Unconventional Sources of Dietary Fiber

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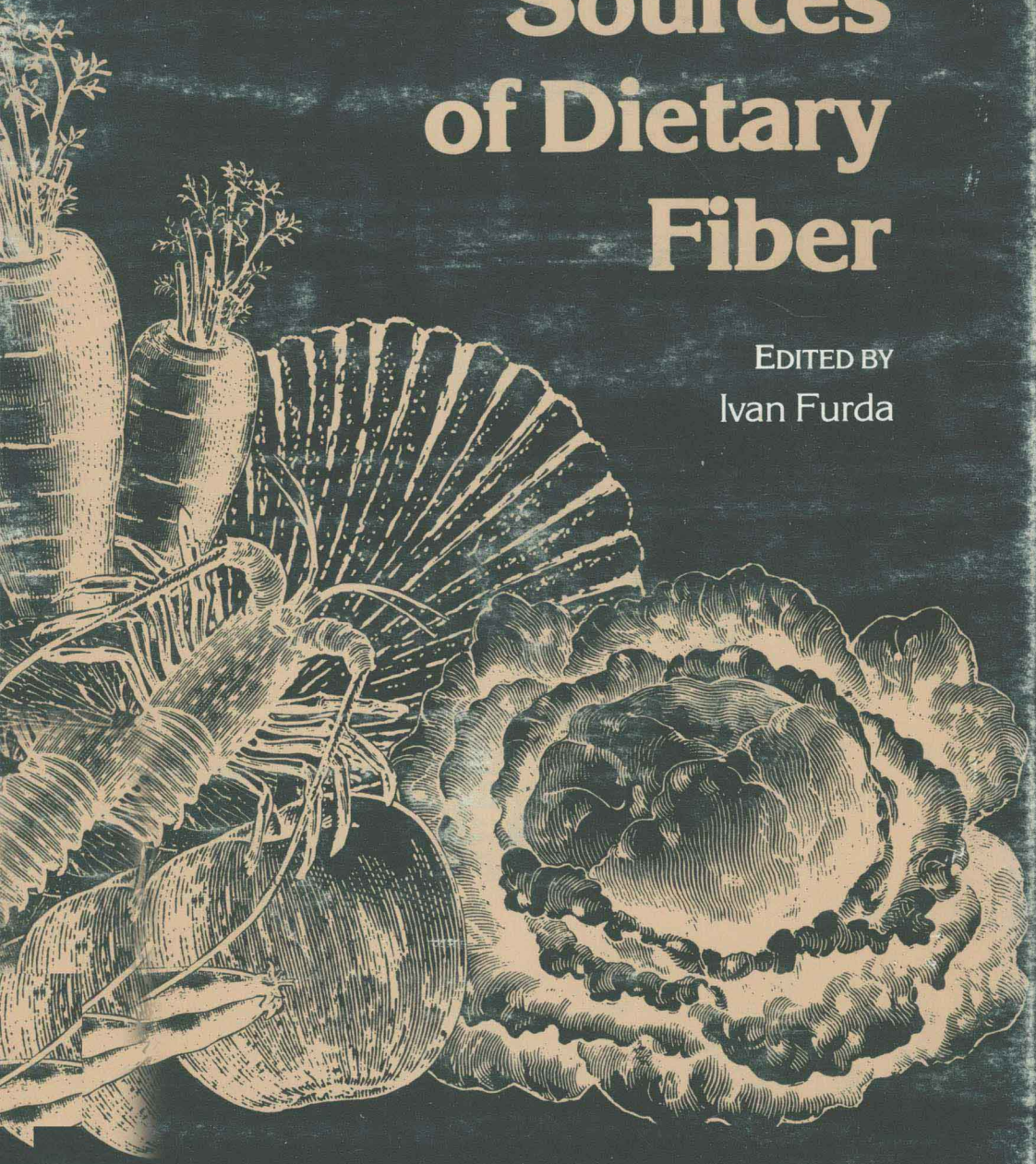
Ivan Furda



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Ivan Furda, EDITOR
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FOREWORD

The ACS SYMPOSIUM SERIES was founded in 1974 to provide a medium for publishing symposia quickly in book form. The format of the Series parallels that of the continuing ADVANCES IN CHEMISTRY SERIES except that in order to save time the papers are not typeset but are reproduced as they are submitted by the authors in camera-ready form. Papers are reviewed under the supervision of the Editors with the assistance of the Series Advisory Board and are selected to maintain the integrity of the symposia; however, verbatim reproductions of previously published papers are not accepted. Both reviews and reports of research are acceptable since symposia may embrace both types of presentation.

