Flaxseed Consumption Influences Endogenous Hormone Concentrations in Postmenopausal Women

Andrea M. Hutchins, Margaret C. Martini, B. Amy Olson, William Thomas, and Joanne L. Slavin

Abstract: Lignans, similar in structure to endogenous sex steroid hormones, may act in vivo to alter hormone metabolism and subsequent cancer risk. The objective of this study was to examine effects of dietary intake of a lignan-rich plant food (flaxseed) on serum concentrations of endogenous hormones and binding proteins (estrone, estrone sulfate, 17β-estradiol, sex hormone-binding globulin, progesterone, prolactin, dehydroepiandrosterone sulfate, dehydroepiandrosterone, androstenedione, testosterone, and free testosterone) in postmenopausal women. This randomized, crossover trial consisted of three seven-week feeding periods, during which 28 postmenopausal women, aged 52–82 yr, consumed their habitual diets plus 0, 5, or 10 g of ground flaxseed. Serum samples collected during the last week of each feeding period were analyzed for serum hormones using standard diagnostic kits. The flaxseed diets significantly reduced serum concentrations of 17β-estradiol by 3.26 pg/ml (12.06 pmol/l) and estrone sulfate by 0.09 ng/ml (0.42 nmol/l) and increased prolactin by 1.92 µg/l (0.05 IU/ml). Serum concentrations of androstenedione, estrone, sex hormone-binding globulin, progesterone, testosterone, free testosterone, dehydroepiandrosterone, and dehydroepiandrosterone sulfate were not altered with flaxseed feeding. In this group of postmenopausal women, consuming flaxseed in addition to their habitual diets influenced their endogenous hormone metabolism by decreasing serum 17β-estradiol and estrone sulfate and increasing serum prolactin concentrations.

Introduction

Lignans, compounds found in plants that possess some estrogenic or antiestrogenic activity (1–3), are believed to play a role in cancer prevention (2,4,5). Similar in structure to endogenous sex steroid hormones (Figure 1), lignans are diphenolic compounds that are hypothesized to act in vivo to alter hormone metabolism and subsequent risk for hormone-dependent cancers (1,4–6). Flaxseed is the most concentrated food source of the plant lignan secoisolariciresinol. Secoisolariciresinol is converted by colonic microflora to the mammalian lignan enterodiol (7). Enterodiol can then be oxidized by colonic microflora to form the mammalian lignan enterolactone (7,8). Flaxseed also contains small quantities of the plant lignan matairesinol, which is also converted by colonic microflora to enterolactone (7,8). Enterodiol and enterolactone, along with their plant lignan precursors, are absorbed by the colon, undergo enterohpatic circulation, and are excreted in the urine and feces (9–11).

Consumption of flaxseed has been shown to reduce early risk markers for and incidence of mammary carcinogenesis in animal models (12–14), affect menstrual cycle length in premenopausal women (6), delay onset of puberty in animal models (15,16), and lengthen estrous cycles in animals (15, 16). Enterolactone and enterodiol have been shown to decrease cell proliferation (17–19) and inhibit aromatase (20, 21), 5α-reductase (22), and 17β-hydroxysteroid dehydrogenase activity (22). Enterolactone excretion has also been positively associated with higher concentrations of sex hormone-binding globulin (SHBG) in epidemiological studies (1,9), and it increased concentrations of SHBG in an in vitro study (23). Flaxseed also contains the n–3 fatty acid α-linolenic acid, which has been shown to reduce the incidence of mammary tumorigenesis (24) and reduce growth of already established mammary tumors (14).

Although flaxseed, its mammalian lignan products (enterolactone and enterodiol), and α-linolenic acid have been the subject of numerous studies examining their effects on hormone metabolism and cancer risk in vitro and in vivo, to our knowledge, no studies have examined the effects of flaxseed consumption on hormone metabolism in postmenopausal women. The objective of this study was to examine effects of dietary intake of a lignan-rich plant food (flaxseed) on the serum concentrations of endogenous hormones and a binding protein [estrone, estrone sulfate, 17β-estradiol, SHBG, progesterone, prolactin, dehydroepiandrosterone...
one sulfate (DHEAS), dehydroepiandrosterone (DHEA), androstenedione, testosterone, and free testosterone] in post-menopausal women.

