

Physiological effects of fibre-rich types of bread

2. Dietary fibre from bread: digestibility by the intestinal microflora and water-holding capacity in the colon of human subjects*

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1. Twelve young adult male volunteers were given a low-fibre white bread diet (9 g neutral-detergent fibre (NDF)/d) and a medium-fibre coarse-bran bread diet (22 g NDF/d), each lasting 20 d. In a third period of 20 d the volunteers were subdivided in groups of four, consuming a high-fibre coarse-bran bread diet (35 g NDF/d), a medium-fibre fine-bran diet (22 g NDF/d, bran particle size > 0.35 mm) or a wholemeal bread diet (22 g NDF/d). Digestion of dietary fibre and its components hemicellulose, cellulose and lignin were determined as well as colonic function.

2. An increase of the amount of dietary fibre (through bran in bread) from 9 to 22 g NDF/d resulted in the following significant changes ($P < 0.01$): increase in faecal wet weight of 63 g/d, decrease in the percentage of faecal dry weight from 27 to 24, increase in defaecation frequency of 0.2 stools/d and reduction of the intestinal transit time of 36 h.

3. Further significant changes with regard to all factors mentioned were observed during the high-fibre diet. Faecal wet weight was significantly ($P < 0.05$) lower with the fine-bran bread diet than with the coarse-bran bread on a similar fibre intake of 22 g NDF/d. Results obtained in the wholemeal-bread period did not show significant differences compared with those from the coarse-bran bread period of 22 g NDF/d.

4. Mean digestibilities for the fibre from bread were: for NDF 0.34, for hemicellulose 0.46, for cellulose 0.20 and for lignin 0.04.

5. The results obtained suggest that the theory of sponge activity of the fibre matrix structure is the predominant factor accounting for the water binding capacity of fibre in the colon.

Many papers have been published on the significance of dietary fibre in human nutrition. It is generally recommended that in the western world dietary fibre intake should be increased, because several diseases are believed to be associated with the consumption of refined carbohydrate-rich foods (Burkitt *et al.* 1972; Burkitt & Trowell 1975; Trowell 1976). However, conclusive evidence of the relative importance of fibre in our diet in the aetiology of diseases, such as appendicitis, cancer of the colon and diabetes, is still lacking.

Because of the beneficial influence dietary fibre, particularly from cereal sources, seems to have on colonic function (Cummings *et al.* 1976a; Mitchell & Eastwood 1976), an increased cereal fibre intake is desirable and this can best be effected by means of bread.

In various review articles (Kelsay 1978; Spiller *et al.* 1978) the following effects of increasing wheat-fibre intake are mentioned: increase in stool volume, shorter intestinal transit time, increase in stool frequency and decrease in absorptions of energy, fat, nitrogen and minerals. The influence of wheat fibre on serum lipids is not clear; both increased and decreased levels have been reported as a result of increased wheat-fibre intake and the effects observed on bile acid excretion are also rather contradictory. Although the definition of dietary fibre states that digestion does not take place in the (human) gastrointestinal tract by alimentary enzymes, it does not imply that dietary fibre or its components (cellulose, hemicellulose, lignin and pectin, the latter not present in wheat fibre) or both are not digested at all. Several of the previously mentioned physiological effects are brought about by the

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action of the bacterial flora of the colon, resulting in partial digestion of dietary fibre components. However, there is little information available on the extent of digestion of the various dietary fibre components (Holloway *et al.* 1978; Heller *et al.* 1980).

In order to determine some of the physiological effects of wheat fibre, as well as the digestibility of dietary fibre components by the bacterial flora of the colon, we carried out a study with subjects who were given three experimental diets, each lasting 20 d, differing in amount and particle size of bran incorporated in the bread. The mineral-balance results and the effects on some serum biochemical indices have been reported previously (Van Dokkum *et al.* 1982). In the present paper the effects are reported of wheat fibre on stool weight, stool frequency, intestinal transit time, bile acid excretion, excretion of volatile fatty acids (VFA), excretion of faecal N and phosphorus and the apparent digestibility by the intestinal flora of hemicellulose, cellulose, lignin and of the total amount of dietary fibre.

METHODS

The experimental design is shown in Table 1. Twelve male volunteers (means and standard deviations: age 23 (2)years, weight 68 (6)kg, height 1.82 (0.06)m, body fat 14 (3)%) were given two experimental diets with different amounts and types of dietary fibre for 20 d each. Results were compared with those on a low-fibre (white bread) diet with 9 g dietary fibre/d.

The volunteers gave informed written consent according to the Institute's procedures and were housed in the Institute's controlled metabolic ward, but they continued their normal daily routines. They passed beforehand a clinical examination and a nutritional evaluation. The routine haematological values were all within normal ranges.

Experimental procedure

The basal diet (the total daily diet without bread), which was constant for each subject throughout the study, consisted of conventional low-fibre foods and provided 7 g neutral-detergent fibre (NDF)/d (see Table 2).

In addition to the basal diet, all volunteers consumed bread, i.e. white bread in the adaptation period A and in the experimental period B and 150 g coarse-bran/kg bread in period C. The amount of dietary fibre in the latter type of bread was the same as that in conventional wholemeal bread; the organoleptic properties were different however. In the third experimental period (period D), three other types of bread were consumed, each type by four of the twelve volunteers. The 150 g fine-bran/kg bread was included to study the effect of the bran particle size on, for example, colonic function. The 320 g coarse-bran/kg bread contained twice the amount of dietary fibre as compared with conventional wholemeal bread, which was also included. The particle size of the coarse and fine brans were > 0.35 mm and < 0.35 mm respectively.