PREFACE

THE IDEA OF ORGANIZING THIS SYMPOSIUM was born at the 179th Annual ACS meeting in Houston in 1980, from discussions with the previous chairman of the Agricultural and Food Chemistry Division, Dr. Robert Ory. It was felt that the subject of dietary fiber should be viewed in a broader spectrum that should not cover excessively the current common, almost classical types, of fiber such as wheat and corn bran or cellulose. It should also encompass less conventional sources of dietary fiber which would be the main topic of this symposium. Because soluble nondigestible plant polysaccharides are now included in the definition of dietary fiber, and because numerous nondigestible polymers of nonplant origin exist, greater attention should be paid to these alternatives. These polymers have a variety of unique physiological, chemical, and functional properties that need to be better understood and utilized. This type of understanding is frequently achieved when different points of view are presented and a variety of scientists engage in discussion.

Presentations in this volume come from chemists, medical researchers, and microbiologists, as well as nutritionists and food scientists. In a few cases, rather exotic fiber types such as tobacco fiber, wheat straw lignin, or shellfish aminopolysaccharides are discussed. Other sources include psyllium, different legumes, and vegetable and fruit fibers. There is no doubt that additional unconventional fiber sources will be continuously identified and increasingly used.

It is my hope and belief that this publication will help in the expansion and utilization of fiber sources in foods, as well as in pharmaceutical and medical preparations. After all, we should recognize that the chance to accomplish this is quite favorable because "complex carbohydrates," which are the main constituents of dietary fiber, represent the only group of major food components for which increased daily intake is repeatedly being recommended.

I would like to express my appreciation and thanks to all participants of this symposium and to the writers of the individual chapters for their efforts and contributions. Without them, this undertaking and publication

would not be possible. Finally, I am grateful to Akiva Pour-El for his administrative help, and to General Mills and The Quaker Oats Company for their generous financial support.

IVAN FURDA
General Mills, Inc.
Minneapolis, MN 55427

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Effect of Conventional and Unconventional Dietary Fibers in Colon Carcinogenesis

BANDARU S. REDDY, KENSHI WATANABE,[1] and HIDEKI MORI[2]

American Health Foundation, Naylor Dana Institute for Disease Prevention, Valhalla, NY 10595

Epidemiologic and animal model studies suggest a protective effect of certain dietary fibers against colon cancer. This protection by the dietary fibers may be mediated through (a) the dilution of tumorigenic compounds in the gut,(b) the binding of tumorigenic compounds to the fiber in the gut, and (c) an indirect effect on the metabolism of carcinogens. Experiments were conducted in animal models to study the effect of certain non-conventional dietary fibers on colon carcinogenesis. Rats were fed the diets containing alfalfa, citrus pulp, pectin, wheat bran, undegraded carrageenan or cholestyramine and treated with azoxymethane (AOM), 3,2'-dimethyl-4-aminobiphenyl (DMAB), or methylnitrosourea (MNU) to induce tumors. Animals fed the wheat bran or citrus pulp and treated with AOM or DMAB had a lower colon tumor incidence than did those fed the control diet and treated with respective carcinogens. Alfalfa diet had no effect on AOM- or MNU-induced colon tumors, whereas the pectin diet inhibited AOM-induced but not MNU-induced colon tumors. Animals fed the diets containing undegraded carrageenan or cholestyramine developed more AOM and/or MNU-induced colon tumors than did the rats fed the control diet. Thus, the protective effect of various dietary fibers in colon carcinogenesis depends on the type of fiber fed, as well as the type of carcinogen used to induce tumors.

[1] Current address: Kagoshima University School of Medicine, Kagoshima, Japan
[2] Current address: Gifu University School of Medicine, Gifu City, Japan

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Rapid progress has been made in basic concepts concerning carcinogenesis (1,2). There are agents which are genotoxic, which by definition interact with the gene to yield an abnormal genetic material and would be considered initiating agents (3). The second broad class of agents act by epigenetic mechanisms and tend to increase the development of lesions initiated by genotoxic carcinogens. Thus, this list includes co-carcinogens which operate at the same time as genotoxic carcinogens and can alter the metabolism of a genotoxic agent with an increased ratio of activation/detoxification metabolites. Such agents can also act as more classic tumor promoters which exhibit their effect after the action of a genotoxic carcinogen.

