The effect of wheat fibre on plasma lipids and faecal steroid excretion in man

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1. The effect of isoenergetic substitution of bran and wholemeal bread for white flour and white bread was investigated in six healthy volunteers in a metabolic unit.

2. A 21 d high-fibre experimental period was preceded and followed by a 14 d control period, so that each experiment lasted 7 weeks. Blood samples were taken on each of the last 3 d and faeces collected (using carmine markers) for the last 6 d of each diet period.

3. Plasma cholesterol, triglycerides and serum calcium concentrations were not altered by the addition of wheat fibre.

4. Wheat fibre caused a significant increase in faecal bulk and frequency. Faecal steroid excretion was the same in the last week of the first control and the high-fibre periods but increased in the subsequent control period.

There has been much recent interest in the hypothesis that dietary fibre may prevent coronary heart disease (CHD) (Trowell, 1972 *a, b*, 1975; Trowell, Painter & Burkitt, 1974). Consumption of wheat fibre is lower in Britain today than it used to be in the nineteenth century (Robertson, 1972) when CHD was uncommon, and we eat less cereal fibre than the populations of many developing countries which have low rates of CHD incidence. If large intakes of cereal fibre do indeed protect against CHD the most likely mechanism would be by reduction of plasma cholesterol concentration resulting from adsorption of bile salts or cholesterol to cereal fibre in the intestine with negative sterol balance. Eastwood & Hamilton (1968) showed that lignin adsorbs bile acids in vitro. Wheat fibre consists of hemicellulose, cellulose and lignin in a ratio of 5:2:1 by vol. (McConnell & Eastwood, 1974). The reported effects of wheat fibre on plasma lipids both in man (Eastwood, 1969; Heaton & Pomare, 1974) and animals (Kay & Truswell, 1975) were conflicting and confusing when we started the present experiments. The practical question being tested was whether plasma cholesterol will fall when people eat brown bread instead of white or take extra bran, or both.

A preliminary report of these results has already appeared (Truswell & Kay, 1975).

EXPERIMENTAL

Subjects

The six subjects were postgraduate students of Queen Elizabeth College, three men and three women, aged 22–27 years. Most were research students in nutrition, and all were interested in the experiment and fully understood the regularity and reliability it required.

Table 1. *Sample menu provided for volunteers on low-fibre and high-fibre experimental diets*

Breakfast (g)	Lunch (g)	Dinner (g)	Evening (g)
150 Orange juice	100 Bread	130 Turkey breast	50 Muffin*
25 Kelloggs Cornflakes	20 Butter	4 Peanut oil	50 Bread
25 Bread	55 Salmon	10 Butter	10 Butter
5 Butter	17 Mayonnaise	10 Maize starch	30 Cheese
30 Sugar (for whole day)	17 Cucumber	170 Potato	20 Pickles
300 Milk (for whole day)	50 Tomato	60 Peas	280 Beer
	150 Yogurt	60 Banana Loaf*	
	50 Muffin*		

* Items in which white flour was isoenergetically replaced by 11·5 g wheat bran in the high-fibre period.

Diets

Three subjects participated together in two separate 7-week experiments, consisting of a 2-week control period followed by a 3-week high-fibre period and a second 2-week control period. The diets were varied daily over a short, repeating cycle; this was a 3 d cycle in the first experiment and a 7 d cycle in the subsequent experiment. A sample menu is given in Table 1. The diets were based on food items which enabled rigid control of fat and cholesterol intake. Alcohol was included in stipulated quantities similar to usual intakes. All food was prepared and eaten in the metabolic unit with the exception of breakfast and a late evening snack which were pre-weighed and packed for home consumption. One subject lived in the metabolic unit full-time. The control diet was adequate in all nutrients but relatively low in cholesterol and crude fibre content. The fibre content of diets was estimated from crude fibre values in food tables (see footnote to Table 2).

Prewett's wheat bran (Prewett's Stone Flour Mills, Horsham, Sussex RH12 1ST) was substituted isoenergetically for white flour, using the Atwater coefficient of digestibility to calculate the energy value of bran (Watt & Merrill, 1963). The substitution was made in various muffins and quickbreads which were distributed throughout the day. Each serving contained 11·5 g bran and the number of servings of bran-substituted muffins was varied according to individual tolerance. Individual subjects took between 23 and 35 g bran/d, except for one who could not tolerate the bran (see later).

In addition, white bread (Sunblest Prairie Gold, sliced) in the control diet was isoenergetically replaced by 100% wholemeal bread (Mother's Pride sliced 100% wholemeal). Intakes of bread ranged from 100 to 200 g/d in different subjects. Changes in fat and protein intake due to these substitutions were negligible.

