Table IX. Fecal Oxalic Acid Excretion of Twelve Men Consuming Different Levels of Fiber from Fruits and Vegetables (Mean ± SEM)[a]

	Study I low fiber	higher fiber
mg/day	41 ± 6[b]	423 ± 53[a]

	diet 1	diet 2	diet 3	diet 4
Study II				
mg/day	21 ± 3[d]	81 ± 8[c]	157 ± 14[b]	210 ± 16[a]

[a] Row means followed by the same letter are not significantly differnet at the 5% level according to Duncan's multiple range comparisons.

IX. In study I, fecal oxalic acid was 10 times as high on the higher fiber diet as on the low fiber diet. In study II, fecal oxalic acid increased with the fiber in the diet. The diets in study II did not include spinach but included other foods that contained oxalic acid in smaller amounts. Oxalic acid excretion on diet 3 in study II was only about one-third of that on the similar diet in study I. The 423 mg of oxalic acid excreted on the higher fiber diet in study I could conceivably tie up 188 mg of calcium, which might have been just enough to result in negative calcium balance.

Further studies are needed to determine whether greater levels of intake, different sources, or different forms of fiber would affect mineral balances. The effects of phytic acid and oxalic acid with and without fiber in the diet should be defined. The time required for adaptation of the mineral balances of subjects to interfering factors should be determined in long-term controlled studies.

ACKNOWLEDGMENT

The authors express their appreciation to Priscilla Steele and Sheryl Cottrell of the Diet Facility for their assistance in conducting this study.

LITERATURE CITED

American Association of Cereal Chemists "Approved Methods of the AACC"; 1978 Revisions; AACC: St. Paul, MN, 1978; Method 32-40.
Basu, K. P.; Ghosh, D. *Indian J. Med. Res.* **1943**, *31*, 37.
Bonner, P.; Hummel, F. C.; Bates, M. F.; Horton, J.; Hunscher, H. A.; Macy, I. G. *J. Pediatr. (St. Louis)* **1938**, *12*, 188.
Brodribb, A. J. M.; Humphreys, D. M. *Br. Med. J.* **1976**, *1*, 424.
Dorfman, S. H.; Ali, M.; Flock, M. H. *Am. J. Clin. Nutr.* **1976**, *29*, 87.
Drews, L. M.; Kies, C.; Fox, H. M. *Am. J. Clin. Nutr.* **1979**, *32*, 1893.
Eastwood, M. A.; Kirkpatrick, J. R.; Mitchell, W. D.; Bone, A.; Hamilton, D. *Br. Med. J.* **1973**, *4*, 392.
Goering, H. K.; Van Soest, P. J. *U.S., Dep. Agric., Agric. Handb.* **1970**, *No. 379*.
Guthrie, B. E.; Robinson, M. F. *Fed. Proc., Fed. Am. Soc. Exp. Biol.* **1978**, *37*, 254.
Harvey, R. F.; Pomare, E. W.; Heaton, K. W. *Lancet* **1973**, *1*, 1278.
Heller, S. N.; Hackler, L. R. *Am. J. Clin. Nutr.* **1978**, *31*, 1510.
Ismail-Beigi, F.; Reinhold, J. G.; Faradji, B.; Abadi, P. *J. Nutr.* **1977**, *107*, 510.
Johnston, F. A.; McMillan, T. J.; Falconer, G. D. *J. Am. Diet. Assoc.* **1952**, *28*, 933.
Kelsay, J. L. *Am. J. Clin. Nutr.* **1978**, *31*, 142.
Kelsay, J. L.; Behall, K. M.; Prather, E. S. *Am. J. Clin. Nutr.* **1978**, *31*, 1149.
Kelsay, J. L.; Behall, K. M.; Prather, E. S. *Am. J. Clin. Nutr.* **1979a**, *32*, 1876.
Kelsay, J. L.; Jacob, R. A.; Prather, E. S. *Am. J. Clin. Nutr.* **1979b**, *32*, 2307.
Kies, C.; Fox, H. M.; Beshgetoor, D. *Cereal Chem.* **1979**, *56*, 133.
McLaughlin, L. *J. Biol. Chem.* **1927**, *74*, 455.
"Recommended Dietary Allowances", 8th ed.; National Academy Sciences, National Research Council: Washington, DC, 1974.
Reinhold, J. G.; Faradji, B.; Abadi, P.; Ismail-Beigi, F. *J. Nutr.* **1976**, *106*, 493.
Sandstead, H. H.; Munoz, J. M.; Jacob, R. A.; Klevay, L. M.; Reck, S. J.; Logan, G. M., Jr.; Dintzis, F. R.; Inglett, G. E.; Shuey, W. C. *Am. J. Clin. Nutr.* **1978**, *31*, S180.
Schlutz, F. W.; Morse, M.; Oldham, H. *Am. J. Dis. Child.* **1933**, *46*, 758.
Sherman, H. C.; Hawley, E. *J. Biol. Chem.* **1922**, *53*, 375.

Received for review June 23, 1980. Accepted November 26, 1980. Presented at the 179th National Meeting of the American Chemical Society, Houston, TX, March 23–28, 1980, Division of Agricultural and Food Chemistry.

Nutritional Significance of Dietary Fiber: Effect on Nutrient Bioavailability and Selected Gastrointestinal Functions

R. Ali,* H. Staub, G. Coccodrilli, Jr., and L. Schanbacher

Fiber in foods varies considerably in composition and physicochemical properties. Water-active and gel-forming polysaccharides modify viscosity of the gut content and may influence bioavailability of nutrients. All undigestible structured fibers reach the large bowel relatively unchanged to exert a direct effect on gut microflora and motility. Conflicting results in the literature do not allow definitive conclusions to be drawn regarding the effect of dietary fiber on requirements of macro- and micronutrients in experimental animals and man. The role of fiber, however, in relieving symptoms associated with chronic constipation and diverticular diseases is well documented. The favorable changes in rate of gastric emptying and intestinal transit time and motility seem to be related mainly to the bulking properties of dietary fiber which decrease the forcefulness of contractile pressure in the lumen. Furthermore, metabolites of bacterial fermentation of dietary polysaccharides which modify the environment in the colon may positively contribute to promotion of regularity.

The significance of dietary fiber in nutrition is the subject of wide scientific review (Heaton, 1979; Kelsay, 1978; Kimura, 1977; Roth and Mehlman, 1978; Spiller and Amen, 1975, 1976, 1978; Staub and Ali, 1981). Little is known about quantitative intake of dietary fiber in relation to health, and qualitative differences in composition and lack of agreement on a single definition makes even current estimates of dietary fiber intake suspect. Although the

Department of Nutrition and Health Sciences, General Foods Technical Center, Tarrytown, New York 10591.

0021-8561/81/1429-0465$01.25/0

mechanism(s) involved is (are) not clearly understood, it is generally accepted that source, level, and chemical composition of dietary fiber contribute to nutrition by influencing nutrient bioavailability and modifying the environment in the colon. This review deals with some nutritional aspects of dietary fiber in relation to selected gastrointestinal functions.