**Materials and Methods**

**Subjects**

Healthy, postmenopausal women, recruited from the Monastery of the Sisters of the Order of St. Benedict in Central Minnesota, were screened with a detailed dietary/medical questionnaire to exclude those who had gastrointestinal disorders, food allergies, alcohol intake greater than two drinks per day (equivalent to 720 ml beer, 240 ml wine, 90 ml hard liquor), smoked, had taken antibiotics within the last six months, used hormone replacement therapy, or had dietary habits that were not representative of the general population (e.g., exclusion of an entire food group from their diet, <20% or >45% of energy from fat, consumption of >3 servings of flaxseed/flaxseed products per week). After the screening, 34 subjects were contacted and agreed to participate by providing informed written consent. The Institutional Review Board: Human Subjects Committee at the University of Minnesota approved the study. All subjects were healthy, nulliparous, and at least one year postmenopausal as determined by subjects reporting the date of their last menses occurring more than one year previous to the start of the study. Seven subjects were not taking any prescription medications but, because of the age of the subjects, it was not possible to exclude for all medications. Of the remaining 27 subjects, 10 were taking thyroid replacement medications, 9 were taking antihypertensives, 7 were taking antihyperlipidemics, 6 were taking diuretics, 3 were taking antidepressants, and 1 was taking a corticosteroid. In all cases, medication use was consistent throughout the study.

Of the 34 subjects who began the study, 32 completed all feeding periods. One subject withdrew when she moved to another state, and another withdrew for medical reasons unrelated to study participation. During the study, four subjects began hormone replacement therapy; their results are not included in the statistical analyses reported here. Results from the remaining 28 subjects who completed the entire study were included in statistical analyses. Baseline values (means ± SD) for age, height, weight, and body mass index of the subjects were 68.3 ± 8.5 years, 161.9 ± 4.3 cm, 62.3 ± 10.8 kg, and 23.9 ± 3.9 kg/m², respectively.

**Experimental Design**

This study was a randomized, crossover trial consisting of three 7-week feeding periods with a 7- or 14-week washout period between feeding periods. One of the feeding periods was used as a control period, during which subjects consumed only their usual diets. During the remaining feeding periods, subjects consumed their usual diets plus either 5 or 10 g of ground flaxseed each day. The 5-g flaxseed supplement provided ~25 kcal, 1.2 g of protein, 1.8 g of fat (50–60% α-linolenic acid), 1.4 g of carbohydrate, and 1.1 g of dietary fiber, including 0.6 g of soluble fiber. The 10-g flaxseed supplement provided twice these amounts. During the flaxseed-supplemented feeding periods, subjects were supplied daily with one tube containing 5 or 10 g of ground flaxseed. The flaxseed was kept frozen at −20°C until consumption, and subjects were instructed to consume the contents of one tube daily in raw form. Subjects typically consumed the flaxseed in one serving at breakfast. Used tubes were collected, and any uneaten flaxseed was measured to monitor subject compliance. Subjects were asked to maintain their usual body weight and exercise habits throughout the study.

Ground flaxseed was prepared weekly from commercially available whole flaxseed (Frontier Whole Flax Seed,
Whole flaxseed was ground to a coarse texture in a household blender for one minute. The flaxseed was then immediately aliquoted into 5- or 10-g doses and frozen at -20°C. To determine the plant lignan content of the flaxseed, the secoisolariciresinol-diglycoside content was measured by high-performance liquid chromatography (25). According to the analysis, the 5 and 10 g of ground flaxseed provided 10 and 20 mg (27.6 and 55.2 μmol) of secoisolariciresinol [2 mg (5.5 μmol) secoisolariciresinol/g ground flaxseed], respectively. Matairesinol was not measured, since it comprises only ~0.3% of the total lignans in flaxseed (26).

**Sample Collection and Analysis**

During the last week of each feeding period, subjects completed self-reported three-day diet records to monitor food intake, body weights were measured, fasted blood samples were collected on two consecutive days of the three-day diet record period, and two 24-hour urine collections were completed. Fasted blood samples were centrifuged at 2,650 rpm (1,500 g) for 20 minutes at 4°C. Plasma was drawn off and placed in cryovials that were flushed with nitrogen and then frozen at -70°C until analysis.

