

ORIGINAL COMMUNICATION

Associations of menstrual pain with intakes of soy, fat and dietary fiber in Japanese women

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Objective: Intakes of soy, fat, and dietary fiber may be associated with the symptoms of dysmenorrhea through their biological effects on estrogens or prostaglandin production. The present study was to examine the relationships between intakes of soy, fat, and dietary fiber and the severity of menstrual pain.

Design: Cross-sectional study.

Setting: Three colleges and two nursing schools.

Subjects: A total of 276 Japanese women aged 19–24 y.

Methods: Intakes of nutrients and foods including soy products, isoflavones, fats and dietary fiber were estimated by a validated semiquantitative food frequency questionnaire. Severity of menstrual pain was assessed by the multidimensional scoring system reported by Andersch and Milson.

Results: Intake of dietary fiber was significantly inversely correlated with the menstrual pain scale ($r = -0.12$, $P = 0.04$) after controlling for age, smoking status, age at menarche and total energy intake. Neither soy nor fat intake was significantly correlated with menstrual pain after controlling for the covariates.

Conclusions: The cross-sectional difference in dietary fiber intake across the level of menstrual pain was small in magnitude but warrants further studies.

Sponsorship: None.

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Dysmenorrhea, or painful menses, is a common gynecological disorder among women in the reproductive age groups. Several theories regarding the causes of primary dysmenorrhea have been presented over the years (Deligeoroglou, 2000). Since elevated levels of prostaglandins have been found in the endometrium and menstrual fluid of dysmenorrheic women (Pickles *et al*, 1965), abundant evidence linking prostaglandins to dysmenorrhea has been accumulated. Increased prostaglandin production is now the most accepted theory to explain the etiology of primary dysmenorrhea

(Deligeoroglou, 2000). Prostaglandin F_{2a} (PGF_{2a}) and PGE₂ stimulate uterine contractions and cervical narrowing and increase vasopressin release, leading to ischemia and pain. Isoflavones, the phytoestrogens found mainly in soybeans, inhibit PGE₂ production (Yamaki *et al*, 2001) and cyclooxygenase activity (Liang *et al*, 1999). Isoflavones also can reduce the responsiveness to PGF_{2a} of rat uterine muscle (Picherit *et al*, 2000) and inhibit contractions of several types of smooth muscle (Steusloff *et al*, 1995). Dietary soy may have a beneficial effect on symptoms of dysmenorrhea by affecting the cyclooxygenase pathway. Estrogen has been suggested to modify PGE₂ production (Miyagi *et al*, 1993; Pavan *et al*, 2001). A high intake of soy isoflavones has been shown to decrease blood estrogen levels in premenopausal women (Kurzer, 2002). Dietary fat and fiber have been also indicated to alter estrogen status (Rose *et al*, 1997; Wu *et al*, 1999). These dietary components may be associated with symptoms of dysmenorrhea through hormonal influence.

Strom *et al* (2001) reported that infant exposure to soy formula vs cow milk formula was not associated with

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menstrual cramps in young adult women. To our knowledge, no other studies have described the association of soy intake and dysmenorrhea. Epidemiological data on dysmenorrhea and intakes of fat and dietary fiber are also scanty. In the present cross-sectional study, we examined the relationships between intakes of soy, fat, and dietary fiber and the severity of menstrual pain among premenopausal Japanese women.

Methods

The study subjects were female students at three colleges and two nursing schools between 1998 and 2001. A total of 362 women agreed to participate in the present study and responded to a self-administered questionnaire that asked about menstrual history, demographic characteristics, smoking and drinking habits, diet, exercise, and past medical and reproductive histories. The response rate was 90.0%. The present study was approved by the institutional review board.

The severity of menstrual pain was measured using the verbal multidimensional scoring system reported by Andersch and Milson (1982). This scoring system grades pain as none, mild, moderate, or severe and takes into account the effect of pain on daily activity, systemic symptoms, and analgesic requirements. Each woman was asked to report the date of the beginning of her last menses, the length of her usual menstrual cycle, and the number of days of bleeding. For woman who reported irregular menstrual cycles, we asked the range of the length of cycles and allotted the median as her cycle length.

Exercise was assessed by asking the average hours per week spent performing various kinds of activities during the past year. The details including its validity are described elsewhere (Suzuki *et al*, 1998).