DIGESTIBILITY AND AVAILABILITY OF DIETARY FIBER

Fiber, whether crude or dietary, is not digested by mammalian enzymes but is acted on by the flora in the colon. Mangold (1934) recognized that digestibility of crude fiber by both animals and man was of great importance in utilization of plant foodstuffs. He reported digestibility coefficients in man ranging from ~25% for lettuce fiber to ~63% for carrots, celery, and cabbage. Digestibility studies reported during the late 30s and 40s confirmed these earlier observations. Williams and Olmstead (1936a) reported that for most of the fiber sources they studied, 60–80% of the hemicellulose fraction and 53–74% of the total indigestible residue disappeared in the gut. Coefficients of digestibility ranged from very low for cereals, 0–6%, to much higher for oranges, 24%, cabbage, 42%, and apples, 57% (Hoppert and Clark, 1945). The most recent work, following the dietary fiber revival of the 70s, confirms partial digestibility of dietary fiber in humans. In ileostomy patients, 84.5% of the cellulose in a known mixed diet was recovered in the ileostomy fluid, while only 22.4% was excreted in the feces of normal subjects. Of the water-insoluble hemicelluloses ingested, 27.5% was collected from the ileostomy while only 4% was present in the feces of the control group. Lignin was nearly completely recovered (Holloway et al., 1978).

In normal adolescent boys fed supplements of three purified fiber fractions, the apparent disappearance of cellulose was 45–46% and hemicellulose 76–90%. Pectin disappeared completely, although some products of its degradation were present in the feces (Fetzer et al., 1979). In studies of seed hull fibers incorporated into bread, great disruptions in fiber morphology and individual variations in fiber digestibility were observed. While cellulose and lignin sometimes could be fully recovered, only 50% of the hemicellulose was found in the feces (Dintzis et al., 1979).

In feeding studies with pectin and cellulose fed at levels up to 20% in the diet, 25% of the cellulose and 75% of the pectin disappeared in the rat gut. Part of the loss could be accounted for by the enlarged ceca and increased volatile fatty acid (VFA) concentration in cecal contents. None of the gel-forming substances studied produced any discernible growth and did not contribute to net energy gain in the rat (Hove and King, 1979). When 36 g/day of pectin was included in the diet of five male human subjects, no significant change in fecal polysaccharide content was observed. Pectin appeared to have been completely digested or metabolized by the gut microflora (Cummings et al., 1979b). The apparent digestibility of Ispaghula husk when included in the diet of four human volunteers ranged from 47 to 80% (Prynne and Southgate, 1979). Since the husk consists almost totally of arabinoxylan, any water-soluble pentoses found in the feces are likely to be from that source. Water-soluble, noncellulosic polysaccharides were virtually absent in stools from the control group.

The role of colon microbial flora in man and other monogastric animals in the breakdown of dietary fiber has been demonstrated by comparing the complex carbohydrate content in the colon to that in the terminal ileum. Concentrations of the sugars characteristic of complex polysaccharides were all higher at the ileocecal junction than in the colon contents (Vercellotti et al., 1977, 1978). Bacteria recovered from human feces demonstrated a variety of polysaccharide-degrading activities (Salyers et al., 1978; Salyers, 1979). Although Smith and Bryant (1979) estimated that 7.4% of the energy requirement can be supplied by fermentation of complex carbohydrates in the colon, direct evidence of availability of VFA for energy in man was shown only recently (McNeil et al., 1978). The rates of absorption of these acids in man were comparable with rates of absorption observed in experimental animals. In the rat, absorption from the colon contributed ~5% of the daily caloric needs, though theoretically it could supply up to 50% of gross energy requirement (Yang et al., 1970). Southgate and Durnin (1970) calculated maximum energy contributions of 0.6–1.2% and 0.03–0.38% for pentosans and cellulose, respectively, and concluded that their contributions in the diet may be ignored.

Reports on the influence of fiber source and level in the diet on species of intestinal flora have been inconsistent (Salyers, 1979). Drasar et al. (1976) and Goldberg et al. (1977) reported that gut flora is not greatly affected by diet. However, Finegold et al. (1977) noted some differences in flora recovered from Seventh-Day Adventists and subjects on conventional diets, but differences between vegetarian and nonvegetarian Adventists were not observed. A change in diet was shown to introduce new substrates resulting in differing levels of microbial enzymes and metabolic activities in the colon (Salyers et al., 1978; Salyers, 1979; Moore et al., 1978).

EFFECT OF DIETARY FIBER ON DIGESTIBILITY AND BIOAVAILABILITY OF NUTRIENTS

Dietary Protein. In the rat receiving a 10% casein diet containing 5% guar, nitrogen in the feces increased, and urinary nitrogen decreased but overall nitrogen retention was not altered compared to that of the controls (Harmuth-Hoene et al., 1978). When a dose of ^{14}C-labeled protein was administered, accelerated amino acid turnover rate was observed only for the first 3 h, in the guar-fed animals. In additional studies including 10% guar gum, carob bean gum, sodium alginate, agar, or carrageenan, apparent protein digestibility was significantly reduced by 5–10% in all experimental treatments compared to that of the controls (Harmuth-Hoene and Schwerdtfeger, 1979). The effect on urinary nitrogen was inconsistent and nitrogen retention was significantly reduced in groups receiving agar or carrageenan. Trypsin activity was found to be slightly lower in all groups except with carrageenan where significant inhibition was noted. Nomani et al. (1979) studied the effects of wheat bran, pectin, and cellulose on growth and protein metabolism in the rat. Their data suggested that dietary fiber improved growth and nitrogen utilization at marginal levels of protein and energy intake. In mice, however, Keim and Kies (1979) reported decreased weight gain and protein efficiency with increasing intake of dietary fiber.

In man, Southgate and Durnin (1970) reported a greater fecal nitrogen loss on high-fiber diet. On a high-fiber fruit and vegetable diet, Kelsay et al. (1978) also reported significantly elevated fecal nitrogen losses. Urinary excretion of nitrogen was not significantly different. Although apparent nitrogen digestibility was significantly lower, nitrogen balance did not differ between diets. Calloway and Kretsch (1978), comparing a Guatemalan diet containing a high dietary fiber level to an egg diet, reported a large fecal nitrogen loss on the Guatemalan diet although the subjects were all in nitrogen balance. Increasing the level of egg protein in the diet alleviated the effects of oat bran on nitrogen balance. Kies and Fox (1978) reported decreased nitrogen balance with increased levels of hemicellulose in the diet supplied from Plantago ovata.

These studies indicate that the various dietary fiber sources do influence nitrogen metabolism in man and other animals. The observed rise in fecal nitrogen excretion is probably due to enhancement of bacterial activity in the colon and increased need for microbial nitrogen. The fecal losses of nitrogen on an adequate protein intake, however, appear to be minimal and are likely to be nutritionally insignificant.

