

## Review:

# A Vicious Cycle: Using Nutrition to Combat the Behavioral Impact of Premenstrual Syndrome and Premenstrual Dysphoric Disorder

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**Premenstrual Syndrome (PMS) and Premenstrual Dysphoric Disorder (PMDD) are mood disorders experienced by women of child-bearing age who are regularly experiencing their menstrual cycles. Symptoms experienced negatively impact women cognitively, socially, emotionally, and physically.**

## ABSTRACT

**Methods:** A literature review was performed relevant to the behavioral effects of PMS and PMDD and symptomatology. Nutritional and lifestyle interventions with evidence were analyzed and reviewed.

**Results:** Studies indicated that intestinal dysbiosis, the Western diet, inadequacies in omega-3 fatty acids, vitamin D, vitamin B6, iron, sun exposure, smoking, alcohol, and physical activity are linked to symptomatology associated with PMS and PMDD.

**Conclusion:** Addressing micronutrient and essential fatty acid deficiencies, intestinal dysbiosis, smoking avoidance, limiting alcohol consumption, promoting physical activity and sunlight exposure, and providing education on PMS and PMDD may improve symptoms associated with these conditions.

**Keywords:** Nutrition, Diet, Supplementation, Behavioral Effects, Premenstrual Syndrome, Premenstrual Dysphoric Disorder

## INTRODUCTION

Premenstrual Syndrome (PMS) and Premenstrual Dysphoric Disorder (PMDD) are mood disorders experienced by women of child-bearing age who are regularly experiencing their menstrual cycles. The menstrual cycle is broken into four main phases: the menstrual phase, or menstruation, the preovulatory phase, or the follicular phase, ovulation, and the postovulatory phase (the luteal phase). The menstrual cycle is governed by the hypothalamus' release of gonadotropin-releasing hormone (GnRH), which stimulates the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which both secrete ovarian hormones, estrogens, and progesterone, throughout the cycle.<sup>1</sup> PMS and PMDD symptoms occur during the postovulatory, or luteal phase, of the menstrual cycle, due to the cyclic rise of the ovarian hormone, progesterone, at this time.<sup>1</sup> PMS varies between women but can be characterized by edema,

weight gain/water retention, breast pain/enlargement, back and joint pain, constipation, extreme fatigue, acne, mood swings, anxiety, irritability, headaches, and insomnia.<sup>1</sup> PMDD has the same symptoms as PMS, but is on the higher end of the continuum of severity.<sup>2</sup> Symptoms of PMS and PMDD subside completely once menstruation begins and progesterone levels return to baseline, indicating an on-off phenomenon.<sup>2</sup> This intermittent experience of symptoms, that fluctuates in correspondence to the menstrual cycle, differentiates PMS and PMDD from other mood disorders and is an important criterion for diagnosis.

Despite the blanketing of symptoms, PMS is broken into four categories based on the symptoms experienced: PMS-A, PMS-C, PMS-D, and PMS-H. PMS-A is the most common, characterized by elevated estrogen and lowered progesterone, manifesting as nervousness, irritability, mood swings, and insomnia. PMS-C symptoms include increased appetite, particularly for sweets, altered glucose tolerance, and fatigue. PMS-D is less common, and differs from PMS-A, as it is characterized by abnormally high levels of serum progesterone and low estrogen. In PMS-D there is also increased steroidal hormone secretion, also seen in PMDD. In PMS-H, there is cyclic weight gain, as this category of PMS is associated with excessive production of aldosterone, causing water retention during the luteal phase.<sup>3</sup>

All types considered, PMS affects 30-40% of child-bearing women.<sup>4</sup> Of premenopausal women, 3-8% are diagnosed with PMDD, and many more women who experience the disorder go undiagnosed.<sup>5</sup> But diagnosed or not, premenstrual symptoms continue to negatively impact women cognitively, socially, emotionally, and physically, as women with PMS/PMDD show cognitive decline, a tendency towards addictive behaviors, social isolation, thoughts of suicide, and other emotional dysregulation during this phase of their cycle.<sup>6-8</sup> Women also experience intense cravings for foods that are highly sweetened and high in fat during the luteal phases of their cycle.<sup>9</sup> Both the lack of self-inhibition and the uptick in cravings during this time affect behavior, as

women with PMS and PMDD consume more calories during the postovulatory phase of menstruation, with the excess energy coming from fat and carbohydrates.<sup>10</sup>

Risk factors for PMS and PMDD include lifestyle factors, such as alcohol-drinking and smoking habits, as well as family history of other psychiatric disorders, including anxiety and depression.<sup>11-12</sup> Women who consumed a primarily Westernized Diet were also positively associated with worsened symptoms of both PMS and PMDD, as are those with heightened stress levels.<sup>13</sup> Genetics also play a vital role in the likelihood of developing the disorder. Research is beginning to see associations between the prevalence of PMDD with specific estrogen receptor genes.<sup>14</sup>

Severe PMS and PMDD do not present evidence of abnormal hormonal regulation, but rather defective serotonin function triggered by these cyclic hormonal events.<sup>15</sup> While hormonal therapy is common in treating PMS and PMDD, it is not the most efficacious, due to the various types of PMS discussed above.<sup>12</sup> Psychiatric drug interventions have shown positive impacts on the emotional symptoms associated with PMS/PMDD; Selective Serotonin Retake Inhibitors (SSRIs) are the most commonly used pharmaceutical to combat PMS/PMDD. Other antidepressants are also used, but SSRIs have been the most successful.<sup>16</sup> SSRIs have also shown efficacy when used intermittently, administered only during the luteal phase of the cycle.<sup>17</sup> Some novel studies inducing menopause in younger women have reported resolution of the symptoms associated with PMS and PMDD.<sup>18</sup> However, given the cost and the uncertainty regarding the long-term effects, particularly fertility, this is not the most practical option for most people.<sup>19</sup> Anti-inflammatory drugs are also commonly used to treat symptoms of the disorder. Nutritional interventions are given little attention compared to pharmaceuticals, but research has indicated that omega-3 fatty acids, vitamin D, calcium, and high doses of vitamin B6 are negatively associated with the severity of PMS and PMDD symptoms.<sup>20</sup>

