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The impact of macronutrient intake on sex steroids during onset of puberty

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Abstract

INTRODUCTION: Increased fiber intake has been associated with decreased breast cancer risk, while increased animal protein intake with increased risk. The objective of this study was to examine the relationship of dietary fiber and protein intake to estrogen and sex hormone binding globulin (SHBG) concentrations at puberty onset.

METHODS: These analyses were conducted using the Cincinnati puberty cohort of the Breast Cancer and the Environment Research Program, with girls followed every six months from ages 6 and 7. The analyses included serum measurements at six-month intervals for estrogen and SHBG concentrations, from 18 months prior to breast stage 2 (onset of puberty). Dietary intake was

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documented via 24-hour dietary recalls every 3 months. Dietary factors of interest included total energy intake; total and animal protein; total, soluble and insoluble fiber; and lignan and flavanol intake.

RESULTS: This study included 260 participants who generated 871 serum specimens and 3000 days of diet intake. In longitudinal models, estradiol was associated inversely with insoluble fiber intake; estrone positively with animal protein intake; SHBG with greater insoluble fiber and lower total protein intake; and ratio of estrone to SHBG, a measure of bioavailable estrogen, positively with animal protein.

DISCUSSION: Greater protein intake, especially animal protein, led to greater estrogen concentrations and lower SHBG; greater animal protein and greater caloric intake led to increased bioavailable estrogen. This relationship may have served an evolutionary advantage in the past for greater fertility with adequate high-quality protein; in contemporary women, a modest decrease in animal protein may be beneficial in reducing breast cancer risk.

Keywords

puberty; sex steroids; macronutrients; dietary fiber

Introduction

Increased fiber intake has been associated with decreased breast cancer risk. Two meta-analyses of prospective observational studies noted a 5%−7% decreased risk of breast cancer with every 10 gm/day of increased fiber intake,[1,2] and a recent study noted a 14% decreased risk of breast cancer with every 10 gm/day increased fiber intake from adolescent diet patterns recalled at ages 33–52 years.[3] The underlying mechanism is uncertain; researchers have proposed that dietary fiber could impact breast cancer risk through decreasing absolute and bioavailable levels of sex steroids through suppression of gonadotropins, decreased circulating estrogen levels or increasing sex hormone–binding globulin (SHBG).[4–10] The majority of studies have investigated adult women. de Ridder[6] reported that adolescent girls with lower fiber intake had greater gonadotropin and estradiol levels. As noted, Farvid reported a decreased risk of breast cancer with greater intake of fruit during adolescence and young adult years; this effect was reduced if controlled for fiber consumption.[3] There is an inconsistent relationship of dietary fat with risk of breast cancer, although a meta-analysis of case-control studies noted a decreased risk of post-menopausal breast cancer with lower saturated fat intake.[11]

Conversely, intake of animal protein is associated with a higher risk for breast cancer. In the Women's Health Initiative, animal protein intake was positively associated, and vegetable protein negatively associated, with breast cancer incidence in post-menopausal women.[12] Similarly, the Shanghai Women's Health Study reported that vegetable protein, specifically soy protein, was associated with lower breast cancer risk in adult women, but they did not report associations with animal protein.[13] The Shanghai study also found that soy protein consumed during adolescence was associated with lower risk, but this result was based on adult participants recalling their diet as adolescents.

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Earlier pubertal maturation is associated with increased risk for hormonal cancers, such as breast cancer. Separate investigations by Rogers and Koo each reported that higher dietary fiber intake in childhood was associated with later menarche,[14,15] while Cheng reported that fiber intake had no impact on pubertal timing.[16] Meanwhile, a review of pubertal timing and nutritional factors reported that high animal protein intake in childhood has been shown to accelerate the onset of puberty by as much as 7 months.[17] Another study found that animal protein intake is inversely associated with age at time of the pubertal growth spurt and time of peak height velocity.[18]

The objectives of our study were to examine the relationship of peripubertal dietary factors to estrogen and SHBG concentrations during onset of puberty, in order to understand better the potential mechanism of diet on breast cancer risk.

Methods

The subjects of this study were part of the Cincinnati puberty study cohort of the Breast Cancer and the Environment Research Program. The participants were recruited between 6 and 7 years of age. Pubertal maturation was assessed every 6 months (until attainment of breast stage 5 and pubic hair 5, then annually) by a female physician or advanced practice nurse trained and certified in maturation assessment, as described previously.[19] Onset of puberty was defined as age at pubertal breast stage 2 (B2, presence of breast buds), using both inspection and palpation.