Commercially available radioimmunoassay kits were used to determine serum concentrations of estrone, estrone sulfate, SHBG, total testosterone (Diagnostic Systems Labs, Webster, TX), 17β-estradiol, DHEAS (Pantex, Santa Monica, CA), androstenedione, and DHEA (Associated Regional and University Pathologists, Salt Lake City, UT). Commercially available microparticle enzyme immunoassay kits were used to analyze progesterone and prolactin (Abbott Laboratories, Abbott Park, IL). Free testosterone was analyzed by equilibrium dialysis with radioimmunoassay by the Fairview-University Medical Center Endocrinology Laboratory. Intra- and interassay coefficients of variation were 6% and 11% for estrone, 2% and 2% for estrone sulfate, 8% and 12% for DHEAS, 3% and 2% for SHBG, 18% and 9% for total testosterone, 2% and 5% for progesterone, 3% and 8% for prolactin, 3% and 6% for androstenedione, and 3% and 4% for DHEA. Interassay coefficients of variation are 6% for 17β-estradiol and 5% for free testosterone; no intra-assay information is available for these hormones. Results given for estrone sulfate are based on triplicate assays, free testosterone on 23 subjects. To correct for data that were not normally distributed, analysis and \( P \) value computations for 17β-estradiol, estrone, estrone sulfate, testosterone, and free testosterone were performed on a logarithmic scale. Analysis and \( P \) value computations for androstenedione, DHEA, DHEAS, prolactin, SHBG, and progesterone were performed on an arithmetic scale, since the data were normally distributed. For reporting purposes, data summaries of logarithmically transformed data were transformed back to the original scale. All comparisons in Table 3 were pre-planned so that multiple comparisons were not used. Results were considered statistically significant at \( p < 0.05 \). Exact \( P \) values for significant results are given in Results.

**Results**

Body weight measurements and body mass index at the end of each feeding period are presented in Table 1. Because the subjects maintained their body weights throughout the duration of the study, there were no significant differences in these measurements among any of the feeding periods.

Intakes of total energy, carbohydrate, protein, fat, and dietary fiber during each feeding period are presented in Table 2. There were no significant differences in total energy, carbohydrate, protein, fat, or total fiber intake among any of the feeding periods. For soluble fiber, intakes for the 5-g flaxseed feeding period (9.0 ± 2.2 g) and the 10-g flaxseed feeding period (9.4 ± 2.2 g) were significantly higher than that for control (8.2 ± 2.0 g, \( p = 0.0182 \) and 0.0012, respectively). The 5- and 10-g flaxseed diets reduced serum concentrations of 17β-estradiol by 2.90 pg/ml (10.7 pmol/l, \( p = 0.0386 \)) and 3.62 pg/ml (13.4 pmol/l, \( p = 0.0096 \)), respectively. The 10-g flaxseed diet reduced serum concentrations of estrone sulfate by 0.10 ng/ml (0.47 nmol/l, \( p = 0.0185 \)) and increased serum prolactin concentrations by 2.20 μg/l (60.6 IU/ml, \( p = 0.0123 \); Table 3). Serum concentrations of estrone, SHBG, progesterone, androstenedione, testosterone, free testosterone, DHEA, and DHEAS were not significantly altered with flaxseed feeding (Table 3).

Consumption of flaxseed significantly increased the excretion of enterodiol, enterolactone, and total lignans on the 5- and 10-g flaxseed diets compared with the control diet (\( p < 0.0001 \)). Matairesinol excretion was not changed with flaxseed consumption. Complete urinary enterodiol, enterolactone, and total lignans in flaxseed feeding.

**Table 1. Subject Weight and BMI During Each Feeding Period**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>5 g</th>
<th>10 g</th>
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<tbody>
<tr>
<td>Weight, kg</td>
<td>62.7 ± 10.5</td>
<td>62.3 ± 11.0</td>
<td>61.9 ± 10.2</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.2 ± 3.9</td>
<td>23.9 ± 4.1</td>
<td>23.7 ± 3.7</td>
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\( a: \) Values are means ± SD; \( n = 28 \). BMI, body mass index. There were no significant differences.

Statistical analyses were performed using the Statistical Analysis System (SAS Proprietary Software release 6.11, copyright 1989–1995, SAS Institute, Cary, NC). Results were analyzed using a repeated-measure analysis of variance within subject. Because of lack of sufficient serum for analysis, results of testosterone are based on 25 subjects and free testosterone on 23 subjects. To correct for data that were not normally distributed, analysis and \( P \) value computations for androstenedione, DHEA, DHEAS, prolactin, SHBG, and progesterone were performed on a logarithmic scale. Analysis and \( P \) value computations for estrone, estrone sulfate, testosterone, and free testosterone were performed on a logarithmic scale. Analysis and \( P \) value computations for androstenedione, DHEA, DHEAS, prolactin, SHBG, and progesterone were performed on an arithmetic scale, since the data were normally distributed. For reporting purposes, data summaries of logarithmically transformed data were transformed back to the original scale. All comparisons in Table 3 were pre-planned so that multiple comparisons were not used. Results were considered statistically significant at \( p < 0.05 \). Exact \( P \) values for significant results are given in Results.
lactone, and total lignan excretion results have been reported previously (28).