Dietary Fat. Keim and Kies (1979) found that total fat excretion was highest in mice receiving diets containing 10% hemicellulose and that fat excretion in the groups consuming hemicellulose was significantly different from that in groups receiving cellulose and lignin. A trend toward lower percent digestibility was seen as mice were fed increasing levels of cellulose and hemicellulose. In man, Southgate and Durnin (1970) found that high-fiber diet significantly reduced the apparent digestibility of fat. Sugar cane bagasse and wheat bran both increased total fat excretion in human subjects (Walters et al., 1975). Conversely, school children receiving high fiber excreted a relatively constant amount of fecal fat which was not affected by the addition of fiber-free foods (Walker, 1975). When a control high-fiber diet was supplemented with oranges, fecal volume was increased, fecal fat concentration reduced, but total fat excreted increased significantly. It was noted that absorption of fat was normal on the control high-fiber diet. A bran-supplemented diet also increased fecal fat excretion in most subjects (Southgate et al., 1976). In the studies of Cummings et al. (1976), fecal fat output was increased by the addition of 28 g of dietary fiber from wheat bran. However, the increase in fecal fat excretion was considered nutritionally insignificant since fat absorption was 97.1% on the high-fiber diet (including the added wheat bran). When 15 g of pectin was added daily to control diets, fecal fat excretion increased by 4.4%.

Kelsay et al. (1978), Calloway and Kretsch (1978), and Robertson et al. (1979) have also supplied evidence in human trials relating increased fiber intake to elevated fat excretion when fruits and vegetables, oat bran, and raw carrot were fed. Reports on no effect of high intake of wheat bran and Ispaghula husk on fat excretion have been submitted by Farrell et al. (1978) and Prynne and Southgate (1979), respectively.

The effect of dietary fiber on fat excretion and digestibility in mixed diets remains a controversial area of research, and its impact on overall energy balance in man and experimental animals needs to be further elucidated.

Dietary Minerals. *Calcium.* There is a consensus that fiber intake influences mineral balance (Kelsay, 1978; Cummings, 1978; Cummings et al., 1976; Davies, 1979). Malabsorption of calcium has been associated with high intakes of phytic acid containing whole grain materials (McCance and Widdowson, 1942–1943; McCance and Walsham, 1948). In in vitro experiments with low phytate dietary fiber preparations, James et al. (1978) reported calcium binding in proportion to the uronic acid content of the dietary fiber. Since the colonic microflora attacks these polysaccharides, calcium may be liberated in the colon and, therefore, adaptability could be a factor in long-term calcium balance studies.

Following the observations by Heaton and Pomare (1974) that wheat bran had a lowering influence on plasma calcium, several workers explored this relationship in experimental animals and in clinical trials. Sina et al. (1976) found no influence on calcium plasma levels when a 20% cellulose diet was fed to rats. In a 19-week clinical trial with young male subjects, Heaton et al. (1976) found no influence on plasma calcium between diets containing whole meal bread (high fiber) vs. white bread. Twenty grams of bran per day consumed over a 6-week period by elderly subjects was found to be without effect on serum calcium level (Persson et al., 1976). That practical fiber levels in the diet have little, if any, consequence on plasma or serum calcium was further substantiated by clinical trials using wheat bran fed at 24 g/day (Weinreich et al., 1977) and studies of standard vs. bran-enriched bread in a 5-week crossover design (Henry et al., 1978).

Although addition of high-methoxy pectin to the diet of young male subjects did not affect calcium balance, decreased urinary calcium and increased fecal calcium excretion was noted (Cummings et al., 1979b). Using a high-fiber diet containing fruit and vegetable sources compared to a low-fiber diet containing fruit and vegetable juices, Kelsay et al. (1979) found that fecal calcium was higher on the high-fiber diet while the urinary calcium did not differ significantly. Subjects on a daily 200-g carrot supplement excreted more calcium. A high-protein diet containing meat and wheat fiber supplement resulted in a negative calcium balance by the third week (Robertson et al., 1979). Similar results were also reported with a bran-supplemented diet (Cummings et al., 1979a), high-fiber Iranian bread (Reinhold et al., 1976), and white bread supplemented with cellulose (Beigi et al., 1977b). In a series of studies conducted recently at the USDA human nutrition laboratory, the dietary calcium requirement was increased by the addition of fiber to a high-protein diet (Sandstead et al., 1979). It should be noted, however, that in an earlier study Farrell et al. (1978) could not demonstrate any effect on fecal excretion of calcium when male subjects were fed 16 g of wheat bran/day as supplements to a conventional Western diet.

Zinc. Tsai and Lei (1979) reported depressed serum zinc concentrations as dietary cellulose was increased to 16%. Zinc distribution in soft tissues, however, was not changed.

Several studies with human subjects have demonstrated changes in zinc nutritional status due to the addition of fiber to the diet. Reinhold et al. (1976) observed negative zinc balances in Iranian subjects consuming a high-fiber native bread. Beigi et al. (1977a,b) confirmed negative balances in human subjects consuming cellulose as a fiber source and provided evidence that the zinc binding was not due to phytate in the cereal flour. In studies with English whole meal bread and wheat bran, the binding of zinc was cited by Reilly (1979).

In an effort to define the fraction of dietary fiber which has a high binding capacity, Drews et al. (1979) fed diets containing relatively purified hemicellulose, pectin, or cellulose as fiber sources to adolescent boys. Hemicellulose supplementation resulted in significantly increased fecal zinc. Directionally the effect of cellulose was the same but not of the same order of magnitude and pectin was the least effective. Significant influences of hemicellulose on zinc balance were also observed by Kies et al. (1979). As the level of hemicellulose supplement was increased, the fecal zinc content increased significantly. Urinary and blood levels of zinc were unaffected. Although earlier data reviewed by Cummings (1978) and Kelsay (1978) consistently showed dietary fiber to influence zinc balance, no effect on the requirement for zinc could be demonstrated in man (Sandstead et al., 1979).

In our laboratory, Coccodrilli et al. (1979) studied the effect of ready-to-eat breakfast cereals, wheat bran, and defatted peanut flour using zinc accummulation in the femur as a measure of zinc bioavailability in the rat.

