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# Diet and Lifestyle Factors Associated with Premenstrual Symptoms in a Racially Diverse Community Sample: Study of Women's Health Across the Nation (SWAN)

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## ABSTRACT

***Aims*: We sought to determine if the frequency of reported physical or emotional premenstrual symptoms (PMSx) was associated with (1) dietary intake of phytoestrogens, fiber, fat, or calcium, (2) consumption of alcohol or caffeine, (3) active or passive smoke exposure or lack of physical exercise, and (4) race/ethnicity or socioeconomic status.**

***Methods*: A cross-sectional analysis was conducted of PMSx and demographic and lifestyle factors reported at baseline in the multiethnic sample of 3302 midlife women in the Study of Women's Health Across the Nation (SWAN). Stepwise multiple logistic regression analyses were performed for the overall sample and for each racial/ethnic group for each of five PMSx groupings.**

***Results*: Most dietary factors were not related to PMSx. Fat intake was negatively associated with craving and bloating (adjusted odds ratio [AOR] = 0.56, $p = 0.024$), and fiber intake was positively associated with breast pain (AOR = 1.39, $p = 0.037$). Alcohol intake was negatively associated with anxiety and mood changes (AOR = 0.63, $p = 0.045$) and headaches (AOR = 0.50, $p = 0.009$). Current smoking (AOR = 1.60, $p = 0.028$) and passive smoke exposure (AOR = 1.56, p = 0.050) were positively associated with cramps and back pain. Symptom reporting differed significantly by race/ethnicity. PMSx were also associated with comorbidities, early perimenopausal status, depressive symptoms, and symptom sensitivity.**

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*Conclusions*: **We found little evidence to support a role for diet in PMSx reporting. However, alcohol intake was positively associated with premenstrual anxiety and mood changes, and active and passive smoke exposure was associated with a number of PMSx. Ethnic differences in symptom reporting and associations of comorbidities, early perimenopausal status, depressive symptoms, and symptom sensitivity with reported PMSx were also observed.**

## INTRODUCTION

Premenstrual symptoms (PMSx) include mood, physical, and cognitive symptoms that begin in the luteal phase of the menstrual cycle and end with, or shortly after, the onset of menstruation.[1] The frequency, type, severity, and combination of symptoms that comprise PMSx vary.[2] The most frequently reported symptoms are irritability, depression, fatigue, water retention, weight gain, breast tenderness, headaches, abdominal cramps, and mood swings.[3] About 80% of women of reproductive age may experience premenstrual emotional and physical changes,[4] and about 50% of women seek medical care for them,[5–7] thus posing a large medical care burden.

The etiology of PMSx appears to be related to ovarian function, as suppression of ovarian hormone secretion markedly attenuates PMSx,[8] although differences in ovarian steroid hormones have not been consistently observed between symptomatic and asymptomatic women. Several biological, social, and behavioral factors have consistently been positively associated with PMSx, whereas demographic factors, education, employment, and marital status have shown inconsistent relationships.[9] Younger age, less education, and higher levels of perceived stress have been reported to be risk factors for premenstrual emotional symptoms.[2,10] Physical inactivity, smoking, caffeine intake, and a low intake of certain micronutrients (particularly calcium and vitamin D) are potentially modifiable risk factors.[10,11]

Several dietary agents and changes in behavioral and lifestyle factors have been recommended for the management of PMSx, including reducing or limiting intake of tobacco, chocolate, caffeine, and alcohol; having small, frequent meals high in complex carbohydrates; and taking vitamin and mineral supplements.[10] Calcium carbonate (1000–1200 mg daily) may improve affect and alleviate water retention, food cravings, and pain after three treatment cycles.[10,11] High-fiber diets reduce serum estrogen concentrations in premenopausal women by altering the enterohepatic circulation of estrogens, leading to elimination of estrogen in feces.[12]

Severe symptoms most commonly appear in the late second decade[9] and may be associated with a history of major depressive and anxiety disorders.[13–15] Women usually seek healthcare for PMSx in their mid- to late 30s.[9] Thus, most published work about the etiology of and risk factors for PMSx has focused on women under age 40, although, during the menopausal transition, PMSx have been associated with menopausal symptoms.[16–19] Thus, identification of modifiable factors related to PMSx during the menopause transition might allow for the development of prevention strategies and might help to improve the quality of life of women during their midlife years.

We undertook the present cross-sectional analysis of PMSx among a racially diverse cohort of midlife women to determine if frequency of reporting physical or emotional PMSx was associated with (1) dietary intake of phytoestrogens, fiber, fat, or calcium, (2) consumption of alcohol or caffeine, (3) exposure to active or passive smoking or lack of physical exercise, and (4) demographic characteristics, such as race/ethnicity or socioeconomic status.

## MATERIALS AND METHODS

### *Study population*

This was a cross-sectional study, using data on PMSx, health, reproductive, demographic, and lifestyle factors from the baseline questionnaires of the Study of Women's Health Across the Nation (SWAN), a longitudinal, multicenter, multiethnic study of midlife women. SWAN is following a cohort of women ($n$ = 3302 at baseline) from five ethnic groups at seven clinical sites located nationwide.[20] Each site recruited community-based cohorts of Caucasians and one non-Caucasian group: African Americans in Pittsburgh, Boston, Detroit, Chicago; Hispanics

(Puerto Rican, Dominican, Cuban, Central and South American) in Newark, New Jersey; Japanese in Los Angeles; and Chinese in the Oakland, California, area. Participants were eligible for inclusion in the cohort if they were aged 42–52 years and premenopausal or early perimenopausal, had not undergone a hysterectomy or bilateral oophorectomy, were not pregnant, and were not using menopausal hormone therapy (HT) or oral contraceptives at baseline. In addition, participants were required to be able to speak English, Spanish, Cantonese, or Japanese and to provide informed consent to participate and comply with the study protocol. All instruments and the study protocol were approved by the institutional review boards at each site, and signed, written informed consent was obtained from all participants.

*Data collection*

All SWAN participants completed a self-administered and interviewer-administered questionnaire at baseline.

*Outcomes*. The present analyses included data from the baseline visit (administered during 1996–1997) at which participants indicated yes or no in response to the following question for each of eight symptoms: During the last year, have you had any of the following during at least half of your menstrual periods or in the week before them? The eight symptoms included abdominal cramps/pain, breast pain/tenderness, weight gain/bloating, mood changes/suddenly sad, increase in appetite or cravings, anxious/jittery/nervous, back/joint/muscle pain, and severe headaches. If a participant answered yes to any one of the symptoms, she was also asked: Did this/these characteristic(s) usually (more than half of the time) disappear within 1–3 days after your period started? Answering yes to this question was used as the criterion for a symptom to be considered premenstrual in the present multivariate analyses; those who answered no or don't know were excluded from multivariate analyses.