The diet characteristics (Table 3) are based on analyses of individual, duplicate, daily samples; the basal diet and the breads were analysed separately.

The energy intake was adjusted to the individual energy requirements derived from a dietary history before the experiment. A constant body-weight during the study was aimed at; reduction of body-weight of more than 2% was corrected by the addition of sugar, soft drinks and other carbohydrate equivalents. This procedure was necessary for four volunteers.

The food was prepared in the diet kitchen according to standard procedures, weighed to the nearest g, packed in individual portions and deep-frozen, when necessary.

Only demineralized water was allowed in restricted amounts (maximum approximately 200 ml daily, apart from water for coffee and tea). All types of bread were prepared from one batch of wheat flour. All meals were served at the Institute.

Table 1. *Experimental design**

Period ...	General adaptation A	First experimental period B	Second experimental period C	Third experimental period D
Duration (d)	8	20	20	20
Type of bread	White bread (entirely white flour)	White bread (entirely white flour)	Bread made from 850 g white flour and 150 g coarse bran/kg	(1) Bread made from 680 g white flour and 320 g coarse bran/kg (n 4) (2) Bread made from 850 g white flour and 150 g fine bran/kg (n 4) (3) Wholemeal bread (n 4)
Approximate total daily fibre intake (g NDF)	9	9	22	(1) 35 (2) 22 (3) 22

NDF, neutral-detergent fibre (Van Soest & Wine, 1967).

* The study was carried out with four volunteers at a time and replicated twice (with other subjects), resulting in three experiments with four volunteers each.

Table 2. *Composition (g) of the basal* diet*
(Mean daily values)

Cheese	60
Smoked beef	15
Ham	15
Orange juice	250
Vegetable margarine	30
Custard (low-fat)	150
Jelly	30
Potatoes	200
Ground beef	100
Ice cream	50
Whipped cream	25
Vegetables†	
Instant tea	0.9
Instant coffee	3
Sugar	20
Soft drinks	400
Whisky	35

* In addition to the basal diet, 240 g bread/d of various types were consumed; for details, see Table 1.

† Rotating order for every 4 d: day 1: 75 g string beans, 5 g margarine, 100 g apple sauce; day 2: 30 g lettuce, 40 g carrot salad, 40 g celeriac salad; day 3: 75 g sliced beans, 5 g margarine, 100 g apple sauce; day 4: 200 g tomatoes, 5 g margarine, 5 g rusk.

Stools were collected in 3 l plastic buckets, one for every 4 d, stored at 4°. Composites (4 d) of stools were made for analysis and stored at -20°.

Analytical procedures

As wheat bran dietary fibre is low in water-soluble components and the basal diet (without bread) was low in dietary fibre, analyses were carried out according to the method of Van Soest & Wine (1967) as an approximation for dietary fibre, applying predigestion with pancreatin to remove residual starch (Terry & Outen, 1973). The method of Van Soest & Wine (1967) was also used for the analysis of acid-detergent fibre (ADF), cellulose and lignin. The hemicellulose content was calculated as the difference between NDF and ADF. Total available carbohydrates were determined, using pancreatic amylase to transform starch into soluble carbohydrates and subsequent hydrolysis of the carbohydrates with hydrochloric acid to glucose; glucose and other monomeric sugars were analysed using the Luff-Schoorl reagent (van de Kamer, 1941). Fat was analysed according to the Weibull-Stoldt method by extraction of the sample with light petroleum (b.p. 60-80°) (Schormüller, 1969). Protein was calculated from the Kjeldahl N determination, using the automated KjellFoss (Noel, 1976) and applying the factor 6.25. Phosphorus was analysed gravimetrically as ammonium molybdophosphate (Schormüller, 1967).

In all stool composites the moisture content was analysed by drying on sand. Stool frequency was computed from information obtained from the diaries of the volunteers; intestinal transit time was measured using radio-opaque pellets (Hinton *et al.* 1969) towards the end of each experimental period and calculated as 80% recovery. In the last two 4 d composites of the stools in each period of 20 d, total bile acid excretion was measured enzymically with 3- α -hydroxy-steroid-dehydrogenase (EC 1.1.1.50) in a faecal extract which was prepared as follows. The faeces were boiled with methanol, the extracted matter was saponified by boiling with 1 M-KOH in ethylene glycol; the free bile acids were extracted

Table 3. Diet characteristics of the various experimental periods*
 (Mean daily values, with values for the contribution of bread (%) to the total daily intake in parentheses)

