricted by its ability to impair value of pectin in the and other nutrients.

In order to investigate this and to determine the reasons why pectin should have so little effect on bowel habit when compared with other types of dietary fibre, we have fed 36 g pectin/d under controlled dietary conditions to a group of five medical students. Only a small change in bowel habit was observed and whilst faecal fat and nitrogen excretion

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increased, Ca balance remained normal. Pectin could not be recovered from the faeces suggesting that it is totally metabolized in the gut.

SUBJECTS AND METHODS

Subjects and protocol

Five healthy male medical students aged 21–24 years were studied for 10 weeks. For 9 weeks they ate a controlled diet to which was added in the final 6 weeks (control period weeks 1–3; pectin period weeks 4–9) 36 g pectin/d (high methoxy N.F. Pure; Bulmers Ltd, Hereford). In week 10 an *ad lib.* diet was taken. The subjects lived in a hostel in the grounds of the hospital and within the constraints imposed by adherence to the protocol, were expected to continue their normal life and activities throughout the study. During week 2 of the control period subject Y had a mild attack of influenza lasting 2 d during which time he stayed in bed for 24 h. This occurred during the first 4 d urine collection of the study and led to a pronounced increase in urine Ca excretion. Results from this subject for week 2 have therefore been excluded. From day 3–7 of week 4 subject D inadvertently took a course of eleven 250 mg tablets of erythromycin prescribed by his family doctor for an infected scratch on the leg acquired playing tennis. The medication had no apparent effect on the subject except to expedite healing of the abrasion. Nevertheless all values from this subject from week 4 have been excluded and none of the values on polysaccharide digestion were included (Table 5).

The general protocol for these metabolic-diet studies was approved by the ethical committee of the hospital and the sub-dean of the medical school.

Diets

All the food for the subjects was prepared in the metabolic kitchen. Three 1 d menus of similar composition were designed and fed in rotation during weeks 1–9, and only deionized water with tea and coffee was allowed. Table 1 shows the individual day's menus. Two complete samples of each day's food were collected and stored at −20° for analysis. Pectin was analysed separately.

The over-all composition of the diet (Table 2) was obtained from food tables (Paul & Southgate, 1978) whilst dietary fibre and pectin (Table 3) were analysed by the method of Southgate (1969). Pectin was taken in divided doses as a powder mixed into either the food or drink of each meal as the subjects preferred. It was introduced gradually into the diet over 3–4 d.

Faeces and urine collections

Throughout the 10 weeks the subjects collected their stools. Each was collected separately into a plastic bag fixed within a toilet bowl. The bag was then sealed, labelled, cooled to −20°, weighed and stored. At the end of the study faeces were collected into either 3, 4 or 7 d pools, allowed to thaw and homogenized whilst still cold, sufficient distilled water being added to ensure good mixing of the homogenate.

Collections of urine (24 h) were made into 1 l bottles containing 25 ml 2 M-hydrochloric acid. Urine was collected for four consecutive days at the end of control weeks 2 and 3, during the first 4 d of the pectin diet (week 4) and the last 4 d of weeks 5, 7 and 9. Urine was frozen after collection, subsequently thawed, each day's collection thoroughly mixed, the volume recorded and 6 × 20 ml portions stored for analysis.

Table 1. *Components (g) of diets*

	Daily allowance
Orange juice†	200
Cornflakes	25
Jam	25
Egg (one medium)	60
Milk (whole)	568
White bread	120
Biscuits (Nice)‡	52
Butter	60
Sugar	100
Lettuce	20
Cucumber	20
Tomato	60
Plain yoghurt	140
Potato	100
Instant coffee	

Main courses

Day ... 1		2		3	
Cod	100	Chicken	75	Beef	100
Green beans	100	Mixed vegetables	50	Tomato	50
Apricots†	120	Sprouts	50	Broccoli	50
Beef	75	Fruit salad†	120	Peaches†	120
		Salmon†	75	Cottage cheese	100

† Tinned. ‡ Waitrose Ltd.