Serum estradiol concentrations were negatively correlated with urinary enterodiol ($R = -0.2226$, $p = 0.0458$), enterolactone ($R = -0.3163$, $p = 0.004$), and total lignan (enterodiol + enterolactone + matairesinol) excretion ($R = -0.3396$, $p = 0.0019$). There were no correlations between serum estrone sulfate or serum prolactin concentrations and excretion of urinary enterodiol, enterolactone, or total lignans.

**Discussion**

Consuming 5 or 10 g of flaxseed per day in addition to their habitual diets decreased serum 17β-estradiol and estrone sulfate concentrations and increased serum prolactin concentrations in this group of postmenopausal women. Although these changes were relatively small, these results suggest that consuming flaxseed may offer protection against the incidence of breast cancer in postmenopausal women.

An increased lifetime exposure to estrogen is a well-recognized risk factor for increased incidence of breast cancer (29–32). Early age at menarche, late age at menopause, late age at first full-term pregnancy, nulliparity, and use of estrogen replacement therapy increase a woman’s risk of breast cancer (29–32) by increasing her lifetime exposure to estrogen; factors that decrease these exposures may offer protection against breast cancer (32,33). The overall role of diet in the incidence of breast cancer has been supported by

<table>
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<th>Table 2. Nutrient Intakes During Diet Treatments$^{a,b}$</th>
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<tbody>
<tr>
<td>Control</td>
</tr>
<tr>
<td>Energy, kcal</td>
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<td>CHO g</td>
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<td>% energy</td>
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<td>Total dietary fiber, g</td>
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<tr>
<td>Soluble fiber, g</td>
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<td>Insoluble fiber, g</td>
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$^a$: Values, which include nutrient contribution of flaxseed, are means ± SD; $n = 28$. CHO, carbohydrate.

$^b$: Significantly different from control: *, $p = 0.0182$; †, $p = 0.0012$.

$^c$: 1 kcal = 4.184 kJ.

<table>
<thead>
<tr>
<th>Table 3. Serum Hormone Concentrations During Each Feeding Period$^{a,b,c}$</th>
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<tr>
<td>Control</td>
</tr>
<tr>
<td>Androstenedione, ng/ml</td>
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<tr>
<td>DHEA, ng/ml</td>
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<tr>
<td>DHEAS, µg/dl</td>
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<td>Estrone sulfate, ng/ml</td>
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<tr>
<td>Progesterone, ng/ml</td>
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<td>Prolactin, µg/l</td>
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<tr>
<td>SHBG, nmol/l</td>
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<td>Testosterone, ng/dl</td>
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$^a$: Values are least-squares means ± SE and geometric means (95% confidence interval). DHEA, dehydroepiandrosterone; DHEAS, DHEA sulfate; SHBG, sex hormone-binding globulin.

$^b$: Where group means differ significantly ($p < 0.04$), groups with the same symbol (*, †) are indistinguishable; where there are no symbols, means are not significantly different.

$^c$: Conversions for conventional units to Système International units: androstenedione, ng/ml * 3.45 = nmol/l; DHEA, ng/ml * 3.44 = nmol/l; DHEAS, µg/dl * 27.1 = µmol/l; 17β-estradiol, pg/ml * 0.0037 = nmol/l; estrone, pg/ml * 3.7 = pmol/l; estrone sulfate, ng/ml * 4.7 = nmol/l; progestosterone, ng/ml * 3.18 = nmol/l; prolactin, µg/l * 0.027 = IU/ml; testosterone (total and free), ng/dl * 34.7 = nmol/l.
numerous epidemiological studies (29,32,34,35), even though some studies have failed to show correlations between individual dietary factors and breast cancer (32,35). Despite the lack of consensus among the epidemiological studies, certain dietary components may influence endogenous hormone concentrations. For example, diets high in fiber, plant products, and phytoestrogens, including lignans, may offer protection by lowering endogenous hormone concentrations (29,32–36), while diets high in fat and animal products may increase risk by increasing endogenous hormone concentrations (29,32–34).

