Emerging Evidence on the Role of Estrogenic Sorghum Flavonoids in Colon Cancer Prevention

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Colon cancer is the third most prevalent type of cancer in the United States, with 136,830 new cases and 50,310 deaths expected in 2014 (57). The probability of developing colon cancer is 5.0% (1 in 20) and 4.2% (1 in 22) for U.S. men and women, respectively (58). Colon cancer is also a common type of cancer worldwide; it is the third most diagnosed cancer in males (663,600 new cases/year) and the second most diagnosed cancer in females (570,100 new cases/year), as well as the most commonly diagnosed malignancy in the digestive tract (29). In 2008, more than 1.2 million new cases and 608,700 deaths are estimated to have occurred worldwide. The incidence of colon cancer in developed regions (e.g., Australia and New Zealand, Europe, and North America) is among the highest in the world, whereas the incidence in Africa and southeastern Asia is the lowest. Over the past decade, the incidence of colon cancer in developed regions has stabilized or decreased, primarily due to early detection and removal of precancerous lesions resulting from annual colon cancer screening (8,20). At the same time, the incidence in some regions where colon cancer used to be low, such as several countries in eastern Asia and eastern Europe, has increased (8,9). Adoption of a Western diet has been well documented in a number of observational studies. The proposed protective mechanisms of whole grains against colon cancer include stimulation of a healthy gut environment, provision of antioxidants (62), carcinogen binding, modulation of glycemic response, and production of hormone-like effects (from phytoestrogens) (59).

Whole Grains and Colon Cancer Prevention

Regular consumption of whole grains has been linked with 21–33% lower risk of colorectal cancer (27,54). The evidence has been well documented in a number of observational studies. The proposed protective mechanisms of whole grains against colon cancer include stimulation of a healthy gut environment, provision of antioxidants (62), carcinogen binding, modulation of glycemic response, and production of hormone-like effects (from phytoestrogens) (59).

Dietary fibers contained in whole grains also lower the risk of colon cancer (54,65). Insoluble nonstarch polysaccharides, resistant starches, and oligosaccharides act as laxatives in the large bowel, microbiota fermentation substrates, and prebiotics that promote gut health (65). Dietary fibers that are fermented in the gut produce short-chain fatty acids (SCFAs), namely acetate, propionate, and butyrate. These SCFAs lower the pH in gut lumen, which significantly reduces the absorption of ammonia (a carcinogen derived from urea) and potentially inhibits the overgrowth of pathogenic bacteria (65). The SCFAs, especially butyrate, also serve as energy sources for colonocytes, thus promoting optimal functioning of the colon epithelium (65). Dietary fibers also protect against colon cancer by diluting or adsorbing carcinogens (35), reducing colonic transit time, and altering bile acid metabolism (34).

Whole grains reduce glycemic index and glycemic loads by contributing dietary fibers with various structures, which may alleviate abnormal glucose metabolism and dyslipidemia due to refined grain consumption (24,42). This protective effect was shown in an Italian study in which researchers observed an inverse association between the glycemic index or load of a diet and colorectal cancer risk (23).

It is clear that the dietary fibers contained in whole grains play a major role in colon cancer prevention. However, evidence indicates that the benefits of whole grains go beyond what their dietary fibers provide. In the NIH-AARP Diet and Health Study involving 489,611 participants aged 50–71 years, the benefits of whole grain fibers in reducing the relative risk of colorectal cancer only accounted for two-thirds of the reduction in relative risk shown with whole grain consumption (54). Bioactive compounds concentrated in the bran fractions of whole grains are among the nonfiber whole grain components that contribute to protection against colorectal cancer.