Experimental period ... Type of bread ...	B White bread (n 12)	C 150 g coarse-bran/kg bread (n 12)	D ₁ 320 g coarse-bran/kg bread (n 4)	D ₂ 150 g fine-bran/kg bread (n 4)	D ₃ Wholemeal bread (n 4)
Energy (MJ)	11.1 (26)	11.1 (24)	11.5 (21)	9.9 (27)	11.5 (24)
Total fat (energy %)	36 (8)	36 (8)	41 (8)	35 (10)	36 (9)
Total available carbohydrates (energy %)	49 (40)	48 (37)	42 (33)	49 (39)	50 (33)
Total protein (energy %)	12 (29)	12 (30)	12 (33)	13 (30)	12 (31)
Phosphorus (mg)	1163 (17)	1530 (36)	1905 (48)	1867 (31)	1622 (40)
NDF (g)	8.7 (25)	21.2 (69)	34.9 (83)	22.8 (65)	22.0 (71)
NDF (g/4.2 MJ)	3.3 (25)	8.0 (69)	12.8 (83)	9.6 (65)	8.0 (71)
ADF (g)	5.8 (21)	9.6 (52)	13.5 (69)	10.2 (53)	10.7 (52)
Cellulose (g)	4.2 (19)	6.9 (51)	10.2 (67)	6.8 (48)	7.1 (51)
Hemicellulose (g)	2.9 (31)	11.6 (83)	21.4 (92)	12.6 (75)	11.3 (90)
Lignin (g)	1.6 (25)	2.7 (56)	3.3 (78)	3.4 (62)	3.6 (53)

NDF, neutral-detergent fibre (Van Soest & Wine, 1967); ADF, acid-detergent fibre (Van Soest & Wine, 1967).
 * For details, see Table 1.

Table 4. Defaecation pattern of twelve human subjects consuming a white bread diet and a 150 g coarse-bran/kg bread diet for a period of 20 d

(Mean values and standard error of differences)

Experimental period ... Type of bread ... NDF intake (g/d) ...	B	C	SED
	White bread 9 Mean	150 g coarse-bran/kg bread 22 Mean	
Faecal wet wt (g/d)	77	140**	5
Faecal dry wt (%)	27	24**	0.8
Defaecation frequency (no. of stools/d)	0.9	1.1**	0.1
Intestinal transit time (h)	88	52**	8
Faecal VFA (mmol/g wet faeces)	0.10	0.11	0.01
Faecal VFA (mmol/d)	8.3	15.2**	1.5
Faecal bile acids: (μ mol/g wet faeces)	12.6	6.2**	0.8
(μ mol/d)	936	806	108
Faecal N (g/d)	1.11	1.58**	0.12
Faecal P (mg/d)	323	590**	36

VFA, volatile fatty acids.

NDF, neutral-detergent fibre (Van Soest & Wine, 1967).

Mean values significantly different from those for period B: ** $P < 0.01$.

with ethyl ether, purified by partitioning between petroleum ether and aqueous methanol (H_2O-CH_3OH , 30:70 v/v) and dissolved in methanol.

Faecal P was determined gravimetrically as ammonium molybdophosphate (Schormüller, 1967), faecal N was analysed (Noel, 1976) and faecal VFA (C_1-C_6) were determined by gas-liquid chromatography (van de Kamer *et al.* 1955); the modified method of Van Soest & Wine (1967) was applied to determine the amount of residual dietary fibre components in the stools.

The apparent digestibilities of dietary fibre and its components were estimated by regression analysis, using the values for intake and faecal excretion (see pp. 69-71).

For statistical evaluation the results, with respect to the defaecation pattern of each dietary treatment, were compared with those of the preceding treatment by means of analysis of variance (Snedecor & Cochran, 1967) using Student's *t*-test for paired observations. Each subject thus served as his own control. The means for period D (four subjects) were compared with those of the corresponding subjects in period C.

This research was approved from an ethical standpoint by a working group responsible for human nutrition studies.

RESULTS

The defaecation pattern is indicated by results presented in Tables 4-7. Substituting 150 g coarse-bran/kg bread (period C) for white bread (period B) resulted in the following significant ($P < 0.01$) changes: an increase in faecal wet weight of 63 g/d, a decrease in the percentage of faecal dry weight of 3, an increase in defaecation frequency from 0.9 to 1.1 stools/d, a decrease in the intestinal transit time of 36 h, an increase in faecal VFA from 8.3 to 15.2 mmol/d and increases in faecal N (by 0.47 g/d) and faecal P (by 0.27 g/d) (see Table 4).

During the 320 g coarse-bran/kg bread period (period D₁, see Table 5) a further significant ($P < 0.01$) increase of faecal wet weight was observed, the defaecation frequency

Table 5. Defaecation pattern of four human subjects consuming a 150 g coarse-bran/kg bread diet and a 320 g coarse-bran/kg bread diet for periods of 20 d

(Mean values and standard error of differences)

Experimental period...	C		D ₁	SED
	150 g coarse-bran/kg bread		320 g coarse-bran/kg bread	
Type of bread...	22		35	
NDF intake (g/d)...	Mean		Mean	
Faecal wet wt (g/d)	137		202**	8
Faecal dry wt (%)	25		23	1
Defaecation frequency (no. of stools/d)	1.1		1.4**	0.03
Intestinal transit time (h)	77		45*	11
Faecal VFA (mmol/g wet faeces)	0.08		0.09	0.01
Faecal VFA (mmol/d)	10.0		16.9	2.7
Faecal bile acids:				
μmol/g wet faeces	7.3		4.6**	0.5
μmol/d	812		888	63
Faecal N (g/d)	1.30		1.84	0.19
Faecal P (mg/d)	524		891*	79

VFA, volatile fatty acids.

NDF, neutral-detergent fibre (Van Soest & Wine, 1967).

Mean values significantly different from those for period C: * $P < 0.05$, ** $P < 0.01$.