*Independent variables*. Usual dietary intake was assessed using a modified 1995 Block Food Frequency Questionnaire (FFQ)[21,22] with 103 core food items, based on responses of African Americans and Caucasians in the Second National Health and Nutrition Examination Survey (NHANES).[21,23] Foods added for the Hispanic version were identified from the Hispanic HANES,[24] and Japanese and Chinese foods were added based on focus groups. Frequently consumed sources of dietary phytoestrogens were also included.[25] Dietary intake of nutrients was computed based on a food composition database from U.S. Department of Agriculture (USDA) data (*www.nal.usda.gov/fnic/foodcomp/Data/SR18/reports/sr18page.htm*)[26] linked to the food frequency data. Because genistein and daidzein are the largest components of phytoestrogen intake and their intakes were highly correlated, final analyses were performed only for genistein. Dietary intake of fat, fiber, and genistein were log transformed for inclusion in the statistical models. Quartiles of dietary intake of calcium, sodium, and calories and percent of calories from carbohydrates and of consumption of caffeine in milligram equivalents of cups of coffee were included as categorical variables. Servings per week of alcoholic beverages were analyzed as none, $\leq 1$, and $>1$ (one serving = 12 oz. beer, 5 oz. wine, or 1.5 oz hard liquor).

Active smoking status was assessed by standard questions.[27] Passive smoke exposure was assessed by the validated instrument of Coghlin et al.[28] Physical activity was measured by a composite score based on the Kaiser Permanente Activity Score,[29] a modification of the Baecke scale[30] assessing three domains: sports, leisure, and household activities. Race/ethnicity was self-identified as Caucasian, African American, Hispanic, Chinese, or Japanese and included both U.S.-born and foreign-born women.

*Covariates*. Age at baseline was analyzed as a continuous variable. Annual household income was evaluated using a three-level categorical variable based on tertiles of total income reported. A binary categorical variable was used for the proportion of women with a college education.

Menopausal status at baseline was defined using a dichotomous variable: (1) premenopausal (menstrual period in the prior 3 months with no change in regularity of periods) or (2) early perimenopausal (menstrual period in the prior 3 months with change in regularity of periods) without use of HT. Parity was self-reported and analyzed as a categorical variable.

Comorbidity consisted of reporting of 1 or more of 10 chronic health conditions (heart dis-

ease, arthritis, high blood pressure, diabetes, high cholesterol, stroke, anemia, migraines, angina, and osteoporosis) during the past year and was treated as a categorical variable. Weight and height were measured using a calibrated balance beam scale and stadiometer, respectively. Body mass index (BMI) (weight in kilograms/(height in meters)$^2$) was computed and analyzed as a four-level categorical variable: low (<18.5), normal (18.5–24.9), overweight (25–29.9), or obese (≥30). Use of five types of complementary and alternative medicines (CAM) (nutritional, herbal, psychological, physical, and folk) (yes/no) in the past year was ascertained by questionnaire and analyzed as a binary variable for each type and for any vs. no use.

Social support was assessed by a summed scale of how often four types of needed emotional and instrumental supports were available, with responses ranging from 0 = none of the time to 4 = all of the time.[31] A measure of the symptom sensitivity trait was measured at follow-up visit 01 using a summed score (degree of awareness of loud noise, hot or cold, hunger, pain, and things happening in one's body, with responses ranging from 1 = not at all true to 5 = extremely true)[32] and analyzed dichotomously as at/above or below 15, the median for the SWAN cohort. Depressive symptoms were assessed by the Center for Epidemiologic Studies Depression (CES-D)[33] scale (score ≥16 on a 20-item scale of the extent to which each item was experienced in the previous week).

### *Statistical analysis*

Women were excluded from analyses who were missing data for any independent or dependent variables ($n = 120$). Women were also excluded who did not complete the FFQ ($n = 12$) or reported consumption of an unreasonable number of foods (<4 or >17 per day), skipped more than 10 foods, or reported consumption of <500 or >5000 calories per day ($n = 157$). Some women met more than one exclusion criterion. After these exclusions, 3013 remained; 2758 remained in the multivariate analyses after also excluding those who answered no or don't know to whether the symptoms disappeared within 1–3 days after their menstrual period started.

Descriptive statistics were computed using bivariate analyses for each symptom grouping and each independent variable and each covariate. Categorical variables were analyzed using chi-square tests or Fisher's exact test for comparison of proportions, and *t* tests and ANOVA were used for comparisons involving continuous variables. Unadjusted odd ratios (ORs) were computed for each symptom by each independent variable.

All eight symptoms underwent factor analyses with Varimax rotations to determine appropriate groupings of symptoms so that a parsimonious set of outcome variables could be evaluated. To determine whether to retain a particular symptom in a symptom grouping, we used factor loadings of 0.40 or more. If items loaded on more than one factor, the item with the highest loading was retained. Factors were accepted with an Eigen value of ≥1.0. The five resulting PMSx groupings were (1) anxiety/jitter/nervous and mood changes, (2) abdominal cramps and back/joint/muscle pain, (3) increased appetite/craving and weight gain/bloating, (4) breast pain/tenderness, and (5) headaches. Associations of the independent variables with the total number of these five symptom groupings (>3 vs. ≤3) were also estimated.

To assess potential confounding variables, we calculated unadjusted ORs and stratum-specific ORs for each racial/ethnic group (compared with Caucasian), one variable at a time. To adjust simultaneously for confounding variables, multiple logistic regression models were developed for each PMSx grouping. Covariates that were associated (at $p < 0.1$) in unadjusted analyses were entered into stepwise multiple logistic regression analyses for the overall sample and for each racial/ethnic group for each PMSx grouping. Independent variables were forced into all multiple logistic regression models. Hosmer-Lemeshow goodness of fit tests were performed for multiple logistic regression models.

## RESULTS

Most demographic and health characteristics of the SWAN cohort differed significantly by race/ethnicity at baseline, with the exception of age and history of thyroid disease (Table (1). African American and Hispanic women were significantly less likely than the other racial/ethnic groups to have a college education, be married or in a committed relationship, be employed, have annual household incomes >$75,000, have a BMI <25, be nulliparous, have no comorbidities, and have a CES-D score <16. Caucasian and Japanese women had higher social support scores.

Significant racial/ethnic differences were also observed in lifestyle characteristics and dietary intake reported at baseline (Table (2). African American and Hispanic women were significantly less likely to report use of CAMs or supplements or to consume genistein. Caucasian and Japanese women had higher physical activity, reported the highest alcohol intake, and had the highest sodium intake. Chinese women were the least likely to be current smokers and to report passive exposure to cigarette smoke.

PMSx reporting at baseline also differed significantly by race/ethnicity (Table 3). Chinese and Japanese women reported the lowest number of total symptoms and each of the individual symptom groupings. Headache was the symptom least reported by all racial/ethnic groups (24.5%), and craving and bloating (80.4%) were reported by the most women.

