Nutrient Metabolism

Dry Beans Inhibit Azoxymethane-Induced Colon Carcinogenesis in F344 Rats

Joe S. Hughes, Chutima Ganthavorn and Susan Wilson-Sanders*

Food and Nutrition Science Program, Health, Physical Education, Exercise Science and Nutrition, Northern Arizona University, Flagstaff, AZ 86011-5095 and *University of Arizona Animal Care Diagnostic Laboratory, Tucson, AZ 85724

ABSTRACT Epidemiological studies show a low incidence of colon cancer in many Latin American countries where the consumption of dry beans (e.g., pinto) is high. The purpose of this study was to use rats as an animal model to obtain experimental data on the inhibition of colon carcinogenesis by dry beans. Fifty-three 5-wk-old weanling male F344 rats were randomly assigned by weight to the following groups: control (11 rats), casein diet (21 rats), and bean diet (21 rats). Animals fed the casein and bean diets were treated with the carcinogen azoxymethane (AOM) once weekly for 2 wk. Rats in the control group also consumed the casein diet but were not exposed to AOM. All diets were isocaloric. The protein concentration of the diets was adjusted to 18 g/100 g with casein, and the fat concentration was adjusted to 5 g/100 g with corn oil. Rats fed the bean diet had significantly fewer colon adenocarcinomas (P < 0.05) than rats fed the casein diet (5 vs. 22 tumors), and significantly fewer rats fed the bean diet (P < 0.05) had colonic tumors than did casein-fed rats (24 vs. 50%). Tumor multiplicity was also significantly lower for the bean-fed rats, and significantly fewer (P < 0.05) tumors per tumor-bearing rat were observed in bean-fed rats than in casein-fed rats (1.0 ± 0.0 vs. 2.5 ± 0.6). This study demonstrates that dry beans contain anticarcinogenic compounds capable of inhibiting AOM-induced colon cancer in rats. However, the specific anticarcinogenic components within dry beans have not been identified, and it is unclear whether dietary fiber, phytochemicals or other components within dry beans are primarily responsible for the anticarcinogenic properties of beans. J. Nutr. 127: 2328–2333, 1997.

KEY WORDS: · F344 rats · colon cancer · anticarcinogenic compounds · dietary fiber · pinto beans

Colon cancer is the second most common cause of cancer-related death (after lung cancer) in the United States (Greenwald et al. 1987). Although the etiology of colon cancer is multifactorial and complex (Rao et al. 1993), epidemiological data suggest that colon cancer is the form of cancer most closely associated with diet (Drasar and Irving 1973). The strong link between dietary factors and colon cancer has caused speculation that significant reductions in colon cancer incidence could be achieved through dietary modification (Doll and Peto 1981). Interest in lowering colon cancer risk by dietary means has also been stimulated by an awareness that existing methods for treating colon cancer are largely ineffective. Surgery is the principal method of treatment, but the surgical success rate for patients with recently diagnosed colon cancer is less than 40% (Bond 1993). The lack of an effective treatment has underlined the importance of developing a better understanding of the role of diet in preventing colon cancer (Willet 1989).

Dry beans (Phaseolus vulgaris) are a good source of both dietary fiber and a wide range of phytochemicals that have been linked to reduced colon cancer risk. Considerable interest has been focused on dietary fiber and colon cancer since Burkitt (1971) initially hypothesized a preventative role for dietary fiber. Although there is substantial epidemiological and experimental evidence to indicate that dietary fiber is protective (Watanabe et al. 1984), conflicting results also exist, emphasizing the need to clarify the link between dietary fiber and colon cancer. Because dietary fiber is not one entity but a complex mixture of chemically different compounds that share the common physiological characteristic of resisting enzymatic digestion, controversy exists as to which form or forms of dietary fiber are most effective (Kritchevsky 1986a, Wynder 1987).