Table 6. Defaecation pattern of four human subjects consuming a 150 g coarse†-bran/kg bread diet and a 150 g fine†-bran/kg bread diet for periods of 20 d

(Mean values and standard error of differences)

Experimental period...	C		D ₂	SED
	150 g coarse-bran/kg bread		150 g fine-bran/kg bread	
Type of bread...	22		22	
NDF intake (g/d)...	Mean		Mean	
Faecal wet wt (g/d)	126		102*	8
Faecal dry wt (%)	26		29	1
Defaecation frequency (no. of stools/d)	1.2		1.1	0.05
Intestinal transit time (h)	44		62	15
Faecal VFA (mmol/g wet faeces)	0.10		0.09	0.01
Faecal VFA (mmol/d)	12.8		10.9	2.2
Faecal bile acids:				
μmol/g wet faeces	6.4		6.7	0.6
μmol/d	818		788	121
Faecal N (g/d)	1.67		1.63	0.16
Faecal P (mg/d)	592		621	17

VFA, volatile fatty acids.

NDF, neutral-detergent fibre (Van Soest & Wine, 1967).

Mean value significantly different from that for period C: * $P < 0.05$.† Coarse-bran, particle size > 0.35 mm, fine-bran, particle size < 0.35 mm.

Table 7. Defaecation pattern of four human subjects consuming a 150 g coarse-bran/kg bread diet and a wholemeal bread diet for periods of 20 d

(Mean values and standard error of differences)

Experimental period...	C	D ₃	
Type of bread...	150 g coarse-bran/kg bread	Wholemeal bread	
NDF intake (g/d)...	22	22	
	Mean	Mean	SED
Faecal wet wt (g/d)	158	143	11
Faecal dry wt (%)	22	24	1
Defaecation frequency (no. of stools/d)	1.1	1.2	0.1
Intestinal transit time (h)	35	45	10
Faecal VFA (mmol/g wet faeces)	0.14	0.14	0.01
Faecal VFA (mmol/d)	22.8	18.8	3.0
Faecal bile acids:			
μmol/g wet faeces	4.8	4.8	0.3
μmol/d	790	645	60
Faecal N (g/d)	1.82	1.48	0.12
Faecal P (mg/d)	653	549	15

VFA, volatile fatty acids.

NDF, neutral-detergent fibre (Van Soest & Wine, 1967).

None of the differences is statistically significant.

increased to 1.4 ($P < 0.01$) and the intestinal transit time was significantly ($P < 0.05$) shorter than during the 150 g coarse-bran/kg bread period. The further increase of faecal VFA and faecal N on the 320 g coarse-bran/kg bread diet was not statistically significant while faecal P increased significantly ($P < 0.05$), which reflects the increased P intake.

A marked influence of the particle size of the bran on the water-holding capacity was observed. The amount of faecal wet weight was significantly ($P < 0.05$) lower on the fine-bran bread (period D₂) than on the coarse-bran bread (period C, see Table 6); the increase of the percentage of faecal dry weight and the intestinal transit time, as well as the decrease of the excretion of faecal VFA on the fine-bran bread diet, were not statistically significant.

No significant differences of the various indices were detectable between the wholemeal bread period (period D₃) and the 150 g coarse-bran/kg bread period (period C, see Table 7), both providing a similar dietary fibre intake of 22 g NDF/d.

Throughout the study no significant changes in the daily excretion of faecal bile acids could be demonstrated; the concentration of total bile acids in the faeces decreased significantly ($P < 0.01$) on an increasing dietary fibre intake (Tables 4 and 5).

Although the amount of faecal VFA increased on the higher bran intake, the concentration in the faeces and the VFA pattern (of which 70% was acetic acid) remained constant.

The levels of intake and faecal excretion of dietary fibre and its components for each volunteer are shown in Figs. 1-4.

The total intake of NDF in the white bread period (period B) was 9 g. Of the total NDF intake 75% originated from the basal diet (see Table 3). In eight subjects NDF was almost completely digested, as shown by the low faecal excretions. For the lower digestibility of NDF in four subjects, cellulose and lignin seem to be responsible; hemicellulose, however, was almost completely degraded in all subjects. When 150 g coarse-bran/kg bread (period C) was substituted for white bread (period B), the NDF intake increased by 13 g (through bran in bread). It appears that in eleven subjects this increase in NDF intake resulted in approximately equal increments in faecal excretion of NDF, independent of a low