### *Adjusted results*

The multiple logistic regression models are shown in Table 4. All variables above comorbidities were forced into the models because they pertained to our hypotheses, whereas those below race/ethnicity only remained in the models if they met the criteria for retention of covariates. In large measure, the results did not support hypothesized associations with dietary phytoestrogens, fiber, fat, or calcium or with consumption of alcohol or caffeine. The only exceptions were that (1) fat intake was modestly negatively associated with premenstrual craving and bloating (adjusted OR [AOR] = 0.56, $p = 0.024$), (2) fiber intake was modestly positively associated with premenstrual breast pain (AOR = 1.39, $p = 0.037$), (3) caffeine consumption was significantly positively associated with premenstrual anxiety and mood changes (but not in a monotonic dose-response), and (4) alcohol intake was modestly negatively associated with premenstrual anxiety and mood changes (AOR = 0.63, $p = 0.045$) and more strongly negatively associated with headaches (AOR = 0.50, $p = 0.009$). With the exception of caffeine, however, these were not in the expected direction.

More consistent support was observed for our third hypothesis, as current smoking (AOR = 1.60, $p = 0.028$) and passive smoke exposure (AOR = 1.56, $p = 0.050$) were associated with premenstrual cramps and back pain. Also, passive smoke exposure among former smokers was associated with premenstrual anxiety and mood changes (AOR = 1.54, $p = 0.031$), craving and bloating (AOR = 2.40, $p = 0.003$), and number of symptoms reported (AOR = 1.55, $p = 0.012$). Among never smokers, passive smoke exposure was significantly associated with premenstrual breast pain (AOR = 1.49, $p = 0.009$) and headache (AOR = 1.64, $p = 0.009$). Physical activity was only significantly positively associated with premenstrual craving and bloating (AOR = 1.13, $p = 0.01$).

A number of significant racial ethnic differences were observed. Most significant racial/ethnic differences were observed for Chinese or Japanese compared with Caucasian women for a number of PMSx. Anxiety and mood changes were reported significantly less by African American (AOR = 0.60, $p = 0.001$) and Japanese non-English speaking (AOR = 0.39, $p = 0.01$) women. Premenstrual cramps and back pain were reported by significantly fewer English-speaking Chinese women (AOR = 0.51, $p = 0.008$). Craving and bloating were reported significantly less by non-English-speaking Chinese and Japanese (AOR = 0.11, $p < 0.001$, and AOR = 0.20, $p < 0.001$, respectively) and significantly more by English-speaking Japanese women (AOR = 2.95, $p = 0.014$). More non-English-speaking Hispanic women reported premenstrual headaches (AOR = 1.95, $p = 0.043$).

Number of comorbidities was also significantly positively associated with all PMSx groupings, with the exception of breast pain, in a dose-response fashion. Being early perimenopausal compared with premenopausal was also significantly positively related to all PMSx groupings. Premenstrual anxiety and mood changes as well as cramps and back pain decreased with increasing age. Having depressive symptoms was positively related to all symptom groupings but most significantly to anxiety and mood changes and headache. Increased symptom sensitivity score was significantly associated with premenstrual anxiety and mood changes (AOR = 1.82, $p < 0.001$) and cramps and back pain (AOR = 1.51, $p = 0.001$) and modestly with breast pain (AOR = 1.26, $p = 0.023$). BMI >25 was significantly positively related to craving and bloating, whereas BMI <18.5 was significantly negatively related.

## DISCUSSION

Our findings provide little support for any relation of dietary intake, with the exception of caf-

TABLE 1. DEMOGRAPHIC AND HEALTH CHARACTERISTICS BY RACIAL/ETHNIC GROUP

| | *Total* (n = *3013*) | | *African American* (n = *836*) | | *Caucasian* (n = *1453*) | | *Chinese* (n = *221*) | | *Hispanic* (n = *267*) | | *Japanese* (n = *236*) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | p *value* |
| Demographics | | | | | | | | | | | | | |
| Mean (SD) age (years) at baseline | 45.8 | 2.69 | 45.8 | 2.67 | 45.8 | 2.7 | 46.0 | 2.59 | 45.8 | 2.75 | 46.14 | 2.7 | 0.302 |
| College or higher education (*n*, %) | 1302 | 43.7 | 275 | 33.3 | 770 | 53.4 | 114 | 51.6 | 27 | 10.5 | 116 | 49.2 | <0.0001 |
| Married/committed relationship (*n*, %) | 2325 | 77.2 | 595 | 71.2 | 1152 | 79.3 | 188 | 85.1 | 185 | 69.3 | 205 | 86.9 | <0.0001 |
| Employed (*n*, %) | 2421 | 80.5 | 659 | 79.0 | 1232 | 84.8 | 200 | 90.5 | 153 | 57.7 | 177 | 75.0 | <0.0001 |
| Annual household income (*n*, %) | | | | | | | | | | | | | <0.0001 |
| <$35,000 | 916 | 31.2 | 330 | 41.1 | 305 | 21.2 | 44 | 20.4 | 208 | 80.6 | 29 | 13.0 | |
| $35–75,000 | 1203 | 41.0 | 337 | 42.0 | 644 | 44.9 | 92 | 42.6 | 39 | 15.1 | 91 | 40.8 | |
| >$75,000 | 816 | 27.8 | 136 | 16.9 | 486 | 33.9 | 80 | 37.0 | 11 | 4.26 | 103 | 46.2 | |
| Interview language (*n*, %) | | | | | | | | | | | | | |
| English | 2627 | 87.2 | 836 | 100.0 | 1451 | 99.9 | 142 | 64.2 | 48 | 18.1 | 150 | 63.6 | |
| Other | 384 | 12.8 | 0 | 0.0 | 2 | 0.14 | 79 | 35.8 | 217 | 81.9 | 86 | 36.4 | |
| Health | | | | | | | | | | | | | |
| Menopausal status (N, %) | | | | | | | | | | | | | |
| Premenopausal | 1567 | 53.3 | 412 | 50 | 742 | 52.4 | 131 | 60.1 | 142 | 57.3 | 140 | 59.8 | 0.0084 |
| Early perimenopausal | 1373 | 46.7 | 412 | 50 | 674 | 47.6 | 87 | 39.9 | 106 | 42.7 | 94 | 40.2 | |
| BMI (kg/m$^2$) (*n*, %) | | | | | | | | | | | | | |
| <18.5 | 47 | 1.58 | 5 | 0.61 | 25 | 1.74 | 4 | 1.83 | 1 | 0.38 | 12 | 5.13 | <0.0001 |
| 18.5–24.9 | 1136 | 38.2 | 147 | 18.0 | 600 | 41.7 | 162 | 74.0 | 58 | 21.8 | 169 | 72.2 | |
| 25–29.9 | 806 | 27.1 | 245 | 30.1 | 372 | 25.8 | 43 | 19.6 | 104 | 39.1 | 42 | 18.0 | |
| >30 | 984 | 33.1 | 418 | 51.3 | 442 | 30.7 | 10 | 4.57 | 103 | 38.7 | 11 | 4.7 | |
| Number of pregnancies (*n*, %) | | | | | | | | | | | | | |
| None | 334 | 11.1 | 45 | 5.38 | 232 | 16.0 | 26 | 11.8 | 7 | 2.62 | 24 | 10.2 | <0.0001 |
| 1–3 | 1648 | 54.7 | 409 | 48.9 | 830 | 57.2 | 134 | 60.6 | 121 | 45.3 | 154 | 65.2 | |
| 4 or more | 1030 | 34.2 | 382 | 45.7 | 390 | 26.9 | 61 | 27.6 | 139 | 52.1 | 58 | 24.6 | |
| Number of comorbidities (*n*, %) | | | | | | | | | | | | | |
| None | 788 | 27.1 | 140 | 17.1 | 415 | 29.8 | 94 | 44.3 | 57 | 23.1 | 82 | 35.5 | <0.0001 |
| 1–2 | 1639 | 56.4 | 466 | 56.8 | 782 | 56.1 | 106 | 50.0 | 155 | 62.8 | 130 | 56.3 | |
| 3+ | 477 | 16.4 | 214 | 26.1 | 197 | 14.1 | 12 | 5.66 | 35 | 14.2 | 19 | 8.23 | |
| Having thyroid disease (*n*, %) | 300 | 10.1 | 78 | 9.44 | 159 | 11.1 | 24 | 11.2 | 25 | 9.4 | 14 | 5.96 | 0.1432 |
| CES-D ≥ 16 (*n*, %) | 739 | 24.5 | 228 | 27.3 | 328 | 22.6 | 28 | 12.7 | 117 | 43.8 | 38 | 16.1 | <0.0001 |
| Mean (SD) social support score | 12.34 | 3.33 | 12.2 | 3.39 | 12.7 | 3.02 | 11.9 | 3.15 | 10.87 | 4.69 | 13.07 | 2.61 | <0.0001 |
| Symptom sensitivity score ≥ 15 (*n*, %) | 1165 | 44.6 | 336 | 50.6 | 558 | 42.5 | 73 | 34.4 | 97 | 48.3 | 101 | 44.7 | 0.0002 |