The race/ethnicity composition of this sample included 32.7% black participants, 4.2% Hispanic, 0.8% Asian, and 61.9% white non-Hispanic. Because of the small numbers of Hispanic and Asian participants, they were incorporated with black participants for the hormone analyses to form two groups, ‘white’ and ‘non-white’. There was a broad socio-economic representation in the Cincinnati cohort, with 11% of families reporting <$25,000 annual income, 18% reporting $25–50,000, and 31% reporting >$100,000.[20]

Serum specimens, drawn at every visit, were used for measurements of reproductive hormones from 18 months prior to and through 6 months following onset of B2. Estradiol and estrone were analyzed by high performance liquid chromatography with tandem mass spectrometry, and SHBG was analyzed by automated immunoradiometric assay (IRMA), as described.[21] Interassay precision, expressed as percent coefficient of variation for low, medium, and high control serum specimens was 4.9%, 4.6%, 4.7% for estrone; and 4.4%, 3.5%, 3.3% for estradiol. The estrogen to SHBG ratio, used in previous studies, was incorporated as a measure of bioavailable estrogen.[22]

Trained interviewers made unannounced phone calls to collect dietary intake via 24-hour diet recalls, using the Nutrition Data System for Research (NDSR; Nutrition Coordinating Center, Minneapolis, MN). Phone calls were made approximately every 3 months, starting at time of recruitment, to account for seasonal variability and included at least one weekend day annually to assess both weekday and weekend intake. We examined the daily mean total energy intake; total and animal protein intake; and total, soluble, and insoluble fiber intake as assessed from dietary data collected in a time window around age of B2. In the

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longitudinal analyses, data from diet recalls were included from 5.5 months prior to 0.5 months after a given study visit.

Independent and dependent variables were examined for normality. Spearman rank-based correlation coefficients were calculated for pubertal parameters, sex steroids, and dietary factors at onset of puberty (age at B2). A mixed-effects model was used to examine the relationship between log-transformed values of hormone measures with dietary intake, and included age at onset of puberty and chronologic age as covariates; subsequent longitudinal analyses added race or nutrient intake adjusted for total caloric intake. The mixed model incorporated variance components to account for repeated longitudinal measures of assays within an individual over study visits. All analyses were performed using SAS, version 9.3 (SAS Institute Inc).

The study was approved by the Institutional Review Board of Cincinnati Children's Hospital Medical Center, with written informed consent from parents, and assent from participants.

Results

This study included 260 participants who generated 871 serum specimens and 3000 dietary recall records. Values for dietary factors at pubertal onset (age B2) are listed in Table 1. Average age at onset of breast development (B2) was 8.4 years in black and 9.2 years in white participants (difference in age at onset, $p < 0.01$). Values for hormone factors at pubertal onset (age B2) are listed in Table 2. Greater intake of total fiber as well as insoluble fiber were correlated with later age of B2 (Spearman Rho = 0.165, p = .008; Rho = 0.173, p = .005, respectively) (Table 3). When stratified by race (white v non-white), the associations of later age of onset of puberty with increased total fiber and insoluble fiber were driven by white participants (Rho = 0.180, p = .015; Rho = 0.180, p = .015) (Supplemental table). When nutrient intake is adjusted for total caloric intake, the impact of total fiber on age of onset of puberty is slightly attenuated (Rho = 0.165, p = .008, unadjusted; Rho = 0.134, p = .03, adjusted).

When examining the association of hormone values at onset of puberty with dietary intake, estrone was associated with total protein (Rho= .152, p = 0.038) and animal protein intake (Rho= .144, p= 0.048). SHBG was negatively associated with energy (Rho= − .177, p = 0.02) and total protein, total fat, and animal protein (Rho= − .151, p = 0.05) (Table 3). The ratio of estrone to SHBG (a measure of bioavailable estrogen) was related to total caloric intake (Rho = 0.167, p = .04), total protein intake (Rho = 0.163, p = .049), and animal protein intake (Rho = 0.167, p = .04). When stratified by race (white v non-white), the relationship of SHBG to protein and animal protein intake was driven by non-white participants (Rho = −0.255, p = .05; Rho = −0.270, p = .04). All other associations between hormone measures and macronutrient intake, adjusted for caloric intake, are greater than p = .05 (except total calories on SHBG, and estrone to SHBG ratio, unchanged) (data not shown).

