

Effect of dairy products intake in women with premenstrual syndrome: a randomized controlled trial

Merve Yurt¹, Seyit Mehmet Mercanhgil^{1,2}, Seray Kabaran¹

¹Department of Nutrition and Dietetics, Faculty of Health Sciences, Eastern Mediterranean University, Famagusta, North Cyprus via Mersin 10, Turkey - E-mail: merve.yurt@emu.edu.tr; ²Cyprus International University, Faculty of Health Sciences, Nutrition and Dietetics Department, Nicosia, Cyprus

Summary. *Aim:* This study was planned and conducted to investigate the effects of adequate dairy products, at least 3 portions, and calcium intake on Premenstrual Syndrome (PMS) symptoms in women with PMS who have inadequate calcium intake. *Methods:* Thirty-one women, aged between 20-28 years who were diagnosed with PMS had participated. All women had inadequate calcium intake and regular menstrual cycles. These participants were randomly allocated into two groups, an intervention (n=16) group and a control (n=15) group. It was ensured that the intervention group consumed foods containing at least 1000 mg calcium for two months. Turkish *kasseri* cheese (50 g) which is made from cow's milk was provided to the intervention group and they were informed to consume at least 400 ml of milk and 150 g of yogurt every day for two months. No specific diet was recommended to the control group. The Premenstrual Syndrome Scale (PMSS) and the Short Form of Quality of Life Scale were administered at the baseline and after the study. Independent t-test and Mann-Whitney-U test were used for group comparisons according to data normality. **Results:** No significant difference in PMSS scores, physical functions and mental health scores on the SF-36 quality of life scale and daily calcium intake between the intervention (500.9±114.6 mg) and the control groups (511.3±149.2 mg) at baseline assessment (p>0.05). The intervention group's total PMSS post-intervention (104.8±21.5) score was found to be significantly lower than their baseline scores (151.9±15.4) (p<0.05). The intervention group's physical functions (47.9±8.0) and mental health (48.6±6.9) post-intervention scores on the SF-36 quality of life scale were found to be significantly higher than their baseline scores (41.2±10.7 and 45.0±10.7 respectively) (p<0.05). **Conclusion:** These results indicate that sufficient dairy and calcium intake affects women's PMS symptoms and improves the quality of life.

Keywords: premenstrual syndrome, calcium, quality of life

Introduction

Premenstrual syndrome (PMS) is characterized by physical, behavioral and psychological symptoms during the luteal phase of menstrual cycle, which ends with onset menstrual flow (1,2). Prevalence of PMS among the women of reproduction age varies between 30% and 73.7% (2-4). PMS not only affects women's mental health and social relationships but also reduces their efficiency at work, family, friends and hobbies

due to its physical and behavioral symptoms. PMS, considered a community health problem, is common among most women of reproduction age (5).

PMS is considered a psychoneuroendocrine disorder. Instabilities of the ovarian hormones such as estrogen and progesterone, and neurotransmitters such as serotonin and γ -amino butyric acid (GABA) can be seen in the pathophysiology of PMS. Estrogen, increasing in the fourteenth day of menstruation, may decrease the amount of calcium in the luteal phase

and increase the parathyroid hormone (hyperparathyroidism). It may also develop PMS symptoms such as mood swings, muscle cramps, tension and migraine (6,7). Therefore, nutritional, hormonal and neurotransmitters such as serotonin treatments are administered to PMS patients (8). The guide published by the Royal College of Obstetrics and Gynecology (RCOG) in 2007 indicates that B6 vitamin supplements, oral progesterone (10mg/day) and selective serotonin reuptake inhibitors should be administered in the luteal phase or continually to reduce PMS symptoms (9).

Formation process of PMS is affected by some nutrients. Insufficient intake of calcium, copper, potassium, iron and group B vitamins, alcohol consumption, smoking and sedentary behaviors increase PMS risk (6,10). Estrogen increases with ovulation, causing fluctuations in calcium, parathyroid hormone and vitamin D levels. Fluctuating calcium levels cause hyperparathyroidism in the luteal phase and PMS symptoms (6,7,11). It was found in the Nurses' Health Study-II that women who consume low amounts of calcium have a higher risk of PMS. Women who consumed high amounts of dietary calcium (1283 mg/day on average) and those who consumed low amounts of calcium (529 mg/day) were compared and it was found that high calcium consumption decreases PMS risk by 30%. Fat free or low-fat milk consumption was also found to be related to lower risk of PMS (12). The symptoms of women with PMS were found to decrease in many studies that calcium supplements were used (13-16). Thys-Jacobs et al. (17,18) reported that a daily supplement of 1200 mg of calcium decreased PMS symptoms by 48%, and a daily supplement of 1000 mg of calcium reduced PMS pains and reduced edema.

Dairy products are good source of riboflavin, niacin and vitamin B₁₂ and calcium, magnesium, phosphorus and zinc. Previous studies indicating the effect of calcium, zinc, vitamin B₆, D and iron supplements on PMS symptoms exist, but there is no study on the effectiveness of dietary dairy and calcium intake. The aim of this study is to examine if increasing dairy product and dietary calcium consumption improves the symptoms of PMS in women with inadequate calcium intake.