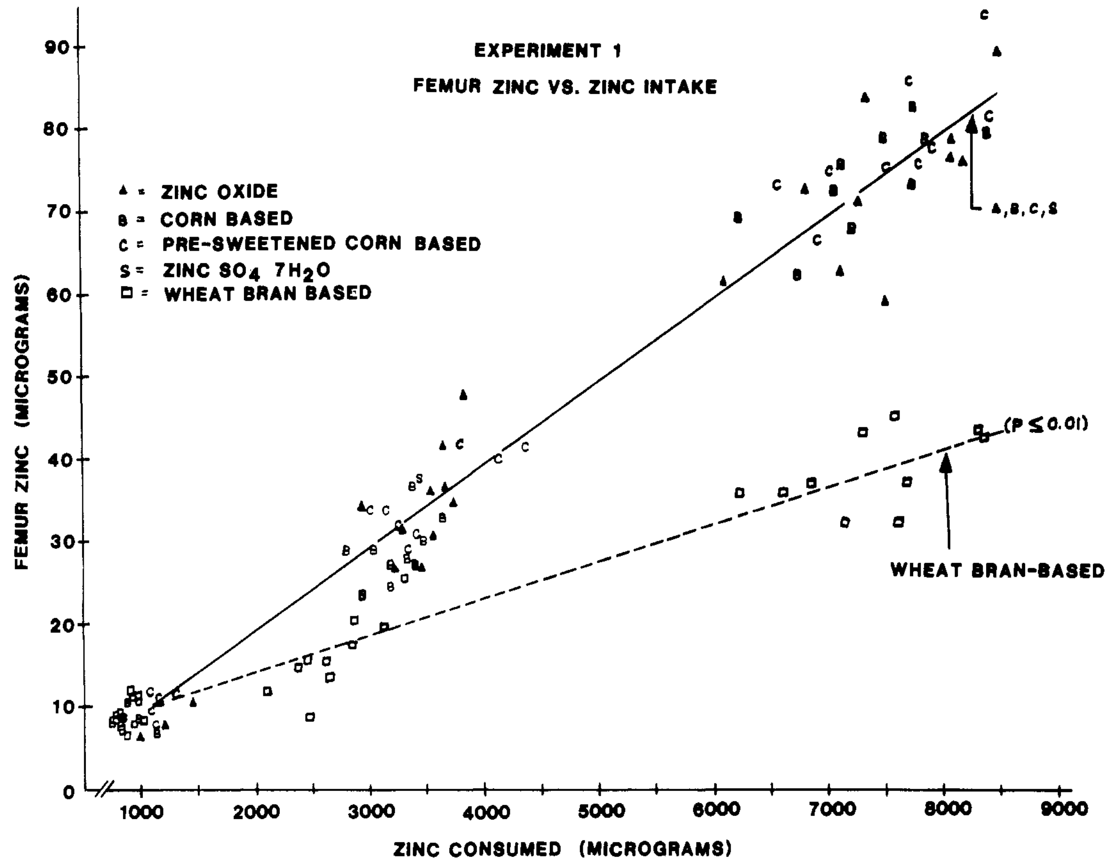

Figure 1. Effect of corn vs. wheat bran based diets on bioavailability of zinc in the rat (Coccodrilli et al., 1979).

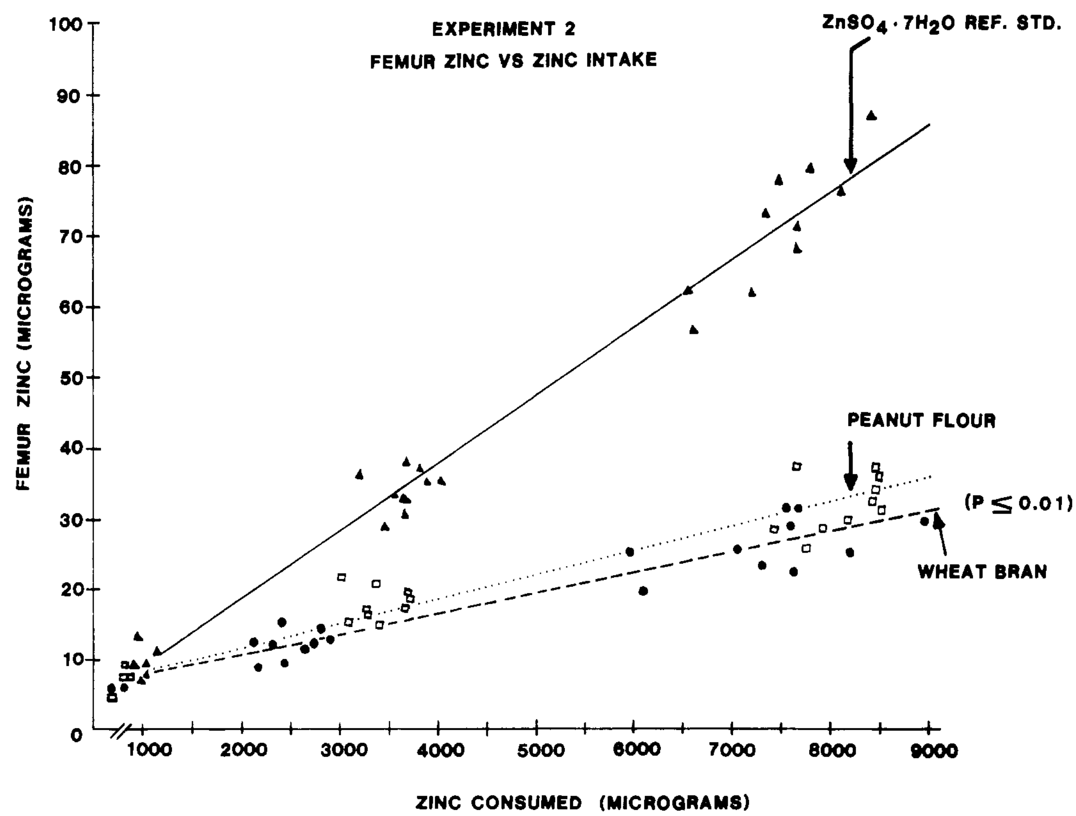

Figure 2. Effects of peanut flour vs. wheat bran based diets on bioavailability of zinc in the rat (Coccodrilli et al., 1979).

Slope-ratio analysis of the data using zinc oxide as a reference standard showed no significant difference in zinc bioavailability between a corn-based cereal and a presweetened corn-based cereal. A wheat bran based processed cereal with no zinc added, however, was significantly different ($p < 0.01$) from zinc oxide treatment, with relative biological values (RBV) of 44.6 and 100, respectively (Figure 1). In a second experiment, zinc inherent to wheat bran and defatted peanut flour was shown to be significantly less bioavailable when compared to zinc sulfate

Table I. Effect of Source and Dietary Matrix on Relative Zinc Bioavailability in the Rat (Coccodrilli et al., 1979)

treatment	RBV[a]	95% confidence limits
Experiment I		
zinc oxide (ref std)	100	
corn-based cereal		
presweetened corn-based cereal		
wheat bran based cereal	44.6	35.6-53.1
Experiment II		
zinc sulfate, heptahydrate (ref std)	100	
peanut flour	36.5	29.8-43.6
wheat bran	30.8	23.6-38.4

[a] Relative biological values.

standard (Figure 2). These results indicate that matrix and fiber composition of the diet influence availability of zinc at marginal zinc intake under controlled experimental conditions (Table I).

Iron. Conflicting results have been reported on the effects of fiber on iron absorption. Earlier reviewers (Cummings, 1978; Kelsay, 1978) concluded that high-fiber diets impaired iron absorption. In studies with elderly subjects receiving up to 20 g of bran daily for 6 weeks, Persson et al. (1976) reported decreased serum iron levels. Ranhotra et al. (1979), studying the influence of cellulose on the bioavailability of iron from bread, found that hemoglobin repletion in rats was faster on the reference diet than on diets containing cellulose. Additional experiments with bread indicated that fiber content was not the sole dependent factor affecting iron bioavailability and that other elements in the baking process were implicated (Ranhotra, 1979).

Conversely, Sandstead et al. (1979) did not observe any influence of dietary fiber upon iron requirement in human subjects.