Blood and faecal samples

Venous blood was taken before breakfast on the last 3 d of each dietary period. Each subject remained in a reclining position for 20–30 min beforehand and minimum venostasis was used.

Five subjects collected faeces corresponding to the last 6 d of each dietary period.

Table 2. *Composition (/d) of control and high-fibre diets given to normal volunteers**

(Mean values with ranges in parentheses)

	Control diet	High-fibre diet
Energy (MJ)	10·14 (8·07–12·08)	10·14 (8·07–12·08)
Protein (g)	90 (78–100)	91 (79–101)
Fat (g)	91 (74–108)	92 (75–109)
Carbohydrate (g)	282 (208–337)	279 (205–334)
Alcohol (g)	18 (4–38)	18 (4–38)
Crude fibre (g)	3·7 (2·7–4·8)	8·6 (6·8–10·6)
Cholesterol (g)	0·52 (0·43–0·72)	0·52 (0·43–0·72)

* Calculated from food tables (McCance & Widdowson, 1960; Watt & Merrill, 1963; Feeley, Criner & Watt, 1972).

Transit time was measured by carmine markers (taken after the evening meal) at the beginning and the end of the 6 d. The samples were individually collected in plastic bags and frozen. The samples for each period were mixed, weighed and two 50 g portions were freeze-dried and ground to a fine powder.

Analyses

Plasma cholesterol was measured by the method of Abell, Levy, Brodie & Kendall (1952). An enzymic method was used for plasma triglyceride determination (Eggstein, 1966) and serum calcium was measured on a Vickers analyser (Vickers Medical Engineering, Basingstoke, Hants). Faecal bile acids and neutral steroids were measured by gas-liquid chromatography using the method of Evrard & Janssen (1968) as modified by Mitchell, Findlay, Prescott, Eastwood & Horn (1973).

Significance levels were calculated by Student's *t* test for paired data. Mean values for the two control periods were compared against values for the wheat-fibre period (except for the values in Table 5).

RESULTS

General

Mean daily intakes of major and relevant nutrients are shown in Table 2.

Changes in body-weight were minimal, ranging from 0 to ±700 g in any dietary period. All subjects remained healthy throughout the experiment. Total body-weight change for the whole of the experiment averaged −0·2 kg (range −1·0 to +0·6 kg).

Table 3. *Fibre consumption by normal volunteers given a high-fibre diet and changes in faecal weight and intestinal transit time*

Subject	Bread (g/d)	Bran (g/d)	Changes in high-fibre period			
			Added crude fibre (g/d)	Change in faecal wet wt (g/d)	Change in mean transit time (h)	Change in mean no. of bowel movements/d
B	200	23·0	5·5	+124	− 9	+0·35
C	150	0	2·6	+ 64	−18	+0·65
I	175	34·5	6·1	+ 55	−20	+0·35
K	175	23·0	5·1	+130	− 3	+0·70
S	100	23·0	3·8	+ 91	−33	+1·70
T	200	34·5	6·5	+100	− 7	+0·60
Mean	167	23·0	4·9	+ 94	−15	+0·73

Fibre intake

Intakes of fibre in individual subjects are given in Table 3.

One subject (C) developed abdominal cramps and nausea on the addition of bran to the diet. Bran was discontinued in this subject and wheat fibre continued only in the form of wholemeal bread (150 g/d) (Table 3).

Faecal weights and transit time

The mean daily faecal weight in the last weeks of the two control periods was 125 g (range 59–194 g). During the bran period, this increased significantly to 225 g (range 154–290 g) ($P < 0·01$). The changes in individual subjects are shown in Table 3. This was largely due to an increase in the water content of the faeces (mean 82 g control, 154 g on bran) while the dry weight of the faeces increased from an average of 32 g control to 53 g on bran. The percentage of water in faeces was 71 % in control periods and 74% on bran.

Mean gastrointestinal transit time decreased during the high-fibre period but this change was not significant and was most notable in those subjects with an initially long transit time.

Bowel movements increased from a mean of 0·95 (range 0·6–1·4)/d in the control periods to a mean of 1·7 (range 1·0–2·7)/d during the high-fibre period. This was statistically significant ($P < 0·02$).

Plasma lipids

Plasma cholesterol and triglyceride concentrations were not altered by the addition of wheat fibre to a low-fibre diet for a 3-week period (Table 4, Fig. 1).

Serum Ca levels were not lowered (Table 4) by the inclusion of wheat fibre. All diets contained over 700 mg Ca/d.