Table 2. Lifestyle and Dietary Characteristics by Racial/Ethnic Group

| *Characteristic* | *Total* (n = *3013*) | | *African American* (n = *836*) | | *Caucasian* (n = *1453*) | | *Chinese* (n = *221*) | | *Hispanic* (n = *267*) | | *Japanese* (n = *236*) | | p *value* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | *Mean or* n | *SD or* % | |
| Any CAM use (*n*, %) | 1463 | 48.7 | 324 | 39.1 | 838 | 57.8 | 102 | 46.2 | 53 | 19.8 | 146 | 61.9 | <0.0001 |
| Mean total physical activity score (*n*) | 7.68 | 1.78 | 7.34 | 1.73 | 8.07 | 1.77 | 7.29 | 1.67 | 6.75 | 1.54 | 7.87 | 1.62 | <0.0001 |
| Current present and past smoking status (*n*, %) | | | | | | | | | | | | | |
| Never smoker and no passive exposure | 884 | 29.7 | 174 | 21.3 | 321 | 22.2 | 162 | 73.3 | 126 | 48.3 | 101 | 43.4 | <0.0001 |
| Never smoker, passive exposure < median | 497 | 16.7 | 136 | 16.7 | 276 | 19.1 | 32 | 14.5 | 20 | 7.66 | 33 | 14.2 | |
| Never smoker, passive exposure ≥ median | 329 | 11.0 | 128 | 15.7 | 142 | 9.81 | 11 | 4.98 | 30 | 11.5 | 18 | 7.73 | |
| Former smoker, no passive exposure | 339 | 11.4 | 75 | 9.2 | 202 | 14.0 | 10 | 4.52 | 19 | 7.28 | 33 | 14.2 | |
| Former smoker with any passive exposure | 420 | 14.1 | 105 | 12.9 | 272 | 18.8 | 2 | 0.9 | 22 | 8.43 | 19 | 8.15 | |
| Current smoker | 510 | 17.1 | 198 | 24.3 | 235 | 16.2 | 4 | 1.81 | 44 | 16.9 | 29 | 12.4 | |
| Dietary variables | | | | | | | | | | | | | |
| Supplement Use (*n*, %) | 1595 | 52.9 | 405 | 48.4 | 827 | 56.9 | 115 | 52.0 | 104 | 39.0 | 144 | 61.0 | <0.0001 |
| Mean (SD) Log transformed fat (g) | 4.11 | 0.45 | 4.2 | 0.49 | 4.12 | 0.44 | 3.97 | 0.38 | 4.03 | 0.4 | 4.01 | 0.39 | <0.0001 |
| Mean (SD) Log transformed dietary fiber (g) | 2.42 | 0.45 | 2.36 | 0.47 | 2.42 | 0.44 | 2.61 | 0.42 | 2.4 | 0.43 | 2.4 | 0.4 | <0.0001 |
| Mean (SD) Log transformed genistein ($\mu$g) | 2.83 | 3.49 | 1.28 | 2.22 | 2.3 | 3 | 7.86 | 1.77 | 1.31 | 2.57 | 8.59 | 1.55 | <0.0001 |
| Mean (SD) % Kcal from fat | 32.8 | 7.42 | 34.1 | 7.33 | 33.0 | 7.44 | 29.0 | 6.99 | 33.41 | 7.04 | 29.94 | 6.55 | <0.0001 |
| Mean (SD) % Kcal from carbohydrates | 50.9 | 8.31 | 51.2 | 8.62 | 50.2 | 8.02 | 54.4 | 7.92 | 49.61 | 8.9 | 52.93 | 7.37 | <0.0001 |
| Mean (SD) Dietary calcium (mg) | 751.5 | 414.6 | 674.6 | 400.6 | 827.0 | 438.4 | 688.7 | 373.7 | 742.58 | 343.0 | 628.67 | 326.4 | <0.0001 |
| Mean (SD) Diet and supplementary calcium (mg) | 933.4 | 538.9 | 780.9 | 476.1 | 1050.9 | 570.5 | 860.3 | 510.8 | 860.16 | 436.5 | 901.84 | 515.3 | <0.0001 |
| Mean (SD) Vitamin D (IU) | 137.2 | 105.4 | 115.8 | 91.6 | 151.7 | 115.9 | 126.6 | 99.0 | 150.9 | 81.3 | 118.21 | 97.4 | <0.0001 |
| Mean (SD) Total calories | 1836.9 | 676.9 | 1961.2 | 807.5 | 1816.2 | 622.8 | 1792.1 | 621.7 | 1639.6 | 548.2 | 1789.7 | 593.2 | <0.0001 |
| Mean (SD) Sodium (mg) | 2260.1 | 897.3 | 2193.2 | 964.0 | 2314.5 | 865.6 | 2219.1 | 922.0 | 2106.4 | 732.4 | 2375 | 953.5 | 2E-04 |
| Weekly alcohol intake (*n*, %) | | | | | | | | | | | | | |
| None | 1488 | 49.4 | 472 | 56.5 | 568 | 39.1 | 173 | 78.3 | 136 | 50.9 | 139 | 58.9 | |
| ≤1 serving[a] | 1356 | 45.0 | 334 | 39.95 | 771 | 53.1 | 44 | 19.9 | 124 | 46.4 | 83 | 35.2 | |
| >1 serving | 169 | 5.61 | 30 | 3.59 | 114 | 7.85 | 4 | 1.81 | 7 | 2.62 | 14 | 5.93 | |
| Mean (SD) Caffeine (mg) | 241.0 | 235.6 | 200.0 | 227.1 | 279.2 | 256.5 | 165.6 | 174.1 | 212.55 | 164.7 | 253.48 | 203.5 | |

