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A MODEL SYSTEM FOR EVALUATING THE ROLE OF DIETARY FIBER IN CHEMICAL CARCINOGENESIS

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Fractions of foods resistant to the digestive enzymes of man have been defined as dietary fiber (1). These include lignins and the polysaccharides, cellulose, hemicelluloses and pectins of the plant cell wall (2). It is estimated that the dietary fiber intake of Western man has declined 50 to 80 percent during the last 100 years (3). Epidemiological evidence shows that a number of diseases of the digestive tract including colon cancer are more common in populations consuming diets low in fiber (2). It is possible that dietary fiber protects intestinal cells by binding carcinogens to enhance their excretion. Various components of fiber have been shown to bind bile acids *in vitro* (4,5) and to enhance excretion of fecal steroids (6,7) and other substances (8).

Presented in this communication is a model system which we believe can be useful in studying the role of dietary fiber and its component fractions in intestinal cancer. It is reasonable to anticipate that as the chemistry of fiber becomes more defined, an increasing number of crude and purified fiber preparations will become available for *in vivo* examination. A simple testing procedure for evaluating fiber preparations for their potentially protective action against carcinogens in the diet would have value for planning longer term more definitive studies.

Polycyclic aromatic hydrocarbons (PAH) are widely distributed in the environment primarily due to anthropogenic factors (9). Many of these compounds are carcinogenic in laboratory animals and man (10). Evidence indicates that extensive exposure to PAH can occur via dietary means, as during the consumption of charcoal broiled or smoked meats (11). Tissues exposed to benzo(a)pyrene respond by increasing the activity of aryl hydrocarbon hydroxylase (AHH), a microsomal mixed function oxidase enzyme which metabolizes PAH (12). Presented in this communication are data on AHH activity in the small intestine of rats fed diets with or without benzo(a)pyrene and with or without wheat bran.

Female Sprague-Dawley rats weighing 150-220 g were maintained in stainless steel cages with wire floors and no bedding. Twelve rats were randomly assigned to each of the

four experimental diets following a 24 hr fast. Treatment C was a control semi-synthetic diet that fulfills the known requirements for all nutrients (Table 1). Treatment C + WB was

Table 1. Percent composition of control diet

Ingredients	Percent
Casein*	20.0
Methionine	0.4
Corn oil	10.0
Vitamin mix+	1.0
Mineral mix‡	5.0
Sucrose	21.2
Dextrin	42.4

*Contains 92% protein.

+Vitamin Fortification Mixture, Teklad Test Diets, Madison, WI.

‡Rogers and Harper Salt Mixture, Teklad Test Diets, Madison, WI.

the control diet diluted with wheat bran† to a final concentration of 10% which also lowered the concentration of all other dietary constituents by 10%. Experience has shown that non-excessive percentages of fiber added to diets by dilution cause rats to increase their total food intake so that ultimately their nutrient consumption is equal to that of control rats given the same feed undiluted. Treatment BP was the control diet containing 1 mg of benzo-(a)pyrene/g of diet and treatment BP + WB was the control diet containing 1 mg BP/g followed by dilution with 10% wheat bran. Feeding was ad lib. for 48 hr; feed jars were removed 3 hr before sacrifice. The microsomal fraction was isolated by differential centrifugation from the first 25 cm of the small intestine (13). AHH was assayed by the method of Nebert and Gelboin (14). Rats fed the BP diet consumed significantly less food than rats fed the other diets ($P < .05$) (Table 2). There was no significant difference in benzo(a)pyrene intake for rats fed the BP or the BP + WB diet because the BP + WB fed rats increased their food intake. There were no significant differences ascribed to treatment in wet weights or in total microsomal protein for the first 25 cm of the small intestine (Table 3).

AHH activity for animals fed no benzo(a)pyrene was not significantly different at 21 ± 7 for C and 13 ± 7 for C + WB respectively (Fig. 1). However, induction of AHH was significantly greater for the BP group compared to the BP + WB, specific activities being 233 ± 25 and 152 ± 20 respectively. Decreased AHH induction indicates that wheat bran prevented

†American Association of Cereal Chemists certified food grade wheat bran RO7-3691.

