Effect of Oat Hull Fiber on Human Colonic Function and Serum Lipids

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ABSTRACT

Oat hull fiber is an insoluble source of dietary fiber, derived from the outermost layer of the oat grain. The effect of oat hull fiber on colonic function and serum lipids was investigated by conducting a controlled study on 10 healthy males, aged 20–37, who ate, for two three-week periods, a controlled low fiber diet (13.1 g of nonstarch polysaccharide [NSP]/day), and the same diet with 25 g of oat hull fiber per day incorporated into foods, providing 17 g of NSP/day. Fecal weight increased from 113 ± 10.4 to 155 ± 10.8 g/day ($P < 0.001$) with no change in transit time or serum lipids. Fermentation of oat hull fiber was studied by analysis of feces for NSP. Excretion of NSP increased from 2.0 g/day excreted to 19.7 g/day, indicating that no degradation had occurred. Oat hull fiber is therefore resistant to fermentation in the human colon, has no effect on serum lipids, and provides no energy to the body

Oat hull fiber is the outermost layer of the oat grain, external to the endosperm and the bran layers. On analysis, oat hull fiber has been shown to contain high amounts of cellulose, insoluble noncellulosic polysaccharides, and lignin (Smith et al 1980, Stanogias and Pearce 1985). Feeding studies in pigs have shown oat hull fiber to be largely indigestible (Stanogias and Pearce 1985). Insoluble fiber is necessary in the human diet to maintain healthy colonic function and reduce constipation. Hence, sources of insoluble fiber, in addition to wheat bran, are being sought by the food industry. Traditionally, oat hulls have been discarded during processing, but the need for concentrated, insoluble fiber sources for human consumption has resulted in the production of oat hull fiber for human food use. Although the effects of feeding oat hull fiber have been studied in pigs (Moser et al 1982, Moore et al 1986), rats (Barke and Harrold 1980), chicks (Thompson and Weber 1981), and cattle (Smith et al 1980), there has not been a controlled study of oat hull fiber as an addition to the human diet. This study examines the effects of oat hull fiber on colonic function and serum lipids in healthy human volunteers. The fermentability of oat hull fiber in the human gastrointestinal tract has also been investigated by analysis of fecal samples for nonstarch polysaccharides (NSP).

METHODS

Ten healthy male volunteers, aged 20–37 years (25.1 ± 5.6), who were free from gastrointestinal symptoms, were recruited through notices around the campus of the University of Saskatchewan. Subjects were of normal body weight, had a body mass index (BMI) of 20.2–26.5 (23.7 ± 2.0), and had not taken antibiotics in the weeks prior to the beginning of the study. Only men were included in the study because of the hormonal influences on gastrointestinal function that women experience during the course of the menstrual cycle (Turnbull et al 1989, Davies et al 1993) which could interfere with the interpretation of results. The subjects took part in a study of eight weeks duration, six weeks of which were a period of strict dietary control, in a double cross-over design. For one week in the month before beginning the dietary control, subjects made a seven-day weighed record of all food consumed using portable electronic tape recording automated scales (PETRA, Cherlyn Electronics, Cambridge, UK), which record all weights of food placed on the scale on tape while the subject verbally describes each food item into a microphone (Bingham et al 1985, Barker et al 1988). Nutrient intake was assessed using a nutritional assessment program (NUTS, Quilchena, Vancouver, BC). The average energy intake for the seven-day assessment was used as a guide for providing the appropriate energy level to maintain body weight of each subject during the period of dietary control.