Other Minerals. (*1*) *Magnesium.* Kelsay et al. (1979), Sandstead et al. (1979), and Reinhold et al. (1976) have provided data from clinical trials indicating magnesium balance and retention to be lower when high-fiber breads, hemicellulose, or fruit and vegetable fiber source were fed. The impact of fiber on magnesium nutrition remained controversial in light of reports by Robertson et al. (1979) and Beigi et al. (1977b), who observed no effect in man.

(*2*) *Phosphorus.* In the Iranian bread studies negative balances for phosphorus were reported. Not only did fecal phosphorus excretion increase but also serum phosphorus levels responded to the dietary changes. With the cellulose-supplemented bread, there were inconsistent phosphorus responses (Reinhold et al., 1976; Beigi et al., 1977b). Cummings et al. (1976) reported a significant increase in fecal output of phosphorus on a wheat bran diet.

(*3*) *Copper.* The copper requirement determined in the Grand Forks studies was 1.24 mg daily and fiber had no apparent effect on copper absorption (Sandstead et al., 1979). In a rat study, addition of cellulose to the diet appeared to have no adverse effect on the distribution of zinc and copper in the tissues (Tsai and Lei, 1979).

(*4*) *Silicon.* Silicon balance was significantly lower on the high fruit and vegetable fiber diet in the USDA experiments. Urinary excretion was 35 vs. 58% while fecal excretions were 97 vs. 60% of intake in the high- and low-fiber diets, respectively (Kelsay et al., 1979). This result is surprising since silicon intake was much higher on the high-fiber diet.

Vitamins. Interactions between fiber and vitamin utilization have not been extensively investigated. Cullen and Oace (1978) studied vitamin B_{12} nutriture of rats fed pectin and cellulose. Both fiber sources exerted negative effects on vitamin B_{12} status with pectin ingestion resulting in larger excretion of methylmalonic acid. Since pectin is degraded in the lower bowel and dietary addition stimulates microbial activity, competition for B_{12} between microbes and the host may play a role in affecting vitamin B_{12} status.

Keltz et al. (1978) showed that hemicellulose supplements increased urinary excretion of ascorbic acid but pectin had an opposite effect.

Pectin at 3% of the diet did not limit the absorption of vitamin A or the utilization of the provitamin β-carotene in the rat (Phillips and Brien, 1970). Measurement of postprandial vitamin A following a vitamin A and fiber-supplemented diet showed serum vitamin A to be higher when the vitamin was administered with the fiber. The fibers studied included cellulose, bran, and some gel-forming fibers (Kasper et al., 1979).

In one study on folate metabolism, Russell et al. (1976) found no interference with folate uptake in experiments with high-fiber breads, and in vitro studies could not demonstrate the formation of insoluble folate–fiber complexes.

With the scanty data available, no definitive interactions between fiber and vitamins can be predicted. However, the hypothesis of how pectin may interfere with the vitamin B_{12} bioavailability serves to emphasize the differences between water-soluble and insoluble dietary fibers in influencing the environment in the lower gut.

EFFECT OF DIETARY FIBER ON GI FUNCTION AND MOTILITY

Upper GI Tract. McCance et al. (1953) evaluated the effect of diets containing bread made from high- and low-fiber extraction flours on gastric emptying and reported that radiopaque materials moved at a 25% faster rate through the small bowel in subjects placed on high-fiber diets. In addition, they showed that the high-fiber bread diet was evacuated from the stomach more rapidly and resulted in decreased mouth to anus transit time and increased stool weights as compared to the low-fiber bread diet. Although fiber content of the diet may play a significant role in regulating gastric emptying, additions of lipid to high-fiber diets was reported to drastically reduce it (Mendeloff, 1978).

The reported rapid rates of gastric emptying associated with increased fiber intake appears to be related to its bulking action which directly promotes distention and efficient motor tone.

Small Bowel. Little information is available on the effect of various dietary fibers on small bowel motility and associated changes in intestinal transit time. Recent quantitative results from our laboratory clearly showed that myoelectric (contractile) activity pattern during the fed state from animals on 20% wheat bran diets was significantly reduced ($p \leq 0.01$). The mean percent slow waves with spike activity recorded in consecutive 2-min intervals was 7.2 compared to the control value of 24.9 (Table II). The data suggest that bulking action of wheat bran in the small intestine promotes motor tone and facilitates organization of contractile movements to effectively move gut contents, with a reduced forceful effort, in a net aboral direction (Schanbacher and Ali, 1979).

McConnell et al. (1974) demonstrated cation-exchange capacity of plant fibers to vary from 0.35 mequiv/g for potato fiber to 3.1 mequiv/g for lettuce fiber. The effect of adding cereal grain fiber, such as bran, to the diet was shown by Eastwood et al. (1976) to facilitate the excretion

Table II. Effect of Level of Wheat Bran in the Diet on the Fasted and Fed Intestinal Motility Pattern in the Rat (Schanbacher and Ali, 1979)

	% slow waves with spikes[a]		burst cycle, min	
diet, %	fasted	fed	fasted	fed
0	40.6 ± 3.5	24.9 ± 1.5	14.3	–
5	48.4 ± 3.6	19.4 ± 2.7	14.5	–
10	49.6 ± 3.6	24.3 ± 4.0	14.6	–
20	47.3 ± 3.9	7.2 ± 1.0[b]	13.7	–

[a] Each value represents the mean ± SE of 12 observations each. [b] $p < 0.01$.

of cations, mainly sodium, potassium, and magnesium. Virtually nothing is known of the effect of modifying cationic binding strength of dietary fiber on the motility functions of the small intestine. By calculation, 16 g of lettuce fiber would bind 5% (49.6 mequiv) of the total cations presented daily, and it is doubtful that this would be sufficient to alter normal functional activity of smooth muscle structurally removed from the lumen of the gut.

Biliary Function. Bile acid excretion is increased with certain dietary fiber. Eastwood and Boyd (1969) reported that the total bile acid pool in the rat small intestine was increased from 7.5 to 15 mg by addition of cellulose or wheat bran to the diet. Kelsay (1978) indicated that dietary fiber from maize, wheat, vegetable, bagasse, Bengal gram, and cellulose increased total fecal bile acid excretion in man. One study reported that Metamucil and cellulose increased bile salt excretion slightly, but the effect of Cholestyramine was many times greater (Stanley et al., 1973). In patients with choleretic enteropathy, and when bile acids were added to the colon of rabbits, the motor activity of the colon was markedly increased (Eastwood and Boyd, 1969).

Large Bowel. Contractions of the large intestine are normally organized to allow for optimal residence time to absorb water and electrolytes, provide for net aboral propulsion of contents, and store residual contents for eventual evacuation associated with mass movement activity of the colon.

The effect of particle size of fiber on producing favorable laxation in the colon is controversial. Kirwan et al. (1974) demonstrated that finely ground wheat bran was not as effective as coarse bran in producing faster transit and reducing intraluminal colonic pressures. Furthermore, Wyman et al. (1976) reported that raw uncooked millers bran was more effective in bulking the stool and increased fecal weights in healthy subjects when compared to a processed ready-to-eat bran cereal.