Current concepts of colon cancer etiology stem from multidisciplinary research efforts based on three major approaches, namely: (1) the variation in incidence of colon cancer as a function of area of residence, with particular regard to migrant population; (2) the changes in incidence as a function of time; and (3) detailed laboratory studies in humans, in animal models, and through *in vitro* systems. The consistency of these findings suggests that environmental factors in general, and dietary factors in particular, play a dominant role in the development of colon cancer in humans.

Epidemiologic studies suggest that diets particularly high in total fat and low in fiber and in certain vegetables as well as high intake of beef are generally associated with an increased incidence of large bowel cancer in man (4-10). Dietary fat may be a risk factor in the absence of factors that are protective, such as use of high fibrous foods and fiber (11,12). As an example, in Finland, where the dietary intake of fat is similar to many of the Western countries and the fiber intake is higher, the incidence of colon cancer is lower than all of the Western countries.

This brief review evaluates current research on the relation between dietary fiber and large bowel cancer in humans, including the use of animal models. This brief review also presents an evaluation of the mechanism whereby certain dietary fibers including conventional and unconventional fibers modify the risk for the development of colon cancer.

Correlation and Case-Control Studies

Cross-national correlations between the incidence of colon cancer and dietary habits have been used to select hypotheses for testing in case-control and cohort studies. These studies have shown that certain food preferences appear to be associated with either a high- or a low-risk for colon cancer. When such correlations are supported by experimental evidence from animal studies, the hypothesis could be attractive.

Burkitt (10,13) first observed the rarity of large bowel cancer in most African populations and suggested that populations consuming a diet rich in fiber have a lower incidence of this type of cancer, while those eating refined carbohydrates

and little fiber have a higher incidence. A recent study comparing low-risk populations in Kuopio, Finland, with those at high risk in Copenhagen indicated that dietary fiber intake is higher in Finland compared with Copenhagen (14). The Finnish population is unique in this respect because in this country the total dietary fat is similar to countries with high rate of colon cancer. The crucial difference in dietary intake between Finland and Denmark and other Western countries may relate to dietary fiber and meat. Our data also suggest that one of the factors contributing to the low risk of large bowel cancer in Kuopio appears to be that a high intake of dietary fiber (mainly cereal fiber) leads to increased stool bulk, in effect diluting tumorigenic compounds in the colon (12). The results are consistent with a possible role for dietary fiber in the prevention of colon cancer in humans.

Case control studies have been conducted to study the possible relation of dietary fiber to large bowel cancer. Recently, Dales et al. (15) found that among American blacks significantly more colon cancer patients than controls reported that their diet was high in saturated fat and low in fibrous foods. Investigating many dietary constituents, Modan et al. (16) discovered that those contributing less to the diets of patients with colon cancer than to the diets of controls were those containing fiber. Bjelke (17) who interviewed hospitalized patients and controls in Minnesota and in Norway, learned that colorectal cancer patients less frequently ate vegetables, in particular the Minnesota patients ate less cabbage. Similarly, Graham et al. (18) found that individuals who ate vegetables such as cabbage, broccoli and Brussels sprouts had a lower risk of colon cancer.

These studies indicate that diets with a high intake of total fat and a low intake of certain fibers and certain vegetables are generally associated with an increased incidence of colon cancer in humans. Even in populations consuming high amount of fat, high dietary fiber acts as a protective factor in colon carcinogenesis.

Possible Mechanism of Protective Effect of Dietary Fiber In Colon Carcinogenesis

Although the concept of fiber involvement in colon carcinogenesis is attractive, the data often appear contradictory and confusing. Discrepancies may have arisen from the general misuse of fiber terminology. As well, experimental design has failed to account for the possible subtle effect of inhibitors, especially in relation to the promoting process. Evaluations of the biologic function of dietary fiber have often lacked complete information on the nature of the fiber.