During the six weeks of diet control, the subjects consumed, for three weeks, a typically North American diet with a three-day diet rotation. The details of the diet are given in Table I. Each subject was given an amount of energy to maintain weight, but all diets had the same content of NSP and starch, both of which affect fecal output (Cummings et al 1978, 1996; Shetty and Kurpad 1986; Stephen et al 1986). Starch intake was calculated using food tables (Holland et al 1989, 1991). Total NSP and NSP components were calculated using published data of Englyst et al (1988, 1989) as shown in Table II. Energy was adjusted using an increment of foods that did not contain fiber or starch but had the same proportion of energy from protein, fat, and carbohydrate as the basal diet. Incremented foods are indicated in Table I. Carbohydrate intake was increased or decreased using refined sugar, grape jelly, or jelly beans, depending on individual preferences. The diet contained no seeds or skins, as these would appear in stools and create sampling

TABLE I
Composition of the Study Diet[a]

Daily	Day 1	Day 2	Day 3
Orange juice, 250 g	Beef, 75 g	Salmon, 60 g	Turkey, 60 g
Sugar, 45 g[b]	Apricots, 120 g	Fruit cocktail, 130 g	Peaches, 140 g
White bread, 50 g	Pork, 75 g[b]	Chicken, 90 g[b]	Beef, 85 g[b]
Butter, 20 g[b]	Broccoli, 50 g	Cauliflower, 60 g	Green beans, 80 g
Cheese, 20 g	Carrots, 50 g	Coleslaw, 69 g	Mushrooms
Cucumber, 20 g			
Canned beets, 20 g			
Iceberg lettuce, 20 g			
Salad dressing, 25 g			
Potato, 100 g			
Apple (no skin), 120 g			
Ice cream, 50 g[b]			
Cornflakes, 20 g			
Milk, 325 g			
Muffins[c]			
Cookies[c]			

[a] For both the control and oat hull fiber periods, the basal diet had an energy value of 1,110 kJ, with 13.4% energy from protein, 37.1% from fat, and 51.2% from carbohydrate. Intake of nonstarch polysaccharide was 13.1 g/day in the control period and 29.9 g/day in the oat hull fiber period.
[b] Items incremented for energy intake.
[c] Muffins and cookies containing oat hull fiber were made with a smaller amount of flour to account for the small amount of starch in the oat hull fiber (5.6%).

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difficulties when analyzing for NSP. For the other three weeks of the diet, the control diet was supplemented with 25 g of oat hull fiber per day supplied by Canadian Harvest Ltd. The oat hull fiber was produced by grinding oat hulls to 35/60 mesh, resulting in a product 0.2–0.4 mm. The oat hull fiber provided 17 g of NSP per day (Table III). NSP is the sum of the constituent monosaccharides, analyzed by the method of Englyst et al (1982). The oat hull fiber was incorporated into muffins and cookies, with adjustments in the quantity of flour used to equalize carbohydrate content. Control cookies and muffins were made with brown sugar so that both types had the same appearance. Subjects and all research personnel, except the principal investigator and the individual distributing the cookies and muffins, were blinded to the order of treatment; the order of the two periods was randomized.

Subjects obtained all food from the metabolic unit of the Division of Nutrition and Dietetics, College of Pharmacy and Nutrition, University of Saskatchewan. They could not consume any food other than that provided, and they had to consume all food given to them. They were not permitted alcohol. They were permitted instant coffee, the amount used being weighed weekly. Subjects collected their food daily from the metabolic unit and took it to their homes, although they often consumed some meals in the unit while on campus.

Fecal Collections

During the six weeks of diet control, subjects took radio-opaque pellets (three capsules each containing 10 markers daily) for the correction of fecal output for intake (Branch and Cummings 1978) and for the measurement of mean transit time (MTT) (Cummings et al 1976b). Subjects made complete fecal collections throughout the study, including one week after the period of diet control to ensure complete recovery of markers. Each stool was collected into a plastic bag and immediately frozen. Weighed frozen stools were then x-rayed in batches in the Department of Medical Imaging, Royal University Hospital, Saskatoon, to determine the excretion of radio-opaque markers. MTT was calculated using the continuous marker method (Cummings et al 1976b). Marker-corrected fecal weights (g/day) = mean daily fecal weight for third week of each three-week period × marker-correction factor. Marker-correction factor = 210 (intake of markers in seven days)/markers excreted in week 3 (Branch and Cummings 1978).

Fecal Sample Analyses

Pooled fecal collections from the third week of each three-week period were freeze-dried for one week (Edwards Supermodulyo freeze dryer). The dried stools were weighed, crushed with a rolling pin, mixed well, and stored for analysis.