Animal models have been widely used to evaluate the anticarcinogenic properties of dietary fiber, with rats being the most commonly used model (Reddy 1987). The majority of animal studies indicate that dietary fiber protects against colon cancer, but numerous inconsistencies have also been reported. Several authors recently reviewed the results of animal studies examining the dietary fiber-colon cancer link in an attempt to clarify inconsistencies (Jacobs 1987, Jenkins et al. 1986, Kritchevsky 1986b). Jenkins et al. (1986) reviewed 25 animal studies of dietary fiber and colon cancer and found that in 11 studies...
The relationship between dietary fiber and colon cancer is complicated by the need to control for other dietary factors, including dietary fat (Kritchevsky 1986a, Nigro and Bull 1987) and anticarcinogenic phytochemicals (Huang et al. 1994). Numerous phytochemicals have been shown to possess anticarcinogenic properties, and several researchers have used animal models to evaluate the ability of phytochemicals to inhibit colon cancer. Phytochemicals that have been shown to be potent colonic anticarcinogens in animals include green tea polyphenolics (Kim et al. 1994), isothiocyanates and other organosulfur compounds (Reddy and Rao 1994), isoflavones (Barnes et al. 1994), phenolic antioxidants (Wattenberg 1983), licorice (Webb et al. 1992) and inositol hexaphosphate (Pretlow et al. 1992, Ullah and Shamsuddin 1990).

Dry beans (P. vulgaris) are a staple food in many Latin American countries where the incidence of intestinal tract cancers is typically low (Correa 1981). Dry beans are among the best-known sources of dietary fiber and contain significant quantities of both soluble and insoluble dietary fiber (Hughes et al. 1996). Dry beans are also a rich source of numerous anticarcinogenic phytochemicals including polyphenolics, which possess both anticarcinogenic and antioxidant properties. The purpose of this research was to use rats as an animal model to obtain experimental data on the ability of whole dry beans to protect against azoxymethane (AOM)-induced colon cancer.

### MATERIALS AND METHODS

**Experimental animals.** A total of 53 male weanling (~3-wk-old) F344 rats from Bantin-Kingman Inc. (Fremont, CA) were used in this study. The rats were housed in individual cages in a temperature-controlled room and were placed under quarantine for 2 wk before the start of the experiment. Rats were given free access to food and water. The care and housing of the rats complied with institutional and NRC (1985) guidelines for animal care and use. After grouping by weight, rats were randomly assigned to one of three groups: control (11 rats), casein-fed (21 rats) and bean-fed (21 rats).

**Composition of diets.** The two semipurified diets used in this study were prepared by Purina Test Diets (Richmond, IN) and were isocaloric (Table 1). Rats in the control and casein-fed groups consumed the same bean-free casein diet, whereas rats in the bean group consumed a diet with dry beans as the primary nutrient source. Both diets were adjusted to 18 g protein/100 g diet with casein and were supplemented with 0.3 g DL-methionine/100 g to ensure adequate protein quality. The fat concentration of both diets was adjusted to 5 g/100 g with corn oil, and vitamin and mineral mixes were added according to American Institute of Nutrition standards (Reeves et al. 1993). Cornstarch made up the remainder of the diets. Every effort was made to ensure that the beans used in this study were similar to those consumed by humans. Pinto beans were purchased from a local grocery store and soaked in water overnight before being cooked until soft. Cooked beans were dried with their cook water overnight in a convection oven at 60°C. Dry cooked beans were provided to Purina Test Diets, where they were ground and incorporated into pellets. Fresh diets were prepared every 6 mo, and all diets were refrigerated to prevent spoilage.

**Experimental design.** The experimental design for this research was modeled after the experimental design of Reddy and Maruyama (1986). Rats were received when they were ~3-wk old. Rats were quarantined for 2 wk. During the 2-wk quarantine period, all rats were fed nonpurified diet (Purina Test Diets). When the rats reached 5 wk of age, the experiment began, with all rats being switched to the bean-casein diet for 3 wk. After the rats had consumed the casein diet for 1 wk, treatment with carcinogen began. Rats in the casein and bean experimental groups received subcutaneous injections (15 mg/kg body wt) of azoxymethane (AOM) (Sigma Chemical, St. Louis, MO) diluted with normal saline once weekly for 2 wk (a total of two injections of AOM). Rats in the control group consumed the casein diet and received injections of normal saline at the same time. One week after final exposure to AOM, rats in the bean-fed group were transferred to the bean diet, whereas rats in the control and casein-fed groups were fed the casein diet for the duration of the study.