Table 2. *Composition (g) of diets*†

Diet‡ ...	Control	+Pectin
Energy (MJ)	11·39	11·39
Carbohydrate	350·7	350·7
Protein	91·8	91·8
Fat	111·3	111·3
Calcium§ (mg)	1280	1357
Dietary fibre§:		
Total	14·9	45·72
Cellulose	6·09	6·09
Lignin	< 0·01	< 0·01
Non-cellulosic polysaccharides:		
Hexose	5·68	11·22
Pentose	1·99	4·37
Uronic acid	1·16	24·06

† From Paul & Southgate (1978).
‡ For details, see Table 1.
§ Determined by procedures detailed on p. 480.

Markers and transit measurement

On each day of weeks 1–9 the subjects took thirty radio-opaque pellets (ten per meal) as a non-absorbable marker. Two types of marker were used, small rods and small circles each made by cutting sections from radio-opaque tubing (Portex Ltd, Hythe, Kent). The type of marker given was changed when the pectin was added to the diet and also at the end of week 5 since the study was also being used as an opportunity to compare the value of radio-opaque pellets with chromium oxide as markers in metabolic-balance studies. Full details and

Table 3. *Pectin analysis (/36 g) of diet*†

Hexose (as galactose)	5·54
Pentose (as arabinose)	2·38
Uronic acid (as galacturonic)	22·9
Moisture	4·14
Methoxyl‡	2·34§
Esterified (%)	72§

† Sum of all components = 37·3, because carbohydrates and uronic acids expressed as weight of monosaccharide.

‡ Calculated at CH_3O.

§ Based on measurements carried out at Unilever Research, Bedford.

validation of these markers and their manufacture are reported elsewhere (Branch & Cummings, 1978). Marker recovery in the stool was measured by X-ray and the values used for the correction of faecal weight and the output of N, fat, Ca and dry matter.

Mean transit time (MTT) being the average time that a substance takes to pass through the gut was calculated for each day of the study from a knowledge of the cumulated number of markers ingested less the total number excreted in the stool at any time. Details of this method are reported by Cummings, Jenkins *et al.* (1976).

Analytical methods

Faecal solids were measured by freeze-drying duplicate portions of homogenate to constant weight, faecal bile acids by a modification of the method of Evrard & Janssen (1968) and fatty acids by gas–liquid chromatography, as previously described by Cummings, Wiggens *et al.* (1978).

Ca was determined by ashing at 400° portions of freeze-dried faeces equivalent to 2·5–5 g of original stool. The ash was extracted with nitric acid, diluted with lanthanum–HCl solution and analysed by atomic absorption spectroscopy (Unicam SP90). Urine was diluted with lanthanum–HCl before analysis.

Faecal polysaccharides were measured by the method of Southgate (1969) and faecal N after acid-digestion by a standard Auto Analyzer method (Technicon).

Serum gamma glutamyl transpeptidase was measured at the start of week 4 and the end of week 9 (beginning and end of pectin period). Analysis was performed in the hospital routine laboratory.

In analysing samples for the study, weeks 3, 6 and 9 were selected as representing the control and pectin periods. Some confusion in the laboratory did arise however, with regard to analysis of faecal polysaccharides and in two subjects weeks 3, 7 and 9 were analysed and in the other two, weeks 3, 6 and 8. Results from weeks 6 and 7 and from weeks 8 and 9 were consistent so it was not felt necessary to analyse further specimens. Additional weeks were examined for calcium balance since it was felt adaptive changes might be occurring between the subjects *ad lib.* diet, controlled diet and on addition of pectin.

Results are given as mean values ± 1 standard error of the mean unless otherwise stated and statistical analysis has been by Student's *t* test for paired values.

RESULTS

Diets

The diets provided 51 % of total energy from carbohydrate, 36 % from fat and 13 % from protein. They contained commonly-eaten foods which were chosen to keep fibre intake in the control period low, yet sufficient to be compatible with a reasonable bowel habit. At

14·9 g/d fibre intakes were approximately 74 % of the national average daily intake (Bingham *et al.* 1979; Southgate *et al.* 1978). They were well tolerated by the subjects. The introduction of pectin into the diet produced fewer problems or symptoms than anticipated, once it had been found that it was best taken mixed in with some orange juice before or at the beginning of meals. The main comments were that it produced a feeling of abdominal distension associated with a considerable increase in flatus production. The excess wind continued throughout the study.