Aliquots (200–300 mg) of freeze-dried stools were analyzed for NSP by the method of Englyst et al (1982). Briefly, this method hydrolyzes the monosaccharides that make up the NSP and analyzes the individual sugars as alditol acetates by gas chromatography. By using three different samples and appropriate conditions, the method separates soluble NSP from insoluble NSP and cellulose from noncellulosic polysaccharide (NCP). Cellulose is calculated as the difference in glucose values with and without the addition of concentrated sulfuric acid to the sample. Excretion in grams per day was calculated by multiplying grams of NSP excreted in 100 g of fecal dry matter by the marker-corrected fecal dry weight. Because fecal NSP includes bacterial polysaccharides, the extent of fermentation of oat hull fiber NSP was calculated by subtraction of fecal cellulose and pentose from the control period from fecal cellulose and pentose from the oat hull fiber period. The difference in total NSP was also calculated, but this includes any changes in bacterial polysaccharides.

Blood Analyses

Fasting blood samples were taken at the beginning of the study and on two days at the end of each three-week period to provide duplicate samples. Serum was analyzed for total cholesterol and triglycerides using the Boehringer Mannheim automated analysis system. High-density lipoprotein (HDL) cholesterol was analyzed using precipitation with phosphotungstic acid and magnesium (Lopes-Virella et al 1977). Low-density lipoprotein (LDL) was calculated using the Friedewald equation (Friedewald et al 1972). Analyses were performed in the Department of Pathology, Royal University Hospital, Saskatoon.

Subjects were weighed weekly. They also kept a diary, noting each marker capsule taken, each stool passed, and any unusual occurrences or symptoms. This study was approved by the University Advisory Committee on Ethics in Human Experimentation of the University of Saskatchewan. All results were compared by paired *t*-tests, unless otherwise stated.

TABLE II
Nonstarch Polysaccharide (NSP) Components in the Control Diet[a,b]

NSP	Total	Cellulose	Rha	Fuc	Ara	Xyl	Man	Gal	Glu	UAc
Daily	4.93	1.47	0.07	0.02	0.74	0.60	0.08	0.67	0.26	0.92
Days 1–3 (average)	4.36	1.53	0.10	trace	0.53	0.28	0.11	0.50	0.13	1.17
Muffin portion	2.06	0.15	trace	trace	0.56	0.68	0.11	0.11	0.23	0.22
Cookie portion	1.79	0.05	trace	trace	0.60	0.75	0.05	0.10	0.25	trace
Total daily	13.14	3.20	0.17	0.02	2.43	2.31	0.35	1.38	0.87	2.31

[a] Rha = Rhamnose, Fuc = Fucose, Ara = Arabinose, Xyl = Xylose, Man = Mannose, Gal = Galactose, Glu = Glucose, UAc = Uronic acids.
[b] Calculated using values of Englyst et al (1988, 1989).

TABLE III
Analysis of Oat Hull Fiber for Nonstarch Polysaccharide (NSP) Components

			Noncellulosic Polysaccharides[a]							
	Total	Cellulose	Rha	Fuc	Ara	Xyl	Man	Gal	Glu	UAc
Oat hull fiber (dry matter)/100 g	...	...	...	...	...	...	...	...	...	...
Soluble NSP	0.1		...	...	...	...	...	...	...	...
Insoluble NSP	70.8	30.9	...	...	3.7	33.2	0.1	1.1	0.6	1.2
Total NSP	70.9	30.9	...	...	3.7	33.2	0.1	1.1	0.7	1.2
Oat hull fiber amount fed (25 g)	...	...	...	...	...	...	...	...	...	...
Soluble NSP	...	...	...	...	...	...	...	...	...	...
Insoluble NSP	16.9	7.4	...	...	0.9	7.9	...	0.3	0.1	0.3
Total NSP	16.9	7.4	...	...	0.9	7.9	...	0.3	0.1	0.3

[a] Rha = Rhamnose, Fuc = Fucose, Ara = Arabinose, Xyl = Xylose, Man = Mannose, Gal = Galactose, Glu = Glucose, UAc = Uronic acids.