Only a few studies have been carried out on the effects of fiber on bowel habits or functions of healthy subjects. Most studies reported results of stool weights without reference to frequency or consistency of stools produced. The original objective studies of Williams and Olmstead (1936b) showed that various plant sources could increase stool weight, with carrot being the most effective followed by cabbage, sugar beet pulp, peas, and wheat bran in order of decreasing potency. McCance et al. (1953) later studied the effect of increasing dietary fiber intake on small intestinal transit time and found that as the dietary intake of fiber increased, the transit time decreased by some 1.5 h. Burkitt et al. (1972) clearly showed that eating diets high in natural amounts of fiber produced large soft stools (275–490 g/day) and passed through the intestinal tract within 33–35 h. Semirefined mixed diets resulted in smaller stools (155–225 g/day) and required 10 h longer to traverse the entire intestinal tract. Individuals ingesting highly refined diets that were not supplemented with fruits had daily stool weights of only 104–110 g and much slower rate of intestinal transit (76–83 h). More recently, Cummings et al. (1976) demonstrated that increasing the dietary intake of fiber from 17 to 45 g/day by addition of cereal fiber increased the daily stool weights of healthy subjects from 79 to 228 g. The total water output of the feces was increased more on the high-fiber diet than were the total solids. The average transit time was reduced from 57.8 to 40.3 h by increasing fiber intakes from 17 to 45 g/day, and total volatile fatty acids and fecal bile acid excretion were also increased.

Spiller et al. (1977) suggested that beyond an average production of 140–150 g of feces/day and a 3-day or less transit time, a further increase in fiber intake was not accompanied by a greater reduction in transit time. Inclusion of fiber in the diet increased stool bulk in 13 studies where the diets were controlled from a relatively low to high fiber intakes (Kelsay, 1978). In 3 of the 13 studies where transit time was measured and the diets controlled, increased transit could be related to the high dietary fiber intakes. It was clear that controlled fiber intake levels should be initially established before one can judge the effective laxation effects of added dietary fiber sources. Cummings et al. (1978) studied the bulking laxation effects of a variety of fiber sources and found that changes in increased fecal weights were highly correlated with an increased intake of pentose-containing polysaccharides from fiber.

The reported therapeutic effects of dietary fiber on two intestinal disease states, irritable colon and diverticular disease, as well as constipation, appear to be favorable. These conditions can collectively be categorized as distal stasis of the colon. According to Painter (1975), diverticular disease of the colon has emerged as a clinical problem during the last 70 years. Diverticula are herniations that may be ascribed either to weakness of the bowel wall, to abnormally high intracolonic pressures, or to a combination of both. Since no congenital weakness has ever been reported in the colonic musculature, it has been suggested that diverticula must be an acquired structural change in the gut wall (Reilly and Kirsner, 1977).

Reported studies of intraluminal colonic pressures demonstrated that increasing intake of natural fiber resulted in lowering of intracolonic pressures. Findlay et al. (1974) studied the effect of adding two 10-g servings of bran to the diet of patients with diverticular disease or constipation. Intraluminal pressures during and after meal stimulation were significantly reduced in patients receiving wheat bran with no change in colonic contractions during the resting premeal period. The increase in pressure activity following prostigmine administration, a direct smooth muscle contractile agent, was less than that in the control subjects. Increased stool weights and some reduction in transit time were also reported.

In a similar study, Brodribb and Humphreys (1976) also demonstrated that the daily addition of ~24 g of wheat bran daily to the diet reduced intracolonic pressures. Additionally, Kirwan et al. (1974) indicated that particle size of coarse bran was more effective in reducing intracolonic pressures, reducing transit time, and relieving the painful cramping symptoms of irritable colon or diverticular disease. Taylor and Duthie (1976) evaluated three bulk laxative agents in 20 patients with symptomatic diverticular disease. Only the bulk laxative, Normacol, and bran tablets (18 g/day) increased stool weights while all three treatments decreased transit time. The bran tablets produced a significant reduction in intracolonic pressure activity, and results of the electrical rhythms of the sigmoid and rectum indicated that the incidence of rapid electrical waves (contractions) was significantly reduced in all three treatments. The results of Hodgson (1972) on the effect of methylcellulose on rectal and colonic pressure supported these observations.

These studies clearly indicate that dietary fiber sources, specific fractions, and particle size are all factors involved in producing functional effects on bowel habit and activity. Relief of constipation and symptoms of irritable bowel syndrome is undoubtedly related to the reduction of painful colonic pressures, increased bulkiness of fecal mass, colonic distention, and easier passage or propulsion of colonic contents in an aboral direction.

ACKNOWLEDGMENT

We thank C. Riley and C. Mondello for their assistance in searching the literature and preparation of the manuscript.