The microbiome affects various mental processes and is a critical part of the bidirectional communication between the central nervous system and the gut. Gut dysbiosis and inflammation affect the mind via the gut-brain axis. Emerging research links the gut microbiome and the depressive symptoms associated with PMDD.<sup>21</sup> Additionally, symptoms and treatment of PMDD mirror those of other psychiatric disorders. Direct comparisons between PMDD and major depressive disorder, anxiety, obsessive-compulsive disorder, bipolar disorder, and schizophrenia appear frequently throughout the current literature. Bipolar disorder, for example, is a comorbidity for PMDD, and vice versa.<sup>22</sup>

Furthermore, researchers are finding an overlapping gene pool between women with PMDD and patients with schizophrenia, suggesting a common genetic predisposition.<sup>23</sup> Low estrogen during different phases of the menstrual cycle has exacerbated symptoms of schizophrenia for some women.<sup>24</sup> Research has illuminated unique gut microbiota behavior in schizophrenics that may provide insight into treatment and diagnosis. Existing links between PMDD and other psychiatric disorders highlight the potential these connections may have in both treatment and prevention. Established similarities in symptoms and treatment between PMDD and other psychiatric disorders warrant the need for more research on the gut's relationship to PMS and PMDD.

The focus of this review will be on the behavioral effects of PMS and PMDD, and how they may perpetuate severity of the disorder through addictive behavior, binge eating, and subsequent gut dysbiosis. Additionally, this paper will discuss the nutritional interventions with evidence that may mitigate the symptoms associated with PMS and PMDD as well as preventive care.

### METHODS

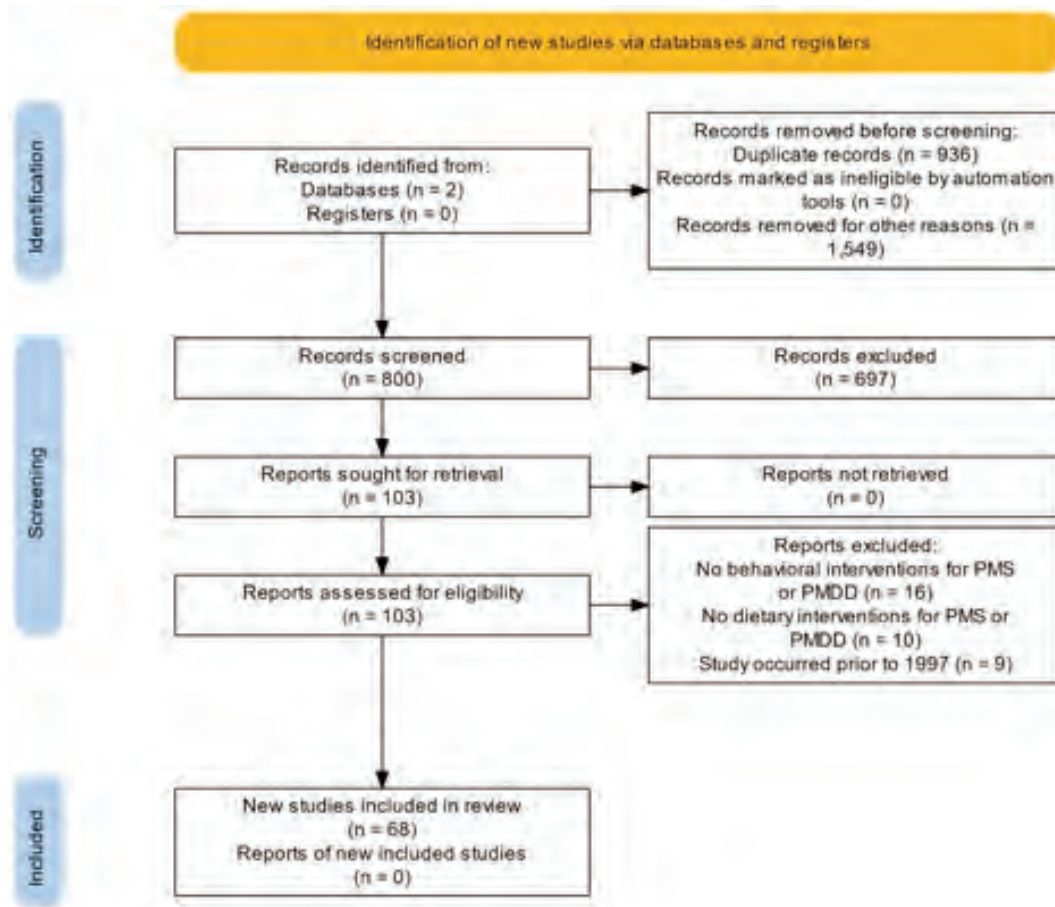
A literature search in Wahlstrom Library, JAMA network, and PubMed was conducted using the search terms “PMS, Premenstrual Syndrome, “Premenstrual Dysphoric Disorder,” “PMS and Depression,” “Eating behaviors PMS,” “Dietary intervention for PMS,” “Leptin levels in PMS and PMDD,” “PMDD Etiology,” “Depression and PMDD,” “Eating Habits and PMDD,” “PMDD treatment,” “PMDD behaviors,” “Vitamin D PMS,” and “B6 and PMDD.” Filters applied included: Clinical trials, meta-analysis, systematic reviews, randomized controlled trials, reviews, and systematic reviews. Cohort, epidemiological, randomized control trials, meta-analyses, and systematic reviews with full-text published between 1997 and 2022 were included. Studies not pertaining to PMS or PMDD were excluded.

### RESULTS

A database search for this review resulted in 3285 abstracts. After accounting for duplicates and abstracts that did not meet inclusion criteria, 800 papers remained. Based on title and abstract provided, 103 papers were considered relevant. After a full-text review, 68 papers were included in this review. Included studies were papers involving behavioral and dietary interventions for treating PMS and PMDD, as well as etiology reviews and population studies discussing the prevalence of PMS and PMDD and the current perspectives on treatment. Study designs include 25 narrative reviews, 9 cross-section studies, 6 systematic reviews, 3

cohort/prospective studies, 5 comparative studies, 2 case/control studies, 8 clinical trials, and randomized controlled trials, 5 meta-analyses, 1 animal study, 4 observational/survey studies, and 2 case reports. The paper also refers to current national nutritional guidelines.