[a]One serving = 12 oz. beer, 5 oz. wine, or 1.5 oz. hard liquor.

feine and alcohol consumption, or physical activity to PMSx reporting in midlife women. Of the six dietary exposures examined (phytoestrogens, calcium, fiber, fat, alcohol, and caffeine), we observed relations between intakes of fat, alcohol, and caffeine and specific PMSx reporting. Active and passive smoke exposure, having a greater number of comorbidities, being early perimenopausal (compared with being premenopausal), reporting depressive symptoms, and having the symptom sensitivity trait were each associated with PMSx.

### *Diet*

A relative excess of high-density lipoprotein cholesterol during the late luteal phase of the menstrual cycle and a higher fat intake throughout the cycle may increase premenstrual symptoms.[34] A low-fat intervention diet to prevent premenstrual symptoms caused reduced breast swelling, tenderness, and nodularity.[35] Our results indicated that greater fat intake was associated with less premenstrual craving and bloating, possibly reflecting ingestion of comfort food to self-treat PMSx. Food cravings may reflect changes in energy needs during the luteal phase of the cycle. Resting metabolic rate is higher in the luteal phase,[36] when 24-hour energy expenditure[37] and sleeping metabolic rate increase.[38] This change in energy expenditure has been reflected in an average increased intake of approximately 500 calories and a 50% increase in carbohydrate consumption during the luteal phase.[39,40]

Consumption of more than one serving of alcohol was associated in our study with significantly lower reporting of headache. Similar to the inverse association observed between fat intake and PMSx, the apparent protective effect of alcohol may represent self-medication. However, only 5.6% of our cohort consumed more than one serving of alcohol per week, and among Chinese and Japanese this represented only 18 participants. One study concluded that PMSx were strongly associated with alcohol consumed in the symptom-free, postmenstrual period, and drinking was unlikely to be a response to PMSx.[41]

Increasing caffeine intake in our study was associated with reporting of premenstrual anxiety and mood changes, but the association was not monotonic. Among Chinese and Japanese participants, the highest level of caffeine intake was associated with significantly increased reporting of premenstrual cramps and back pain. These results agree in general with one report of a dose-dependent relation between caffeine and PMSx in which ORs ranged from 1.3 for consumers of 1 cup of a caffeine-containing beverage per day and increased steadily to 7.0 for consumers of 8–10 cups per day.[42] However, another study reported no significant difference by total caffeine intake or by the individual caffeinated beverages consumed during either the postmenstrual or the premenstrual period.[41]

We postulated, but did not observe, that PMSx would be associated with dietary intakes of genistein (a phytoestrogen of the isoflavone class), fiber, or calcium. Phytoestrogens occur in plants and plant products (legumes, grains, nuts, and fiber-rich foods), are functionally and structurally similar to 17-beta estradiol (isoflavones), and may influence or interfere with estrogen receptors and reproductive processes, alter the pattern of synthesis and metabolism of endogenous estrogen, and modify hormone receptor values.[12] Endogenous estrogen levels and progesterone levels in the luteal phase are inversely related to soy intake.[43,44]

Dietary fiber enhances the excretion of endogenous estrogens and speeds up intestinal transit, which may lower serum estrogen concentrations,[45] which may prevent its metabolites from being absorbed back into the bloodstream, possibly leading to a reduction in PMSx. In our results, however, fiber intake was positively associated with breast pain, contrary to this hypothesis.

Calcium supplementation may reduce physical and emotional symptoms.[8,46] Estrogen regulates calcium metabolism, intestinal calcium absorption, and parathyroid gene expression and secretion, triggering fluctuations across the menstrual cycle.[8] The symptoms of hypocalcaemia are similar to PMSx (cramps, abnormal nerve impulses), and a recent analysis from the Nurses' Health Study suggested that lower calcium intake and vitamin D intake was associated with premenstrual syndrome (PMS),[47] although we found no association of calcium intake with any symptom reporting.

### *Smoking*

Active and passive smoke exposures were positively associated in SWAN participants with total number of reported PMSx. In prior studies, current smokers, compared with nonsmokers, were at increased risk of cramps or pain requiring medication or time off work, after adjusting