Subjects and methods

This study was planned as a randomized controlled trial consisting of intervention and control groups. Resource screening and background processes began in April, 2014. The sample consists of voluntary students studying at the Eastern Mediterranean University who meet the study inclusion criteria. This study was conducted between January 2015 and January 2016 with thirty-one women between 20 and 28 years of age who were consuming less than the recommended dietary allowance (RDA) of calcium and had been diagnosed with PMS by a doctor. This study is registered at Trials.Gov (NCT02809066).

All of the voluntary students were diagnosed with PMS and had been consuming less calcium than the RDA. The inclusion criteria for the intervention and control groups include: regular menstrual cycle (lasting for three to eight days between the range of 22 to 35 days), non-smoker, no systemic diseases (diabetes, chronic renal failure, hypertension, hyperlipidemia, PCOS, coronary heart disease, hyperthyroid, renal or hepatic diseases), no use of oral contraceptives, antidepressant and vitamin/mineral supplements, and no history of hormonal treatment. These criteria were observed while selecting the sample. The research procedures were approved by the Clinical Research Ethics Committee of Eastern Mediterranean University and written informed consent was obtained from all participants (2015/10-03).

The sample size was calculated using G*Power software 3.1.9 and assuming a nonparametric test was used in comparison of two groups with 0,05 alpha value, 0,80 power and Cohen d :1 effect size, total sample size was calculated as at least 15 for each group. With an expected drop out, 20 participants per group was considered adequate.

The participants were allocated into two groups randomly by simple randomization using random number charts, an intervention group of twenty and a control group of twenty. It was ensured that the intervention group consumed the RDA of 1000 mg of calcium (70-80% minimum from dairy products, 20-30% from other food groups) for two months. Turkish *kasseri* cheese (50 g) which is made from cow's milk was given to the intervention group every day for two months, and they were informed to consume at least

400 ml of milk and 150 g of yogurt each day. The nutrition composition of Turkish *kasseri* cheese is summarized in Table 1. The calcium consumption of the control group was not altered.

Interviews were conducted with the participants and a questionnaire was administered including questions about their general backgrounds, nutritional status and physical activities, the Premenstrual Syndrome Scale (PMSS) and the short form of the Quality of Life Scale (SF-36). The Premenstrual Syndrome Evaluation Scale is a five-point Likert type scale with forty-four items that was developed in 2006 by Gençdoğan using DSM-III and DSM-IV-R. This scale was administered to the participants both at the beginning and at the end of the study. The PMSS has nine subscales: depressive mood, anxiety, tiredness, anger bursts, depressive thoughts, pain, appetite changes, sleep disturbances and abdominal bloating. Exceeding half of the maximum score on the subscale of this scale determines whether an individual has PMS (19). Total scores on the PMSS and its nine subscales were calculated. The SF-36 quality of life scale was administered to the participants at the beginning and end of the study (20). The participants were followed up for two months, and at the end of two months 31 participants finished the study: 16 in the intervention group and 15 in the control group.

Dietary assessment

3-day dietary record was recorded twice, once at the beginning of the study and once at the end. Dietary assessments were recorded using the Photography Catalogue of Food and Dishes: Portion Sizes and Amounts (21). Portions were calculated using the Standard Recipes book (22). After specifying the daily amounts of the consumed foods, daily energy and macro- and micro-nutrient intakes were calculated using the Nutritional Information Systems Package Software (BEBİS) 7.2 professional edition (23). It was evaluated whether energy and nutrient intakes met the RDA (24).

Anthropometric Measures

Anthropometric measurements such as waist and hip circumference, height, weight and body composition (body fat mass, fat-free mass, total body water and abdominal fat volume) were performed by the researcher at the beginning and end of the study. The

Table 1. Kasseri cheese nutrition composition of edible portion (23).

Nutrient	Value per 100 g
Energy (kcal)	452.2
Protein (g)	19.1
Total lipid (fat) (g)	39.1
Carbohydrate (g)	0.0
Minerals	
Calcium (mg)	600.0
Magnesium (mg)	50.0
Phosphorus (mg)	400.0
Potassium (mg)	100.0
Iron (mg)	0.4
Zinc (mg)	3.0
Vitamins	
A (µg)	468.0
E (mg)	1.2
B ₂ (mg)	0.4
B ₃ (mg)	6.4
B ₆ (mg)	0.1
B ₁₂ (µg)	2.0

Table 2. Demographic and anthropometric characteristics of participants at baseline

	Intervention Group (n=16)	Control Group (n=15)	p
	Mean±SD (% 95 CI)	Mean±SD (% 95 CI)	
Age (years)	22.3±1.84	22.9±2.06	0.425
Weight (kg)	57.0± 9.40	54.9±5.65	0.621
BMI (kg m ⁻²)	21.3±3.08	21.0±2.13	0.767
Menarche age (years)	13.5±1.03	13.4±1.12	0.902
Menstrual duration (day)	5.4±1.09	5.1±0.74	0.529
Body composition			
Lean body mass (kg)	42.8±4.66	41.2±2.62	0.251
Soft lean mass (kg)	39.5±4.18	38.1±2.34	0.220
Skeletal muscle mass (kg)	18.9±4.06	17.3±2.25	0.363
Body fat (kg)	14.2±5.4	13.7±3.7	0.906
Total body water (kg)	30.8±3.35	29.4±2.5	0.206

p value was calculated Mann-Whitney U test

body weights of the participants were measured using the Jawon X Scan Plus 2 bioelectrical impedance analyzer. The measurements were carried out in the fasting state. They were wearing light clothes and no shoes.