**FIGURE 1. PRISMA FLOW DIAGRAM.82**



**DISCUSSION**

**Behavioral Effects of PMDD**

Serotonin dysregulation during the premenstrual cycle contributes to depression, mood swings, and food cravings women experience during the luteal phase of the menstrual cycle.<sup>25</sup> The luteal-phase depression experienced by women with PMDD has been shown to influence dietary behaviors, exacerbating the depression and emotional dysregulation, and therefore perpetuating the psychological symptoms of the disorder. Women with PMS and PMDD have been shown to consume more calories, particularly from lipids, during the luteal phase when compared to women without PMDD.<sup>9</sup> Women with PMS/PMDD have also been shown

to consume more carbohydrates, particularly simple sugars, during the luteal phases of their cycles when compared to controls.<sup>10</sup> Multiple studies have linked depression and anxiety with increased intake of refined sugars and fats and have noted that these eating behaviors may serve as a coping mechanism for these psychological ailments.<sup>26-27</sup> The relationship is not mutually exclusive; researchers have also

found that increased intake of processed foods may exacerbate the symptoms of depression.<sup>28</sup> Women who increase caloric intake with highly processed foods may experience more severe bouts of psychological distress during this phase. In addition, the replacement of nutrient-dense foods with highly processed foods can result in a loss of vitamins and amino acids that serve as precursors to serotonin.

Serotonin dysregulation can contribute to excessive food consumption due to its strong influence on dietary behavior. Serotonin communicates with both the homeostatic and hedonic circuitry that regulates energy consumption.<sup>25</sup> Weight gain and obesity are positively associated with decreased serotonin

signaling and activity, as serotonin is highly involved with these processes.<sup>25</sup> The homeostatic circuitry matches energy expenditure with energy consumption, while the hedonic is involved with satiety and reward-signaling from food. Reduced activity in both serotonin and/or serotonin receptors results in increased consumption due to lower homeostatic and hedonic satisfaction.

Women with PMDD are more likely to be overweight or obese.<sup>26</sup> Leptin, a hormone regulating satiety, also fluctuates throughout the cycle and is understood to influence eating behaviors and the reward-effect of food in women with PMS and PMDD.<sup>27</sup> It is unknown whether the excess weight on these women is causing exacerbated premenstrual symptoms, such as serotonin dysregulation and dramatic leptin

fluctuations, or if the serotonin and leptin dysregulation during the premenstrual phase induces obesogenic eating behaviors. It is not elucidated in the literature as to which comes first, or whether there is a bidirectional influence.<sup>27</sup>

Women are more likely to increase addictive behaviors during the luteal phase of their cycles.<sup>6</sup> The dysregulation of the reward-effect of food, as governed by serotonin and leptin can also influence addictive behaviors around food, causing an increased consumption of highly palatable, dopaminergic foods, as we see in foods high in refined sugars and fats. A case report looking at a woman with co-occurring PMDD and binge eating disorder was cured of her binge-eating behavior after receiving medically induced menopause, suggesting that the binge eating behavior was a byproduct of her PMDD.<sup>18</sup> When evaluating the relationship between women with eating disorders, specifically bulimia and binge eating disorder, researchers found 16.6% similarities between women with PMDD and eating disorders (ED), particularly in serotonin mediation and a tendency towards harm avoidance, as compared to the 2.3% similarities they found between women with ED and normal population.<sup>28</sup> It is also important to note that both PMS/PMDD and ED occur when a woman reaches puberty. Women of the same age at the onset of their periods, are within the same time frame where they commonly develop patterns of disordered eating behaviors.<sup>29</sup> Cross-sectional studies looking at the epigenetics of eating disorders have come up inconclusive. However, research has suggested that variations within the brain-derived neurotrophic factor (BDNF) gene may contribute to the likelihood of ED onset.<sup>30</sup> While emergent, this research has potential for future ED treatment.<sup>31</sup> PMDD has also been linked with specific gene variations and their interactions with BDNF. These variations affect the depressed mood associated with PMDD, as it causes more sensitivity to the rise of ovarian steroids experienced during the menstrual cycle.<sup>32</sup> More research is necessary to elucidate the onset of disordered eating, and whether disordered eating perpetuates symptoms of PMS or whether they co-occur because of common genetic and environmental factors underlying these pathologies that may predispose women to PMS/PMDD and ED.<sup>32</sup>

### **Gut Dysbiosis**

Gut dysbiosis and the resultant disruption in intestinal barrier function have been linked with a variety of psychiatric issues, including those associated with PMDD, such as anxiety and depression.<sup>21</sup> Gut dysbiosis involves increased intestinal permeability by loosening tight junctions; this causes disruptions to the gut microbiome, which interferes with the bi-directional communication of the gut-brain axis via the vagus nerve.<sup>33</sup>

The behavioral effects of PMS/PMDD, including depression, binge eating, and inclination towards addictive behaviors like smoking, drinking, and excessive highly palatable foods, can alter the gut microbiome and increase intestinal permeability.<sup>33</sup> This dysbiosis, via the bidirectional communication between the gut and the brain, can perpetuate depression and anxiety experienced in women with PMDD, along with other neuropsychiatric disorders.<sup>33</sup> In vivo studies using fecal transplants to alter the microbiome of rats with dysbiosis have demonstrated that once the restoration of a healthy microbiome was restored, rats ceased to show depressed behaviors.<sup>33</sup> Within the same study, they were able to induce depression in rats through fecal transplants in the other direction.<sup>33</sup> Subsequently, this negative mood can increase the individual's drive to eat to soothe emotions, as depressive symptoms have been shown to influence eating behavior.<sup>34,35</sup> Recent evidence has demonstrated differences in the gut microbiome of obese subjects, suggesting that obesity is a risk factor for gut dysbiosis.<sup>36</sup> The process of deriving pleasure from food occurs through the dopaminergic response. With a steep rise in dopamine obtained from highly palatable foods also comes a drop that brings the individual below their baseline levels; they would require more of the substance of choice to get the same dopaminergic effect.<sup>37</sup> This behavior can be seen in the excessive consumption of highly palatable foods, foods high in sugar and fat, that leads to obesity and is a risk factor for dysbiosis. The Western diet is a contributor to altered gut microbiota.<sup>38</sup> The excessive consumption of processed food is closely linked to dysbiosis, depression, and eating behaviors. This cycle of eating, not unlike the menstrual cycle, becomes a vicious cycle, as overeating drives the depression that drives overeating.