Various phytochemicals and minerals present in whole grains also provide antioxidant benefits in the gastrointestinal tract epithelia. Important groups of phytochemicals found in whole grains include...
phenolics, carotenoids, vitamin E compounds, phytates, and oligosaccharides (e.g., inulin) (44). Major antioxidant minerals found in cereals include zinc and selenium (1). The most common and abundant phenolic compounds contained in whole grains are phenolic acid derivatives (mainly benzoic and cinnamic acid derivatives, e.g., caffeic and ferulic acid esters of long-chain mono- and dialcohols). Other phenolic compounds include flavonoids, lignans (phytoestrogen), and condensed tannins (proanthocyanidins), which are found in specific grains (18). Most of these phytochemicals are bound to cell wall materials (44) and, thus, survive digestion in the upper gastrointestinal tract and reach the colon. The colonic microbiota fermentation of insoluble fibers may release these phenolics and make them bioavailable (39). For example, ferulic and diterolic acids from cereal bran can be released by gastrointestinal esterase from intestinal mucosa and microbiota (2). Dietary fibers, condensed tannins, and phytic acids may bind excess iron and heavy metals contained in the diet and, thus, provide antioxidant benefits (59).

Whole grain polyphenols play many protective roles, such as antioxidant, anti-inflammatory, and tumor suppression. The ability to activate estrogen receptor-β (ERβ) and associated protective mechanisms (i.e., exert hormone-like effects) is increasingly recognized as a major mechanism by which whole grains protect against colon cancer.

**Estrogen Signaling and Receptors**

The female steroid hormone estrogen is essential for the development and functions of the female reproductive system and related mammary glands. Estrogen also plays an important role in other endocrine-responsive systems, such as brain, bone, cardiovascular system, and adipose tissue (45). Estrogen also is believed to participate in the development and progression of various diseases: for example, certain cancers (breast, ovarian, colorectal, and prostate), osteoporosis, cardiovascular, and cognition (13). The function of estrogen is mainly mediated through the estrogen receptor (ER).

The ER is a ligand-binding inducible transcription factor that after activation regulates the expression of target genes (45). Two ER subtypes have been discovered in humans and rodents: ERα and ERβ. The two ER subtypes are ubiquitously expressed in many tissues but have distinct tissue distribution patterns and physiological functions. ERα is the major ER found in reproductive systems (i.e., endometrium, breast, ovarian stroma, and hypothalamic tissue), and ERβ is the major ER found in all other nonreproductive system-related tissues (e.g., kidneys, brain, bone, heart, lungs, intestinal mucosa, and endothelial cells) (13).

The genomic (classical) action of estrogen is ligand binding. Estrogen binds to the ligand-binding domain of the ER and then induces ligand-specific conformational changes in the protein (termed “activation” of the ER) (64). The ligand-binding receptor (estrogen-ER) then dimerizes and binds to a specific DNA-binding domain, estrogen-responsive element (ERE), located within the promoter region of the target genes. The estrogen-ER binding to ERE results in recruitment of coregulatory proteins (coactivators or corepressors) to the promoter, an increase or decrease in mRNA levels and associated protein production, and, finally, a physiological response (13). The two ER subtypes lead to different signaling pathways: cell growth for ERα and apoptosis for ERβ (56). ERα is the main ER mediating the essential function of estrogen in the development of female reproductive systems. ERβ is believed to prevent or protect against disease in tissues where it is predominantly expressed, such as the colon (28). ERβ is prevalent in normal colon mucosa, and it is capable of modulating colonic physiological functions (41). The major form of estrogen, 17β-estradiol (E2), reduces nonmalignant colonocyte growth in vitro and colonic tumor formation in vivo through ERβ-mediated apoptosis (67) and p53 activation (68). This suggests that ERβ is important in the early stages of colon carcinogenesis and that activation of ERβ could be a mechanism in chemoprevention.

**Role of Phytoestrogens in Colon Cancer Prevention**

Due to the ligand-binding activation mechanism of ER, estrogens are not the only group of compounds that can bind to ER. Other compounds capable of binding to ER are termed selective ER modulators, and they include phytoestrogens. Phytoestrogens are a group of plant-derived polyphenolic compounds that can modulate the activation of ERs, i.e., they possess a function similar to the major form of estrogen, 17β-estradiol (E2) (31,33). Estrogenic compounds have been documented in more than 300 plant species, but only a few of these compounds are found in the diets of humans and animals. Common dietary phytoestrogens include isoflavones and coumestans found in legumes (especially soybeans), alfalfa sprouts, and clover; lignans found in oilseeds (especially flaxseeds and sesame seeds) and whole grains; prenyllflavonoids found in hops and beer; and flavonoids and stilbenes. Among phytoestrogens, isoflavones (e.g., genistein, daidzein, and their glycosides) and lignans (e.g., secoisolariciresinol and matairesinol) are the two most recognized groups in the human diet (15).