Statistical Analysis

Statistical analysis of the data obtained from the study was performed using SPSS (Statistical Package for Social Sciences) Version 20.0. The Kolmogorov-Smirnov test was used to see whether the numerical data had a normal distribution. The data with normal distributions were evaluated with parametric tests, and those that did not were evaluated using non-parametric statistical tests.

To compare the numerical data of the intervention and control groups, the independent two-sample t-test was used when the parametric conditions were met, and the Mann-Whitney U test was used when they were not. Baseline and post-intervention results were

compared using dependent two-sample t-test when the parametric conditions were met, and the Wilcoxon test was used when they were not. For the categorical data and comparisons between the intervention group and control group, 2*2 and 2*3 chi-square tests were used. P value could not be specified when sufficient frequency was not ensured in the categorical variables. The threshold for significance was $p < 0.05$ in all statistical test results. P-value was adjusted for potential confounders, such as the baseline values of daily intake of energy and macro- and micro-nutrients in Table 3 and Table 4.

Table 3. Evaluation of the premenstrual syndrome scale (PMSS) total subscale mean scores and quality of life (SF-36) scale

	Intervention Group (n=16)			Control Group (n=15)			p ^a	p ^b
	Baseline Mean±SD (% 95 CI)	Post-intervention Mean±SD (% 95 CI)	p	Baseline Mean±SD (% 95 CI)	Post-intervention Mean±SD (% 95 CI)	p		
PMSS subtitles								
Depressive mood	24.7±3.9 (22.6-26.7)	17.5±4.5 (15.2-19.8)	<0.001*	23.1±4.0 (21.0-25.2)	23.0±4.5 (20.6-25.4)	0.688	0.306	0.003*
Anxiety	19.0±3.3 (17.3-20.8)	13.2±3.9 (11.2-15.2)	0.001*	20.0±3.3 (18.2-21.8)	20.0±3.9 (18.0-21.1)	0.951	0.446	<0.001*
Tiredness	21.1±3.9 (19.1-23.2)	14.7±4.6 (12.3-17.0)	0.001*	21.7±3.9 (19.6-23.8)	22.4±4.6 (20.0-24.8)	0.688	0.725	<0.001*
Angry	20.5±3.3 (18.8-22.2)	12.1±4.5 (9.8-14.4)	0.001*	17.5±3.3 (15.7-19.3)	19.0±4.5 (16.6-21.3)	0.674	0.026*	0.001#
Depressive thoughts	22.3±4.7 (19.8-24.7)	15.7±4.5 (13.4-18.0)	<0.001*	22.9±4.7 (20.3-25.4)	24.2±4.5 (21.8-26.6)	0.680	0.744	<0.001*
Pain	11.6±2.9 (10.0-13.1)	6.6±2.5 (5.3-7.9)	0.001*	9.2±3.0 (7.6-10.8)	11.1±2.5 (9.8-12.5)	0.078	0.047*	<0.001#
Appetite changes,	11.9±3.5 (10.1-13.7)	8.8±3.5 (7.0-10.6)	0.007*	10.1±3.5 (8.2-11.9)	10.9±3.5 (9.0-12.8)	0.031*	0.188	0.119
Sleep disturbances	10.3±2.4 (9.0-11.6)	7.6±2.5 (6.3-8.8)	0.003*	10.4±2.5 (9.1-11.7)	10.9±2.5 (9.6-12.3)	0.783	0.898	0.001*
Abdominal bloating	10.8±2.2 (9.6- 11.9)	7.7±2.9 (6.2-9.2)	0.001*	10.1±2.2 (9.0-11.3)	10.6±3.0 (9.1-12.2)	0.506	0.453	0.014*
PMSS total scores	151.9±15.4 (143.9-159.8)	104.8±21.5 (93.8-115.8)	<0.001*	145.0±15.5 (136.8-153.2)	151.1±21.5 (139.7-162.5)	0.320	0.254	<0.001*
SF-36 quality of life scale								
Physical functions	41.2±10.7 (35.7-46.7)	47.9±8.0 (43.8-51.9)	0.041*	45.0±10.7 (39.2-50.7)	40.9±8.0 (36.7-45.1)	0.222	0.364	0.027*
Mental health	40.3±9.2 (35.6-45.0)	48.6±6.9 (45.0-52.1)	0.002*	37.6±9.2 (32.7-42.5)	38.7±6.9 (35.0-42.3)	0.078	0.445	0.001*