The Western Diet is characterized by the overconsumption of highly processed and refined foods. The Western Diet amplifies intestinal permeability, making the individual more vulnerable to pathogenic bacteria.<sup>39</sup> Gut dysbiosis is associated with the overgrowth of yeasts in the gut that help to facilitate the growth of *Candida* organisms.<sup>40</sup> These *Candida* organisms produce thiaminase, which destroys thiamin and B1, causing various neuropsychiatric pathologies.<sup>40</sup>

The inefficiency of the gut also obstructs the conversion of various amino acids, particularly tryptophan, an important amino acid to produce serotonin, the "feel good" neurotransmitter.<sup>40</sup> Hormone-replacement therapy has been shown to stimulate candida growth in the gut.<sup>40</sup> Likewise, vulvovaginal candidiasis, often co-occurring with candida growth in the gut, is commonly seen in women taking oral contraceptives, a common treatment to manage symptoms of PMS and PMDD.<sup>41,42</sup> Women taking oral contraceptives also show

higher rates of depression and lower levels of tyrosine, a dopamine precursor.<sup>43</sup> Additionally, oral contraceptives impede the absorption of vitamin B6, a precursor to serotonin, contributing to the increased depression seen among women on birth control medications.<sup>44</sup>

### **Nutritional Interventions**

The Western Diet, as previously discussed, is associated with overconsumption of processed foods, salt, and refined carbohydrates with underconsumption of vitamins and minerals. Approximately 75% of Americans do not consume the recommended intake of fruits, and 80% of individuals do not consume the recommended daily amounts of vegetables.<sup>45</sup> Due to the Westernized diet, more than 40% of Americans do not reach micronutrient requirements from food alone.<sup>46</sup> This could be a major puzzle piece in managing PMS and PMDD; in addition to the lack of nutrient consumption we see in Americans on a regular basis, women's eating styles tend to change along with the phases of their menstrual cycles, leading to poorer food choices and exacerbated symptoms of the condition. Women experience an uptick in hunger and cravings during the luteal phase of their cycles. This contributes to the physical and psychological ailments of the disorder via gut dysbiosis, and the subsequent nutritional inefficiencies mentioned above. By addressing the body's nutrient depletions during this time of the month, women can mitigate those cravings by providing the nutrients their bodies require instead of perpetuating their symptoms with refined sugars and processed fats.

### **Omega-3s**

Across all ages and ethnicities, omega-3 levels in Americans are well below the recommended intake.<sup>47</sup> Omega-3s play an important role in the management of C-reactive protein, a marker for systemic inflammation in the body.<sup>48</sup> Randomized controlled trials indicate other markers of inflammation, lipopolysaccharides (LPS) and Interleukin-6 (IL-6), also drop with eicosapentaenoic acid (EPA) and docosahexaenoic (DHA) supplementation, with corresponding reductions in anxiety for subjects.<sup>49</sup> Meta-analyses confirm that there is efficacy in the utilization of EPA and DHA in treating depression and anxiety.<sup>50, 51</sup> Including omega-3 fatty acids regularly in the diet can play an important role in managing the depressive symptoms of PMDD. Intake of EPA and DHA and fish oil, particularly in women, was negatively associated with depression and anxiety.<sup>52</sup> The Mediterranean Diet, which is high in omega-3s is noted as the best dietary approach to combating psychological symptoms.<sup>52</sup> Researchers found that the benefits of EPA/DHA supplementation came with a decrease in the omega-6:omega-3 ratio.<sup>49</sup>

### **Vitamin D**

Epidemiological studies suggest that most Americans are deficient in vitamin D with 81% of children and adolescents and 95% of adults not meeting the estimated average requirement (EAR) of vitamin D.<sup>46</sup> Vitamin D and calcium play an important role in estrogen balance, and adequate intake is essential for adequate hormonal health.<sup>53</sup>

Cyclic changes in estrogen that occur throughout the menstrual cycle affect calcium balance within the body by altering absorption, which impedes vitamin D metabolism.<sup>54</sup> This dysregulation of calcium and vitamin D are suspected culprits for the various symptoms experienced in women with PMDD, such as mood disturbances, cramps, low back pain, and insomnia.<sup>55</sup> Supplementing with calcium and vitamin D can be important steps to mitigate symptoms and prevent long-term effects, like osteoporosis. Vitamin D supplementation can help to regulate serum levels of vitamin D and alleviate symptoms, particularly mood dysfunction, experienced in PMDD.<sup>56</sup> High doses of vitamin D, 50,000 IU per week, have shown to significantly reduce the severity of both physical and psychological premenstrual symptoms in adolescent women.<sup>57</sup> The current RDI for vitamin D for menstruating women is 600 IU/day, which is not being met in the United States. Women can increase their amounts of vitamin D through supplementation or by incorporating 2 teaspoons of cod liver oil or one serving of fatty fish, such as trout, salmon, or sardines into their daily diet.<sup>58</sup> This would have an additional benefit of also increasing omega-3 levels.

Natural food sources, most fortified food sources, and UV light exposure utilize vitamin D3, or cholecalciferol, which the body later converts to 25-hydroxy-vitamin D.<sup>59</sup> Vegan sources of vitamin D are synthetically produced from fungi and yeast as vitamin D2. These sources are not as efficacious in improving serum vitamin D as D3 supplementation, due to their inability to bind with serum vitamin D binding protein. Thus, vitamin D3 through food and sun exposure is the most bioavailable source for improving serum vitamin D.<sup>60,61</sup> Oral D3 supplementation, natural sunlight, and artificial UV exposure are all viable ways to improve serum 25 (OH)D.<sup>59</sup> Meeting adequate levels with oral exposure alone can lead to toxicity if more than 10,000 IU/day is consumed, so it is important to also balance UV exposure with supplementation.<sup>59</sup>

### **Vitamin B6**

Although the recommended intake of vitamin B6 is only 1.5mg/day for women, most people who did not supplement with B6 were deficient in this micronutrient.<sup>62</sup> Vitamin B6, a precursor for the neurotransmitter serotonin, can help to

alleviate mood symptoms associated with PMS and PMDD. In a double-blind randomized control trial, B6 alleviated the severity of PMS/PMDD symptoms in women over the course of two menstrual cycles.<sup>63</sup> The results from B6 supplementation alone produced comparable results to the control group, which received broad-spectrum micronutrients. Therefore, the incorporation of a diet rich in a variety of micronutrients is recommended.