RESULTS

All subjects complied with the six weeks of dietary control, taking of markers, blood sampling, and complete fecal collections. Compliance with the diet was checked by informal questioning of subjects during meals consumed on campus. Compliance with fecal collections was determined by radio-opaque marker excretion; complete fecal collections are indicative of good compliance with other aspects of the study. Mean body weight was 72.4 ± 2.8 kg at the beginning of the study and 71.7 ± 2.8 kg at the end (not significant). There was no effect on body weight of the oat hull fiber treatment: control period initial weight 72.0 ± 2.9 kg, final weight 72.0 ± 2.8 kg; oat hull fiber period initial weight 72.0 ± 2.8 kg, final weight 71.8 ± 2.8 kg. All subjects except one lost a little weight during the study, usually <1 kg. No subjects reported any adverse gastrointestinal effects of eating oat hull fiber, although several felt more constipated on the control diet than on their usual diets.

Fecal Weights

The marker-corrected fecal weights, fecal dry weights, and MTT are shown in Table IV. The individual changes in fecal weight are shown in Fig. 1. These results demonstrate an increase in fecal output in each subject, with an average increase of 42 g/day or 37.2%. There was no effect of the order of treatment. The five subjects who had the control diet first had an increase in fecal weight of 51 g/day on average. The subjects that had the fiber diet first had an increase in fecal weight of 33 g/day (not significant). Fecal dry weight also increased in each subject; the average increase was 22.8 g/day (19.0–25.0 g/day) (Table V).

NSP Excretion

The individual results for the fecal excretion of total NSP for both the control and oat hull fiber periods are given in Table V. The average excretion of NSP for the control diet was 2.0 g/day (1.1–4.0 g/day), of which 26.2%, on average, was soluble NSP and 73.8% was insoluble NSP. This corresponds to an average 85% being fermented. Excretion of NSP components and their fermentation for the control period are shown in Table VI. Fecal polysaccharide excretion includes rhamnose and fucose, which are monosaccharides present in fecal bacteria, as is some of the fecal glucose. Mannose and galactose are also present in bacteria, but in much smaller amounts. Uronic acids might also be present, but in extracellular polysaccharide (Salton 1964). The pentoses, arabinose and xylose, as well as cellulose, are mainly present in plant material. It is therefore appropriate to consider them separately. Fig. 2 shows the extent of fermentation of cellulose and pentose for the control diet in relation to MTT. Both cellulose and pentose show maximum fermentability >90%. Reduced fermentation occurred in two individuals with very fast transit times. Fermentation of cellulose was more affected by very fast transit than was fermentation of pentose. The two subjects with a MTT of <30 hr had <60% of cellulose fermented.

The fermentation of the NSP, and of the pentose and cellulose in particular, for the oat hull fiber period are shown in Table VII. The average excretion with oat hull fiber was 19.7 g/day (17.3–23.2 g/day), of which only 3.3%, on average, was soluble NSP. By subtracting the total NSP excretion from the control period, a figure of 17.7 g/day is obtained, which most closely represents the amount of added oat hull fiber remaining. When this is converted to the extent of oat hull fiber fermented, a negative value is obtained,

TABLE IV
Effect of Oat Hull Fiber on Gastrointestinal Function[a]

	Control	Oat Hull Fiber
Marker-corrected fecal weight (g/day)	113 ± 10.4	155 ± 10.8***
Marker-corrected fecal dry weight (g/day)	22.2 ± 1.3	45.0 ± 1.3***
Mean transit time (hr)	44.3 ± 4.1	42.0 ± 3.9 (NS)

[a] Mean ± standard error of the mean. *** = $P < 0.0001$.