### TABLE 1

**Composition of experimental diets fed to rats**

<table>
<thead>
<tr>
<th>Casein diet</th>
<th>Bean diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>g/100 g</td>
<td></td>
</tr>
<tr>
<td>Beans (pinto)</td>
<td>0.0</td>
</tr>
<tr>
<td>Cornstarch</td>
<td>52.0</td>
</tr>
<tr>
<td>Casein</td>
<td>20.0</td>
</tr>
<tr>
<td>Dextrose</td>
<td>13.0</td>
</tr>
<tr>
<td>Corn oil</td>
<td>5.0</td>
</tr>
<tr>
<td>Alphacel</td>
<td>5.0</td>
</tr>
<tr>
<td>DL-Methionine</td>
<td>0.3</td>
</tr>
<tr>
<td>Choline bitartrate</td>
<td>0.2</td>
</tr>
<tr>
<td>Vitamin mix</td>
<td>1.0</td>
</tr>
<tr>
<td>Mineral mix</td>
<td>3.5</td>
</tr>
</tbody>
</table>

1 The casein diet was consumed by rats in both the control and casein groups, whereas rats in the bean group consumed the bean diet.
2 Composition of the 59.1 g of beans was ~31.6 g starch, 13.6 g dietary fiber, 13.0 g protein and 0.8 g fat.
3 Vitamin mix AIN-93-VX (Reeves et al. 1993).
4 Mineral mix AIN-93G-MX (Reeves et al. 1993).

### TABLE 2

**Distribution of hyperplastic lesions and tumors in the intestinal tract (small intestine and colon) of azoxymethane-treated rats fed casein and bean diets**

<table>
<thead>
<tr>
<th>Hyperplastic lesions</th>
<th>Adenomas</th>
<th>Adenocarcinomas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small I</td>
<td>Colon</td>
<td>Small I</td>
</tr>
<tr>
<td>Casein</td>
<td>10</td>
<td>17</td>
</tr>
<tr>
<td>Bean</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>33</td>
</tr>
</tbody>
</table>

1 Twenty rats consumed the casein diet, and 21 rats consumed the bean diet. * Significantly different from the corresponding value for the casein group (P < 0.05). Small I = small intestine.
study. The experiment was terminated 34 wk after final exposure to carcinogen.

**Histological analysis.** At 34 wk after carcinogen treatment, all rats were killed by carbon dioxide inhalation and necropsied. Intestinal tracts were opened longitudinally, examined grossly for lesions and tumors, and fixed in 10% neutral buffered formalin for further detailed histological examination. Other internal organs were also examined grossly for tumors and, if necessary, fixed in 10% neutral buffered formalin for further examination. Histological examination of the tissues was conducted, and abnormal growths were categorized as being hyperplasia, adenomas or adenocarcinomas. Tissues with hyperplasia exhibited abnormal growth but had not formed a tumor, and showed no evidence of invading surrounding tissues. Adenomas were tumors with no evidence of invasion of surrounding tissues, and whose cell types resembled normal cells. Adenocarcinomas were tumors that were spreading to surrounding tissues and that showed cellular or nuclear abnormalities as well as increased mitotic rates.

**Statistical analysis.** Fisher’s exact test was used to assess statistical differences in tumor incidence among groups (Steel and Torrie 1980). Analysis of variance was used to test for differences in tumor multiplicity, and ANOVA and Duncan’s multiple range test for means separation (Steel and Torrie 1980) were used to assess differences in body weight. The chi-square test was used to assess differences in numbers of tumors (Steel and Torrie 1980). A probability of $P \leq 0.05$ was used for assessing statistical significance. Values in the text are means ± SD.

### RESULTS

**Food consumption and weight gain.** The body weights of the rats in the three different experimental groups did not differ throughout the experiment, and no significant differences were observed in body weights at the end of the study. The mean body weights for rats in the control, casein and bean groups at the end of the study were 440 ± 26, 441 ± 26 and 430 ± 25 g, respectively. Because the bean diet contains more dietary fiber (13.6 compared with 5 g/100 g), we anticipated that rats fed the bean diet might gain weight more slowly and have lower body weights. However, this concern was unfounded, and although the rats fed the bean diet did have lower body weights, no significant differences were observed when comparing the body weights of the three groups.

**Tumor distribution.** The incidence of intestinal tumors (adenomas and adenocarcinomas) was the primary endpoint of interest in this study, but data on hyperplastic lesions (Table 2) have also been included for purposes of comparison. Considerably more intestinal tumors were observed in the casein group than in the bean group (28 vs. 5), and the casein group consistently had more instances of both adenomas (5 vs. 0) and adenocarcinomas (23 vs. 5). The majority of tumors induced by exposure to AOM (Table 2) were adenocarcinomas (28/33; 85%), with only a few noninvasive adenomas being observed (5/33; 15%). Considerably more tumors were observed in the colon than in the small intestine (30 vs. 3), and this was true primarily for adenocarcinomas (27 vs. 1) but also for adenomas (3 vs. 2). The one adenocarcinoma observed in the small intestine was in the duodenal portion of a casein-fed rat. No adenomas were observed in rats fed the bean diet, but five adenomas were found in rats fed the casein diet: three in the colon and two in the small intestine.