Vitamin B6 deficiencies can cause depression and confusion. Maintenance of adequate levels of B6 can help to distinguish depression from deficiency which may contribute to depression and brain fog associated with PMS/PMDD. Randomized controlled trials prescribing 80mg (53x the recommended daily amount) to women with PMS showed a significant improvement in symptoms, particularly mood and cognitive-related symptoms.<sup>62</sup> Further research is necessary to elucidate the importance of vitamin B6 in PMS and PMDD. However, it is important to note that although the current Recommended Daily Allowance (RDA) of vitamin B6 is less than 2 milligrams per day, Americans still consume inadequate levels.<sup>64</sup> Additionally, researchers suggest that due to hindrance of absorption from oral contraceptives, women taking oral contraceptives have higher Vitamin B6 requirements.<sup>65,66</sup> People eating a Western Diet, especially women taking oral contraceptives should be diligent to incorporate a variety of animal and plant-based foods rich in B6, such as liver, tuna, salmon, chickpeas, and foods fortified with B6. Research has shown efficacy with large doses of vitamin B6 (up to 80mg/day), but the research is not clear as to whether it is better to supplement externally or to get it from nutrient-dense food. The research done on B6 shows comparable results to broad-spectrum micronutrients, therefore it is important for women who are not taking external supplementation to include a wide variety of these pyridoxine-rich foods throughout the day to ensure adequate dosage and a variety of other micronutrients.

### *Iron*

Iron is the most common nutrient deficiency in the United States.<sup>67</sup> Iron is essential in the transport of oxygen, DNA regulation/repair, and protection against reactive oxygen species (ROS).<sup>68</sup> Iron deficiency can cause anemia, poor thyroid function and subsequent hormone dysregulation, and immune system impairment. Of menstruating women, 9.8% meet at least two of the three markers of iron deficiency.<sup>68</sup> Given the blood loss during the menstrual cycle, it is imperative that these women increase their iron, especially during the menstrual phase of their cycles. Including foods rich in iron is important to prevent anemia during menstruation, particularly for women experiencing menorrhagia. Iron and zinc

intake has also been linked to lower rates and lower severity of PMS.<sup>69</sup>

However, in the case of iron, individuals need to be cautious of iron overload, which results from an accumulation of excessive dietary iron in parenchymal cells. This accumulation can lead to tissue damage, particularly in the liver, heart, and pancreas.<sup>67</sup> Most individuals should strive to meet the recommended daily intake of iron, which is 18mg per day for women. This can be achieved through just one serving of fortified cereal or 2 cups of white beans. One 3-oz serving of beef liver provides about one-third of the daily value. Women should also be mindful to include spinach and lentils into their diets, each containing 3mg of iron per serving.<sup>70</sup> Incorporating these foods throughout the day will help to add enough iron to meet the adequate levels for most people.

### *Lifestyle Recommendations*

There are lifestyle factors outside of nutrition that help to combat the symptoms of PMS/PMDD. Physical activity has been proven to alleviate physical symptoms of the pathology. Randomized controlled trials demonstrate that physical symptoms including headaches, nausea, and GI distress, were reduced significantly after a consistent 8-week aerobic exercise program.<sup>71</sup> In this study, participants saw benefits from 20 minutes of aerobic exercise three times per week. Yoga is also an efficacious practice in reducing physical symptoms, more so than aerobic exercise, though both are effective.<sup>72</sup> A meta-analysis looking at various types, frequencies, and durations of exercises and their effects on PMS/PMDD found that exercise has a net positive effect on PMS symptoms after 8-12 weeks of regimented physical activity lasting 20-60 minutes, whether it be aerobic, resistance-training, or yoga.<sup>73</sup> It was noted that along with statistically significant changes in the physical symptoms of the exercise groups, they also experienced significant improvements in the psychological symptoms associated with PMS/PMDD as compared to the controls.<sup>73</sup> However, because information bias was present in most of the studies reviewed, this is an area in need of more robust research.<sup>73-75</sup> The American College of Sports Medicine also recommends regular exercise to prevent obesity, which can exacerbate PMS symptoms.<sup>76</sup>

Additionally, one should avoid smoking and limit alcohol consumption, particularly during the luteal phase, as both behaviors are associated with PMS/PMDD.<sup>13</sup> A meta-analysis looking at alcohol consumption found a moderate association between drinking and PMS; little to moderate drinking (0-1 drinks per day) was not associated with increased PMS severity, but heavy drinking (more than one

drink per day) was associated with increased PMS severity.<sup>77</sup> Additionally, because alcohol influence can impact eating behavior, women should be mindful of foods they consume while drinking, as discussed earlier.

Exposure to sunlight is recommended, as it may help to combat depression associated with PMDD. Additionally, exposure to sunlight early in the day can help regulate circadian rhythms by signaling the body to release melatonin. This exposure is particularly important upon awakening, as a signal to the body that the day has started. This regulation may help to combat insomnia associated with PMS/PMDD.<sup>78</sup>

## Education

The effects of PMS and PMDD greatly impede the everyday lives of women. A cross-sectional, nationwide survey demonstrated that 38% of women could not complete daily tasks due to premenstrual symptoms, and only 48% of women disclosed to the family that their inability and discomfort were due to menstrual symptoms.<sup>79</sup> The stigma and shame surrounding these issues are preventing women from seeking adequate help. This has resulted in the unfortunate consequence of cases of PMDD getting either ignored or misdiagnosed as other mental health disorders.<sup>80</sup> The self-silencing theory attests to the notion that women's health issues are often brushed off as a mere part of the process of becoming a woman; they are surrounded by secrecy and shame that prevents women from getting adequate help, seeing the disorder as part of growing up that needs to be dealt with rather than a pathology that needs to be treated. Research is ongoing into various methods to implement education for adolescent girls to provide a better understanding of the symptoms of PMS and its management.<sup>81</sup> This research can be an important step in the next generation's experience with PMDD, as it will not only give them the appropriate tools to combat the physical and emotional symptoms they will encounter during their cyclic fluctuations, but it can empower them to start the conversation, ask questions, and deepen the shallow pool of research done on premenstrual dysphoric disorder.

## CONCLUSION

PMDD and PMS are pervasive conditions that affect the physical and psychological health of premenopausal women. The lack of self-inhibition and the poor coping strategies for the psychological triggers of PMDD/PMS may affect the physiology via the gut microbiome and subsequent inflammation. Ensuring adequate micronutrients, essential fatty acids, sun exposure, and physical activity, as well as avoiding processed foods, smoking, and alcohol may improve symptoms associated with these conditions.