Dietary fiber comprises a heterogenous group of carbohydrates, including cellulose, hemicellulose and pectin, and a noncarbohydrate substance, lignin (19). According to Van Soest (20), fibers can be classified into three groups: vegetable

fibers, which are highly fermentable and have little indigestible residue; brans, which are less fermentable; and chemically purified fibers, such as cellulose, which are relatively nonfermentable. Pectins and gums, soluble substances that are not true fibers, are considered part of the dietary fiber complex because of the similar effects they can elicit in the diet. Wheat bran and vegetable and fruit fibers have different percentages of cellulose, hemicellulose and lignin.
Carrageenan, a broad generic class of sulfated polysaccharides derived from a wide range of seaweed species, can be classified as non-conventional fiber and is used in food as an emulsifier, stabilizer, thickener and gelling agent.

The protective effect of dietary fiber may be due to adsorption, dilution or metabolism of cocarcinogens, promoters and yet-to-be-identified carcinogens by the components of the fiber (12,21,22). There is evidence that alfalfa, wheat straw and some other fibers can bind considerable amounts of bile acids *in vitro* (23). This indicates that the different types of nonnutritive fibers possess specific binding properties. Dietary fiber could also affect the enterohepatic circulation of bile salts (24) which act as colon tumor promoters (6). Fiber not only influences bile acid metabolism (12,22) thereby reducing the formation of potential tumor promoters in the colon, but also exerts a solvent-like effect in that it dilutes potential carcinogens and cocarcinogens by its bulking effect (12) and is able to bind bile acids and certain carcinogenic compounds (23,25,26). On the other hand, dietary undegraded carrageenan markedly enhances the bile acid content in the colon, thereby increasing the potential colon tumor promoters in the gut (6).

Smith-Barbaro *et al.* (26) in our laboratory determined the capacity of various fibers to bind the colon carcinogen 1.2-dimethylhydrazine (DMH) in vitro. The percent of DMH bound to wheat bran, corn bran, alfalfa fiber and dehydrated citrus pulp was dependent on pH of the medium as well as the type of fiber examined. Results from this study show that at colonic pH, a greater percent of DMH was bound by wheat bran than by citrus pulp. Therefore, it is possible that certain fibers bind carcinogen at colonic pH, thus making it unavailable for contact with the colonic mucosa. Other fibers such as pectin (soluble fiber) do not bind DMH at colonic pH, but may modify the metabolism of carcinogen via activation/deactivation steps either in the liver and/or in the colonic mucosa.

Investigations have been carried out in several laboratories to determine whether there are differences in fecal constituents between populations at high and low risk of colon cancer, and whether changes in the fiber content of the diet would alter the concentration of fecal bile acids that act as colon tumor promoters and the activity of fecal microflora. Recently, we studied healthy individuals in Kuopio, Finland, an area of low risk for the development of colon cancer (12). Dietary

histories indicated that the total fat consumption is similar to that in the United States but the intake of cereal fiber in Finland is higher and the daily output of feces three times higher than that of healthy individuals in the United States. The concentration of fecal secondary bile acids, mainly deoxycholic acid and lithocholic acid is less in Kuopio than in the United States, but the total daily output is the same in the two populations because of the threefold greater daily output of feces in Kuopio. This suggests that increased fecal bulk dilutes suspected carcinogens and promoters that may be in direct contact with the large bowel mucosa. Cummings (27) demonstrated that fiber from carrot, cabbage, apple, bran and guar gum produces different responses in fecal weight in humans related to the intake of pentose-containing polysaccharides in the fiber. The fecal weight increased by 127% when bran was added to the diet and 20% when guar gum was added; carrot, cabbage and apple produced intermediate changes.

In another study, Cummings (28) reported that an increase in cereal fiber intake from 17 to 45 g/d increased the fecal weight from 79 to 228 g/d and diluted the fecal bile acids. Kay and Truswell (29) showed that adding wheat fiber to the diet decreased the concentration of fecal bile acids and neutral steroids because of the bulking effect of fiber, whereas the addition of pectin to the diet increased the fecal steroid and bile acid output. These results suggest that the effect on fecal bile acid excretion may depend on the type of fiber consumed.