Considerably more hyperplastic lesions were observed in the casein-fed rats (Table 2) than in the bean-fed rats (27 vs. 17). The majority of the hyperplasia occurred in the colon as opposed to the small intestine (33 vs. 11). An approximately equal number of hyperplastic lesions were found in the colon of rats consuming the casein and bean diets (17 vs. 16). However, in the small intestine 10 hyperplastic lesions were observed in rats fed the casein diet, whereas only one lesion was found in the bean-fed rats.

A majority of rats in the bean group showed no evidence of lesions or tumors (13/21; 62%), whereas only five rats in the casein group (5/20; 25%) were without lesions or tumors (Table 3). Only one rat had an adenocarcinoma without other hyperplastic tissues also being present, and that rat had con-

<table>
<thead>
<tr>
<th>Group</th>
<th>Rats with hyperplasia only</th>
<th>Adenocarcinomas only</th>
<th>Hyperplasia and adenocarcinomas</th>
<th>Hyperplasia, adenomas, and adenocarcinomas</th>
<th>No lesions or tumors observed</th>
<th>Total rats in group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casein-fed</td>
<td>5</td>
<td>1</td>
<td>6</td>
<td>3</td>
<td>5</td>
<td>20</td>
</tr>
<tr>
<td>Bean-fed</td>
<td>3</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>13</td>
<td>21</td>
</tr>
</tbody>
</table>

### TABLE 4

Percentages of azoxymethane-treated rats fed casein and bean diets and developing colonic tumors

<table>
<thead>
<tr>
<th>Group</th>
<th>Adenomas</th>
<th>Adenocarcinomas</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casein-fed</td>
<td>5% (1/20)</td>
<td>45% (9/20)</td>
<td>50%</td>
</tr>
<tr>
<td>Bean-fed</td>
<td>0% (0/21)</td>
<td>24% (5/21)</td>
<td>24%*</td>
</tr>
</tbody>
</table>

1 Twenty rats consumed the casein diet, and 21 rats consumed the bean diet. *Significantly different from the value for casein-fed rats ($P < 0.05$).
suggested the casein diet. The majority of rats with adenocarcinomas in both the bean (5/5; 100%) and the casein (9/10; 90%) groups also had other hyperplastic tissues present. Relatively few rats in both the casein (5/20; 25%) and the bean groups (3/21; 14%) had hyperplastic lesions without also having tumor formation. Fifteen of the 41 rats exposed to AOM developed tumors, but only one developed an adenoma without also developing an adenocarcinoma, and that animal had been fed the casein diet. Consequently, the vast majority of rats with tumors in this study developed adenocarcinomas (93.3%).

**Percentage of rats with tumors.** The endpoint of primary interest in this study was colonic adenocarcinomas. Significantly more rats fed the casein diet (P < 0.05) had colonic adenocarcinomas (45%) than those fed the bean diet (24%; Table 4). Colonic adenomas were observed in only one rat fed the casein diet. The incidence of total tumors (adenomas and adenocarcinomas) was significantly greater for the casein group (50%) than for the bean group (24%; Fig. 1), with twice as many rats in the casein group having tumors as rats in the bean group (Table 4). Similar results were obtained for tumors of the small intestine, but the low incidence of small intestine tumors in this study made comparisons difficult. Only one rat developed an adenoma in the small intestine, and one developed a small intestine adenocarcinoma (Table 5). Both of these rats consumed the casein diet, resulting in 10% of the rats in the casein group having small intestine tumors, whereas no small intestine tumors were observed in the bean group (Table 5).

**Tumor multiplicity.** The number of tumors per tumor-bearing rat is generally regarded as the best indicator of tumor multiplicity, but in this study the number of tumors per rat was also calculated for purposes of comparison (Table 6). Tumor multiplicity was lower for the rats fed the bean diet. Rats fed the casein diet had more than two adenocarcinomas per tumor-bearing rat (2.2 ± 0.5), whereas rats fed the bean diet had only one adenocarcinoma per tumor-bearing rat (P < 0.05, Table 6). Similar results were obtained for tumors per rat. Slightly more than one adenocarcinomas per rat (1.1 ± 0.3) was observed among rats consuming the casein diet, whereas significantly fewer adenocarcinomas per rat (P < 0.05) were observed for the bean group (0.2 ± 0.1).