Phytoestrogens have been reported in observational studies to play protective roles in preventing colon cancer. For example, a case-control study in Toronto found higher intake of lignans from a typical Western diet (>0.255 mg/day) was associated with a significant (27%) reduction in colorectal cancer risk compared with the lowest dietary consumption of lignans (0–0.158 mg/day); the same study also observed a similar reduction in colorectal cancer risk in people who consumed higher amounts of dietary isoflavones (>1.097 versus 0–0.289 mg/day) (12). Similarly, a recent case-cohort study of Danish men and women examined plasma concentrations of enterolactone, the microbial metabolites of lignans, and observed that with each doubling of enterolactone concentration, the relative risk for colon cancer in women decreased by 24%. However, no protective effect was found in men (30).

Some cell-culture and animal studies have linked the colon cancer prevention capacity of phytoestrogens with activities related to ERβ, such as restoring the expression of ERβ (52) and inhibiting tumor progression through ERβ-mediated G2/M cell cycle arrest (5).

**Role of Sorghum Polyphenols in Cancer Prevention**

Sorghum (Sorghum bicolor) production ranks fifth among cereal crops globally, with more than 50 million tons produced in 2012 (21). The agronomic advantages of sorghum (primarily drought and heat tolerance) make it suitable for growing in most arid and semiarid areas around the world. Sorghum grain is a staple food and important cereal in many parts of Africa, Asia, and the Middle East (59). In these areas sorghum is consumed mainly in couscous, porridge, baked goods (e.g., leavened breads, flat breads, cookies, and...
steamed breads), and fermented foods and beverages. Sorghum is also an important alternative grain used in gluten-free diets (55). In addition to the grain, the leaf sheaths of some red sorghum varieties are used as food colorants in traditional African cooking (32).

In Africa and parts of Asia, evidence suggests that regular consumption of sorghum and millets (40, 66), as well as the amount consumed (11), is associated with lower incidence of esophageal cancer compared with areas where wheat and corn are the major cereals consumed. A study by Isaacson (26) suggests increased incidence of esophageal cancer among South African blacks is due to substitution of sorghum with corn as a diet staple (26). These observations provide evidence that sorghum may have cancer chemopreventive properties that other grains do not possess due in part to its unique phenolic profile.

Sorghum grain has a higher polyphenol content and antioxidant capacity than other major cereal grains such as wheat, barley, millet, and rye (51). Sorghum also contains phenolic compounds not commonly found in other cereal grains. For example, the extractable phenolic acids found in sorghum are mostly esters of glycerol (Fig. 1) (63), whereas in other grains phenolic acids are generally non-esterified. Flavanoids in general are not the major groups of phenolics found in cereal grains; however, they tend to be the most abundant groups found in sorghum. Among the common flavonoids found in sorghum are 3-deoxyanthocyanin pigments (in pigmented grains), flavones (high content in grains with a tan secondary plant color), flavanones (high content in grains with a lemon-yellow pericarp), and polymeric flavonoids (condensed tannins in grains with a pigmented testa) (17).

Animal and lab experiments suggest that the unique profile of polyphenols in sorghum is partly responsible for its greater cancer chemoprotective properties relative to other grains. In a 2008 study, black and tannin sorghum brans (6% in diet) decreased the number of azoxymethane-induced premalignant lesions in the co-

![Fig. 1. Skeletal structures of major sorghum phenolic compounds.](attachment:image1.png)

![Fig. 2. Effects of phenolic extracts from black, white, and red sorghum varieties, alone and cotreated with 1 μM ICI 182, 780 (estrogen receptor antagonist), on growth of nonmalignant, young adult mouse colonocytes. Data are expressed as means ± SEM from three separate experiments. Dunnett’s t test was used to compare the means of each treatment with the control after one-way analysis of variance. Asterisks indicate treatments differed significantly from the control (DMSO) (P < 0.05). Reproduced with permission from Yang et al. (71).](attachment:image2.png)