LITERATURE CITED

Beigi, F. I.; Faraji, B.; Reinhold, J. G. *Am. J. Clin. Nutr.* **1977a**, *30*, 1721.
Beigi, F. I.; Reinhold, J. G.; Faraji, B.; Abadi, P. *J. Nutr.* **1977b**, *107*, 510.
Brodribb, A. M. J.; Humphreys, D. M. *Br. Med. J.* **1976**, *1*, 424.
Burkitt, D. P.; Walker, A. R. P.; Painter, N. S. *Lancet* **1972**, *2*, 1408.
Calloway, D. H.; Kretsch, M. J. *Am. J. Clin. Nutr.* **1978**, *31*, 1118.
Coccodrilli, G., Jr.; Shah, N.; Sommer, S.; Ali, R. *Fed. Proc., Fed. Am. Soc. Exp. Biol.* **1979**, *38* (3), 558.
Cullen, R. W.; Oace, S. M. *J. Nutr.* **1978**, *108*, 640.
Cummings, J. H. *Am. J. Clin. Nutr.* **1978**, *31*, 521.
Cummings, J. H.; Branch, W.; Jenkins, D. J. A.; Southgate, D. A. T.; Houston, H.; James, W. P. T. *Lancet* **1978**, *1*, 5.
Cummings, J. H.; Hill, M. J.; Jenkins, D. J. A.; Pearson, J. R.; Wiggens, H. S. *Am. J. Clin. Nutr.* **1976**, *29*, 1468.
Cummings, J. H.; Hill, J. M.; Jivray, T.; Houston, W. J.; Branch, W. J.; Jenkins, D. J. A. *Am. J. Clin. Nutr.* **1979a**, *32*, 2086.
Cummings, J. H.; Southgate, D. A. T.; Branch, W. J.; Wiggins, H. S.; Houston, H.; Jenkins, D. J. A.; Jivray, T.; Hill, M. H. *Br. J. Nutr.* **1979b**, *41*, 477.
Davies, N. T. *Proc. Nutr. Soc.* **1979**, *38*, 121.
Dintzis, F. R.; Legg, L. M.; Deatherage, W. L.; Baker, F. I.; Inglett, G. E.; Jacobs, R. A.; Peck, S. J.; Munoz, J. M.; Klevay, L. M.; Sandstead, H. H.; Shuey, W. C. *Cereal Chem.* **1979**, *56*, 123.
Drasar, B. S.; Jenkins, D. J. A.; Cummings, J. H. *J. Med. Microbiol.* **1976**, *9*, 423.
Drews, L. M.; Kies, C.; Fox, H. M. *Am. J. Clin. Nutr.* **1979**, *32*, 1893.
Eastwood, M. A.; Boyd, G. S. *Biochim. Biophys. Acta* **1969**, *137*, 3939.
Eastwood, M. A.; Mitchell, W. D.; Pritchard, J. L. *Proc. Nutr. Soc.* **1976**, *35* (2), 77.
Farrell, D. J.; Girle, L.; Arthur, J. *Aust. J. Exp. Biol. Med. Sci.* **1978**, *56*, 469.
Fetzer, S. G.; Kies, C.; Fox, H. M. *Cereal Chem.* **1979**, *50*, 34.
Findlay, J. M.; Smith, A. N.; Mitchell, N. D.; Anderson, A. J. B.; Eastwood, M. A. *Lancet* **1974**, *1*, 146.
Finegold, S. M.; Sutter, V. L.; Sugihara, P. T.; Elder, H. A.; Lehmann, S. M.; Phillips, R. L. *Am. J. Clin. Nutr.* **1977**, *30*, 1781.
Goldberg, M. J.; Smith, J. W.; Nichols, R. L. *Ann. Surg.* **1977**, *186*, 97.
Harmuth-Hoene, A. E.; Jakubick, V.; Schelenz, R. *Nutr. Metab.* **1978**, *22*, 32.
Harmuth-Hoene, A. E.; Schwerdtfeger, E. *Nutr. Metab.* **1979**, *23*, 399.
Heaton, K. W., Ed. "Dietary Fiber: Current Developments of Importance to Health"; Food and Nutrition Press: Westport, CT, 1979.
Heaton, K. W.; Manning, A. P.; Hartog, M. *Br. J. Nutr.* **1976**, *35*, 55.
Heaton, K. W.; Pomare, E. W. *Lancet* **1974**, *1*, 49.
Henry, R. W.; Stout, R. W.; Love, A. H. G. *Ir. J. Med. Sci.* **1978**, *147*, 249.
Hodgson, J. *Br. Med. J.* **1972**, *3*, 729.
Holloway, W. D.; Tasman-Jones, C.; Lee, S. P. *Am. J. Clin. Nutr.* **1978**, *31*, 927.
Hoppert, C. A.; Clark, A. J. *J. Am. Diet. Assoc.* **1945**, *51*, 157.
Hove, A. E. L.; King, S. *J. Nutr.* **1979**, *109*, 1274.
James, W. P. T.; Branch, W. J.; Southgate, D. A. T. *Lancet* **1978**, *1*, 638.
Kasper, H.; Rabast, V.; Fassl, H.; Fehle, F. *Am. J. Clin. Nutr.* **1979**, *32*, 1847.
Keim, K.; Kies, C. *Cereal Chem.* **1979**, *56*, 73.
Kelsay, J. L. *Am. J. Clin. Nutr.* **1978**, *31*, 142.
Kelsay, J. L.; Behall, K. M.; Prather, E. S. *Am. J. Clin. Nutr.* **1978**, *31*, 1149.
Kelsay, J. L.; Behall, K. M.; Prather, E. S. *Am. J. Clin. Nutr.* **1979**, *32*, 1876.
Keltz, F. R.; Kies, C.; Fox, H. M. *Am. J. Clin. Nutr.* **1978**, *31*, 1167.
Kies, C.; Fox, H. M. *Cereal Foods World* **1978**, *23*, 249.
Kies, C.; Fox, H. M.; Beshgetoor, D. *Cereal Chem.* **1979**, *56*, 133.
Kimura, K. K., Ed. "Nutritional Significance of Dietary Fiber"; LSRO, FASEB for the FDA, Bureau of Foods, 1977, PB-275672.
Kirwan, W. O.; Smith, A. N.; McConnel, A. A.; Mitchell, W. D.; Eastwood, M. A. *Br. Med. J.* **1974**, *4*, 187.
Mangold, E. *Nutr. Abstr. Rev.* **1934**, *3*, 647.
McCance, R. A.; Prior, K. M.; Widdowson, E. M. *Br. J. Nutr.* **1953**, *7*, 98.
McCance, R. A.; Walsham, C. M. *Br. J. Nutr.* **1948**, *2*, 26.
McCance, R. A.; Widdowson, E. M. *J. Physiol.* (*London*) **1942–1943**, *101*, 44.
McConnell, A. A.; Eastwood, M. A.; Mitchel, W. D. *J. Sci. Food Agric.* **1974**, *25*, 1457.
McNeil, N. I.; Cummings, J. H.; James, W. P. T. *Gut* **1978**, *19*, 819.
Mendeloff, A. I. *Am. J. Clin. Nutr.* **1978**, *31*, 5145.
Moore, W. E. C.; Cato, E. P.; Holdeman, L. V. *Am. J. Clin. Nutr.* **1978**, *31*, 533.
Nomani, M. Z. A.; Fashandi, E. F.; Davis, G. K.; Bradoc, C. J. *J. Food Sci.* **1979**, *44*, 745.
Painter, N. S. "Fiber Deficiency and Colonic Disorders"; Reilly, R. W.; Keisner, J. B., Eds.; Plenum Press: New York, 1975.
Persson, I.; Raby, K.; Ønss-Bech, P.; Jensen, E. *J. Am. Geriatr. Soc.* **1976**, *24*, 334.
Phillips, W. E. J.; Brien, R. L. *J. Nutr.* **1970**, *100*, 289.
Prynne, C. J.; Southgate, D. A. T. *Br. J. Nutr.* **1979**, *41*, 495.
Ranhotra, G. S. *Cereal Foods World* **1979**, *24*, 252.
Ranhotra, G. S.; Lee, C.; Gelrotts, J. A. *Cereal Chem.* **1979**, *56*, 156.
Reilly, C. *Biochem. Soc. Trans.* **1979**, *7*, 202.
Reilly, R. W.; Kirsner, J. B. "Progress in Gastroenterology"; George, B.; Glass, J., Eds.; Grune and Stratton: New York, 1977; Vol. III.
Reinhold, J. G.; Faradji, B.; Abadi, P.; Beigi, F. *J. Nutr.* **1976**, *106*, 493.
Robertson, J.; Brydon, W. G.; Tadesse, K.; Wenham, P.; Wallis, A.; Eastwood, M. A. *Am. J. Clin. Nutr.* **1979**, *32*, 1889.
Roth, H. P.; Mehlman, M. A. *Am. J. Clin. Nutr.* **1978**, *31* (10) (Suppl.).
Russell, R. M.; Beigi, F. I.; Reinhold, J. G. *Am. J. Clin. Nutr.* **1976**, *29*, 799.
Salyers, A. A. *Am. J. Clin. Nutr.* **1979**, *32*, 158.
Salyers, A. A.; Palmer, J. K.; Wilkens, T. K. *Am. J. Clin. Nutr.* **1978**, *31*, 5128.
Sandstead, H. H.; Klevay, L. M.; Jacob, R. A.; Munoz, J. M.; Logan, G. M., Jr.; Reck, S. J.; Dintzis, F. R.; Inglett, G. E.; Sjuey, W. C. "Dietary Fibers, Chemistry and Nutrition"; Inglett, G. E.; Falkehag, S. I., Eds.; Academic Press: New York, 1979.
Schanbacher, L. M.; Ali, R., unpublished data, 1979.
Sina, P. A.; Provenchi, R. R.; Cantone, A. *Biochem. Exp. Biol.* **1976**, *12*, 321.
Smith, C. J.; Bryant, M. P. *Am. J. Clin. Nutr.* **1979**, *32*, 149.
Southgate, D. A. T.; Brand, W. J.; Hill, M. J.; Drasar, B. S.; Walters, R. L.; Davies, P. S.; Baird, I. M. *Metab., Clin. Exp.* **1976**, *25*, 1129.
Southgate, D. A. T.; Durnin, J. G. V. A. *Br. J. Nutr.* **1970**, *24*, 517.