Markers

Of the 9460 markers taken by the subjects during the study thirty-three were not recovered, an average of 6·6 ± 0·5 per subject. Over-all recovery was 99·7 %.

Bowel habit and transit time

On the control diet the subjects' stool frequency (4·2 ± 0·8 stools/week) and faecal weight (107 ± 25 g/d) was low and MTT long (77 ± 18 h) as would be expected with a relatively low fibre intake (Table 4). Addition of 36 g pectin to the diet produced no immediate change. A small increase in faecal weight was observed by week 6 (33 %) and in week 9. Otherwise pectin was without significant effect on bowel habit.

Faecal polysaccharide excretion

Faecal polysaccharide excretion showed marked variation between subjects (Table 5). In the control period cellulose excretion for example was 0·5, 0·6, 0·8, 2·6, 3·6 g/d in the five subjects, whilst the range for non-cellulosic hexose excretion was 0·7–2·0 g/d, for pentose 0·4–1·0 g/d and uronic acids 0·2–0·7 g/d. Over-all polysaccharide recovery in the faeces was low ranging from 17–23 % of the control dietary intake suggesting extensive degradation of dietary fibre. When pectin was added to the diet no significant change in polysaccharide excretion was seen. Particularly notable was the absence of any rise in uronic acid excretion in view of the major increase in dietary uronic acid intake (+22·9 g/d). Hexose excretion increased slightly but was not significantly different from the control period. Pectin appeared to have been completely digested or fermented in the gut.

Ca balance

Ca intake from all sources was 1280 mg/d from the control diet and rose to 1357 mg/d with pectin as the pectin powder contained some Ca. Within the limitations of the techniques the subjects were all in Ca balance during the control period and this was not altered by the addition of pectin (Table 6). There was however a small but significant change in the route of Ca excretion after pectin had been taken. The average daily Ca excretion for all urine samples collected during the control periods was 201 ± 41 mg/d. This fell to 179 ± 39 mg/d for all urine samples collected during the pectin period (t 3·5295, $P < 0·025$). Similarly faecal Ca excretion increased significantly from 1114 ± 27 mg/d to 1178 ± 28 mg/d (t 4·4939, $P < 0·025$).

Faecal composition

Despite being totally digested in the gut and altering bowel habit and Ca balance only minimally pectin produced significant increases in faecal fatty acid excretion (80 %) and faecal N (47 % week 6, 24 % week 9) (Table 4). In addition faecal dry matter excretion rose (28 % week 6, 18 % week 9) but the proportion of faecal moisture remained unchanged. Faecal bile acid excretion increased by 35 %. The composition of the faecal fatty acids excreted was unchanged.

Table 4. *Bowel habit and faecal composition of five healthy male subjects given pectin during the last 6 weeks of a 9-week controlled-diet study*

(Mean values ± 1 standard error of the mean)

Week no.	3		6		9		
Diet†	Control		+ Pectin (36 g/d)		+ Pectin (36 g/d)		
	Mean	SE	Mean	SE	Mean	SE	*n*
Stool frequency (stools/week)	4·2	0·8	3·8	0·2	4·2	0·5	5
Stool wt (g/d)	107	25	138	24**	123	25*	5
Mean transit time (h)	77	18	62	14	70	16	5
Faecal solids (g/d)	26·6	3·0	34·1	2·7**	31·5	3·5**	5
Water (g/kg)	727	31	731	30	723	29	5
Fatty acids (g/d)	1·47	0·11			2·65	0·17**	4
Nitrogen (g/d)	1·27	0·16	1·87	0·21*	1·58	0·17**	4
Bile acids (mg/d)	239	42			322	37***	5

Significance of difference from control week: * $P < 0·001$; ** $P < 0·01$, *** $P < 0·05$.
n, number of subjects.
† For details, see Tables 1–3.