<table>
<thead>
<tr>
<th>Cereal Grain</th>
<th>Lignans</th>
<th>Flavones</th>
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<tbody>
<tr>
<td>Sorghum</td>
<td>-c</td>
<td>3.5–386d</td>
</tr>
<tr>
<td>Wheat</td>
<td>7–15</td>
<td>Very lowa</td>
</tr>
<tr>
<td>Rye</td>
<td>27.5–37.5</td>
<td>-</td>
</tr>
<tr>
<td>Oats</td>
<td>13–19.5</td>
<td>Very lowf</td>
</tr>
<tr>
<td>Corn</td>
<td>9</td>
<td>-</td>
</tr>
<tr>
<td>Millet</td>
<td>6</td>
<td>1.9–123.5g</td>
</tr>
<tr>
<td>Brown rice</td>
<td>4</td>
<td>-</td>
</tr>
</tbody>
</table>

a Sources: Smeds et al. (60, 61).
b Flavanones were detected only in sorghum at 134–1,380 μg/g (16, 19).
c Information not available.
d Sources: Dykes et al. (16, 19).
e Sources: Dinelli et al. (14) and Liu et al. (38).
f Source: Dykes and Rooney (18).
g Source: Chandrasekara and Shahidi (10).
The sorghum bran lowered proliferation and induced apoptosis in colonocytes, possibly due to their ability to induce murine endogenous antioxidant enzyme activities (superoxide dismutase, catalase, and glutathione peroxidase) (37). Park et al. (48) found that Hwanggeumchal (which means "golden" in color and "sticky" in texture) sorghum extracts reduced the growth of human breast tumor xenografts in mice and blocked metastasis to the lungs. The mechanism was linked to cycline D1-induced G1 cell cycle arrest and suppressed tumor growth by downregulation of Jak2/STAT pathways (48). Moreover, this sorghum extract possessed a higher capacity to decrease expression of oncogenic proteins than extracts from other grains, i.e., wheat and millet (48). However, the composition of the sorghum extract was not provided. In a 2011 study by Wu et al. (69) a procyanidin-rich sorghum extract inhibited tumor growth in C57BL/6j mice with Lewis lung cancer by suppressing vascular endothelial growth factor; in addition the sorghum extract improved the activity of antioxidant enzymes (superoxide dismutase and glutathione peroxidase) after d-galactose–induced oxidative stress. These studies show that sorghum phenolics can suppress tumor progression in mouse models; however, the active components have not been characterized.

We previously demonstrated that phenolic extracts from sorghum grains with various phenolic profiles inhibited human esophageal (OE33) and colon adenocarcinoma (HT-29) cell growth in vitro at low concentrations (IC50 [concentrations that inhibit 50% of cell growth] of 49.7–883 µg/mL). The antiproliferative activity was correlated with the antioxidant capacity of the phenolic extracts: tannin sorghum extracts were the most potent inhibitors followed by black sorghum rich in 3-deoxyanthocyanin pigments (4). In addition, we showed that specific nontannin sorghum extracts induced quinone oxidoreductase (a phase II protective enzyme) activity in murine liver cells (Hepa1c1c7): sorghum high in 3-deoxyanthocyanins was the most potent inducer. Further research with pure 3-deoxyanthocyanidins revealed a structure–activity relationship: O-methyl substitution on the A ring increased the quinone oxidoreductase–inducing capacity of 3-deoxyanthocyanidins (71). This evidence indicates that the phenolic composition of sorghum affects its cancer chemopreventive potential (4).