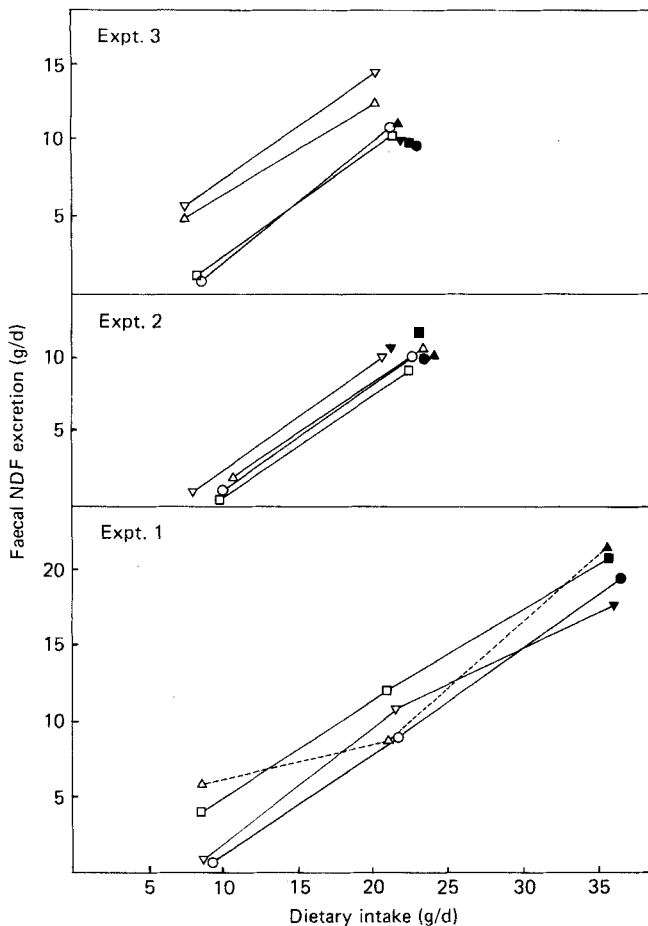


Fig. 1. Dietary intake and faecal excretion of neutral-detergent fibre (NDF). Each symbol indicates one subject. Open symbols on the left side of the graphs relate to the white-bread period (n 12) and those on the right side to the 150 g coarse-bran/kg bread period (n 12). Closed symbols refer in Expt 1 to the 320 g coarse-bran/kg bread period (n 4), in Expt 2 to the 150 g fine-bran/kg bread period (n 4) and in Expt 3 to the wholemeal-bread period (n 4).

or high faecal excretion in period B; this seems to be applicable also for hemicellulose, cellulose and lignin. For one subject (Δ in Expt 1) the results were contradictory, suggesting an analytical error.

The increase in fibre intake in period D_1 (320 g coarse-bran/kg bread diet) resulted in almost similar increments in faecal fibre excretions (four subjects) when compared with those after the substitution of 150 g coarse-bran/kg bread for white bread (Figs. 1–4).

When 150 g fine-bran/kg bread (period D_2) was substituted for 150 g coarse-bran/kg bread, the total NDF intake remained unchanged (22 g NDF/d). The faecal excretion of the fibre components was also similar for the four subjects.

When wholemeal bread was substituted for 150 g coarse-bran/kg bread the faecal excretion pattern of NDF and the components was not essentially altered, although small differences were observed in the digestion of all fractions of dietary fibre.

Faecal excretion of fibre components with the faeces can be expressed as a fraction of the intake. These coefficients of indigestibility were estimated by regression analysis. For

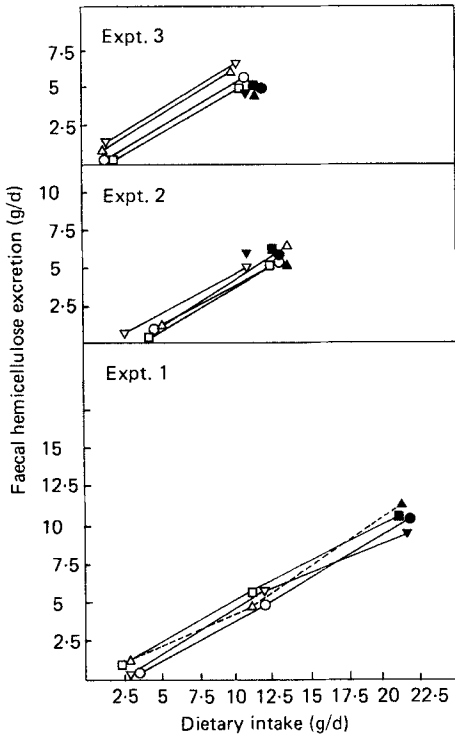


Fig. 2. Dietary intake and faecal excretion of hemicellulose. For details, see legend to Fig. 1.

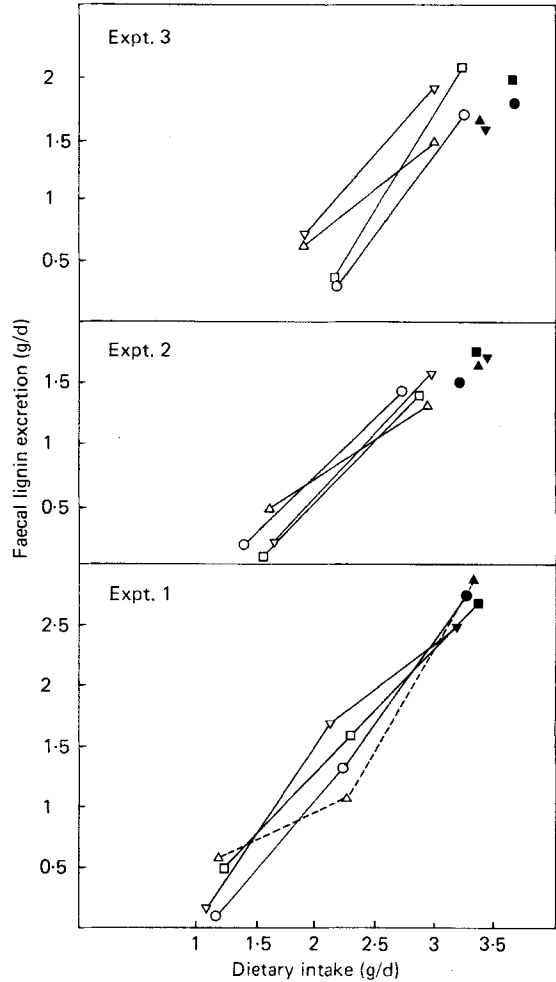


Fig. 3. Dietary intake and faecal excretion of lignin. For details, see legend to Fig. 1.

every subject in each period the following data were available:

Amount in the basal diet	(x_0)
Amount in the bread	(x_1)
Amount excreted with the faeces	(y)

The excretion can be described by the regression equation:

$$y_i = \beta_0 x_{0i} + \beta_1 x_{1i} + e_i$$

where $i = 1, \dots, 36$, β_1 is the coefficient of indigestibility of fibre from the bread and β_0 is the coefficient of indigestibility of fibre from the basal diet. In this simplified model, β_1 is assumed to be the same for all types of bread and β_0 and β_1 are assumed to be subject-independent.