Faecal steroids

The addition of wheat fibre to the diet resulted in a decreased concentration of faecal bile acid and neutral steroids due to the bulking effect of fibre. The total daily

Table 4. *Plasma concentrations of cholesterol and triglycerides and serum concentrations of calcium in normal volunteers given control and high-fibre diets*

(Mean values with ranges in parenthesis for six determinations)

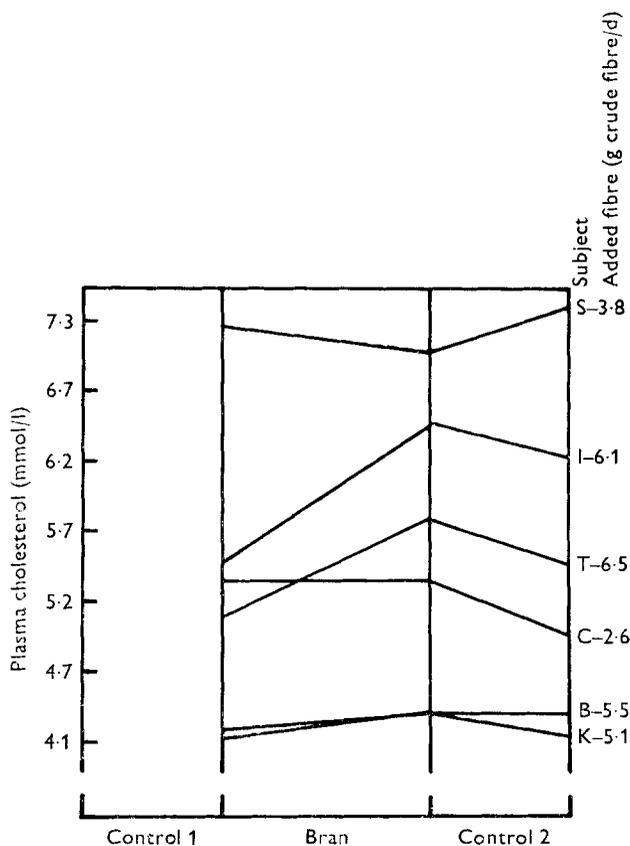|	Control period 1	High-fibre diet	Control period 2
Cholesterol (mmol/l)	5·26	5·57	5·41
	(4·17–7·23)	(4·35–7·02)	(4·35–7·36)
Triglycerides (mmol/l)	0·95	0·90	0·93
	(0·68–1·13)	(0·72–1·07)	(0·80–1·05)
Serum Ca (mmol/l)	2·43	2·45	2·50
	(2·38–2·48)	(2·35–2·58)	(2·42–2·53)

Fig. 1. Changes in plasma cholesterol concentrations in individual subjects on changing from the control to the high fibre (bran) diet and back again to the control diet. Each inflexion of an individual's line is the mean of three determinations on consecutive days.

Table 5. *Faecal steroid excretion (mg/d) by normal volunteers given control and high-fibre diets during the last 6 d of each dietary period*

(Mean values with ranges in parenthesis for five determinations)

	Control period 1	High-fibre diet	Control period 2
Lithocholic acid	77 (46–120)	72 (55–83)	111 (53–174)
Deoxycholic acid	165 (50–226)	150 (112–204)	250 (11–353)
Total bile acids	251[a] (103–353)	233[b] (186–294)	374[ab] (234–545)
Coprostanol	141 (11–244)	126 (12–218)	183 (221–268)
Cholesterol	124 (28–321)	130 (25–277)	128 (17–319)
Neutral steroids	265[c] (175–335)	256[d] (173–335)	311[cd] (235–406)

a, b, c, d, indicate significant differences ($P < 0.05$ or better) in paired t tests.

faecal steroid excretion did not change during the high-fibre period but increased in the second control period (Table 5). Although the means of the values for the 1st and 2nd control periods were not significantly different from those on wheat fibre, both neutral steroid and total bile acid excretions were significantly larger in the second control period than in the high-fibre period.

There was no change in the relative proportions of faecal bile acids.

DISCUSSION

Although a reduction in serum Ca concentration in subjects taking wheat bran has been recorded (Heaton & Pomare, 1974), such an effect was not confirmed by subsequent experiments (Connell, Smith & Somsel, 1975; Heaton, Manning & Hartog, 1976) or in the present study. The phytic acid of bran could interfere with Ca absorption and, in the present study Ca intake was lower when wholemeal bread replaced white. The former contains only 26 mg/100 g compared with 90 mg/100 g in the latter (McCance & Widdowson, 1960).

However, over a relatively short period of time bran and wholemeal bread did not lower the serum Ca concentration. Long-term studies would be required to determine whether continuous consumption of large amounts of bran would lead to a negative Ca balance.