These observational studies with animals and cell cultures suggest, directly and indirectly, that phenolics present in sorghum grains contribute significantly to cancer chemoprevention. Possible mechanisms involved include antioxidant activity, activation of endogenous detoxification enzymes, antiproliferation (induction of apoptosis or cell cycle arrest), and anti-inflammation activity by specific polyphenols in sorghum. Emerging evidence suggests that estrogenic activity may be one of the mechanisms by which sorghum contributes to greater cancer chemoprevention relative to other grains. Two groups of flavonoids, flavones and flavanones, identified in sorghum have been reported as estrogenic. Apigenin and luteolin (and their glycosides) and naringenin and eriodictyol (mainly in the form of glycosides) are the major flavones and flavanones, respectively, detected in sorghum (17). In cells expressing ERα or ERβ, apigenin, luteolin, and naringenin were able to activate ER (25,43). In addition, flavones in general showed better estrogenic activity than flavanones, and among flavones, monohydroxylated B-ring compounds (such as apigenin) showed better estrogenic activity than their catechol substituted counterparts (such as luteolin) (25,43). Table I summarizes the amounts of flavones and flavanones detected in sorghum compared with other major cereal grains.
flavonoid composition that targets. We have confirmed using additional sorghum varieties with specific differences in flavonoid composition that flavones (e.g., apigenin) are the most active phytoestrogens in sorghum and that flavanones are also active, but at much higher concentrations.

Estrogenic potency among flavones also differs. As reported by Harris et al. (25) and in many other studies, in cells transfected with ERβ apigenin is a more potent phytoestrogen than other flavones, such as luteolin. This trend is reflected in the estrogenic activity of sorghum that contains flavones. For example, among extracts from white and black sorghum varieties, the white sorghum extract had a much higher apigenin content than the black sorghum extract, even though they contain similar amounts of total flavones (Table II). As can be seen in Figure 2, the ER antagonist ICI did not completely block the growth-inhibiting effect of black sorghum extract on YAMC, suggesting growth inhibition by black sorghum is only partially attributable to ER activation. This indicates the flavones in the black sorghum extract produced less ER-specific activity than the flavones in the white sorghum extract. Thus, even with sorghum varieties that contain similar amounts of flavones, the specific composition of the flavones has a major impact on their ability to activate ER. Our investigations with pure forms of these compounds have revealed similar patterns (data not shown), confirming the potentially significant role of flavones, especially apigenin, in ER activation.

An important question is whether in vitro observations would be reflected in vivo. We used ovariectomized mice (animals with their ovaries removed by surgery) injected with azoxymethane (AOM) to evaluate the effects of extracts from white and black sorghum varieties on colon carcinogenesis. Rodents (rats and mice) injected with colon-specific carcinogens (e.g., AOM) are the most widely used models to study the pathogenesis of sporadic (adenoma–carcinoma sequence) colorectal cancer (53). The in vivo metabolized form of AOM is a DNA-methylating agent that leads to the formation of preneoplastic lesions, aberrant crypt foci (ACF), in the colon in a relatively short time (6). ACF has been used previously as a biomarker to evaluate the functions and mechanisms of chemopreventive agents against colon cancer (7,47). Ovariectomized animals are used in estrogenic compound studies to remove the effects of endogenous estrogens.

Compared with the control group (standard test diet), white sorghum extract fed at the level of 1% in the diet significantly reduced the total number of ACF in the colon by 39.3% (P < 0.001). Black sorghum extract fed at the same level (1%) also significantly reduced the total number of ACF but to a smaller extent (14.7% reduction; P < 0.044) (Fig. 4). The ability of these two estrogenic sorghum extracts to reduce the number of ACF formed in distal colons of ovariectomized mice suggests protection against colon carcinogenesis via their estrogen-like activities. This finding agrees with the results obtained from our cell culture experiments (Fig. 2) (72). The white sorghum extract contained 4.31 mg of total flavones/g, with 3.93 mg of apigenin/g, whereas the black sorghum extract contained 2.26 mg of total flavones/g, with only 0.26 mg of apigenin/g (Table II). The higher concentration of apigenin in white sorghum extract (3.93 mg/g of extract) was likely related to its stronger inhibiting effect against ACF formation compared with black sorghum extract (0.26 mg of api-genin/g) fed at the same concentration. A higher dose of black sorghum extract (1.5%) did not produce an effect that was significantly different from the 1% black sorghum extract (Fig. 4).