Diet including soy, fat, and dietary fiber intakes was assessed by a semiquantitative food-frequency questionnaire. The women were asked to indicate the average frequency that they consumed 169 food items during the year prior to the study and the usual serving size of each item. We included nine food items for soy products (miso soup, tofu, deep-fried tofu, fried bean curd, dried bean curd, fermented soy beans, houba-miso, soymilk, and boiled soy beans). These nine items and some other dishes including soy products as ingredients were taken account for to obtain the estimates for total amount (g) of soy products and isoflavone intake. Isoflavone intake (mg/day) from soy products was estimated using isoflavone concentration in these soy foods (Wakai *et al*, 1999). The intakes of foods and nutrients were estimated from the frequency of ingestion and portion size using the Japanese Standard Tables of Food Composition, 4th and 5th editions, published by the Science and Technology Agency of Japan (2001). Fatty acid composition was evaluated using data published by Sasaki *et al* (1999). Detailed information on the questionnaire including its validity and reproducibility has been described elsewhere

(Shimizu, 1996; Shimizu *et al*, 1999; Nagata *et al*, 2001). For example, the Spearman correlation coefficients comparing estimates of soy product intake from this questionnaire with the estimates from 12 daily diet records kept over a year period was 0.68. The corresponding figures for total fat and dietary fiber were 0.52 and 0.60.

Because of incomplete or unreliable responses to the dietary questionnaire (criteria shown in the reference by Shimizu, 1996), we did not assess the diets of 44 women. The response to the menstrual pain scale was missing for six women. One woman did not report her age. Therefore, the ultimate response rate was 77.4%. We restricted study subjects to women aged 24y or less, because the frequency of secondary dysmenorrhea is likely to be higher in the elder women (Wentz, 1988). Therefore, 29 women were excluded. We further excluded women who had been taking steroid hormones during the previous 6 months ($n=9$) or who had a history of thyroid diseases ($n=1$) or other endocrine diseases ($n=2$). No one reported ovariectomy or use of oral contraceptives. The remaining 276 women aged 19–24y consisted of the present study. Age distribution of the study subjects were 81 (29.3%), 106 (38.4%), 70 (25.4%), 11 (3.9%), and 8 (2.9%) for 19, 20, 21, 22, 23 + y of age, respectively.

Spearman's correlation coefficients were used to examine the associations of severity of menstrual pain with study variables. Dietary values were log-transformed and adjusted for total energy using the method proposed by Willett (1990). Adjustment for potential confounders of the associations between dietary variables and the severity of menstrual pain was accomplished by regressing the menstrual pain scale and dietary values separately upon confounders. Spearman's correlation coefficients were then calculated. Several nondietary factors including weight, height, body mass index, smoking, exercise, marital status, age at menarche, menstrual cycle, days of bleeding, and number of births or pregnancies and intakes of macro- and micro-nutrients were examined as potential confounders. Age was always included in the model as a covariate to calculate the partial correlation coefficients. All statistical analyses were performed using SAS programs (Version 8, SAS Institute, Cary, NC, USA).

Results

The distribution of menstrual pain scores among the study subjects was 46 (16.7%), 111 (40.2%), 95 (34.4%), and 24 (8.7%) for grades 0–3 (none, mild, moderate, and severe), respectively. Characteristics of subjects according to menstrual pain scale are shown in Table 1. Group comparison for any variable except age at menarche did not reveal a significant association with menstrual pain scale.

Table 2 shows the correlation coefficients between selected nondietary variables and the menstrual pain scale. Age at menarche was significantly inversely correlated with the menstrual pain scale. Smoking status was positively asso-