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## REFERENCES

1. Tortora GJ, Derrickson BH. Principles of Anatomy and Physiology. 15th ed. Hoboken, NJ: Wiley Global Education; 2016.
2. Hantsoo L, Epperson CN. Premenstrual dysphoric disorder: epidemiology and treatment. *Curr Psychiatry Rep.* 2015; 17(11):87. doi:10.1007/s11920-015-0628-3
3. Ronzio Robert. Premenstrual Syndrome (PMS) and Nutrition. Facts On File; 2017. Accessed April 18, 2022. <https://search-ebsohost-com.libproxy.bridgeport.edu/login.aspx?direct=true&db=edsrc&AN=edsrc.27457382&site=eds-live&scope=site>
4. Ryu A, Kim TH. Premenstrual syndrome: a mini review. *Maturitas.* 2015;82(4):436-440. doi:10.1016/j.maturitas.2015.08.010
5. Futterman LA, Rapkin AJ. Diagnosis of premenstrual disorders. *J Reprod Med.* 2006;51(4 Suppl):349-358.
6. Joyce KM, Good KP, Tibbo P, Brown J, Stewart SH. Addictive behaviors across the menstrual cycle: a systematic review. *Arch Womens Ment Health.* 2021;24(4):529-542. doi:10.1007/s00737-020-01094-0
7. Cobanoglu C, Karabekiroglu K, Usta MB, et al. The effects of PMS/PMDD on attention and short-term memory in adolescent girls. *Dusunen Adam: Journal of Psychiatry & Neurological Sciences* 2021;34(3):289-301. DOI: 10.14744/DAJPNS.2021.001
8. Durairaj A, Ramamurthi R. Prevalence, pattern and predictors of premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD) among college girls. *New Indian J OBGYN.* 2019;5(2):93-8. doi: 10.21276/obgyn.2019.5.2.6.
9. Reed SC, Levin FR, Evans SM. Changes in mood, cognitive performance and appetite in the late luteal and follicular phases of the menstrual cycle in women with and without PMDD (premenstrual dysphoric disorder). *Hormones and Behavior.* 2008;54(1):185-193. doi:10.1016/j.yhbeh.2008.02.018
10. Cross GB, Marley J, Miles H, Willson K. Changes in nutrient intake during the menstrual cycle of overweight women with premenstrual syndrome. *British Journal of Nutrition.* 2001;85(4):475-482. doi:10.1079/BJN2000283
11. Fernández MDM, Montes-Martínez A, Piñeiro-Lamas M, Regueira-Méndez C, Takkouche B. Tobacco consumption and premenstrual syndrome: A case-control study. *Plos One.* 2019;14(6):e0218794. doi:10.1371/journal.pone.0218794
12. Limosin F, Ades J. Aspects psychiatriques et psychologiques du syndrome prémenstruel [Psychiatric and psychological aspects of premenstrual syndrome]. *Encephale.* 2001;27(6):501-508.
13. Hashim MS, Obaideen AA, Jahrami HA, et al. Premenstrual syndrome is associated with dietary and lifestyle behaviors among university students: a cross-sectional study from Sharjah, UAE. *Nutrients.* 2019;11(8):1939. doi:10.3390/nu11081939
14. Huo L, Straub RE, Roca C, et al. Risk for premenstrual dysphoric disorder is associated with genetic variation in ESR1, the estrogen receptor alpha gene. *Biol Psychiatry.* 2007;62(8):925-933. doi:10.1016/j.biopsych.2006.12.019
15. Rapkin AJ, Akopians AL. Pathophysiology of premenstrual syndrome and premenstrual dysphoric disorder. *Menopause Int.* 2012;18(2):52-59. doi:10.1258/mi.2012.012014
16. Freeman EW, Rickels K, Sondheimer SJ, Polansky M. Differential response to antidepressants in women with premenstrual syndrome/premenstrual dysphoric disorder: a randomized controlled trial. *Arch Gen Psychiatry.* 1999;56(10):932-939. doi:10.1001/archpsyc.56.10.932
17. Jarvis CI, Lynch AM, Morin AK. Management strategies for premenstrual syndrome/premenstrual dysphoric disorder. *Ann Pharmacother.* 2008;42(7):967-978. doi:10.1345/aph.1K673
18. Dahlgren CL, Qvigstad E. Eating disorders in premenstrual dysphoric disorder: a neuroendocrinological pathway to the pathogenesis and treatment of binge eating. *J Eat Disord.* 2018;6:35. Published 2018 Oct 25. doi:10.1186/s40337-018-0222-2
19. Lang UE, Beglinger C, Schweinfurth N, Walter M, Borgwardt S. Nutritional aspects of depression. *Cell Physiol Biochem.* 2015;37(3):1029-1043. doi:10.1159/000430229
20. Martins LB, Braga Tibães JR, Sanches M, Jacka F, Berk M, Teixeira AL. Nutrition-based interventions for mood disorders. *Expert Rev Neurother.* 2021;21(3):303-315. doi:10.1080/14737175.2021.1881482
21. Dash S, Clarke G, Berk M, Jacka FN. The gut microbiome and diet in psychiatry: focus on depression. *Curr Opin Psychiatry.* 2015;28(1):1-6. doi:10.1097/YCO.0000000000000117
22. Cirillo PC, Passos RB, Bevilacqua MC, López JR, Nardi AE. Bipolar disorder and Premenstrual Syndrome or Premenstrual Dysphoric Disorder comorbidity: a systematic review. *Braz J Psychiatry.* 2012;34(4):467-479. doi:10.1016/j.rbp.2012.04.010
23. Ullah A, Long X, Mat WK, et al. Highly recurrent copy number variations in GABRB2 associated with schizophrenia and premenstrual dysphoric disorder. *Front Psychiatry.* 2020;11:572. doi:10.3389/fpsy.2020.00572
24. Seeman MV. Menstrual exacerbation of schizophrenia symptoms. *Acta Psychiatr Scand.* 2012;125(5):363-371. doi:10.1111/j.1600-0447.2011.01822.x
25. van Galen KA, Ter Horst KW, Serlie MJ. Serotonin, food intake, and obesity. *Obes Rev.* 2021;22(7):e13210. doi:10.1111/obr.13210
26. Hashim MS, Obaideen AA, Jahrami HA, et al. Premenstrual Syndrome Is Associated with Dietary and Lifestyle Behaviors among University Students: A Cross-Sectional