Table 5. *Faecal polysaccharide excretion (g/d) and apparent digestibility* $\left(\frac{intake-output}{intake}\right)$ *of four healthy male subjects given pectin during the last 6 weeks of a 9-week controlled-diet study*

(Mean values ± 1 standard error of the mean)

					Non-cellulosic polysaccharides					
			Cellulose		Hexose		Pentose		Uronic acids	
Diet†	Week no.		Mean	SE	Mean	SE	Mean	SE	Mean	SE
Control	3	Faecal excretion	1·12	0·49	0·97	0·28	0·46	0·06	0·21	0·01
		Digestibility	0·82	0·08	0·83	0·05	0·77	0·03	0·82	0·01
+ Pectin	6 or 7	Faecal excretion	0·80	0·08	1·22	0·14	0·48	0·04	0·16	0·04
		Digestibility	0·87	0·01	0·89	0·01	0·89	0·01	0·99	0·01
+ Pectin	8 or 9	Faecal excretion	0·89	0·36	1·17	0·10	1·41	0·07	0·31	0·03
		Digestibility	0·85	0·06	0·90	0·01	0·91	0·02	0·99	0·01

† For details, see Tables 1–3.

Table 6. *Calcium balance (mg/d) in five healthy male subjects given pectin during the last 6 weeks of a 9-week controlled-diet study*

(Mean values ± 1 standard error of the mean)

Week no.	2		3		5		7		9	
Diet†	Control				+ Pectin (36 g/d)					
Calcium intake (mg/d)	1280				1357					
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Faecal excretion	1108	27	1120	35	1207	17	1197	43	1211	42
Urine	207	47	195	34	195	46	164	38	184	43
Total	1315	27	1315	21	1401	38	1312	48	1394	35
Net balance	−36	26	−35	21	−44	38	−5	48	−37	35
Number of subjects	4		5		5		5		5	

† For details, see Tables 1–3.

Serum gamma glutamyl transpeptidase (γ-GT)

Since pectin degradation in the human gut is likely to lead to the production of methanol from the methoxyl group serum γ-GT, a marker of hepatic microsomal enzyme activity, was measured in all subjects before and after the pectin period. No significant change was observed. γ-GT week 4 (start) 9·5 ± 2·1 mu/l; week 9 (end) 8·0 ± 1·3 mu/l (*t* 0·5781).

DISCUSSION

This study shows that pectin added to the normal diet of young men disappears during its passage through the gut probably as the result of bacterial fermentation. Despite this a significant increase in faecal fat, N, bile acids and total solids excreted was seen. No over-all effect on Ca balance was observed and only a small change in bowel habit occurred.

Using the same method of analysis for both food and faeces it may be seen from Table 5 that faecal cellulose + non-cellulosic polysaccharide (NCP) excretion rose by only 0·3 g or less per d when 30·8 g pectin fibre (all NCP) was added to the diet. Assuming that the amount of fibre remaining in the faeces from the control diet (2·76 g/d) was the same during the pectin period then this is strong presumptive evidence for the complete breakdown of pectin in the human gut. Using less precise methods Werch & Ivy (1941) made a similar observation in man. From in vitro fermentation studies (Werch *et al.* 1942) and studies of ileostomists (Werch & Ivy, 1941) they suggested colonic fermentation of pectin was occurring.

It is also evident from the amount of faecal polysaccharide excreted that a large proportion (approximately 85 %) of the fibre in the control diet was also metabolized during passage through the gut. This fibre was taken in a finely-divided state and, apart from the tomato skins, was neither heavily lignified nor cutinized. Taken together with the relatively long residence time of the food in the gut (approximately 70 h) this would favour its extensive degradation. Similar digestibility values have however been observed in the ruminant (Michaux, 1950, 1951). Digestion of dietary fibre occurs extensively in the animal kingdom in both the rumen (Raymond, 1969) and the caecum and colon of both herbivores and other single-stomached species (Keys *et al.* 1969; McBee, 1970). Fibre digestion has also been shown to occur in man by others (Williams & Olmsted, 1936; Hummel *et al.* 1943; Southgate & Durnin, 1970) but has received much less attention because it is not of any apparent physiological importance except possibly with regard to laxation. There seems little doubt however that man can digest dietary fibre presumably in his caecum or colon.

The fate of dietary fibre in the large bowel has recently assumed greater importance since its presence in or absence from the diet has been related to the development of several large bowel diseases and it is clearly a major factor determining bowel habit (Cummings, Southgate *et al.* 1978; Burkitt *et al.* 1972). Fibre is metabolized by bacteria to short chain fatty acids, hydrogen, carbon dioxide and methane. Short-chain fatty acids are absorbed from the colon in man (McNeil *et al.* 1978) and although they may not contribute significantly to total energy they could be important metabolically. The precise way in which dietary fibre controls faecal bulk is unknown but the extent to which it is digested must in some way affect this.