Table 1 Characteristics of study subjects according to menstrual pain score

Variables	Score 0	Score 1	Score 2	Score 3	P-value ^a
Age (y)	20.6	20.5	20.7	20.8	0.52
BMI (kg/m ²)	20.0	20.7	20.0	20.3	0.12
Age at menarche (y)	12.4	12.2	12.0	12.0	0.04
Menstrual cycle length (days)	30.3	31.4	30.3	28.5	0.38
Days of bleeding (days)	5.5	5.7	5.9	6.1	0.25
Exercise (METs h/week)	25.9	20.1	24.0	39.0	0.23
Alcohol (ml/day)	11.0	5.4	4.4	7.3	0.09
Married (%)	0.4	0.4	0.0	0.0	0.56
Current smokers (%)	8.9	8.1	16.8	16.7	0.20
Ex-smokers (%)	0.0	5.4	7.4	4.2	0.28
<i>Daily dietary intakes</i>					
Total energy (kcal)	2056	2090	1934	1870	0.29
Soy products (g)	57.5	60.8	59.2	55.3	0.50
Isoflavones (mg)	25.6	27.7	21.6	24.4	0.20
Dietary fiber (g)	13.9	14.1	12.4	11.5	0.13
Total fat (g)	59.8	63.2	59.2	56.1	0.55
Saturated fat (g)	17.8	19.5	18.3	15.6	0.20
Monounsaturated fat (g)	21.0	22.0	20.6	20.2	0.68
Polyunsaturated fat (g)	13.8	14.1	13.0	14.1	0.61

METs = metabolic equivalents.

^aFor differences between the groups by ANOVA or Fisher's exact test.

Table 2 Correlations of selected nondietary variables with menstrual pain scale

Variables	Mean (s.d.)	Range	Spearman r	P-value
Age (y)	20.6 (1.0)	19–24	0.05	0.37
BMI (kg/m ²)	20.3 (2.2)	15.8–33.7	–0.04	0.51
Age at menarche (y)	12.1 (1.1)	9–16	–0.17	0.005
Menstrual cycle length (days)	30.6 (7.6)	10–103	–0.03	0.61
Days of bleeding (days)	5.8 (1.2)	3–11	0.10	0.09
Exercise (METs h /week)	24.0 (41.8)	0–323	–0.03	0.60
Alcohol (ml/day)	6.2 (17.6)	0–201	–0.03	0.57
Married ^a (%)	0.7		–0.09	0.15
Current smokers ^b (%)	12.0		0.11	0.06
Exsmokers ^b (%)	5.1		0.08	0.18

METs = metabolic equivalents.

^aCorrelation is based on dummy variable (coded as 0 if not married and 1 if married).

^bCorrelation is based on two dummy variables (coded as 0 and 0 if never-smokers, 1 and 0 if current smokers and 0 and 1 if exsmokers).

Table 3 Correlation coefficients between dietary intakes and menstrual pain scale

Variables	Mean (s.d.)	Range	r1 ^a	P-value	r2 ^b	P-value
Total energy (kcal)	2,012 (696)	704–6060	–0.11	0.08	–0.08	0.17
Soy product (g)	55.8 (54.3)	3.1–539	–0.03	0.67	–0.05	0.39
Soy isoflavone (mg)	25.0 (20.6)	1.6–155	–0.06	0.29	–0.08	0.22
Dietary fiber (g)	13.3 (6.5)	3.6–51	–0.13	0.03	–0.12	0.04
Fat (g)	60.6 (26.1)	15.2–208	0.07	0.30	0.08	0.19
Saturated fat (g)	18.5 (8.6)	4.1–56	0.08	0.23	0.11	0.08
Monounsaturated fat (g)	21.2 (9.2)	5.6–78	0.04	0.53	0.06	0.37
Polyunsaturated fat (g)	13.7 (6.1)	3.7–49	0.10	0.10	0.07	0.22

^aAdjusted for total energy except for total energy.

^bAdjusted for age, total energy, smoking status, and age at menarche.

ciated with the menstrual pain scale, but this association was of borderline significance ($P=0.06$).

Dietary fiber was significantly inversely correlated with the menstrual pain scale after controlling for age, smoking status and age at menarche ($r=-0.12$, $P=0.04$) (Table 3). There were no significant correlations between the menstrual pain scale and intakes of soy product or isoflavone as well as any type of fat. The positive association between saturated fat intake and the menstrual pain scale was of borderline significance ($P=0.08$). The additional adjustment for marital status and numbers of days of bleeding did not alter the results substantially (for example, the correlation coefficient between dietary fiber and the menstrual pain scale was -0.13 , $P=0.03$). Reanalysis restricting the subjects to those who reported a regular menstrual cycle with length of 25–35 days ($n=156$) did not attenuate the association between dietary fiber intake and the menstrual pain scale ($r=-0.14$, $P=0.10$).