TABLE V
Fecal Excretion of Nonstarch Polysaccharide (NSP)

	Control			Oat Hull Fiber		
Subject	MCDW[a] (g/day)	% Total NSP	Fecal NSP (g/day)	MCDW[a] (g/day)	% Total NSP	Fecal NSP (g/day)
1	19.1	7.5	1.4	43.8	44.1	19.3
2	29.9	13.3	4.0	51.4	42.1	21.6
3	21.7	6.4	1.4	44.1	42.1	18.6
4	20.5	7.9	1.6	42.8	44.3	19.0
5	23.3	14.1	3.3	47.3	49.0	23.2
6	15.1	7.2	1.1	39.2	43.9	17.2
7	22.3	7.4	1.6	41.3	41.9	17.3
8	25.2	7.8	2.0	50.2	39.2	19.7
9	19.6	8.3	1.6	42.3	45.1	19.1
10	25.6	8.6	1.6	47.9	45.0	21.6
Mean	22.2	8.8	2.0	45.0***[b]	43.7***	19.7***
SEM[c]	1.3	0.8	0.3	1.3	0.8	0.6

[a] Marker-corrected dry weight.
[b] *** = $P < 0.001$.
[c] Standard error of the mean.

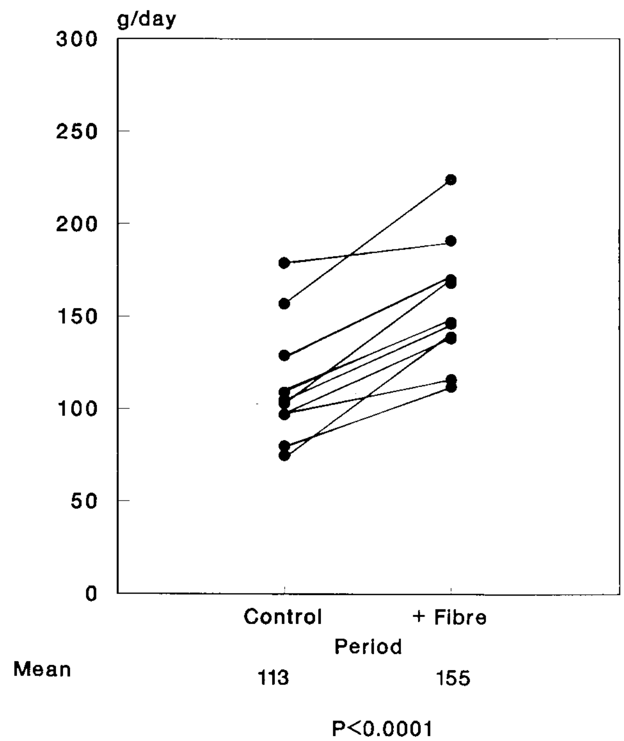

Fig. 1. Effect of oat hull fiber on fecal weight in individual subjects.

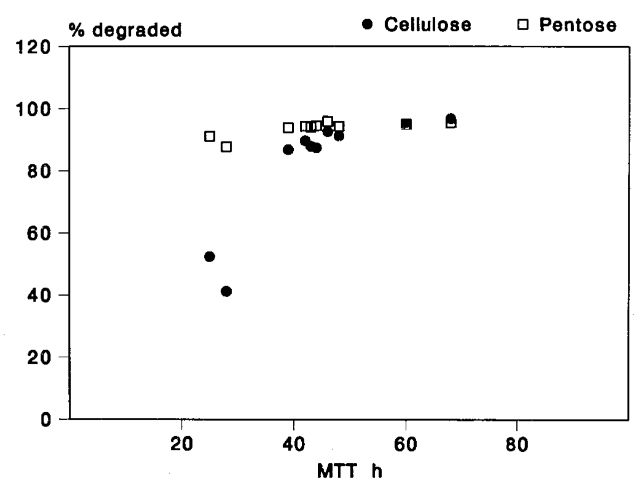

Fig. 2. Fermentation of cellulose and pentose in individual subjects in relation to mean transit time (MTT) for the control period.