Estimates of β_0 and β_1 and their confidence intervals for NDF, hemicellulose, lignin and cellulose are given in Table 8. These values were based on the analysis of the regression

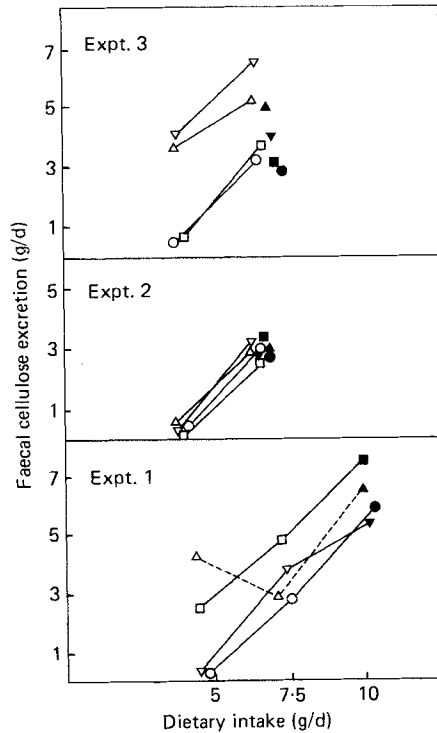


Fig. 4. Dietary intake and faecal excretion of cellulose. For details, see legend to Fig. 1.

Table 8. Coefficients of indigestibility and digestibility of dietary fibre and the components hemicellulose, lignin and cellulose from the basal diet and from bread

	NDF		Hemicellulose		Lignin		Cellulose	
	95% confidence intervals		95% confidence intervals		95% confidence intervals		95% confidence intervals	
β_0	0.09	(0.02, 0.37)	0.06	(0.01, 0.33)	0.0	(0.0, 0.0)	0.23	(0.08, 0.53)
β_1	0.66	(0.59, 0.73)	0.54	(0.51, 0.57)	0.96	(0.83, 0.99)	0.80	(0.50, 0.94)
$1 - \beta_1$	0.34		0.46		0.04		0.20	
RMS	3.1		0.33		0.081		1.6	
PVA	91		97		87		57	

β_0 = coefficient of indigestibility of fibre from the basal diet.
 β_1 = coefficient of indigestibility of fibre from the bread.
 $1 - \beta_1$ = coefficient of digestibility of fibre from the bread.
 RMS = Residual mean squares.
 PVA = Percentage variance accounted for.

equation in the form:

$$y_i = \frac{1}{1 + \exp(-\gamma_0)} \cdot x_{0i} + \frac{1}{1 + \exp(-\gamma_1)} \cdot x_{1i} + e_i$$

where $\gamma_j = \log(\beta_j/1 - \beta_j)$ and $j = 0, 1$. This approach was chosen because β_0 and β_1 must lie between 0 and 1. Different coefficients of indigestibility for the different types of bread did not result in a better fit for all four types of fibre. The relatively large 95% confidence

intervals for β_0 suggest a large variability in the excretion of fibre originating from the basal diet between subjects. Applying a variability between subjects in the regression analysis yields individual β_0 's and actually results in a better description of the excretion for NDF ($P < 0.05$) and cellulose ($P < 0.01$), but not for hemicellulose and lignin. Similarly, assuming a variability between subjects for β_1 does not result in a better fit for all four types of fibre. In addition, in Table 8 the coefficients of digestion for the different types of bread are presented, calculated as the complement of the coefficients of indigestibility.

DISCUSSION

The changes in colonic function following the ingestion of wheat fibre observed in the present study are not in all respects in agreement with the results of other investigators. The observed significant increase in the percentage of faecal water after adding fibre to the diet was also reported by Thomas & Elchazly (1976), whereas other investigators found no significant change (Cummings *et al.* 1976*b*; Floch & Fuchs, 1978). Fantus *et al.* (1941) were unable to find any influence of particle size of the bran on faecal weight, while we observed a smaller faecal weight with the ground bran. In more recent studies, Heller *et al.* (1980) and Smith *et al.* (1981) also found a more significant effect of coarse-bran than of fine-bran on colonic function. The longer intestinal transit time associated with a smaller bran particle size was also observed by Kirwan *et al.* (1974). An increasing wheat fibre intake does not always seem to result in a shorter intestinal transit time; other factors, both physical and psychological ones, are also likely to influence the transit time (Cummings *et al.* 1976*b*; Tucker *et al.* 1981). The influence of dietary fibre on defaecation frequency is also contradictory; the significant increase with an increased wheat fibre intake in our experiment was similar to that reported by Kay & Truswell (1977), while others did not find significant changes (Payler *et al.* 1975; Wymann *et al.* 1976; Heller *et al.* 1980).