* $p < 0.05$; p value was calculated Wilcoxon test; p^a: Comparing the early data of the Intervention and control groups; (p-value was adjusted for baseline protein, carbohydrate, vitamin E and zinc intake); p^b: Comparing the post-intervention data of the Intervention and control groups (p-value was adjusted for post-intervention potassium intake; # p-value was adjusted for baseline scores and post-intervention potassium intake

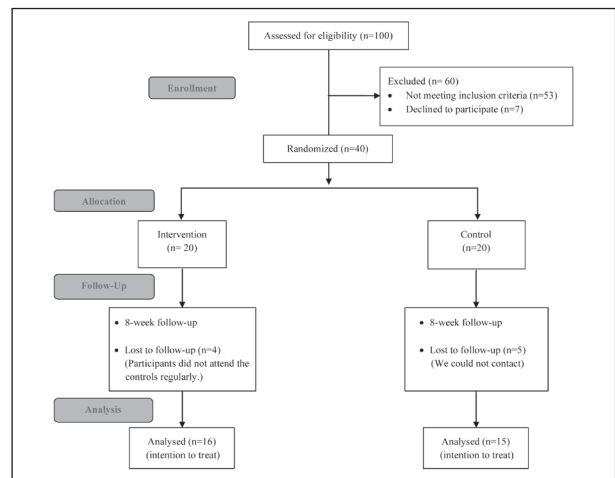
Table 4. Individuals' daily intake of energy and macro- and micro-nutrients

	Intervention Group (n=16)			Control Group (n=15)			p ^a	p ^b
	Baseline	Post-intervention	P	Baseline	Post-intervention	p		
	Mean±SD (% 95 CI)			Mean±SD (% 95 CI)				
Energy†	1608.2±371.9 (1410.0-1806.3)	1733.0±319.5 (1562.8-1903.3)	0.034*	1313.6±218.3 (1192.7-1434.5)	1351.1±349.3 (1157.8-1544.8)	0.955	0.09	<0.001*
Protein (g)†	54.1±9.3 (49.1-59.1)	69.7±9.6 (64.8-74.6)	<0.001*	47.5±7.6 (43.3-51.7)	47.6±9.6 (42.5-52.7)	0.650	0.040*	<0.001#
Carbohydrate (g)†	200.2±57.6 (169.5-230.8)	173.9±34.7 (156.1-191.7)	0.088	160.7±41.0 (137.9-183.4)	164.7±34.8 (146.3-183.2)	0.532	0.037*	0.488#
Fat (g)†	63.2±19.2 (52.9-73.4)	77.1±21.1 (65.9-88.4)	0.013*	51.1±11.6 (44.6-57.5)	54.3±17.2 (44.8-63.9)	0.570	0.078	0.003*
Vit. A (µg)•	653,32 (339,5-1383,5)	838,59 (584,8-2426,9)	0,005*	697,16 (330,56-1174,44)	659,49 (343,57- 954,13)	0,088	0,626	0,003*
Riboflavin (mg)†	1.1±0.2 (0.9-1.2)	1.8±0.2 (1.7-1.9)	<0.001*	1.0±0.3 (0.8-1.1)	1.0±0.3 (0.8-1.2)	0.513	0.299	<0.001*
Niacin (mg)†	19.7±5.2 (16.9-22.5)	23.8±5.7 (20.8-26.9)	0.009*	18.2±4.4 (15.7-20.6)	16.7±5.1 (13.9-19.6)	0.307	0.338	0.001*
Vitamin B6 (mg)•	1.4±0.4 (1.2-1.7)	1.5±0.4 (1.3-1.7)	0.623	1.3±0.4 (1.1-1.6)	1.3±0.5 (1.1-1.6)	0.463	0.175	0.313
Vitamin B12 (µg)†	2.8±0.8 (2.4-3.3)	5.3±1.3 (4.6-6.1)	<0.001	2.1±1.3 (1.4-2.8)	2.7±1.5 (1.9-3.5)	0.177	0.066	<0.001*
Vitamin E (mg)•	16,0±4.7 (7.4-24.6)	14,9±7.9 (10.9-18.9)	0,877	12.7±4.8 (6.9-25.9)	15,9±7.9 (11.9-20.0)	0.394	0.027*	0,751#
Potassium (mg)•	2216.1±554.8 (1920.5-2511.7)	2630.1±613.7 (2303.4-2957.5)	0.003*	2156.9±699.1 (1769.7-2544.0)	2054.1±762.2 (1632.0- 2476.2)	0.570	0.379	0.016*
Calcium (mg)†	500.9±114.6 (439.9-562.0)	1284.8±140.3 (1210.1-1359.5)	<0.001	511.3±149.2 (428.7-593.9)	496.7±158.3 (409.1-584.4)	0.570	0.953	<0.001*
Magnesium (mg)†	236.7±61.6 (203.9-269.6)	271.4±55.8 (241.6-301.1)	0.015*	214.3±63.2 (179.4-249.3)	197.8±96.3 (144.4-251.1)	0.394	0.216	0.009*
Phosphorus (mg)†	927.7±147.9 (848.9-1006.6)	1387.2±140.8 (1312.2-1462.2)	<0.001*	841.6±160.1 (712.9-930.2)	825.5±254.1 (584.8-966.3)	0.570	0.163	<0.001*
Iron (mg)•	9.5±2.3 (8.3-10.7)	8.6±2.7 (7.1-10.1)	0.215	8.5±2.9 (6.9-10.1)	7.8±3.5 (5.9-9.7)	0.334	0.318	0.213
Zinc (mg)•	7.1±1.6 (6.3-8.0)	8.7±1.7 (7.8-9.5)	0.001*	6.0±1.6 (5.2-6.9)	6.7±1.7 (5.8-7.5)	0.977	0.030*	0.003#