The effect of dietary wheat bran and alfalfa at 15% level on the composition of fecal bile acids was studied in rats fed a semipurified diet (30). Diets containing wheat bran and alfalfa caused a significant increase in stool weight. The concentration of fecal bile acids, particularly hyodeoxycholic acid, β-muricholic acid, deoxycholic acid and lithocholic acid was lower in rats fed wheat bran, compared to those fed a control diet,but the daily output of these bile acids was the same in both groups. Alfalfa had no effect on the concentration of fecal bile acids, but the daily excretion of deoxycholic acid, lithocholic acid and 12-ketolithocholic acid was increased compared to the control diet. It is apparent from this study that the fecal excretion of bile acids varies with the type and amount of dietary fiber.

Until recently, the nature of the carcinogens responsible for colon cancer not only was obscure, but there were no real leads. Because of potential importance of fecal mutagens in the genesis of large bowel cancer and of possible role of dietary factors in the induction of colon cancer, the fecal mutagenic activity of various population groups with distinct dietary habits and varied colon cancer incidences was determined by several investigators. Ehrich *et al.* (31) have demonstrated that the stools of South African urban whites who consume a high-fat, low-fiber diet and who are at high risk for colon

cancer development were higher (17% of the individuals) in mutagenic activity with Salmonella typhimurium strains TA98 and TA100 without microsomal activation compared to South African urban and rural blacks (0-5% of the individuals) who consume a low-fat, high-fiber diet and who are at low-risk. Bruce et al. (32) were the first to show that the feces of some normal humans consuming a high-fat, low-fiber diet contained compounds that caused direct mutagenesis of TA98 and TA100 in the Ames assay. They have also demonstrated that increased dietary fiber, -tocopherol or vitamin C reduced fecal mutagens. Kuhnlein et al. (33) compared fecal mutagens (water extracts) from a group of vegetarians consuming high-fiber diets with those from persons on typical North American diets containing meat. On TA100 and TA98, ovo-lactovegetarians and strict vegetarians had lower levels of fecal mutagens than non-vegetarians. Correlation studies between the pH of the fecal homogenate and mutagenicity indicate the presence of several fecal mutagens.

Recently, we have studied fecal mutagens of 3 populations with distinct risk for the development of colon cancer, a high-risk population in New York (non-SDA) consuming a high-fat, low-fiber, mixed-Western diet, a low-risk vegetarian SDA (Seventh-Day Adventists) and a low-risk population in Kuopio, Finland consuming a high-fat, high-fiber diet (34). Fecal samples of non-SDA were highly mutagenic in TA98 without microsomal activation, followed by TA100 without activation and TA100 with activation. None of the samples of SDA tested showed mutagenic activity in any of the tester systems, whereas Kuopio samples exhibited activity only in TA98 with microsomal activation.

Animal Model Studies in Colon Carcinogenesis: Effect of Conventional and Unconventional Fibers

Research on the mechanisms of cancer causation in the large bowel has been assisted by the discovery of several animal models that mirror the type of lesions seen in man. These models include (a) induction of large bowel cancer in rats through chemicals such as 3-methyl-4-aminobiphenyl, or 3-methyl-2-naphthylamine; (b) derivatives and analogs of cycasin and methylazoxymethanol (MAM) such as azoxymethane (AOM and 1,2-dimethylhydrazine (DMH), which work well in rats and mice of selected strains; and (c) intrarectal administration of direct-acting carcinogens of the type of alkylnitrosoureas, such as methylnitrosourea (MNU) or N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) which lead to cancer of the descending colon in every species tested so far.

The relation between dietary fiber consumption and colon cancer has been studied in experimental animals. Wilson et al. (35) found that Sprague-Dawley rats fed a diet containing 20% corn oil or beef fat and 20% wheat bran had fewer benign colon tumors induced by DMH given by gastric intubation than rats fed a control diet containing 20% fat and no bran. It is possible that with additional time, a number of tumors that were classi-

fied as benign might have developed a more invasive character. There was no difference in the incidence of colon cancer between the rats fed corn oil and those fed beef fat. In another study, Clapp et al. (36) found that DMH-induced colon tumors were increased in BALB/C mice fed a semisynthetic diet containing 5% fat and 20% W/W corn bran, soybean bran and soft winter wheat bran. DMH-induced colon tumor incidence in mice fed the control diet was surprisingly low, about 12%. In these studies, the experimental diets containing high-fat/no bran and high-fat/high bran, (35) or low-fat/no-bran and low-fat/high bran (36) differed substantially in caloric density. Animals on high bran diets might have consumed quantitatively different levels of vitamins and minerals compared to those fed high fat-no bran diet or low fat-no bran diets because the rats adjust their food intake to compensate for non-digestible fiber in high bran diet. It is also possible that certain fibers may cause abnormal histology and damage to colonic mucosa, thereby altering the absorptive capacity of the intestinal tract as well as bile acid-binding capacity (37,38) which have bearing on colon carcinogenesis.