In longitudinal models examining dietary intake with sex steroid and SHBG levels, incorporating data from 18 months before until 6 months following pubertal onset, after

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adjusting for age at pubertal onset, estradiol levels were associated with time relative to puberty and lower intake of insoluble fiber (beta= −.0211, p= .029). Estrone levels were associated with time relative to puberty and greater intake of animal protein (beta= .00199, p=.049). SHBG levels were associated with time relative to puberty, greater intake of insoluble fiber (beta= .0111, p= .0082) and lower intake of total protein (beta= −.0016, p= .049) (Table 4). The estrone to SHBG ratio was greater with increased intake of animal protein (beta= .0035, p = .04) (Table 4). When adjusted for caloric intake, this relationship was stronger (beta= 0.718, p = .026). Additionally, the association of SHBG with fiber intake was strengthened after adjustment for caloric intake (beta= 17.08, p = .0013).

Discussion

In longitudinal models incorporating peripubertal data, we found that greater fiber intake was associated with lower estradiol concentrations; greater animal protein intake with greater estrone concentrations; and greater fiber as well as lower protein intake with increased SHBG concentrations. Greater estrone or estradiol concentrations, coupled with lower SHBG levels, would increase bioavailable estrogen,[23] and, consistent with these findings, greater fiber intake delayed onset of breast development. We noted a difference between the two race/ethnicity groups (white and non-white). White participants had a greater delay in age at onset of breast development with total and insoluble fiber intake, whereas non-white participants had a greater decrease in SHBG with increased protein intake. Future studies could examine differences in itemized intake, or perform nutrient density substitution analyses.

There are several mechanisms that could account for the relationship between amount of dietary fiber, levels of sex steroids, and timing of maturation. Greater fiber consumption is associated with an increased volume of feces; increased fecal excretion of estrogens results in lower serum estrogens in adult women,[4,5] possibly through increased bacterial β-glucuronidase activity.[4] Gonadotropins and estrogen levels are lower in adults with greater consumption of fiber,[6,9] potentially through inhibition of the hypothalamic-pituitary-ovarian axis due to higher fiber intake. Hughes[24] proposed that the fiber-fertility link would be a protective mechanism by delaying reproduction during periods associated with greater consumption of fiber, given that greater fiber intake would be associated with vegetable rather than animal protein and resultant lower-quality nutrition. Another mechanism that could impact sex steroid levels and maturation through greater fiber intake is raising SHBG levels,[7,8,10] thereby decreasing bioavailable sex steroids.

Several studies have noted the relationship between consumption of animal protein with earlier pubertal maturation.[6,14,18,25] There are several mechanisms proposed to explain this relationship, including Hughes[24] and higher-quality nutrition (vide supra). Another proposed mechanism is the stimulation of IGF1 secretion by animal protein, with greater IGF1 concentration leading to earlier maturation.[26] This effect may be mediated through the effect of milk on IGF1 secretion rather than other constituents of animal protein.[27,28] Additionally, dietary fats may impact DNA methylation patterns (reviewed by Donovan).[29]

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There are several potential limitations with this study. Our study population, although not representative of the US population, has racial and economic diversity. Although we tried to capture dietary intake through 24-hour diet recalls around time of pubertal onset, these recall records reflected typical patterns at these ages but may not have reflected intake at critical earlier time periods. It is possible that increased fiber intake would also be associated with increased phytoestrogen intake, and it may be difficult to isolate the effects of phytoestrogens from the effects of fiber.[30] Our group reported previously[31] that urinary concentrations of enterolactones, the major metabolite of dietary lignans, were modestly correlated with fiber intake (r = .13–.19), and others have noted even higher associations. Lampe[32] noted a significant correlation between dietary fiber and urinary lignan levels (r = .36, p = .0003). This correlation could serve as another mechanism for the impact of increased fiber on sex steroid level and action as well as decreased breast cancer risk. Of note, dietary recalls measure phytoestrogen consumption but not necessarily the biologic impact of phytoestrogen; to form the more biologically potent phytoestrogen, equol, dietary phytoestrogens need conversion by the gut microflora.[33] The mechanism of the protective effects of phytoestrogens is not fully understood. Potential mechanisms include competition with endogenous estrogens at the estrogen receptor, inhibition of aromatase, or antiproliferative and/or antioxidant properties (discussed in Mervish).[31] A future study could examine the impact of converted phytoestrogens through incorporating biomarkers of phytoestrogen intake. An important strength of our study is that it not only examined cross-sectional relationships at onset of puberty but also incorporated both longitudinal data collection and analytic approaches to help examine these complex temporal relationships.