- Study from Sharjah, UAE. *Nutrients*. 2019;11(8):1939. Published 2019 Aug 17. doi:10.3390/nu11081939
27. McNeil J, Doucet É. Possible factors for altered energy balance across the menstrual cycle: a closer look at the severity of PMS, reward driven behaviors and leptin variations. *Eur J Obstet Gynecol Reprod Biol*. 2012;163(1):5-10.
  28. Verri A, Nappi RE, Vallero E, Galli C, Sances G, Martignoni E. Premenstrual dysphoric disorder and eating disorders. *Cephalalgia*. 1997;17 Suppl 20:25-28. doi:10.1177/0333102497017S2008
  29. Frank GK, Shott ME, DeGuzman MC. The Neurobiology of Eating Disorders. *Child Adolesc Psychiatr Clin N Am*. 2019;28(4):629-640. doi:10.1016/j.chc.2019.05.007
  30. Ceccarini MR, Tasegian A, Franzago M, et al. 5-HT2AR and BDNF gene variants in eating disorders susceptibility. *Am J Med Genet B Neuropsychiatr Genet*. 2020;183(3):155-163. doi:10.1002/ajmg.b.32771
  31. Thaler L, Gauvin L, Joobar R, et al. Methylation of BDNF in women with bulimic eating syndromes: associations with childhood abuse and borderline personality disorder. *Prog Neuropsychopharmacol Biol Psychiatry*. 2014;54:43-49. doi:10.1016/j.pnpbp.2014.04.010
  32. Marrocco J, Einhorn NR, Petty GH, et al. Epigenetic intersection of BDNF Val66Met genotype with premenstrual dysphoric disorder transcriptome in a cross-species model of estradiol add-back. *Mol Psychiatry*. 2020;25(3):572-583. doi:10.1038/s41380-018-0274-3
  33. Capuco A, Urits I, Hasoon J, et al. Current perspectives on gut microbiome dysbiosis and depression. *Adv Ther*. 2020;37(4):1328-1346. doi:10.1007/s12325-020-01272-7
  34. AlAmmar WA, Albeesh FH, Khattab RY. Food and mood: the correlative effect. *Curr Nutr Rep*. 2020;9(3):296-308. doi:10.1007/s13668-020-00331-3
  35. Loxton NJ, Dawe S, Cahill A. Does negative mood drive the urge to eat? The contribution of negative mood, exposure to food cues and eating style. *Appetite*. 2011;56(2):368-374. doi:10.1016/j.appet.2011.01.011
  36. Weiss GA, Hennet T. Mechanisms and consequences of intestinal dysbiosis. *Cell Mol Life Sci*. 2017;74(16):2959-2977. doi:10.1007/s00018-017-2509-x
  37. Lembke A. Dopamine Nation: an age of indulgence. City of Westminster, London: Penguin Publishing Group; 2021.
  38. Tomasello G, Mazzola M, Leone A, et al. Nutrition, oxidative stress and intestinal dysbiosis: influence of diet on gut microbiota in inflammatory bowel diseases. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2016;160(4):461-466. doi:10.5507/bp.2016.052
  39. Zinöcker MK, Lindseth IA. The western diet-microbiome-host interaction and its role in metabolic disease. *Nutrients*. 2018;10(3):365. doi:10.3390/nu10030365
  40. Rogers, S.A. Using organic acids to diagnose and manage recalcitrant patients. *Altern Ther Health Med*. 2006;12(4):44-53. Accessed March 2022.
  41. Lin XL, Li Z, Zuo XL, Zhonghua Fu Chan Ke Za Zhi. 2011;46(7):496-500.
  42. Gonçalves B, Ferreira C, Alves CT, Henriques M, Azeredo J, Silva S. Vulvovaginal candidiasis: epidemiology, microbiology and risk factors. *Crit Rev Microbiol*. 2016;42(6):905-927. doi:10.3109/1040841X.2015.1091805
  43. Parry BL, Rush AJ. Oral contraceptives and depressive symptomatology: biologic mechanisms. *Compr Psychiatry*. 1979;20(4):347-358. doi:10.1016/0010-440x(79)90006-3
  44. Prasad AS, Lei KY, Moghissi KS, Stryker JC, Oberleas D. Effect of oral contraceptives on nutrients. III. Vitamins B6, B12, and folic acid. *Am J Obstet Gynecol*. 1976;125(8):1063-1069. doi:10.1016/0002-9378(76)90809-7
  45. US Department of Health and Human Services and US Department of Agriculture. 2015-2020 Dietary Guidelines for Americans. <https://health.gov/our-work/nutrition-physical-activity/dietary-guidelines/previous-dietary-guidelines/2015>. Updated December 2015. Accessed March 2022.
  46. Fulgoni VL, 3rd, Keast DR, Bailey RL, Dwyer J. Foods, fortificants, and supplements: Where do Americans get their nutrients? *J Nutr*. 2011;141(10):1847-1854.
  47. Murphy, RA., Devarshi PP, Ekimura S, et al. Long Chain omega-3 fatty acid serum concentrations across life stages in the USA: an analysis of NHANES 2011-2012. *BMJ Open* 2021;11:e043301. doi: 10.1136/bmjopen-2020-043301
  48. Huffman FG, Vaccaro JA, Exebio JC, Ajabshir S, Zarini GG, Shaban LH. Relationship of omega-3 fatty acids on C-reactive protein and homocysteine in haitian and African Americans with and without Type 2 Diabetes. *J Nutr Food Sci*. 2013;3(1):180. doi:10.4172/2155-9600.1000180
  49. Kiecolt-Glaser JK, Belury MA, Andridge R, Malarkey WB, Glaser R. Omega-3 supplementation lowers inflammation and anxiety in medical students: a randomized controlled trial. *Brain Behav Immun*. 2011;25(8):1725-1734. doi:10.1016/j.bbi.2011.07.229
  50. Deacon G, Kettle C, Hayes D, Dennis C, Tucci J. Omega 3 polyunsaturated fatty acids and the treatment of depression. *Crit Rev Food Sci Nutr*. 2017;57(1):212-223. doi:10.1080/10408398.2013.876959
  51. Liao Y, Xie B, Zhang H, et al. Efficacy of omega-3 PUFAs in depression: A meta-analysis [published correction appears in *Transl Psychiatry*. 2021 Sep 7;11(1):465]. *Transl Psychiatry*. 2019;9(1):190. Published 2019 Aug 5. doi:10.1038/s41398-019-0515-5