* $p < 0.05$; p value was calculated Wilcoxon test; p^a : Comparing the early data of the Intervention and control groups; p^b : Comparing the post-intervention data of the Intervention and control groups; † The data were distributed normally. p value was calculated Two-sample t -test, • The data were not distributed normally. p value was calculated Mann-Whitney U test; # p -value was adjusted for baseline scores

Results

Thirty-one women with PMS, 16 in the intervention group and 15 in the control group, participated in the study. A Consolidated Standards of Reporting Trials (CONSORT) flow chart of the randomization procedure can be seen in Figure 1. Their ages ranged from 20 to 28, and the mean ages of the intervention and control groups were found to be 22.3±1.8 and 22.9±2.1, respectively. The menarche ages of the intervention and control groups are 13.5±1.0 and 13.4±1.1, respectively. The menstruation durations of the intervention group and the control group were 5.4±1.1 days and 5.1±0.7 days, respectively. There was no signifi-

**Figure 1.** CONSORT flow diagram of the study

cant difference between the ages, menarche ages and menstruation durations of the intervention and control groups ($p>0.05$) (Table 2).

The baseline mean Body Mass Index of the intervention and control groups were 21.3 ± 3.1 kg/m² and 21.0 ± 2.1 kg/m², respectively. They were 21.5 ± 3.1 kg/m² and 21.1 ± 2.1 kg/m² at the end of the study. There was no significant difference between the BMIs of the intervention and control groups at the beginning and end of the study ($p>0.05$). A statistically significant increase was determined in the BMIs of the participants in the intervention group ($p<0.05$). A statistically significant increase was determined in the baseline (42.8 ± 4.6) and post-intervention (43.1 ± 4.6) fat-free body masses of those in the intervention group ($p<0.05$). There were no differences in the baseline and post-intervention body composition values of the control group (Table 2).

Dairy products are sources of calcium, B2, B3, B12, magnesium, zinc, and phosphorus. In addition, the recommended consumption of milk and dairy products to the intervention group resulted in increased energy, protein and fat consumption of individuals. For this reason, PMSS total score, PMSS sub-scale scores and SF-36 quality of life scores were adjusted at the baseline and post-intervention.

PMSS score and sub-scale scores except the appetite change score were found to have decreased between the baseline and post-intervention in the intervention group ($p<0.05$). There is no significant difference between the baseline and post-intervention PMSS scores and PMSS sub-scale scores of the control group ($p>0.05$). The intervention group's baseline and post-intervention physical functions and mental health scores on the SF-36 quality of life scale were increased ($p<0.05$) (Table 3). The changes of the baseline and post-intervention PMSS scores of the intervention and control groups are shown in Figure 2.

At the end of the study, daily calcium intakes of the intervention and control groups were 1284.81 ± 140.28 g and 496.74 ± 158.34 g respectively ($p<0.05$) (Table 4). Post-intervention intake of the intervention group's daily energy, protein, fat, riboflavin, niacin, B₁₂, potassium, calcium, magnesium, phosphorus, zinc, and magnesium was found to be higher than that of the control group ($p<0.05$) (Table 4).

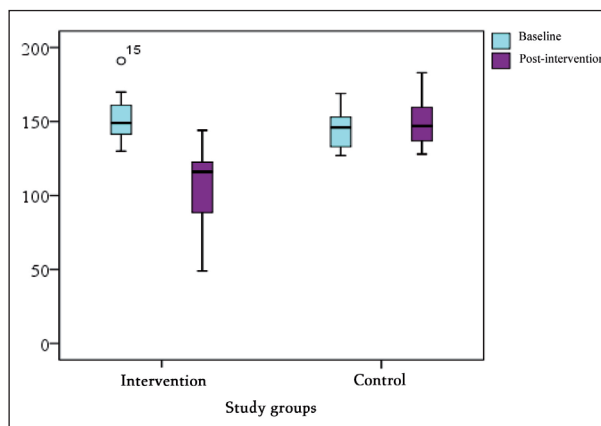


Figure 2. Distribution of the total baseline and post-intervention PMSS scores of the intervention and control groups

Discussion

The intervention group received significantly higher amounts of energy, protein, fat, riboflavin, niacin, vitamin B12, potassium, calcium, magnesium, phosphorus, and zinc due to macro and micro-nutrients having positive effects on PMS symptoms and quality of life.