Serum triglycerides were lower in one study, reported by Heaton & Pomare (1974) at a mean intake of 38 g bran/d. Some of their patients had initially high triglyceride concentrations and might also have had disturbances of bile acid metabolism, as indicated by the presence of gall-stones or previous cholecystectomy. A later trial by Heaton's group (Durrington, Wicks & Heaton, 1975) demonstrated that 30 g bran/d had no effect on the serum triglycerides of subjects with initially low concentrations

similar to those in the present communication. Heaton & Pomare (1975) suggested that only patients with hypertriglyceridaemia may respond to bran. But Bremner, Brooks, Third & Lawrie (1975) in a recent paper reported that 50 g bran/d did not lower serum lipids in five patients with type IV hyperlipidaemia. Other studies have confirmed the lack of effect of wheat fibre on serum triglycerides (Connell *et al.* 1975; Jenkins, Hill & Cummings, 1975; Persson, Ruby, Fønns-Bech & Jensen, 1975; Heaton *et al.* 1976).

As to plasma cholesterol, Jenkins *et al.* (1975) found no reduction in six male volunteers consuming 36 g wheat fibre/d for a 3-week period. Similarly, Connell *et al.* (1975) reported that the addition of 2·5–4·5 g crude fibre/d in the form of a bran cereal did not alter cholesterol concentration in twenty-two healthy students participating in an 11-week trial. It was further demonstrated by Eastwood *et al.* (1973) that 16 g bran/d fed to eight normal subjects for a 3-week period did not lower serum cholesterol and Heaton *et al.* (1976) administered wheat fibre in the form of wholemeal bread for 19 weeks without finding any reduction in blood lipids. Only Persson *et al.* (1975) have reported that plasma cholesterol was reduced after 6 weeks of bran consumption. However, the mean change was only −7 %. Control values after bran consumption ceased were not reported and the patients' average age was 76 years.

The wheat fibre in our subjects came from wholemeal bread in all six subjects and also from bran in five of the six. Most commercially available crude brans are similar in composition except for one that we did not use (Allinson's, which is finely ground and contains substantial amounts of germ and endosperm as well).

The study described here has shown, in agreement with the majority of other reports, that the addition of wheat fibre equivalent to 153–382 g of whole-wheat bread or 28–71 g bran/d, to a controlled diet for a 3-week period does not lower plasma lipid levels in healthy young adults. The amount of fibre added was sufficient to affect stool bulk and intestinal motility.

It has been suggested that certain classes of dietary fibre may alter faecal steroid excretion. However, Pomare, Heaton, Low-Beer & White (1974) found no change in the half-life of labelled cholic and deoxycholic acids administered to subjects consuming 57 g bran/d. This indicates that bran has little if any bile-acid binding capacity in vivo. Walters, McLean-Baird, Davies, Hill, Drasar, Southgate, Green & Morgan (1975) reported that neither faecal bile acid nor neutral steroid excretion increased in subjects receiving 39 g bran/d. A slight but significant increase in bile acid excretion in normal volunteers taking 20 g bran/d was found by Eastwood's group (Findlay, Smith, Mitchell, Anderson & Eastwood, 1974). In the same study, patients with diverticular disease tended to have lower bile acid excretion when they were given bran.

In a separate experiment Eastwood, Kirkpatrick, Mitchell, Bone & Hamilton (1973) reported a rebound phenomenon in bile acid excretion after stopping bran. Mean faecal bile acid excretion was virtually the same in the last week of the first control and the last week of a 3-week bran period but increased in the succeeding control period. In our study, faecal steroid excretion showed a similar pattern and these results suggest that some alteration in the enterohepatic circulation may have occurred

as a consequence of bran consumption, but the mechanism for such an effect is unclear. It has been demonstrated by Pomare & Heaton (1973) that bile acid patterns may be modified in the upper intestine by feeding bran. They report an increase in the ratio, chenodeoxycholate:deoxycholate in duodenal aspirates, implying that bran reduced the degradation of bile salts by colonic bacteria. The results reported here, however, do not indicate any difference in the proportions of faecal bile acids before or after bran.

A possible important effect of bran, observed in the present and other studies, is a significant increase in faecal bulk resulting in a dilution of faecal bile acids. Hill (1974) suggests that if certain bile acids, such as deoxycholic acid, are potential carcinogens or co-carcinogens, their concentration in the faeces is of more importance than the total daily excretion.

The effect of wheat fibre on stool bulk and laxation is direct and unequivocal. This form of dietary fibre, however, does not appear to reduce plasma lipids, at least in trials of a few weeks. This is not to say that other types of dietary fibre may not affect plasma lipid concentrations. We and other workers have been investigating pectin, for example, from this point of view (Truswell & Kay, 1975; Jenkins et al. 1975).

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