The results obtained with the ground bran with respect to colonic function may give some indications as to the mechanism by which dietary fibre from bread effects increase in stool weight. An increased stool weight depends on the production of VFA in the colon (Williams & Olmsted, 1936) as a result of bacterial digestion of cellulose and hemicellulose, suggesting an osmotic action of the VFA (Bustos Fernandez *et al.* 1971; Forsyth *et al.* 1978). The constant concentration of VFA in wet faeces observed by us and also reported by others (Bustos Fernandez *et al.* 1971; Fordtran, 1971; Cummings *et al.* 1976*a*; Forsyth *et al.* 1978), supports the hypothesis of equilibrium concentration. VFA are, however, reported to be absorbed in the human colon (Dawson *et al.* 1964; McNeil *et al.* 1978). Van Soest & Robertson (1977) estimated that bran consumption leads to the absorption of 65% of the VFA produced by the bacterial flora. Applying the same method of calculation, we found 77% VFA absorbed by the colon. Consequently, the VFA in the colon can only partly account for the observed increase in water binding on an increased wheat fibre intake.

Another suggested reason for the increase in water binding is that undigested dietary fibre promotes gel-formation in the colon. The hemicellulose fraction appears to be particularly important in this respect (Jelaca & Hlynka, 1971; Eastwood, 1974). When we compare the 150 g coarse-bran/kg bread with the 150 g fine-bran/kg bread, the hemicellulose intake and the faecal output appear to be the same; the colonic function was, however, different.

The increased stool weight associated with an increased wheat fibre intake could also be accounted for by the increased bacterial mass (as a result of fibre fermentation) which may hold water as well (Stephen & Cummings, 1979). Since a similar degradation of dietary fibre by the bacterial flora was observed in the coarse-bran period and in the fine-bran bread period, the difference in faecal wet weight is not likely to be caused by a difference in bacterial mass, since the faecal N output did not change either.

Water binding is finally ascribed to the capillary structure and sorptive properties of

dietary fibre. Eastwood & Kay (1979) concluded that the matrix of the fibre particles is responsible for sponge action, which is partly destroyed when the particle size is small. This fits well with our findings.

From the present study it can be concluded that both the VFA hypothesis and the non-digested hemicellulose theory (gel formation) may be important factors in accounting for faecal bulk, but that the matrix structure of dietary fibre seems to be the most important determinant of the observed increase in stool weight. The other indices of colonic function (transit time and defaecation frequency) are probably a result of water binding.

Although the concentration of total bile acids in the faeces decreased with an increasing amount of bran in bread, the total daily excretion of bile acids did not change significantly, which reflects the dilution of the bile acids in the increased faecal bulk. Some authors indicate an increase in faecal bile acid excretion in man, others found decreased values following wheat bran feeding (Kay, 1982).

By means of regression analysis an estimate can be made of the apparent digestibility of fibre in the basal diet and in bread separately. The digestibility of the fibre components in the basal diet appears to differ from that in bread; moreover, the digestibility of NDF and cellulose in the basal diet is variable in different subjects. Differences in the degradability of fibre fractions from different sources have been observed by other investigators. Van Soest & Robertson (1977) suggested that the cell wall of vegetables is more fermentable than bran. Holloway *et al.* (1978) observed a bacterial degradation of 78% for cellulose and 96% for hemicellulose on a normal, low-fibre diet. Heller *et al.* (1980) reported a digestibility of 0.35 for NDF and of 0.50 for hemicellulose on a coarse-bran diet; their findings for cellulose (digestibility 0.06) and lignin (a strongly negative digestion) differ from our observations.

Inter-individual variations have been reported as well, e.g. by Dintzis *et al.* (1979). Furthermore, it is remarkable that the degradation of fibre from bread appears to be the same in (almost) all subjects, independent of the amount of wheat fibre in the diet and also independent of the intestinal transit time, the latter being shortest in the period in which 320 g coarse bran/kg bread was consumed. This suggests a maximal digestion of wheat fibre under the various circumstances, possibly because of the typical physical structure of wheat fibre which does not enable bacteria to ferment the fibre component further than to the maximal value observed by us. In experiments with rats (Sinkeldam, unpublished results) similar values for the digestibility of dietary fibre from wheat were found using the same types of bread as in our human experiments. In addition, it is surprising that the digestibility is the same for all types of bread tested in the present study, viz. white bread with practically no visible fibre, wholemeal bread and bread prepared from coarse and fine brans. In particular, a more extensive degradation might have been expected for fine bran, because of the larger surface area of the particles.

Summarizing, we observed a nearly complete degradation in the gut of the small amount of fibre in our basal diet in most of the subjects, whereas in all subjects approximately two-thirds of the fibre from bread were recovered from the faeces. The sorptive properties of fibre in the large intestine seem to be mainly responsible for the bulking effect, which is considered to be beneficial to the colon function (Kay, 1982). Consequently, the importance of including an ample amount of bread, preferably high in coarse fibre, in the diet should be emphasized.