Table 2. Food intake, benzo(a)pyrene intake and weight gains in rats fed the experimental diets

Treatment	Feed intake (g/day)	Benzo(a)pyrene intake (mg/day)	Weight gain (g/day)
C	12.0 ± 0.6[+]	0.0	6.1 ± 0.8
C + WB	12.5 ± 0.7[+]	0.0	8.8 ± 1.4
BP	10.8 ± 0.4[‡]	10.8 ± 0.4	6.5 ± 0.8
BP + WB	12.4 ± 0.5[+,‡]	11.6 ± 0.5	7.6 ± 0.9

[+,‡]Statistically significant from each other at $P < .05$.

Table 3. Wet weight and total microsomal protein in the first 25 cm of the small intestine in rats fed the experimental diets

Treatment	Intestinal weight (g)	Total intestinal microsomal protein (mg)
C	2.2 ± 0.1	17.6 ± 1.6
C + WB	2.1 ± 0.1	15.8 ± 1.1
BP	2.2 ± 0.1	15.9 ± 0.6
BP + WB	2.1 ± 0.1	15.0 ± 0.7

exposure of the intestinal epithelium to benzo(a)pyrene. These results cannot be attributed to differences in intake of calories, other nutrients, or benzo(a)pyrene.

Further research is necessary to determine if wheat bran and other fiber sources decrease absorption, enhance fecal excretion, and ultimately decrease the in vivo carcinogenic action of dietary PAH. We feel that this model system can be expanded to all segments of the intestinal tract including the colon. It has potential for evaluating the ability of other crude or purified fiber preparations in preventing intestinal exposure to carcinogenic PAH.

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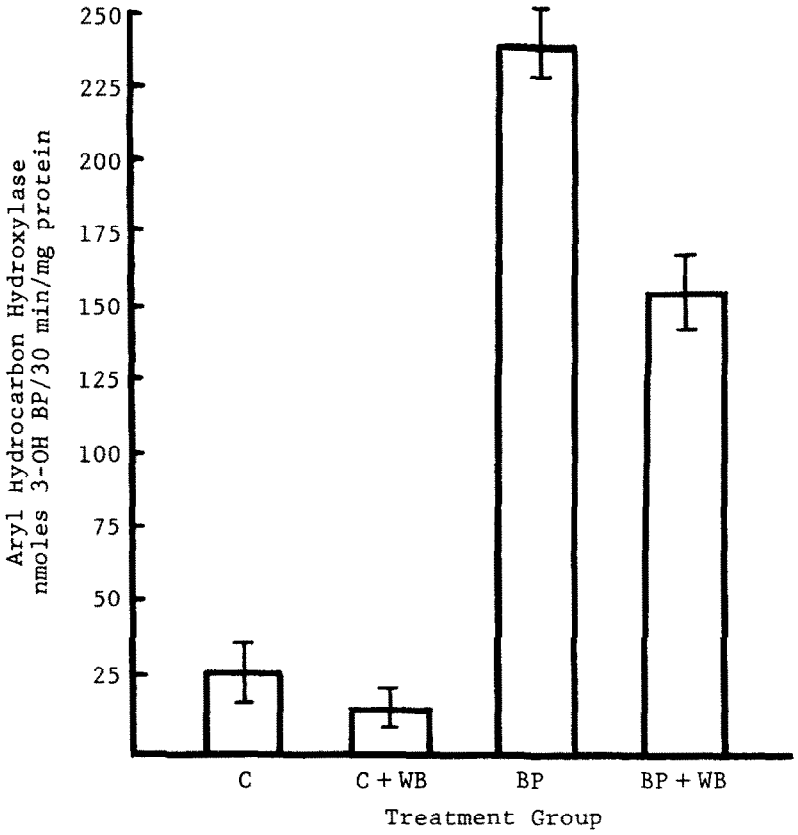

Fig. 1. Specific activity of aryl hydrocarbon hydroxylase in the small intestine of rats fed the experimental diets.

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