Spiller, G. A.; Amen, R. J. *CRC Crit. Rev. Food Sci. Nutr.* **1975**, *7*, 39.
Spiller, G. A.; Amen, R. J., Eds. "Fiber in Human Nutrition"; Plenum Press: New York, 1976.
Spiller, G. A.; Amen, R. J., Eds. "Topics in Dietary Fiber Research"; Plenum Press: New York, 1978.
Spiller, G. A.; Cernoff, M. C.; Shipley, E. A.; Beigler, M. A.; Briggs, G. M. *Am. J. Clin. Nutr.* **1977**, *30*, 659.
Stanley, M. M.; Paul, D.; Gacke, D.; Murphy, J. *Gastroenterology* **1973**, *65*, 889.
Staub, H. W.; Ali, R. "Food Hydrocolloids"; Glicksman, M., Ed.; CRC Press: Boca Raton, FL, 1981.
Taylor, I.; Duthie, H. L. *Br. Med. J.* **1976**, *1*, 988.
Tsai, R. C. Y.; Lei, K. Y. *J. Nutr.* **1979**, *109*, 1117.
Vercellotti, J. R.; Salyers, A. A.; Bullard, W. S.; Wilkins, T. D. *Can. J. Biochem.* **1977**, *55*, 1190.
Vercellotti, J. R.; Salyers, A. A.; Wilkins, T. D. *Am. J. Clin. Nutr.* **1978**, *31*, 586.
Walker, A. R. P. *Am. J. Clin. Nutr.* **1975**, *28*, 1161.
Walters, R. L.; Baird, I. M.; Davies, P. S.; Hill, M. J.; Drasar, B. S.; Southgate, D. A. T.; Green, J.; Morgan, B. *Br. Med. J.* **1975**, *2*, 536.
Weinreich, J.; Pedersen, O.; Kinesen, K. *Acta Med. Scand.* **1977**, *202*, 125.
Williams, R. D.; Olmstead, W. H. *Ann. Intern. Med.* **1936a**, *10*, 717.
Williams, R. D.; Olmstead, W. H. *J. Nutr.* **1936b**, *11*, 433.
Wyman, J. B.; Heaton, K. W.; Manning, A. P.; Wicks, A. B. C. *Br. Med. J.* **1976**, *2*, 944.
Yang, M. G.; Manoharan, K.; Mickelsen, O. *J. Nutr.* **1970**, *100*, 545.

Received for review June 23, 1980. Accepted February 26, 1981. Presented at the Symposium on Dietary Fiber: Sources and Physiological Effects, Division of Agricultural and Food Chemistry, 179th National Meeting of the American Chemical Society, Houston, TX, March 1980.

Effects of Autohydrolyzed Lignin and Lactulose on Gallbladder Bile Composition in Hamsters

Ori D. Rotstein, Ruth M. Kay,* Morris Wayman, Kin Po Siu, and Steven M. Strasberg

Autohydrolyzed lignin was found to bind bile acids in vitro, an effect which was enhanced at low pH. Lignin with and without lactulose was fed to Golden Syrian hamsters to determine whether it could improve gallbladder bile composition by inducing small increases in bile acid excretion/synthesis. Lignin-supplemented diets increased daily fecal lithocholate excretion in association with a significant reduction in biliary cholesterol saturation. Lignin and lactulose did not affect daily neutral steroid excretion, but the combination decreased cholesterol degradation in the intestine. Further experiments are required to assess the efficacy of autohydrolyzed lignin and lactulose in the prevention of cholesterol gallstone disease.

Cholesterol gallstones may be dissolved by lowering the cholesterol saturation of the bile. Agents presently in use such as chenodeoxycholic acid accomplish this by reducing cholesterol synthesis (Coyne et al., 1976) and cholesterol secretion into the bile (LaRusso et al., 1974). Cholesterol secretion into the bile may also be diminished by controlled stimulation of bile acid synthesis, presumably by diverting cholesterol into the bile acid synthetic pathway (Strasberg et al., 1976). Several forms of fiber mildly stimulate bile acid synthesis by causing an increase in fecal bile acid loss (Kay and Strasberg, 1978), and gallstones are rare in populations with high fiber intake (Burkitt and Painter, 1974). The purpose of this experiment was to determine whether lignin, a component of dietary fiber, could reduce the cholesterol saturation of bile in the hamster by increasing fecal bile acid excretion and bile acid synthesis. A highly purified lignin extracted from aspen wood chips by autohydrolysis was used (Lora and Wayman, 1978). This lignin binds bile acids avidly in vitro, an effect enhanced by lowering the pH (Kay et al., 1979). In vivo acidification of the cecum can be induced by lactulose, a nonabsorbable carbohydrate (Bown et al., 1974). Lactulose was added to lignin in one group to determine if it augmented the effects of lignin on fecal bile acid excretion in vivo. In this initial study, the effects of these agents were tested in hamsters on normal diets with low cholesterol content.

Department of Surgery, Toronto Western Hospital, and Department of Chemical Engineering, University of Toronto, Toronto, Ontario, Canada M5T 2S8.

EXPERIMENTAL SECTION

Experimental Protocol. Twenty four mature male Golden Syrian hamsters were caged singly and randomly divided into four groups. The animals were allowed free access to food and water and were maintained in this manner for 21 days. All animals were fed a nutritionally complete dry semisynthetic diet containing cholesterol (0.2 g/kg of diet) supplemented with either cellulose (5% w/w; control), lignin (5% w/w; Lig), lignin (4.1% w/w) plus lactulose (13.6% w/w; Lig–Lac), or lactulose (13.6% w/w; Lac). This amount of lactulose approximates the upper limit of the usual dose in humans. Water was permitted ad libitum.

Food intake and weights were recorded. Total fecal collection was performed from day 16 to day 21.

At the end of the experimental period, after an overnight fast, the animals were anesthetized with nitrous oxide and halothane, and a laparotomy was performed. The cystic duct was ligated with a metal clip and the gallbladder bile was aspirated. A sample of venous blood was taken from the inferior vena cava, and the pH of the cecal contents

0021-8561/81/1429-0472$01.25/0