Discussion

In spite of the relatively low intake levels of dietary fiber in our study subjects, we found a moderate but significant inverse association between dietary fiber intake and menstrual pain. It is well known that primary dysmenorrhea occurs only in ovulatory cycles (Friederich, 1983), indicating that adequate uterine exposure to estrogen and then to progesterone is necessary. Studies have suggested that fiber intake decreases blood estrogen levels in women (Kaneda *et al*, 1997; Rose *et al*, 1997). Although fat intake has been associated with increased estrogen levels (Wu *et al*, 1999), we failed to find a significant positive association between fat intake and menstrual pain. Neither soy product nor isoflavone intake was associated with menstrual pain. We expected that dietary soy would be inversely associated with menstrual pain through its effects on estrogens or on the cyclooxygenase pathway. However, such effects did not appear to be clinically relevant regarding dysmenorrhea. It is also possible that a limited range of soy intake as well as fat intake among the study subjects may have obscured a real association. Additional findings on smoking and age at

menarche in relation to menstrual pain were consistent with previous results from other studies (Klein & Litt, 1981; Sundell *et al*, 1990; Parazzini *et al*, 1994; Harlow & Park, 1996; Hornsby *et al*, 1998).

So far, to our knowledge, five studies have assessed the relationship between diet and menstrual pain (Deutch, 1995; Harel *et al*, 1996; Di Cintio *et al*, 1997; Balbi *et al*, 2000; Barnard *et al*, 2000). One of them (Harel *et al*, 1996) was based on dietary intervention using supplementation of n-3 fatty acids. In two other studies (Di Cintio *et al*, 1997; Balbi *et al*, 2000), dietary fiber as well as fat intake could not be estimated because the questionnaires used for measuring diet, which were apparently not validated, included a limited number of food items. Barnard *et al* (2000) reported that a low-fat vegetarian diet with a change of total fiber from 26.7 to 31.3 g was associated with an increase in sex hormone-binding globulin levels and with reductions in dysmenorrhea duration and intensity. Their findings are not contradictory with our results. In the remaining study reported by Deutch (1995), diet was measured by a 4-day diet record, and fat and dietary fiber intakes were not significantly associated with menstrual pain after controlling for covariates.

One of the limitations of our study is that we could not perform physical imaging and surgical examinations, such as uterasonography and laparoscopy, to rule out secondary cause of dysmenorrhea. The frequency of secondary dysmenorrhea is much lower than that of primary dysmenorrhea in this age group (Balbi *et al*, 2000). However, we cannot deny the possibility that our findings from a study with a small number of subjects were due to the inclusion of secondary dysmenorrhea.

The lack of endocrinologic measures of ovarian activity was another limitation of the present study. Women with anovulatory cycles do not experience menstrual pain. We could not determine whether each woman was ovulatory or not. Thus, there might be a concern that dietary fiber intake may be associated with anovulation rather than menstrual pain. However, when we reanalyzed data restricted to subjects with normal cycle lengths of 25–35 days, whom we thought to be ovulatory (Harlow & Ephross, 1995), the association between dietary fiber and the menstrual pain scale was not altered.

We employed a widely used scaling system to assess menstrual pain. However, pain is difficult to measure because it cannot be confirmed by any instrumental or clinical evaluation. Therefore, measurement error may have affected the results. However, it seems unlikely that women who had a lower intake of dietary fiber reported their pain more inaccurately or perceived more pain than those with a higher intake of dietary fiber. The food-frequency questionnaire, like all methods of dietary assessment, is subject to measurement error. Our questionnaire was designed to measure an individual's relative intakes of foods and nutrients rather than absolute values. The data presented for soy products may have been overestimated because soy product intake estimated from the questionnaire was 40%

higher than that estimated from the 12 daily diet records. The estimate for dietary fiber was 8% higher than that from the diet records. However, again, it is likely that this measurement error was unrelated to menstrual pain and led to an underestimation of the true associations.

Owing to the cross-sectional study design, we can only infer associations. Neuroendocrine functions of the body, mental attitude, and food choice may be mutually related. It is possible that the decreased intake of dietary fiber might be a consequence of menstrual pain. However, if this is true, intakes of other nutrients should also have been affected. None of the other measured nutrients or food groups was associated with the menstrual pain scale. Although the cross-sectional differences in dietary fiber intake across the level of menstrual pain was small in magnitude, more attention should be paid to the role of diet, including soy, fat, and dietary fiber, in the etiology of dysmenorrhea cases that are amenable to public health intervention.

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