A recent study by Fleiszer et al. (39) indicated that the incidence of colon tumors induced by DMH in rats decreases as the dietary intake of fiber increases. The number of animals used in this study was small. The diets in that study, namely high bran diet (28% fiber), special chow (15% fiber), rat chow (5% fiber) Flexical diet (0% fiber) differed not only in consistency (that is, solid or liquid) but also in the proportions of protein and fats, which have been shown to have an independent effect on colon carcinogenesis induced by DMH. However, the colon cancer incidence in fiber-free (Flexical) group was lower than in rat chow group. Some reduction in tumor incidence in the rats ingesting a high-fiber diet might be expected on the basis of reduced energy intake. Although the study's findings suggest that reduced intake alone cannot account for the significant protective effect of dietary bran, a better experimental design might have strengthened the results.

In another study, Cruse et al. (40) found that a diet containing 20% wheat bran had no effect on colon carcinogenesis induced by DMH in rats. Not only were the number of animals used in this study small (10 rats/group), but also the doses of the chemical in their experiment were so high (40 mg/kg body weight-/week for 13 weeks) that any protective effect of bran might have been unobservable. In a study of the effect of diet on chemical carcinogenesis, it is important to avoid exposing the animal to an excessive level of carcinogen for a long period, as this may obscure more subtle changes induced by certain dietary modifications. In addition, differences in caloric density of experimental diets attributable to the dilutional effect of added fiber further complicated the interpretation of the data. In fact, the data presented by Cruse et al.(40) suggest that a high-fiber diet reduces the frequency of death due to DMH in rats.

The effect of a diet containing 15% alfalfa, pectin or wheat bran on colon carcinogenesis by MNU or AOM was studied in F344 rats by Watanabe et al. (41). In this study, the experimental diets were not adjusted isocalorically. The addition of pectin or wheat bran to the diet greatly inhibited colon tumor incidence induced by AOM, a carcinogen requiring host-mediated metabolic activation (Table I). However, the incidence of AOM-induced colon tumors was not influenced by the addition of alfalfa to the diet. The diets containing wheat bran and pectin did not protect against MNU-induced colon carcinogenesis.

Table I

Colon Tumor Incidence in Female F344 Rats Fed Diets Containing Pectin, Alfalfa, or Wheat Bran and Treated with Azoxymethane or Methylnitrosourea

Diet	% Animals Colon Tumors	
	Azoxymethane treated	Methylnitrosourea treated
Control	57	69
Pectin	10[a]	59
Alfalfa	53	83[b]
Wheat Bran	33[a]	60

[a] Significantly different from the groups fed the control diet or alfalfa diet by X^2 test, $P<0.05$.

[b] Significantly different from the other groups, $P<0.05$.

Thus, modifying effect of pectin on AOM-induced colon carcinogenesis might be explained in terms of diet-dependent intestinal mucosal, as well as hepatic, microsomal carcinogen-metabolizing enzyme inhibitors or inducers in the diet that modify the capacity of the animal to metabolize the carcinogen (42). However, this factor would presumably not play a role in MNU-induced carcinogenesis because MNU does not require metabolic activation either in the liver or in the intestine. These results thus indicate that the protective effect of fiber in colon carcinogenesis depends on the type of carcinogen and the source of fiber.

The effect of alfalfa, wheat bran and cellulose on the incidence of intestinal tumors induced by AOM was further studied in Sprague-Dawley rats fed diets containing 10% alfalfa, wheat bran or cellulose and 30% beef fat, 20% alfalfa, bran or cellulose and 6% beef fat, or 30% alfalfa, bran or cellulose and 6% beef fat (21). The presence of 10% fiber in the high fat diet did not reduce the frequency of intestinal tumors. However, in the same study, presence of 20% bran or cellulose or 30% of any