Table 3. Symptom Outcomes by Racial/Ethnic Group

| | *Total* (n = *3012*[a]) | | *African American* (n = *836*) | | *Caucasian* (n = *1453*) | | *Chinese* (n = *221*) | | *Hispanic* (n = *267*) | | *Japanese* (n = *236*) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | % | n | % | n | % | n | % | n | % | n | % | p *value* |
| Number of total symptom groupings | | | | | | | | | | | | | |
| 0–1 | 440 | 14.61 | 112 | 13.40 | 191 | 13.16 | 62 | 28.05 | 20 | 7.49 | 55 | 23.31 | <0.0001 |
| 2 | 374 | 12.42 | 96 | 11.48 | 163 | 11.23 | 41 | 18.55 | 28 | 10.49 | 46 | 19.49 | |
| 3+ | 2197 | 72.97 | 628 | 75.12 | 1097 | 75.60 | 118 | 53.39 | 219 | 82.02 | 135 | 57.20 | |
| Anxiety and mood changes | | | | | | | | | | | | | |
| No | 736 | 24.44 | 214 | 25.60 | 300 | 20.68 | 81 | 36.65 | 47 | 17.60 | 94 | 39.83 | <0.0001 |
| Yes/no, DK[b] | 124 | 4.12 | 36 | 4.31 | 67 | 4.62 | 4 | 1.81 | 10 | 3.75 | 7 | 2.97 | |
| Yes/yes[c] | 2151 | 71.44 | 586 | 70.10 | 1084 | 74.71 | 136 | 61.54 | 210 | 78.65 | 135 | 57.20 | |
| Cramps and back pain | | | | | | | | | | | | | |
| No | 691 | 22.94 | 147 | 17.58 | 323 | 22.25 | 99 | 44.80 | 36 | 13.48 | 86 | 36.44 | <0.0001 |
| Yes/no, DK[b] | 133 | 4.42 | 41 | 4.90 | 68 | 4.68 | 4 | 1.81 | 12 | 4.49 | 8 | 3.39 | |
| Yes/yes[c] | 2188 | 72.64 | 648 | 77.51 | 1061 | 73.07 | 118 | 53.39 | 219 | 82.02 | 142 | 60.17 | |
| Craving and bloating | | | | | | | | | | | | | |
| No | 453 | 15.04 | 97 | 11.60 | 163 | 11.23 | 90 | 40.72 | 36 | 13.48 | 67 | 28.39 | <0.0001 |
| Yes/no, DK[b] | 137 | 4.55 | 43 | 5.14 | 74 | 5.10 | 3 | 1.36 | 11 | 4.12 | 6 | 2.54 | |
| Yes/yes[c] | 2422 | 80.41 | 696 | 83.25 | 1215 | 83.68 | 128 | 57.92 | 220 | 82.40 | 163 | 69.07 | |
| Breast pain | | | | | | | | | | | | | |
| No | 947 | 31.44 | 289 | 34.57 | 419 | 28.86 | 96 | 43.44 | 53 | 19.85 | 90 | 38.14 | <0.0001 |
| Yes/no, DK[b] | 115 | 3.82 | 28 | 3.35 | 64 | 4.41 | 3 | 1.36 | 10 | 3.75 | 10 | 4.24 | |
| Yes/yes[c] | 1950 | 64.74 | 519 | 62.08 | 969 | 66.74 | 122 | 55.20 | 204 | 76.40 | 136 | 57.63 | |
| Headache | | | | | | | | | | | | | |
| No | 2204 | 73.17 | 617 | 73.80 | 1073 | 73.90 | 178 | 80.54 | 154 | 57.68 | 182 | 77.12 | <0.0001 |
| Yes/no, DK[b] | 69 | 2.29 | 20 | 2.39 | 32 | 2.20 | 2 | 0.90 | 10 | 3.75 | 5 | 2.12 | |
| Yes/yes[c] | 739 | 24.54 | 199 | 23.80 | 347 | 23.90 | 41 | 18.55 | 103 | 38.58 | 49 | 20.76 | |

[a]One participant was missing information on whether symptoms disappeared within 1–3 days of start of period.
[b]Yes/no, DK, symptom reported but answered "no" or "don't know" to symptom disappeared within 1–3 days of start of period.
[c]Yes/yes, symptom reported and answered "yes" to symptom disappeared within 1–3 days of start of period.

TABLE 4. ADJUSTED OR FROM MULTIPLE LOGISTIC REGRESSIONS FOR FACTORS ASSOCIATED WITH EACH PREMENOPAUSAL SYMPTOM GROUP, *N* = 2758[a]