### Additional information

Only eight tumors were observed in tissues other than the intestinal tract in this study, resulting in the majority of tumors (80%) being found in the intestinal tract, and only one tumor outside of the intestinal tract was malignant. The one malignant tumor was situated in muscle tissue adjoining the colon of a rat fed the bean diet, and it appeared to have spread to the muscle tissue from a nearby colonic adenocarcinoma. The remaining seven adenomas were distributed as follows: one adenoma in the seminal vesicle and one lipoma in the control group not exposed to AOM; two seminal vesicle adenomas in the casein group; and three lipomas in the bean group. Of the 42 experimental rats exposed to AOM, only one had to be killed before the experiment was completed. This animal was consuming the casein diet and was killed 4 wk before the end of the study because it was not eating and was losing weight. An autopsy revealed a large hair ball in the stomach, but no tumors were observed. The data for this rat were not included in the results.

### DISCUSSION

The results of this study show that whole dry beans are capable of inhibiting AOM-induced colon cancer in rats. Rats consuming the bean diet had a lower incidence of adenomas and adenocarcinomas in both the colon and small intestine. Several investigators have shown energy consumption to be an important variable influencing chemically induced cancers, with rats fed energy-restricted diets consistently having fewer tumors than rats given free access to food (Kritchevsky 1986a,b, Kumar et al. 1990). However, the absence of significant differences in body weight in this study indicates that energy intake had no apparent influence on the results. The fact that rats consuming the bean diet were able to thrive and gain weight in a manner similar to that of rats fed the casein diet also indicates that diets with high levels of beans (59%) are well tolerated by rats.

The results of this study are in agreement with the results of other studies using a similar experimental design. Other researchers exposing male F344 rats to similar levels of AOM (two injections of 15 mg/kg body wt) have typically observed a similar percentage of rats with colonic tumors, but they have often observed fewer adenocarcinomas. In the current study, 50% of rats fed the casein diet developed colon tumors (Table 4), with the majority of these rats (45%) having adenocarcinomas; only 5% had adenomas. In contrast, Kumar et al. (1990) fed male F344 rats a similar diet and reported that 56% of the rats developed colon tumors, but fewer rats developed adenocarcinomas (22%) than developed adenomas (44%). Similarly, Reddy and Maruyama (1986) observed colonic tumors in 40% of male F344 rats fed a diet similar to the casein diet in this study, with 37% of those tumors being adenomas and only 3% being adenocarcinomas.

Exposing male F344 rats to two doses of AOM (15 mg/kg body wt) is a commonly used model for evaluating the colon

### TABLE 5

<table>
<thead>
<tr>
<th>Group</th>
<th>Adenomas</th>
<th>Adenocarcinomas</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casein-fed</td>
<td>5% (1/20)</td>
<td>5% (1/20)</td>
<td>10%</td>
</tr>
<tr>
<td>Bean-fed</td>
<td>0% (0/21)</td>
<td>0% (0/21)</td>
<td>0%</td>
</tr>
</tbody>
</table>

1 Twenty rats consumed the casein diet, and 21 rats consumed the bean diet.

### TABLE 6

<table>
<thead>
<tr>
<th>Group</th>
<th>Adenomas/rat</th>
<th>Adenocarcinomas/rat</th>
<th>Total tumors/rat</th>
<th>Adenocarcinomas/ tumor-bearing rat</th>
<th>Total tumors/ tumor-bearing rat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Casein-fed</td>
<td>0.1 ± 0.1</td>
<td>1.1 ± 0.3</td>
<td>1.3 ± 0.4</td>
<td>2.2 ± 0.5</td>
<td>2.5 ± 0.6</td>
</tr>
<tr>
<td>Bean-fed</td>
<td>0.0 ± 0.0</td>
<td>0.2 ± 0.1*</td>
<td>0.2 ± 0.1*</td>
<td>1.0 ± 0.0*</td>
<td>1.0 ± 0.0*</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM for 20 rats in the casein-fed group and 21 rats in the bean-fed group. *Significantly different from the corresponding value for the casein-fed group (P < 0.05).
cancer promoting or inhibiting capacity of dietary components (Holt et al. 1996). The results of the current study indicate that this methodology has good specificity for colon carcinogenesis: 27 of 29 (93%) adenocarcinomas occurred in the colon. One adenocarcinoma was observed in the small intestine, and another seemed to have spread from the colon to muscle tissue adjacent to the colon. Azoxymethane is well known for its ability to induce tumors in the colon and small intestine (Holt et al. 1996) and has also been observed to produce liver (Reddy et al. 1985) and kidney (Clinton et al. 1992, Sugie et al. 1992) tumors. However, no liver or kidney tumors were observed in the current study. The male F344 rat seems to be a better model for studying colon carcinogenesis than the female F344 rat, because researchers have reported a greater than 50% incidence of kidney tumors in female F344 rats exposed to AOM (Hughes and Ganthavorn 1994, Reddy et al. 1985).