Vitamins and minerals play an active role in the hormonal and neurotransmitter metabolisms related to PMS. Therefore, consuming less than the RDA of vitamins and minerals is a risk factor for PMS (6, 25).

Insufficient dietary intake of minerals is related to depressive symptoms (26, 27). Davison et al. (26) found that insufficient intake of minerals such as calcium, potassium, phosphorus, magnesium and iron, and vitamins such as pantoic acid, B₂, B₃, B₆, B₁₂, and folate is related to depression and mood disorders. Chocano-Bedoya et al. (25) found that group B vitamins (thiamine, niacin, riboflavin, folic acid, B₆, and B₁₂) might have a role in PMS development by affecting neurotransmitters such as serotonin and GABA. The participants in this study consumed less than the RDA of thiamine and folic acid, which play a role in PMS. Vitamin B₆ serves as a cofactor in the transformation of tryptophan amino acid to serotonin, and riboflavin is necessary for B₆ activation (25). The participants' baseline and post-intervention dietary assessments revealed that they consumed sufficient vitamin B₆. Both groups' baseline riboflavin intake was less than the RDA. After the study, an increase in ribofla-

vin intake was found only in the intervention group. This increase in the riboflavin intake is thought to reduce some of the PMS symptoms such as depressive feelings, depressive thoughts, and anger bursts.

Chronic zinc deficiency reduces zinc in the hippocampus and glucocorticoid secretions, causing neurological symptoms (28, 29). The Nurses' Health Study-II data indicate that those diagnosed with PMS consume less zinc than those who are not diagnosed. Insufficient zinc intake is reported to increase PMS risk (6). Our study found that all the participants had low baseline zinc intakes. The intervention group's post-intervention zinc intake increased significantly. This increase may have caused a significant decrease in their PMSS score and PMSS sub-scale scores, and a significant increase in their SF-36 quality of life scores. The control group's post-intervention zinc intake was found to be significantly lower than their baseline results. Iron is the cofactor of tryptophan hydrolysis enzyme that plays a role in the formation of preliminary serotonin substances (30). Especially the parts of the brain that receive signals from GABA are rich in iron. Low intake of iron causes depression arising from the changes in the ovarian hormones (31). The baseline and post-intervention iron intakes of all the participants were lower than the RDA. Insufficient iron intake may have affected their PMS.

Magnesium reduces menstrual pains, relaxes muscles and ensures vasodilation by inhibiting prostaglandin F₂ (PGF₂) (32). The intervention group's baseline magnesium intake was lower than the RDA. The post-intervention increase in their magnesium intake may have reduced their PMS symptoms and increased their physical functions scores on the SF-36 quality of life scale.

Excessive increase of estrogen in the ovulation and insufficient calcium intake can cause hypocalcaemia. Bocchieri et al. (33) stated that hypocalcaemia is one of the reasons of PMS is seen particularly in luteal phase. Hypocalcaemia can cause similar symptoms that arise from PMS like anxiety, emotional fluctuations, and depression. (34, 35). Also, these symptoms arise with a hypocalcaemia trigger during PMS (34). The body increases parathormone secretion to prevent hypocalcaemia if dietary calcium intake is low. Borer et al. (33) stated that hyperparathyroidism causes neu-

ropsychiatric disorders such as tiredness, concentration deficit, stress and sadness. Moreover, calcium plays a role in the synthesis of serotonin. The level of serotonin synthesis decreases with hypocalcaemia. King et al. (34) found that hypocalcaemia might lead to depression. Sufficient dietary calcium intake in the intervention group caused the decrease in total PMSS score and increase in quality of life score.

Penland and Johnson (37) compared the effect of the daily intake of 1336 mg of calcium (calcium lactate) and 5.6 mg of manganese (manganese sulphate) on PMS symptoms to that of 587 mg of calcium and 1.0 mg of manganese. They found that increased serum calcium concentration affected symptoms such as pain, behavioral symptoms, and positive mood. Thys-Jacobs et al. (6) noted that serum calcium concentration decreases in the luteal phase, as a result of this, PMS symptoms increase. Calcium supplementation inhibits the decline of serum calcium concentration in the luteal phase.

Bae and Kim found daily intake of calcium to be related to depression (36). Miki et al. (37) compared the calcium intakes of those with and without depressive symptoms and found that low intake of (220 mg/1000 kcal/day) calcium increased the depressive symptom development by 36%. Participants' calcium intakes were low in our study, so this might have increased the depressive mood.

Sutariya et al. (38) found that a 500 mg/day calcium supplement (calcium carbonate or glutamate) reduced symptoms such as mood disorder, depression, anger bursts, crying attacks, edema, abdominal cramps and abdominal bloating, back pain, headaches, appetite, acne, and desire for sweets. Dairy products consumption rates were increased in this study to enhance the daily calcium intakes of the participants in the intervention group. Moreover, depending on this increase, there was an increase in the intake of calcium, phosphorus, B₂, and B₁₂. This may have reduced the intervention group's PMSS sub-scale scores for depressive mood, anxiety, anger bursts, and depressive thoughts, and increased their SF-36 mental health scores.