| | *Anxiety and mood change* | | *Cramps and back pain* | | *Craving and bloating* | | *Breast pain* | | *Headache* | | *>3 vs. ≤3 symptom groups reported* | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* |
| % of calories from fat | | | | | | | | | | | | |
| >38 | 1.36 | 0.27 | 0.88 | 0.57 | 1.18 | 0.58 | 1.29 | 0.23 | 1.02 | 0.93 | 1.13 | 0.57 |
| 34–38 | 1.10 | 0.67 | 0.92 | 0.65 | 0.98 | 0.94 | 1.03 | 0.86 | 0.86 | 0.41 | 0.94 | 0.71 |
| 29–33 | 1.05 | 0.80 | 1.10 | 0.59 | 0.85 | 0.47 | 1.09 | 0.57 | 0.88 | 0.45 | 0.97 | 0.84 |
| ≤28 | 1.0 | | 1.0 | | 1.0 | | 1.0 | | 1.0 | | | |
| Log transformed fat intake (g) | 0.71 | 0.32 | 1.11 | 0.61 | 0.56 | 0.024 | 0.91 | 0.68 | 0.92 | 0.66 | 0.98 | 0.92 |
| Log transformed fiber intake (g) | 0.97 | 0.85 | 0.88 | 0.49 | 1.14 | 0.57 | 1.39 | 0.037 | 0.98 | 0.89 | 1.08 | 0.63 |
| Log transformed genistein intake ($\mu$g) | 0.97 | 0.20 | 0.97 | 0.15 | 0.96 | 0.16 | 0.98 | 0.43 | 0.97 | 0.23 | 0.96 | 0.093 |
| Total calcium intake (mg) | | | | | | | | | | | | |
| ≥1121 | 1.05 | 0.79 | 0.95 | 0.78 | 0.99 | 0.97 | 0.90 | 0.52 | 1.38 | 0.06 | 0.83 | 0.27 |
| 789–1121 | 1.04 | 0.80 | 0.95 | 0.79 | 1.06 | 0.82 | 0.90 | 0.52 | 1.25 | 0.19 | 1.05 | 0.76 |
| 531–789 | 1.25 | 0.20 | 1.07 | 0.72 | 0.98 | 0.92 | 1.05 | 0.76 | 1.21 | 0.23 | 0.97 | 0.84 |
| <531 | 1.0 | | | | | | | | | | | |
| Caffeine intake (mg) | | | | | | | | | | | | |
| ≥321 | 1.13 | 0.44 | 1.10 | 0.56 | 1.31 | 0.21 | 0.96 | 0.76 | 1.18 | 0.29 | 1.15 | 0.36 |
| 241–320 | 1.76 | 0.004 | 1.20 | 0.32 | 1.15 | 0.57 | 1.26 | 0.16 | 1.07 | 0.71 | 1.32 | 0.10 |
| 161–240 | 1.58 | 0.016 | 1.18 | 0.37 | 1.29 | 0.29 | 1.10 | 0.56 | 1.15 | 0.42 | 1.20 | 0.29 |
| 81–160 | 1.32 | 0.11 | 1.04 | 0.84 | 1.12 | 0.61 | 1.13 | 0.44 | 1.03 | 0.86 | 1.30 | 0.10 |
| ≤80 | 1.0 | | | | | | | | | | | |
| Weekly alcohol intake | | | | | | | | | | | | |
| >1 serving[b] | 0.63 | 0.045 | 0.76 | 0.25 | 1.24 | 0.50 | 1.24 | 0.33 | 0.500 | 0.009 | 0.84 | 0.41 |
| ≤1 serving | 1.06 | 0.65 | 1.17 | 0.22 | 1.18 | 0.30 | 0.98 | 0.86 | 0.85 | 0.16 | 0.98 | 0.90 |
| None | 1.0 | | 1.0 | | 1.0 | | 1.0 | | 1.0 | | | |
| Physical activity | 1.04 | 0.32 | 1.01 | 0.70 | 1.13 | 0.01 | 1.03 | 0.36 | 1.01 | 0.84 | 1.04 | 0.25 |
| Smoking | | | | | | | | | | | | |
| Current smoker | 0.81 | 0.26 | 1.60 | 0.028 | 1.39 | 0.21 | 1.28 | 0.15 | 1.22 | 0.27 | 1.339 | 0.108 |
| Former smoker, any passive exposure | 1.54 | 0.031 | 1.22 | 0.28 | 2.40 | 0.003 | 1.21 | 0.24 | 1.37 | 0.072 | 1.554 | 0.012 |
| Former smoker, no passive exposure | 1.11 | 0.60 | 1.25 | 0.25 | 1.10 | 0.71 | 1.34 | 0.092 | 1.28 | 0.19 | 1.330 | 0.110 |
| Never smoker, passive exposure ≥ median | 1.31 | 0.21 | 1.56 | 0.050 | 1.12 | 0.67 | 1.26 | 0.21 | 1.64 | 0.009 | 1.357 | 0.130 |
| Never smoker, passive exposure < median | 0.90 | 0.51 | 1.12 | 0.49 | 1.14 | 0.56 | 1.49 | 0.009 | 1.10 | 0.56 | 1.281 | 0.114 |
| Never smoker and no passive exposure | 1.0 | | 1.0 | | 1.0 | | 1.0 | | 1.0 | | | |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ethnicity and language | | | | | | | | | | | | |
| African American | 0.60 | 0.001 | 1.35 | 0.068 | 0.80 | 0.28 | 0.87 | 0.30 | 0.97 | 0.85 | 0.786 | 0.106 |
| Chinese, English speaking | 1.23 | 0.49 | 0.51 | 0.008 | 1.18 | 0.64 | 0.62 | 0.05 | 1.02 | 0.95 | 0.774 | 0.395 |
| Chinese, no English speaking | 0.52 | 0.058 | 0.79 | 0.52 | 0.11 | <0.0001 | 0.83 | 0.58 | 1.23 | 0.62 | 0.331 | 0.007 |
| Hispanic, English speaking | 0.45 | 0.15 | 4.83 | 0.13 | 1.56 | 0.57 | 1.68 | 0.36 | 2.18 | 0.15 | 0.508 | 0.268 |
| Hispanic, no English speaking | 1.17 | 0.62 | 1.26 | 0.42 | 0.70 | 0.27 | 1.29 | 0.28 | 1.95 | 0.043 | 0.712 | 0.358 |
| Japanese, English speaking | 0.65 | 0.12 | 0.74 | 0.26 | 2.95 | 0.014 | 0.94 | 0.80 | 1.42 | 0.27 | 0.760 | 0.368 |
| Japanese, no English speaking | 0.39 | 0.01 | 1.38 | 0.42 | 0.20 | <0.0001 | 0.85 | 0.65 | 1.06 | 0.90 | 0.129 | <0.0001 |
| Caucasian | 1.0 | | 1.0 | | 1.0 | | 1.0 | | 1.0 | | | |
| Number of comorbidities | | | | | | | | | | | | |
| 3+ | 1.75 | 0.004 | 1.83 | 0.003 | 2.52 | 0.001 | | | 2.62 | <0.0001 | | |
| 1–2 | 1.41 | 0.008 | 1.26 | 0.070 | 1.44 | 0.024 | | | 1.45 | 0.005 | | |
| None | 1.0 | | 1.0 | | 1.0 | | | | 1.0 | | | |
| Menopausal status (Early perimenopausal vs. premenopausal) | 1.54 | 0.000 | 1.31 | 0.019 | 0.86 | <0.0001 | 1.28 | 0.014 | 1.47 | 0.000 | 1.388 | 0.0002 |
| Study site | | | | | | | | | | | | |
| Michigan | | | | | | | | | 0.55 | 0.002 | 0.898 | 0.576 |
| Boston | | | | | | | | | 0.49 | 0.001 | 0.819 | 0.300 |
| Chicago | | | | | | | | | 0.73 | 0.094 | 1.267 | 0.240 |
| Davis | | | | | | | | | 0.80 | 0.35 | 0.739 | 0.206 |
| LA | | | | | | | | | 0.74 | 0.22 | 1.310 | 0.261 |
| NJ | | | | | | | | | 0.83 | 0.53 | 1.325 | 0.378 |
| Pittsburgh | | | | | | | | | 1.0 | | | |
| Age (years) at baseline | 0.92 | <0.0001 | 0.93 | 0.001 | | | | | 0.97 | 0.096 | 0.907 | <0.0001 |
| College and above education (yes vs. no) | | | 0.74 | 0.018 | | | | | | | 0.802 | 0.059 |
| Being in committed relationship (yes vs. no) | | | 1.28 | 0.085 | | | 1.30 | 0.030 | | | | |
| CES-D score ≥ 16 | 2.93 | <0.0001 | 1.40 | 0.032 | 1.57 | 0.025 | 1.39 | 0.010 | 1.76 | <0.0001 | 1.752 | <0.0001 |
| BMI ($kg/m^2$) | | | | | | | | | | | | |
| <18.5 | | | | | 0.19 | 0.000 | 0.39 | 0.019 | | | 0.232 | 0.002 |
| 18.5–24.9 | | | | | 1.0 | | 1.0 | | | | 1.0 | |
| 25–29.9 | | | | | 1.85 | 0.002 | 0.90 | 0.41 | | | 0.978 | 0.867 |
| 30+ | | | | | 1.86 | 0.002 | 0.79 | 0.079 | | | 1.160 | 0.289 |
| Number of pregnancies | | | | | | | | | | | | |
| None | | | 1.67 | 0.010 | | | | | | | | |
| 1–3 | | | 1.0 | | | | | | | | | |
| 4+ | | | 1.06 | 0.66 | | | | | | | | |

(*continued*)