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REFERENCES

- Burkitt, D. P. & Trowell, H. C. (1975). *Refined Carbohydrate Foods and Disease: Some Implications of Dietary Fibre*. London: Academic Press.
- Burkitt, D. P., Walker, A. R. P. & Painter, N. S. (1972). *Lancet* **ii**, 1408–1412.
- Bustos Fernandez, L., Gonzalez, E., Marzi, A. & Ledesma de Paolo, M. I. (1971). *New England Journal of Medicine* **284**, 295–298.
- Cummings, J. H., Hill, M. J., Jenkins, D. J. A., Pearson, J. R. & Wiggins, H. S. (1976a) *American Journal of Clinical Nutrition* **29**, 1468–1473.
- Cummings, J. H., Jenkins, D. J. A. & Wiggins, H. S. (1976b). *Gut* **17**, 210–218.
- Dawson, A. M., Holdsworth, C. D. & Webb, J. (1964). *Proceedings of the Society for Experimental Biology and Medicine* **117**, 97–100.
- Dintzis, F. R., Legg, L. M., Deatherage, W. L., Baker, F. L., Inglett, G. E., Jacob, R. A., Reck, S. J., Munoz, J. M., Klevay, L. M., Sandstead, H. H. & Shuey, W. C. (1979). *Cereal Chemistry* **56**, 123–127.
- Eastwood, M. A. (1974). *Journal of the Science of Food and Agriculture* **25**, 1523–1527.
- Eastwood, M. A. & Kay, R. M. (1979). *American Journal of Clinical Nutrition* **32**, 364–367.
- Fantus, B., Hirschberg, N. & Frankl, W. (1941). *Review of Gastroenterology* **8**, 277–280.
- Floch, M. H. & Fuchs, H. M. (1978). *American Journal of Clinical Nutrition* **31**, S185–S189.
- Fordtran, J. S. (1971). *New England Journal of Medicine* **284**, 329–330.
- Forsyth, W. A., Chenoweth, W. L. & Bennink, M. R. (1978). *Journal of Food Science* **43**, 1470–1472.
- Heller, S. N., Hackler, L. R., Rivers, J. M., Van Soest, P. J., Roe, D. A., Lewis, B. A. & Robertson, J. (1980). *American Journal of Clinical Nutrition* **33**, 1734–1744.
- Hinton, J. M., Lennard-Jones, J. E. & Young, A. C. (1969). *Gut* **10**, 842–847.
- Holloway, W. D., Tasman-Jones, C. & Lee, S. P. (1978). *American Journal of Clinical Nutrition* **31**, 927–930.
- Jelaca, S. L. & Hlynka, I. (1971). *Cereal Chemistry* **48**, 211–222.
- Kay, R. M. (1982). *Journal of Lipid Research* **23**, 221–242.
- Kay, R. M. & Truswell, A. S. (1977). *British Journal of Nutrition* **37**, 227–235.
- Kelsay, J. L. (1978). *American Journal of Clinical Nutrition* **31**, 142–159.
- Kirwan, W. O., Smith, A. N., McConnell, A. A., Mitchell, W. D. & Eastwood, M. A. (1974). *British Medical Journal* **iv**, 187–189.
- McNeil, N. I., Cummings, J. H. & James, W. P. T. (1978). *Gut* **19**, 819–822.
- Mitchell, W. D. & Eastwood, M. A. (1976). In *Fiber in Human Nutrition*, pp. 185–206 [G. A. Spiller and R. J. Amen, editors]. New York: Plenum Press.
- Noel, R. J. (1976). *Journal of the Association of Official Analytical Chemists* **59**, 141–147.
- Payler, D. K., Pomare, E. W., Heaton, K. W. & Harvey, R. F. (1975). *Gut* **16**, 209–213.
- Schormüller, J. (1967). *Handbuch der Lebensmittelchemie*, vol. 2, p. 77. Berlin: Springer Verlag.
- Schormüller, J. (1969). *Handbuch der Lebensmittelchemie*, vol. 4, p. 423. Berlin: Springer Verlag.
- Smith, A. N., Drummond, E. & Eastwood, M. A. (1981). *American Journal of Clinical Nutrition* **34**, 2460–2463.
- Snedecor, G. W. & Cochran, W. G. (1967). *Statistical Methods*, 6th ed. Ames, Iowa: Iowa State University Press.
- Spiller, G. A., Shipley, E. A. & Blake, J. A. (1978). *Critical Reviews in Food Science and Nutrition* **10**, 31–91.
- Stephen, A. M. & Cummings, J. H. (1979). *Gut* **20**, A457.
- Terry, R. A. & Outen, G. E. (1973). *Chemistry and Industry, London* **23**, 1116–1117.
- Thomas, B. & Elchazly, M. (1976). *Qualitas Plantarum—Plant Foods for Human Nutrition* **26**, 211–216.
- Trowell, H. (1976). *American Journal of Clinical Nutrition* **29**, 417–427.
- Tucker, D. M., Sandstead, H. H., Logan, G. M., Klevay, L. M., Mahalko, J., Johnson, L. K., Inman, L. & Inglett, G. E. (1981). *Gastroenterology* **81**, 879–883.
- van de Kamer, J. H. (1941). *Chemisch Weekblad* **38**, 286–288.
- van de Kamer, J. H., Gerritsma, K. W. & Wansink, E. J. (1955). *Biochemical Journal* **61**, 174–176.
- Van Dokkum, W., Wesstra, A. & Schippers, F. A. (1982). *British Journal of Nutrition* **47**, 451–460.
- Van Soest, P. J. & Robertson, J. B. (1977). *Nutrition Reviews* **35**, M12–22.
- Van Soest, P. J. & Wine, R. H. (1967). *Journal of the Association of Official Analytical Chemists* **50**, 50–55.
- Williams, R. D. & Olmsted, W. H. (1936). *Journal of Nutrition* **11**, 433–449.
- Wymann, J. B., Heaton, K. W., Manning, A. P. & Wicks, A. C. B. (1976). *American Journal of Clinical Nutrition* **29**, 1474–1479.