Table II. Summary of phenolic compound compositions in phenolic extracts from white, red, and black sorghum varieties

<table>
<thead>
<tr>
<th>Phenolic Compound</th>
<th>Cell Culture Study</th>
<th>Animal Study</th>
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<tbody>
<tr>
<td></td>
<td>White</td>
<td>Red</td>
</tr>
<tr>
<td>Total phenols* (Folin-Ciocalteu method)</td>
<td>24.9 ± 4.3</td>
<td>88.4 ± 5.2</td>
</tr>
<tr>
<td>Total flavones†</td>
<td>2.27 ± 0.77</td>
<td>ND</td>
</tr>
<tr>
<td>Apigenin</td>
<td>1.70 ± 0.59</td>
<td>ND</td>
</tr>
<tr>
<td>Luteolin</td>
<td>0.57 ± 0.18</td>
<td>ND</td>
</tr>
<tr>
<td>Methylated flavones</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Total flavones*</td>
<td>ND</td>
<td>0.52 ± 0.002</td>
</tr>
<tr>
<td>Naringenin glycosides</td>
<td>0.52 ± 0.002</td>
<td>ND</td>
</tr>
<tr>
<td>Naringenin</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Total 3-deoxy-anthocyanins*</td>
<td>ND</td>
<td>1.29 ± 0.29</td>
</tr>
<tr>
<td>Total phenolic acids and esters†</td>
<td>26.8 ± 10.2</td>
<td>119.7 ± 10.8</td>
</tr>
</tbody>
</table>

* All values are expressed as mg/g (mean ± SD of two separate runs), as quantified by HPLC with appropriate standards. ND = not detected.
† Expressed as mg of gallic acid equivalent/g of extract.
‡ Sum of major peaks identified and quantified by HPLC. Adapted in part from Yang et al. (71).

The protective effect of apigenin in reducing AOM-induced formation of premalignant lesions (ACF) in the colons of rodent models has been reported previously (3,36). The proposed mechanisms include suppression of colonocyte proliferation (36) and colonic ornithine decarboxylase activity (a rate-limiting enzyme in polyamide biosynthesis) (3). Interestingly, the white sorghum extract fed at 1% had an effect similar to that previously reported for apigenin fed at 0.1% (3,36) in reducing the total number of ACF (approximately 40% versus 50% reduction). However, the white sorghum extract only contained 3.93 mg of api-
genin/100 g of diet (0.004%), which is orders of magnitude lower than an active concentration of pure apigenin, even considering all the flavones added together. This suggests two possibilities: 1) there may be other unknown phytoestrogens in sorghum; or 2) there is significant synergistic activity among different forms of estrogenic flavonoids in sorghum. Our ongoing investigations suggest the latter.

Conclusions

In summary, the evidence presented from cell culture and ovariectomized mice models suggests that the inherent estrogenic activity of specific sorghum flavonoids may contribute to colon cancer prevention at concentrations that are achievable through diet. It also suggests that the composition of phenolic compounds, not content, has a major effect on the estrogenic activity and protective efficacy of sorghum. Flavones, especially apigenin, are the most potent estrogenic components of sorghum polyphenols. This information provides an important insight that improves our understanding of the specific mechanisms by which sorghum phenolics influence prevention of certain cancers. Sorghum grain is clearly a source of unique bioactive polyphenols. As new evidence emerges, the protective effect of sorghum against gastrointestinal cancers previously reported in epidemiological studies appears to be linked to its unique phenolic profile.

References


An ad appeared here in the print version of the journal.

![Fig. 4. Effects of crude extracts of white and black sorghum varieties fed in the diet on total numbers of premalignant lesions, aberrant crypt foci (ACF), identified in distal colons of ovariectomized mice injected with azoxymethane. Data are expressed as numbers identified per animal (mean ± SEM). Student’s t test was used to compare the means of each treatment with the control after one-way analysis of variance. Treatments with the same letter do not significantly differ (P < 0.05).](image-url)
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52. Raju, J., Bielecki, A., Caldwell, D., Lok, E., Taylor, M., Kapal, K., Curran, I., Cooke, G. M., Bird, R. P., and Mehta, R. Soy isoflavones modulate azoxymethane-induced rat colon carcinogenesis exposed pre- and...


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