Obesity changes the neurotransmitter functions that affect estrogen and progesterone hormones (39). Bertone-Johnson et al. (40) noted that every increase

of 1 kg/m² in BMI increased PMS risk by 3%. In another study, a significant relation was found between a BMI higher than 30 kg/m² and PMS occurrence (39). The participants in this study had intense PMS symptoms although their BMIs were in the normal range. Insufficient consumption of the nutrients related to PMS may have caused PMS symptoms.

Limitations of our study are as follows:

Individuals who were consuming low amounts of dietary calcium and using no supplements were included in the study to see the effect of dairy intake on PMS symptoms. However, it was not possible to determine whether participants had hypocalcaemia since their blood findings such as serum calcium, ionized calcium, vitamin D, and parathormone were not examined.

In conclusion, sufficient and balanced nutrition is always important, as it is for PMS, too. Dairy products are sources of calcium, B₂, B₃, B₁₂, magnesium, zinc, phosphorus. The previous studies showed that these vitamins and minerals could decrease the PMS symptoms. We concluded that daily sufficient intake of calcium from food sources has positive effects on depression, anxiety, fatigue, anger, depressive thoughts, pain, appetite changes, sleep disorders and swelling of extremities symptoms in women with PMS whose calcium intakes are lower than recommended.

Acknowledgments

The authors are thankful to the valuable support of Ceren Gezer and Halit Tanju Besler, Faculty of Health Sciences, Nutrition and Dietetics Department.

References

- Biggs WS, Demuth RH. Premenstrual syndrome and premenstrual dysphoric disorder. *Am Fam Physician* 2011;8:918-24.
- Erbil N, Karaca A, and Kınış T. Investigation of premenstrual syndrome and contributing factors among university students. *Turk J Med Sci* 2010;4:565-573.
- Baker LJ, O'Brien PM. Premenstrual syndrome (PMS): a peri-menopausal perspective. *Maturitas* 2012;2:121-5.
- Duenas JL, Lete I, Bermejo R et al. Prevalence of premenstrual syndrome and premenstrual dysphoric disorder in a representative cohort of Spanish women of fertile age. *Eur J Obstet Gynecol Reprod Biol* 2011;1:72-7.
- Maharaj S, Trevino K. A Comprehensive Review of Treatment Options for Premenstrual Syndrome and Premenstrual Dysphoric Disorder. *J Psychiatr Pract* 2015;5:334-50.
- Thys-Jacobs S, McMahan D, Bilezikian JP. Cyclical changes in calcium metabolism across the menstrual cycle in women with premenstrual dysphoric disorder. *J Clin Endocrinol Metab.* 2007; 8: 2952-9.
- Saha S, Goswami R. Menstruation associated hypocalcemic symptoms and serum calcium in patients with idiopathic hypoparathyroidism. *BMC Endocr Disord.* 2014; 14: 28.
- Panay N. Management of premenstrual syndrome. *J Fam Plann Reprod Health Care*, 2009; 3:187-94.
- Arrigo T, De Luca F, Antoniazzi F et al. Menstrual cycle pattern during the first gynaecological years in girls with precocious puberty following gonadotropin-releasing hormone analogue treatment. *Eur J Pediatr.* 2007; 1: 73-4.
- Chocano-Bedoya PO, Manson JE, Hankinson SE et al. Intake of selected minerals and risk of premenstrual syndrome. *Am J Epidemiol* 2013;10:1118-27.
- Thys-Jacobs S. Micronutrients and the premenstrual syndrome: the case for calcium. *J Am Coll Nutr* 2000;2:220-7.
- Bertone-Johnson ER, Hankinson SE, Bendich A, Johnson Sr, Willett WC, Manson JE. Calcium and vitamin D intake and risk of incident premenstrual syndrome. *Arch Intern Med* 2005;11:1246-52.
- Shobeiri F, Araste FE, Ebrahimi R, Jenabi E, Nazari M. Effect of calcium on premenstrual syndrome: A double-blind randomized clinical trial. *Obstet Gynecol Sci* 2017;60:100-5.
- Ghanbari Z, Haghollahi F, Shariat M, Foroshani AR, Ashrafi M. Effects of calcium supplement therapy in women with premenstrual syndrome. *Taiwan J Obstet Gynecol* 2009;2:124-9.
- Masoumi SZ, Ataollahi M, Oshvandi K. Effect of Combined Use of Calcium and Vitamin B6 on Premenstrual Syndrome Symptoms: a Randomized Clinical Trial. *J Caring Sci* 2016;1:67-73.
- Shehata NA. Calcium versus oral contraceptive pills containing drospirenone for the treatment of mild to moderate premenstrual syndrome: a double blind randomized placebo controlled trial. *Eur J Obstet Gynecol Reprod Biol* 2016;1:100-4.
- Thys-Jacobs S, Ceccarelli S, Bierman A, Weisman H, Cohen MA, Alvir J. Calcium supplementation in premenstrual syndrome: a randomized crossover trial. *J Gen Intern Med* 1989;3:183-9.
- Thys-Jacobs S, Starkey P, Bernstein D, Tian J. Calcium carbonate and the premenstrual syndrome: effects on premenstrual and menstrual symptoms. *Premenstrual Syndrome Study Group.* *Am J Obstet Gynecol* 1998;2:444-52.
- Gençdoğan B. A New Scale for Premenstrual Syndrome. *Turk Psikiyatri Derg* 2006;2:82-87.
- Demiral Y, Erdor G, Unal B et al. Normative data and discriminative properties of short form 36 (SF-36) in Turkish urban population. *BMC Public Health* 2006;6:247-55.