Increased concentrations of estrone sulfate, the primary circulating estrogen in postmenopausal women, and 17β-estradiol, the most biologically active estrogen in the circulation of pre- and postmenopausal women, may increase a woman’s risk of developing breast cancer (31,37,38). Epidemiological studies have suggested that women who develop breast cancer have estrogen concentrations, in particular estradiol concentrations, that are 10–20% higher than those of comparable control subjects (31,37,38). Therefore, factors that decrease circulating concentrations of these two estrogens would be considered to be protective against breast cancer. In this study, consumption of 5 or 10 g of flaxseed per day for seven weeks decreased the concentrations of estrone sulfate and 17β-estradiol by 0.09 ng/ml (0.42 nmol/l) and 3.26 pg/ml (12.1 pmol/l), respectively. Compared with the epidemiological studies, the decrease in estradiol concentrations, 10% and 13% on the 5- and 10-g flaxseed diets, respectively, suggests that these changes are biologically as well as statistically significant.

In a similar study conducted with premenopausal women, Phipps and co-workers (6) reported that consumption of 10 g of flaxseed per day did not alter serum concentrations of estradiol or estrone sulfate. Consequently, the effects of flaxseed on serum estradiol and estrone sulfate concentrations may depend, in part, on the endogenous hormone concentrations of the population being studied. On the basis of the reductions in estradiol and estrone sulfate observed in this study, consumption of flaxseed, a concentrated source of plant lignans, theoretically may offer protection against breast cancer by reducing concentrations of biologically active endogenous hormones in postmenopausal women.

The exact mechanism by which flaxseed, its component plant lignans secoisolariciresinol and matairesinol, or the mammalian lignan products enterolactone and enterodiol lower the concentrations of estrone sulfate and 17β-estradiol is not known. However, enterolactone and secoisolariciresinol have been shown to inhibit aromatase activity in vitro (20,21,39). Aromatase is a principal regulator of estrogen biosynthesis in humans, including the conversions of androstenedione to estrone and testosterone to 17β-estradiol (40,41). Inhibition of aromatase by enterolactone or secoisolariciresinol would decrease the amount of 17β-estradiol formed from testosterone, thereby explaining the decrease in 17β-estradiol concentrations found in this study. The conversion of estrone to estrone sulfate is catalyzed by a sulfokinase. No studies have examined the effects of secoisolariciresinol, matairesinol, enterolactone, or enterodiol on sulfokinase activity. On the basis of the results of this study, we hypothesize that one or more of these plant or mammalian lignans may inhibit this enzyme, decreasing the conversion of estrone to estrone sulfate.

Enterolactone and enterodiol have also been shown to inhibit 17β-hydroxysteroid dehydrogenase, the family of enzymes that catalyze the reversible conversion of 17β-estradiol to estrone, in vitro (22). Inhibition of 17β-hydroxysteroid dehydrogenase could explain the decrease in 17β-estradiol concentrations observed with the consumption of flaxseed in this group of postmenopausal women. The effects on estrogen metabolism may also be due to the combined effect of the lignans, not the effect of one or two individual lignans. Evans and colleagues (22) reported that a cocktail containing 10 μM of each of seven phytoestrogens, including enterolactone and enterodiol, reduced 17β-hydroxysteroid dehydrogenase activity by 94%, with a half-maximal effect produced with 0.7 μM of each of the compounds in the cocktail. Therefore, phytoestrogens, including the lignans enterolactone and enterodiol, have been shown to affect enzyme activity within the currently known plasma concentrations of the compounds (42,43).

The decrease in estrone sulfate concentrations reported here might also be due in part to the increased conversion of estrone to 2-hydroxyestrogen. Haggans and associates (44) reported that, in this same group of subjects, flaxseed consumption increased the excretion of 2-hydroxyestrogen, but not 16α-hydroxyestrone. This suggests that flaxseed, or one of its components, may increase the activity of the cytochrome P-450 enzymes responsible for catalyzing the conversion of estrone to 2-hydroxyestrogen. Thus, if more estrone is converted to 2-hydroxyestrogen, less is available for conversion to estrone sulfate.

The role of prolactin in the risk for and incidence of breast cancer is controversial. Researchers have suggested that increased concentrations of prolactin may promote (45,46), protect against (47,48), or be neutral (48,49) in relation to the incidence of breast cancer.