Table 4. Adjusted OR from Multiple Logistic Regressions for Factors Associated with Each Premenopausal Symptom Group, $N = 2758$[a] (Cont'd)

| | *Anxiety and mood change* | | *Cramps and back pain* | | *Craving and bloating* | | *Breast pain* | | *Headache* | | *>3 vs. ≤3 symptom groups reported* | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* | *OR* | p *value* |
| Social support score | | | | | | | | | | | | |
| <11 | 1.0 | | | | | | | | | | | |
| 11–12 | 0.90 | 0.60 | | | | | | | | | 0.745 | 0.050 |
| 13–14 | 0.83 | 0.32 | | | | | | | | | 0.818 | 0.211 |
| ≥15 | 0.54 | 0.000 | | | | | | | | | 1.079 | 0.642 |
| Use of CAM (yes vs. no) | | | | | 1.30 | 0.094 | 1.20 | 0.08 | 1.22 | 0.072 | 1.232 | 0.061 |
| Any supplement use (yes vs. no) | | | 1.26 | 0.065 | | | | | | | | |
| Sodium intake (mg) | | | | | | | | | | | | |
| <1610 | | | | | | | 1.0 | | | | | |
| 1610–2126 | | | | | | | 0.68 | 0.011 | | | | |
| 2127–2719 | | | | | | | 0.84 | 0.34 | | | | |
| ≥2720 | | | | | | | 0.69 | 0.099 | | | | |
| Total calorie intake | | | | | | | | | | | | |
| ≤1356 | 1.0 | | | | | | | | | | | |
| 1357–1722 | 0.93 | 0.70 | | | | | | | | | | |
| 1723–2192 | 1.52 | 0.10 | | | | | | | | | | |
| >2192 | 1.32 | 0.44 | | | | | | | | | | |
| Symptom sensitivity score (15 + vs. <15) | 1.82 | <0.0001 | 1.51 | 0.001 | | | 1.26 | 0.023 | | | 1.534 | <0.0001 |

[a]Because of the large number of variables, those with $p \leq 0.01$ are highlighted. Also, all variables above comorbidities were forced into the models to address the hypotheses; those below that variable only remained in the models if they met the criteria for retention of covariates.

[b]One serving = 12 oz beer, 5 oz wine, or 1.5 oz hard liquor.

for age, race, and pay grade.[48] Toxic effects of smoking on the ovary, enhancement of estrogen metabolism, and effects on central nervous system hormone release have been proposed as mechanisms of this relationship, but none has been fully investigated.[49] Current cigarette smokers are about four times more likely to have a diagnosis of premenstrual dysphoric disorder (PMDD),[50] possibly related to the strong correlation between cigarette smoking and lifetime prevalence of depression.

*Physical activity*

With the exception of the slight but significant positive association with premenstrual craving and bloating, we did not observe any significant associations between physical activity and PMSx. This is consistent with at least two other relatively large, community-based studies[7,49] but differs from several smaller studies of more select samples that found fewer PMSx in women who exercised.[51–53] Still others have observed a direct association between physical activity and PMSx.[54,55] Women who exercise are likely to be a heterogeneous group, with some being active for non-PMSx-related reasons while others participate in exercise to ameliorate PMSx.[7,56] Exercise produces endorphins that relieve pain and improve mood[52,57] and alleviates symptoms of major depressive disorder, but no definitive evidence shows that it improves PMDD.[58]

*Depression*

A high level of depressive symptoms (CES-D score $\geq$16) was consistently associated with all PMSx groups in our community-based sample, similar to results from clinic-based studies. In perimenopausal women attending a menopausal mood clinic, who were categorized as depressed if their Beck Depression Inventory score was $\geq$10, symptoms consistent with PMS were reported by 26% of depressed but only 9% of nondepressed women.[59] The authors speculated that the menstrual cycle may modulate the appearance of PMSx in some women with perimenopausal depression, with exacerbations occurring during the premenstrual phase of the cycle.

*Sociodemographic and other factors*

Increasing age was significantly associated with decreased reporting of premenstrual anxiety and mood changes and of cramps and back pain. A similar relation of age to PMDD has been observed.[50] PMSx reporting also differed significantly by race/ethnicity, with Chinese and Japanese women reporting the lowest number of total symptoms and each of the individual symptom groupings. For some PMSx, the relative odds were significantly decreased in non-English speaking women, whereas for other symptoms, they were somewhat increased. Birthplace outside the United States was only related to increased reporting of headaches (data not shown). The ethnic differences in symptom reporting are unlikely to be related to acculturation but may be related to cultural differences. A recent publication[60] suggested, for example, that in traditional Japanese society, personal well-being is subordinated to the well-being of the group, so that reporting of symptoms may be suppressed to maintain social harmony.

Because PMSx consist of a number of symptoms, women may use a variety of CAM therapies to treat them. Nationally, women with PMSx use a variety of nutritional, physical, and spiritual or psychological therapies.[61] Reviews of existing clinical trials have concluded that none of those for herbal medicine, nutritional supplements, dietary interventions, exercise, homeopathy, progressive muscle relaxation, massage, reflexology, chiropractic, or biofeedback demonstrated any effectiveness for reducing PMS[62] or PMSx.[63] However, the latter review concluded that calcium, magnesium, vitamin $B_6$ and chastetree berry (*Vitex agnus-castus*), reducing dietary fat, and increasing exercise may have positive effects on PMS.

*Strengths and limitations*

The present study had several significant strengths. First, the sample comprised a large, racially/ethnically diverse, community-based sample of women. Thus, the results are likely to have fairly good generalizability. Second, the assessment of diet and other risk factors used standardized, validated instruments and was made independently of symptom reporting. Third, we simultaneously statistically controlled for a number of potential risk factors so that we could assess the independent effects of each while controlling for the effects of others.

The study also had some limitations. First, multiple statistical comparisons were made, so that

some of the observed associations may have occurred by chance or represent markers for other, uncontrolled factors. Thus, caution must be used for interpreting marginally significant results. Second, the study was cross-sectional; thus, the temporal relation of potential risk factors to symptom reporting cannot be adequately assessed, and some associations may have resulted from some factors being used for self-medication for or be a consequence of symptoms rather than being causally related (e.g., alcohol, caffeine, depressive symptoms). Third, all of the factors examined were recalled by participants and, thus, may lack accuracy of recall. Fourth, the factors were not examined in relation to the timing of their occurrence (e.g., diet during certain phases of the menstrual cycle), which may have diluted our ability to see effects. Fifth, due to time limitations for administration of the study instruments, we were not able to include an exhaustive list of symptoms so that some, such as irritability, were not included. Further, the outcomes are not rare, and the ORs may overestimate risk. Finally, we examined PMSx, so our findings are unlikely to apply to PMS.

In conclusion, we found little evidence to support a significant role for dietary intake in affecting premenstrual symptom reporting. We did find significant positive associations of caffeine intake with premenstrual anxiety and mood changes, as well as ethnic differences in symptom reporting and significant associations of comorbidities, early perimenopausal status, depressive symptoms, and symptom sensitivity with premenstrual symptom reporting. These findings can provide some guidance to women and their healthcare providers in identifying subgroups of women who may have increased susceptibility to certain premenstrual symptoms, as well as provide further reasons for limiting caffeine intake and exposure to active and passive cigarette smoke.

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