The pronounced effect which wheat fibre has on faecal weight has led to the view that all types of fibre increase stool output. That this is not so was shown by Williams & Olmsted (1936) and recently it has been observed that fibre from fruit and vegetables varies in its effect on bowel habit in relation to the pentose content of the NCP (Cummings, Southgate *et al.* 1978). The large dose of pectin in this study produced only a 33 % increase in stool weight and no effect on stool frequency or MTT. Similar small increases in faecal weight

were found by Durrington *et al.* (1976) of 24 % with 12 g pectin/d and 20 % by Kay & Truswell (1977) with 15 g/d. These changes can be predicted from the low pentosan content of pectin (Table 3). Dietary fibre (36 g) from bran would contain eight times as much (Southgate, 1978) and is much more effective at increasing stool weight.

It is difficult to imagine a mechanism whereby the 2·4 g pectin NCP-pentose could lead to even a 33 % increase in stool weight when it is completely digested in the gut. Wheat bran is digested to a lesser extent and produces a much greater increase in stool weight (Southgate *et al.* 1976). These observations suggest that whilst what remains of dietary fibre in the stool may contribute to the increase in faecal weight, by water-holding for example, this is not the only factor. Whatever the mechanism the role of digestion of fibre could be of crucial importance in controlling bowel habit.

On the control diet there was some variation between individuals in the extent to which fibre was digested. In general however the subjects digested the major part of it and no relationship between other variables of bowel function such as MTT with digestion could be established.

Throughout the study the subjects were on average within 44 mg of ideal Ca balance. Adding pectin to the control diet did not significantly alter Ca balance although there was an over-all fall in urine Ca excretion of 21 mg/d.

A possible disadvantage of dietary fibre is its potential for impairing mineral absorption (Cummings, 1978). Originally McCance & Widdowson (1942*a*) showed that high-wheat-fibre diets could lead to significant Ca imbalance but thought this was due to its phytate content (McCance & Widdowson, 1942*b*). More recently Rheinhold *et al.* (1975) have suggested that fibre itself might be partly responsible for this effect and James *et al.* (1978) have shown in vitro that fibre from commonly-eaten foods binds Ca in direct proportion to its uronic acid content. Pectin has a very high uronic acid content and we expected it to affect Ca balance. There were two possible reasons why this did not occur. The pectin was a high methoxyl type in which approximately 80 % of the uronic acids were methyl esterified. Some binding should have occurred. Alternatively Ca may have been bound in the upper gut but released in the large bowel as the pectin was metabolized and then the Ca absorbed from the colon. Colonic absorption of Ca has not been demonstrated for man but it is recognized in animals (Harrison & Harrison, 1969; Petith & Schedl, 1976). Further studies are needed to identify the role fibre plays in vivo in mineral metabolism.

Pectin produced significant changes in faecal composition notably an increase (%) in the excretion of fatty acids (80), N (47), bile acids (35) and total solids (28). Similar changes have been observed in other human studies (Kay & Truswell, 1977; Chenoweth & Leveille, 1975; Miettinen & Tarpila, 1977). In the absence of an increase in faecal fibre excretion the extra faecal solids may represent an increase in faecal bacterial mass. This would explain the increase in fat and N excretion without invoking any suggestion of pectin impairing small intestinal digestion and absorption of these nutrients. The mechanism for the increase in bile acid excretion is more difficult to explain. It is of a similar order to that which we have observed when feeding similar amounts of wheat bran (Cummings, Hill *et al.* 1976). The pronounced cholesterol-lowering effect of pectin seen both in these subjects (Jenkins, Reynolds, Leeds, Waller & Cummings, unpublished results) and observed in other studies (Durrington *et al.* 1976; Kay & Truswell, 1977) is in marked contrast to the lack of effect wheat bran has on blood lipids and suggests that a simple increase in faecal bile acid excretion is unlikely to be the explanation for the cholesterol-lowering properties of dietary fibre.

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