TABLE VI
Recovery and Fermentation of Nonstarch Polysaccharide (NSP) Components from the Control Diet[a]

Subject	Total	Cellulose	Rha	Fuc	Ara	Xyl	Man	Gal	Glu	UAc
1	1.43	0.30	0.11	0.04	0.10	0.15	0.08	0.21	0.29	0.15
2	3.98	2.00	0.12	0.06	0.18	0.36	0.30	0.39	0.33	0.24
3	1.39	0.17	0.17	0.04	0.08	0.13	0.07	0.26	0.35	0.11
4	1.62	0.45	0.10	0.04	0.10	0.16	0.08	0.20	0.33	0.14
5	3.28	1.63	0.12	0.05	0.12	0.28	0.21	0.26	0.47	0.16
6	1.09	0.11	0.20	trace	0.08	0.12	0.05	0.18	0.26	0.11
7	1.65	0.25	0.47	trace	0.09	0.09	0.07	0.29	0.22	0.13
8	1.97	0.35	0.40	trace	0.10	0.15	0.10	0.38	0.28	0.20
9	1.63	0.43	0.18	0.04	0.10	0.16	0.08	0.25	0.20	0.20
10	1.64	0.41	0.31	0.04	0.10	0.18	0.08	0.26	0.18	0.13
Average	1.97	0.61	0.22	0.03	0.16	0.18	0.11	0.27	0.29	0.16
SEM[b]	0.29	0.21	0.04	0.01	0.03	0.03	0.03	0.02	0.03	0.01
% Fermented	85.0	80.9	nc[c]	nc	93.4	92.2	nc	nc	nc	nc

[a] Rha = Rhamnose, Fuc = Fucose, Ara = Arabinose, Xyl = Xylose, Man = Mannose, Gal = Galactose, Glu = Glucose, UAc = Uronic acids.
[b] Standard error of the mean.
[c] Fermentation not calculated as these monosaccharides are present in fecal bacteria.

TABLE VII
Recovery of Nonstarch Polysaccharide (NSP) from Oat Hull Fiber[a]

	NSP Recovered (g/day)			NSP Fermented (%)		
Subject	Total	Cellulose	Pentose	Total	Cellulose	Pentose
1	17.9	8.3	8.7	–5.3	–12.2	1.1
2	17.6	7.7	8.7	–10.7	–4.1	1.1
3	17.2	7.5	8.7	–1.8	–1.4	1.1
4	17.4	8.2	8.8	3.0	–10.8	0
5	19.9	7.1	8.1	–4.7	4.1	8.0
6	16.1	7.4	8.1	10.7	0	8.0
7	15.7	7.2	7.4	7.1	2.7	15.9
8	17.7	8.0	9.1	–4.7	–8.1	–3.4
9	17.5	7.6	9.2	–3.6	–2.7	–4.5
10	20.0	8.9	10.3	–18.3	–20.3	–17.0
Mean	17.7	7.8	8.7	–2.8	–5.3	1.0

[a] NSP fed at 16.9 g/day (7.4 g of cellulose, 8.8 g of pentose).

suggesting that the NSP in the oat hull fiber remains totally undegraded, and that the fermentation of the fiber from the control diet is slightly reduced when oat hull fiber is fed. Table VII also shows that, like total NSP, cellulose in the added fiber is completely undegraded, and more cellulose from the control diet also remains undegraded. Pentose in the oat hull fiber was degraded an average of 1.0%.

Serum Lipids

The results for serum lipids are shown in Table VIII. Oat hull fiber had no effect on serum total cholesterol, triglycerides, HDL, LDL, or VLDL cholesterol.

DISCUSSION

Oat hull fiber is a highly insoluble fiber source, made up mainly of cellulose and pentoses, particularly xylose. Like the chemical profile of the fiber in wheat bran, it is high in insoluble pentoses and cellulose, although as a proportion of total NSP, pentose content is slightly lower and the cellulose slightly higher in oat hull (Englyst et al 1989).

Oat hull fiber had a significant effect on fecal output, increasing wet and dry stool weight in every subject. The size of the bulking effect (37%) is somewhat lower than has been shown for an equivalent amount of wheat bran (Cummings et al 1976a, 1978), but the oat hull fiber was of a considerably smaller particle size than the wheat bran normally fed in fiber supplementation studies. Particle size is known to affect the extent of bulking, smaller particles having a small effect (Wrick et al 1983). Where wheat bran has been fed at a similarly small particle size, the extent of bulking was similar to that shown here for oat hull fiber (Stephen et al 1986).