21. Rakıcıoğlu N, Tek NA, Ayaz A, Pekcan G. Photograph Catalog of Food and Dishes: Portion Sizes and Amounts. Ankara: Hazırlık Ofset Pub; 2015.
22. Kutluay TM. Standart Recipes for Catering Establishments. Ankara: Şahin Ofset Pub; 2003.
23. Bebis (Beslenme Bilgi Sistemi). Nutrition Data base Software Data base: The German Food Code and nutrition Data Base with additions from USDA-sr and other sources. İstanbul; 2004.
24. NAS. IOM. Food and Nutrition Board(1997). Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. <https://fnic.nal.usda.gov> (accessed September 2018)
25. Chocano-Bedoya PO, Manson JE, Hankinson SE et al. Dietary B vitamin intake and incident premenstrual syndrome. *Am J Clin Nutr* 2011;5:1080-6.
26. Davison, KM, Kaplan BJ. Nutrient intakes are correlated with overall psychiatric functioning in adults with mood disorders. *Can J Psychiatry* 2012;2:85-92.
27. Kaplan BJ, Crawford SG, Field CJ, Simpson JS. Vitamins, minerals and mood. *Psychological bulletin*. 2007; 5: 747.
28. Sowa-Kucma M, Legutko B, Szewczyk B et al. Antidepressant-like activity of zinc: further behavioral and molecular evidence. *J Neural Transm* 2008;5:1621-8.
29. Takeda A, Tamano H. Insight into zinc signaling from dietary zinc deficiency. *Brain Res Rev* 2009;1:33-44.
30. Toxqui L, De Piero A, Courtois V, Bastida S, Sanchez-Muniz F, Vaquero MP. Iron deficiency and overload. Implications in oxidative stress and cardiovascular health. *Nutr Hosp* 2010;25:350-65.
31. Albacar G, Sans T, Martin-Santos R et al. An association between plasma ferritin concentrations measured 48 h after delivery and postpartum depression. *J Affect Disord* 2011;131:136-42.
32. Seifert B, Wagler P, Dartsch S, Schmit U, Nieder J. Magnesium-a new therapeutic alternative in primary dysmenorrhea. *Zentralbl Gynakol* 1989;11:755-60.
33. Bocchieri E, Thys-Jacobs S. Role of calcium metabolism in premenstrual syndrome. *Expert Rev Endocrinol Metab* 2008;3:645-55.
34. King RD, Wiest MC, Montague PR. Extracellular calcium depletion as a mechanism of short-term synaptic depression. *J Neurophysiol* 2001;5:1952-9.
35. Bae YJ, Kim SK. Low dietary calcium is associated with self-rated depression in middle-aged Korean women. *Nutr Res Pract* 2012;6:527-33.
36. Borer MS, Bhanot VK. Hyperparathyroidism: neuropsychiatric manifestations. *Psychosomatics* 1985; 7: 597-601.
37. Penland JG, Johnson PE. Dietary calcium and manganese effects on menstrual cycle symptoms. *Am J Obstet Gynecol* 1993;5:1417-23.
38. Miki T, Kochi T, Eguchi M et al. Dietary intake of minerals in relation to depressive symptoms in Japanese employees: the Furukawa Nutrition and Health Study. *Nutrition* 2015;5:686-90.
39. Sutariya S, Talsania N, Shah C, Patel M. An Intervention Study (Calcium Supplementation & Health Education) On Premenstrual Syndrome- Effect On Premenstrual And Menstrual Symptoms. *Natl J Community Med* 2011;1:100-104.
40. Masho SW, Adera T, South-Paul J. Obesity as a risk factor for premenstrual syndrome. *J Psychosom Obstet Gynaecol* 2005;1:33-39.
41. Bertone-Johnson ER, Hankinson SE, Walter CW, Susan RJ, JoAnn EM. Adiposity and the development of premenstrual syndrome. *J Womens Health (Larchmt)* 2010;11:1955-62.

Correspondence:

Merve Yurt

Eastern Mediterranean University, Faculty of Health Sciences, Nutrition and Dietetics Department, Mersin 10, Turkey

E-mail: merve.yurt@emu.edu.tr