The relationship of high fiber and lower available sex steroids may have served an evolutionary advantage given the relationship between greater fiber intake and lower-quality protein in traditional societies.[24] The analogous current findings of greater fiber intake protecting against greater caloric intake and contributing to later onset of puberty and lower levels of bioavailable estrogen may serve as an advantage in contemporary society. Additionally, increased dietary fiber may help prevent the metabolic syndrome.[34] A modest decrease in animal protein, as well as increase in fiber intake, may be beneficial in reducing breast cancer risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations:

SHBG	sex hormone binding globulin
B2	pubertal breast stage 2
IRMA	immunoradiometric assay
NDSR	Nutrition Data System for Research
IGF 1	insulin-like growth factor 1

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Implications & Contribution

This study examined peripubertal dietary factors associated with estrogen and SHBG. Greater protein intake, particularly animal protein, led to greater bioavailable estrogen. Total and insoluble fiber decreased estrogen, raised SHBG, and raised age of puberty; total caloric intake raised bioavailable estrogen. Modest peripubertal dietary changes could impact breast cancer risk.

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Table 1.

Values of dietary factors at onset of puberty (N=260)

Variable	Mean	Std Dev
Energy, kcal	1764	434.2
Total fat, gm	64.2	22.5
Total carb, gm	241.2	58.8
Total protein, gm	61.7	19.4
Animal protein, gm	40.8	16.9
Total fiber, gm	12.6	4.3
Soluble fiber, gm	4.1	1.3
Insoluble fiber, gm	8.4	3.2

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Table 2.

Values of hormone measures at onset of puberty

Variable	N	Mean	Std Dev
Estradiol, pmol/l	202	4.91	7.05
Estrone, pmol/l	192	5.27	3.33
SHBG, nmol/l	168	101.42	43.72
Log of Estradiol	202	1.05	0.94
Log of Estrone	192	1.51	0.53
Log of SHBG	168	4.52	0.46

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Table 3.

Correlation of hormone values to dietary factors at onset of puberty

Spearman Correlation Coefficients Probability value								
	Energy, calories	**Total fat, gm**	**Total carbs, gm**	**Total protein, gm**	**Animal protein, gm**	**Total fiber, gm**	**Soluble fiber, gm**	**Insoluble fiber, gm**
Estradiol	0.0233 0.74	0.038 0.60	0.0096 0.89	0.053 0.46	0.047 0.51	0.052 0.47	0.030 0.68	0.050 0.48
Estrone	0.111 0.13	0.108 0.14	0.082 0.26	0.152 0.038	0.144 0.048	0.128 0.08	0.096 0.19	0.122 0.09
SHBG	−0.177 0.02	−0.151 0.05	−0.112 0.15	−0.151 0.05	−0.151 0.05	0.008 0.92	−0.057 0.46	0.032 0.68
Estrone:SHBG	0.167 0.04	0.134 0.11	0.132 0.11	0.163 0.49	0.167 0.04	0.100 0.23	0.115 0.17	0.083 0.32
age of onset of puberty	0.060 0.33	0.054 0.39	0.056 0.37	0.093 0.14	0.035 0.57	0.165 0.008	0.112 0.07	0.173 0.005

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Table 4.

Longitudinal models for sex hormone concentrations

HORMONE	VARIABLE	ESTIMATE	STANDARD ERROR	P-VALUE
LOG [ESTRADIOL]	Months of age	0.0384	0.0033	<.0001
	Age onset puberty	0.2054	0.0369	<.0001
	Insoluble fiber	−0.0211	0.00968	0.029
LOG [ESTRONE]	Months of age	0.0304	0.00161	<.0001
	Age onset puberty	0.1573	0.0235	<.0001
	Animal protein	0.00199	0.00101	0.049
LOG [SHBG]	Months of age	−0.0177	0.00127	<.0001
	Insoluble fiber	0.0111	0.00417	0.0082
	Total protein	−0.0016	0.000812	0.049
LOG E1: SHBG	Months of age	0.04830	0.002459	<.0001
	Age onset puberty	0.1955	0.03879	<.0001
	Animal protein	0.003535	0.001739	0.043
	Total energy	0.000004806	0.000062	0.93

LOG= log transformation of outcome variable; SHBG= sex hormone binding globulin. Months of age= chronologic age at time of sample. Age onset of puberty= age at onset of breast development.