Dry beans are a good source of both soluble and insoluble dietary fiber (Hughes and Swanson 1989), are low in fat, and are generally regarded as a nutritious food. Dry beans have previously been linked to reduced risk of coronary heart disease (Anderson et al. 1984), diabetes and obesity (Geil and Anderson 1994), but little is known about their ability to inhibit colon cancer. Limited epidemiological data are available concerning dry bean consumption and disease, but some researchers have reported that colon cancer incidence is lower in countries with high levels of dry bean consumption (especially Latin America) and higher in countries such as the United States, where dry bean consumption is low. Correa (1981) reported a statistically significant negative correlation of \(-0.68\) \((P < 0.05)\) between dry bean consumption and colon cancer in 15 countries for which data on dry bean consumption were available. This study provides experimental data to support existing epidemiological research linking high levels of dry bean consumption with reduced colon cancer risk.

Although some controversy exists, dietary fiber has long been linked to reduced colon cancer risk, and dry beans are among the richest sources of dietary fiber. Current knowledge of the physiological effects of dietary fiber indicates that to provide maximum protection against colon cancer a dietary fiber should 1) be rich in insoluble dietary fiber, particularly cellulose; 2) contain sufficient soluble dietary fiber to produce butyrate upon fermentation; and 3) not increase bile acid concentrations in the feces (Kritchevsky 1986a). The dietary fiber in beans seems to be a good candidate for the anticarcinogenic properties of dry beans because bean dietary fiber (1) is among the best-known sources of insoluble dietary fiber and contains more insoluble fiber than most cereals (Hughes and Swanson 1989); 2) is rich in soluble dietary fiber that has been shown to be readily converted to butyrate in the colon (Fleming et al. 1985); and 3) has been shown to be equally as effective as oat bran in lowering blood cholesterol concentrations (Anderson et al. 1984). Also, unlike oat bran, dry beans do not significantly increase fecal bile acid concentrations (Anderson et al. 1984).

Besides being a good source of dietary fiber, dry beans are also rich in many phytochemicals with anticarcinogenic properties. Dry beans have been shown to contain considerable quantities of inositol hexaphosphate or phytate, polyphenolics, flavonoids, protease (trypsin) inhibitors, indoles and lignans. Although little is known about the anticarcinogenic properties of dry bean phytochemicals, other researchers have studied the anticarcinogenic properties of these phytochemicals obtained from other sources. Pretlow et al. (1992) found that phytate from either corn or rice inhibited colon carcinogenesis in F344 rats, and Kim et al. (1994) also used F344 rats to show that polyphenolics could inhibit colon carcinogenesis. Similarly, flavonoids from several plant sources have been shown to inhibit colon carcinogenesis (Singleton 1981), as have protease inhibitors (St. Clair et al. 1990, Weed et al. 1985) and indoles (Willett 1989). Also, Harris et al. (1994) reported that lignans isolated from flaxseed are potent broad-range anticarcinogens. In future research, we hope to clarify the relative contribution of dietary fiber and phytochemicals to the anticarcinogenic capacity of dry beans.

In conclusion, in this study a diet consisting of 59% dry beans consistently reduced the incidence of AOM-induced colon carcinogenesis in rats. Significantly fewer rats fed the bean-based diet had colonic tumors, and tumor multiplicity was also significantly reduced. The specific components responsible for the potent anticarcinogenic properties of dry beans are difficult to identify, because dry beans are a rich source of both dietary fiber and numerous phytochemicals with anticarcinogenic properties. Future research should clarify the role of dietary fiber and phytochemicals in contributing to the anticarcinogenic action of dry beans.

ACKNOWLEDGMENTS

We would like to thank Jessie Loganbill and Part Allen for their expert technical assistance.

LITERATURE CITED

BEANS INHIBIT COLON CARCINOGENESIS