The analysis of fecal samples in this study indicated that oat hull fiber is a highly resistant material, and it travels through the entire gastrointestinal tract undigested and nonfermented. Moreover, it appeared to reduce the fermentability of the NSP in the control diet. The speed of transit through the intestinal tract affects NSP fermentability, particularly when transit is fast (Stephen et al 1987). In this study, conducted on young, fairly active men, transit times were fast even on the control diet, with a mean value of 44 hr (25–68 hr). This may explain why the oat hull fiber had no significant effect on transit, because it is difficult to speed up an already fast transit time, and it may also explain why the oat hull fiber was, in most cases, totally undegraded. It may be possible that some fermentation of oat hull fiber might occur in individuals who have very slow transit times (e.g., >100 hr), but this is unlikely to be extensive given the chemical composition of oat hull fiber. The NSP on the control diet was largely fermented, including the cellulose component. Cellulose is often considered to be inert and nonfermentable because purified cellulose is excreted largely unchanged (Slavin et al 1981, Stephen 1989), but the extent of fermentation clearly depends on the foods in which cellulose is located. The cellulose in the control diet is distributed among a variety of foods and, in that form, can be seen to be fermented.

The role of fermentation in overall health is an area of considerable interest at present. Short-chain fatty acids, particularly acetate, propionate, and butyrate, the products of anaerobic colonic fermentation, are hypothesized to have a number of beneficial effects, both in the colon and elsewhere in the body (Cummings 1981, Roediger 1982, Chen et al 1984, Amaral et al 1993). Propionate, for example, has been shown to lower cholesterol in animals (Chen et al 1984) and human subjects (Amaral et al 1993), and it has been put forward as a possible mechanism whereby soluble fiber, as in pectin and oat bran, lowers cholesterol (Anderson and Gustafson 1988). Butyrate has been suggested as an important energy source for the colonic mucosal cells (Roediger 1982), and

TABLE VIII
Effect[a] of Oat Hull Fiber on Serum Lipids

Lipids[b] (mmol/L)	Background	Control	Oat Hull Fiber
Total cholesterol	5.1 ± 0.1	5.2 ± 0.2	5.2 ± 0.2
HDL cholesterol	1.2 ± 0.1	1.2 ± 0.1	1.2 ± 0.1
LDL cholesterol	3.5 ± 0.2	3.6 ± 0.2	3.5 ± 0.2
VLDL cholesterol	0.4 ± 0.05	0.4 ± 0.02	0.4 ± 0.03
Triglycerides	1.05 ± 0.12	0.82 ± 0.06	0.83 ± 0.07

[a] Mean ± standard error of the mean.
[b] HDL = high-density lipoprotein; LDL = low-density lipoprotein; VLDL = very low-density lipoprotein.

it may have anticarcinogenic effects (Cummings 1981). Short-chain fatty acids may also supply energy to the body. Acetate, for example, may be a contributor to the energy supply because it is incorporated through the normal metabolic processes into long-chain fatty acids; propionate may be an additional although lesser source of energy. The energy value of dietary fiber is being debated at present; clearly, the energy content of any particular fiber source will be determined by the extent of its fermentation.

The present study indicates that oat hull fiber is not fermented in the human large intestine and passes through to the feces intact. Any possible contributions of short-chain fatty acids to overall energy supply or to other processes cannot occur with oat hull fiber because no fermentation takes place. An effect on serum cholesterol would therefore not be expected, and none occurred. Furthermore, oat hull fiber cannot supply energy to the body and may even reduce slightly that energy available from fermentation of the fiber in the control diet. In determining the energy provided by each new fiber source, the extent of fermentation must be determined, because each source will contribute a different amount of energy. One way to assess this is to measure accurately the fecal excretion of NSP and compare the excretion with NSP provided in the fiber material as eaten.

Insoluble fiber sources that resist fermentation, like oat hull fiber, have benefits for human health in that they increase stool bulk and thus reduce constipation. Fiber sources vary dramatically in their effects on the body; the extent to which each is fermented will determine the response to any particular source.

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