The synthesis of prolactin is stimulated by estrogens and inhibited by dopamine (47,48). Therefore, a decrease in estrogen concentrations, as was reported with 17β-estradiol and estrone sulfate in this study, would be expected to decrease the production of prolactin. However, we observed an increase in prolactin concentrations with flaxseed consumption. To our knowledge, the effects of flaxseed, its component plant lignans secoisolariciresinol and matairesinol, or the mammalian lignans enterolactone and enterodiol on prolactin production have not been studied. Other studies have reported that serum prolactin concentrations are positively correlated with a high-fat diet (50), protein intake (50,51), total fatty acid intake (50,51), and saturated fatty acid intake (50,51). However, because these factors did not change between diet periods in this study, we did not observe similar correlations. Studies have reported that enterolactone, en-
terodiol, secoisolariciresinol, and matairesinol exhibit estrogenic activity (1–3). Therefore, it is possible that the estrogenic activity of these compounds may have compensated for the decreased endogenous estrogen concentrations observed in this study. This compensation may provide an explanation for the increase in prolactin concentrations.

Previous studies have reported that enterolactone stimulates SHBG synthesis in vitro (9), and increased excretion of enterolactone is correlated positively with SHBG concentrations (9,52) in epidemiological studies. However, in studies in which flaxseed was fed as part of a controlled or uncontrolled diet, the same results have not been observed. In our study, flaxseed consumption was not associated with an increase in SHBG concentrations. In agreement with our results, Shultz and others (53) fed six men 13.5 g flaxseed/day and also found no significant changes in SHBG concentrations. No change in SHBG concentrations was also reported by Phipps and co-workers (6) when 18 premenopausal women consumed 10 g flaxseed/day for three menstrual cycles.

We found no change in total testosterone or free testosterone concentrations after flaxseed consumption. In contrast to our results, Phipps and co-workers (6) reported that in premenopausal women, whose testosterone concentrations are similar to those of postmenopausal women, consuming 10 g flaxseed/day increased midfollicular phase, but not early follicular or luteal phase, testosterone concentrations. However, Shultz and others (53) also found no change in total testosterone or free testosterone concentrations in men, whose testosterone concentrations are ~10 times those of postmenopausal women, after flaxseed consumption. The conflicting results of these studies point to the need for further research that examines the role of flaxseed consumption in the metabolism of the androgens.

We found no significant changes in the concentrations of androstenedione or DHEA with flaxseed consumption in this group of postmenopausal women. To our knowledge, changes in the concentrations of these hormones as the result of flaxseed consumption have not been reported by any other research group. We also found no significant changes in the concentrations of estrone, DHEAS, or progesterone, results supported by the study of Phipps and co-workers (6), who reported that consumption of flaxseed did not change the concentrations of these hormones in premenopausal women.

In conclusion, consumption of 5 or 10 g of flaxseed per day for seven weeks influenced estrogen and prolactin concentrations, but not androgen or binding protein concentrations, in this group of postmenopausal women. The decreases in 17β-estradiol and estrone sulfate concentrations reported here suggest that consuming flaxseed may offer protection against breast cancer. However, the increase in prolactin concentrations may negate the beneficial effects of flaxseed consumption in relation to breast cancer if prolactin is determined to promote the disease. Therefore, further research is needed to determine whether consumption of flaxseed as a dietary supplement, or nutraceutical, may provide chemoprotective effects against the incidence of breast cancer in postmenopausal women.

Women are also taking flaxseed for its perceived ability to mitigate menopausal symptoms and decrease the risk for osteoporosis and heart disease in addition to or in place of traditional hormone replacement therapy. Although the results of our study suggest that the lignans associated with flaxseed do have estrogenic activity in vivo, further research is needed to determine whether this activity is sufficient to decrease a postmenopausal woman’s risk for osteoporosis and heart disease or alleviate the symptoms of menopause. Studies are also needed to determine the interactions between traditional hormone replacement therapy and the lignans.

If flaxseed is determined to be beneficial in the prevention of cancer, further research is also needed to determine whether the beneficial effects of flaxseed are due to its lignan content, α-linolenic acid content, or the presence of as yet undetermined chemoprotective agents. Additionally, the mechanism by which flaxseed exerts its effects on serum estrogen metabolism in this population remains to be elucidated.

Acknowledgments and Notes

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References