  52. Pizzorno, J., Katzinger, J. *Clinical Pathophysiology: A Functional Perspective*. Coultam, BC. Mind Pub; 2012.
  53. Jiao L, Machuki JO, Wu Q, et al. Estrogen and calcium handling proteins: new discoveries and mechanisms in cardiovascular diseases. *Am J Physiol Heart Circ Physiol*. 2020;318(4):H820-H829. doi:10.1152/ajpheart.00734.2019
  54. Thys-Jacobs S, McMahon D, Bilezikian JP. Cyclical changes in calcium metabolism across the menstrual cycle in women with premenstrual dysphoric disorder. *J Clin Endocrinol Metab*. 2007;92(8):2952-2959. doi:10.1210/jc.2006-2726
  55. Bocchieri E, Thys-Jacobs S. Role of calcium metabolism in premenstrual syndrome. *Expert Rev Endocrinol Metab*. 2008;3(5):645-655. doi:10.1586/17446651.3.5.645
  56. Tartagni M, Cicinelli MV, Tartagni MV, et al. Vitamin D supplementation for premenstrual syndrome-related mood disorders in adolescents with severe hypovitaminosis D. *J Pediatr Adolesc Gynecol*. 2016;29(4):357-361. doi:10.1016/j.jpag.2015.12.006
  57. Bahrami A, Avan A, Sadeghnia HR, et al. High dose vitamin D supplementation can improve menstrual problems, dysmenorrhea, and premenstrual syndrome in adolescents. *Gynecol Endocrinol*. 2018;34(8):659-663. doi:10.1080/09513590.2017.1423466
  58. National Institutes of Health. Vitamin D. <https://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/>. Published 2022. Accessed April 15, 2022.
  59. Cannell JJ, Hollis BW. Use of vitamin D in clinical practice. *Altern Med Rev*. 2008;13(1):6-20.
  60. Chun RF, Hernandez I, Pereira R, et al. Differential responses to vitamin D2 and vitamin D3 are associated with variations in free 25-hydroxyvitamin D. *Endocrinology*. 2016;157(9):3420-3430. doi:10.1210/en.2016-1139
  61. Shieh A, Chun RF, Ma C, et al. Effects of high-dose vitamin D2 versus D3 on total and free 25-hydroxyvitamin D and markers of calcium balance. *J Clin Endocrinol Metab*. 2016;101(8):3070-3078. doi:10.1210/jc.2016-1871
  62. National Institutes of Health. Iron. <https://ods.od.nih.gov/factsheets/Iron-HealthProfessional/>. Published 2022. Accessed April 15, 2022.
  63. Retallick-Brown H, Blampied N, Rucklidge JJ. A pilot randomized treatment-controlled trial comparing vitamin B6 with broad-spectrum micronutrients for premenstrual syndrome. *J Altern Complement Med*. 2020;26(2):88-97. doi:10.1089/acm.2019.0305
  64. Morris MS, Picciano MF, Jacques PF, Selhub J. Plasma pyridoxal 5-phosphate in the US population: the National Health and Nutrition Examination Survey, 2003-2004. *Am J Clin Nutr*. 2008 (May); 87: 1446-54.
  65. Leklem JE, Brown RR, Rose DP, Linkswiler HM. Vitamin B6 requirements of women using oral contraceptives. *Am J Clin Nutr*. 1975;28(5):535-541. doi:10.1093/ajcn/28.5.535
  66. Palmery M, Saraceno A, Vaiarelli A, Carlomagno G. Oral contraceptives and changes in nutritional requirements. *Eur Rev Med Pharmacol Sci*. 2013;17(13):1804-1813.
  67. Nieman, David. *Nutritional Assessment*. 7th ed. New York, NY:McGraw-Hill Higher Education;2018.
  68. Linus Pauling Institute. Iron. <https://lpi.oregonstate.edu/mic/minerals/iron>. Published 2022. Accessed February 22, 2022.
  69. Nierenberg, C., 2022. Iron and zinc may prevent PMS. [online] [livescience.com](https://www.livescience.com/27486-iron-zinc-premenstrual-syndrome.html). Available at: <https://www.livescience.com/27486-iron-zinc-premenstrual-syndrome.html> [Accessed 30 January 2022].
  70. National Institutes of Health. Iron. <https://ods.od.nih.gov/factsheets/Iron-HealthProfessional/>. Published 2022. Accessed April 15, 2022.
  71. Mohebbi Dehnavi Z, Jafarnejad F, Sadeghi Goghary S. The effect of 8 weeks aerobic exercise on severity of physical symptoms of premenstrual syndrome: a clinical trial study. *BMC Womens Health*. 2018;18(1):80. Published 2018 May 31. doi:10.1186/s12905-018-0565-5
  72. Vaghela N, Mishra D, Sheth M, Dani VB. To compare the effects of aerobic exercise and yoga on Premenstrual syndrome. *J Educ Health Promot*. 2019;8:199. Published 2019 Oct 24. doi:10.4103/jehp.jehp\_50\_19
  73. Pearce E, Jolly K, Jones LL, Matthewman G, Zanganeh M, Daley A. Exercise for premenstrual syndrome: a systematic review and meta-analysis of randomized controlled trials. *BJGP Open*. 2020;4(3):bjgpopen20X101032. Published 2020 Aug 25. doi:10.3399/bjgpopen20X101032
  74. Dunn AL, Trivedi MH, O'Neal HA. Physical activity dose-response effects on outcomes of depression and anxiety. *Med Sci Sports Exerc*. 2001;33(6 Suppl):S587-610. doi:10.1097/00005768-200106001-00027
  75. Daley A. Exercise and premenstrual symptomatology: a comprehensive review. *J Womens Health (Larchmt)*. 2009;18(6):895-899. doi:10.1089/jwh.2008.1098
  76. American College of Sports Medicine. *Physical Activity Guidelines Resources*. <https://www.acsm.org/education-resources/trending-topics-resources/physical-activity-guidelines>. Published 2022. Accessed April 15, 2022.
  77. Fernández MDM, Saulyte J, Inskip HM, Takkouche B. Premenstrual syndrome and alcohol consumption: a systematic review and meta-analysis. *BMJ Open*. 2018;8(3):e019490. Published 2018 Apr 16. doi:10.1136/bmjopen-2017-019490
  78. Huberman, A. *Toolkit for Sleep*. Huberman Lab. <https://hubermanlab.com/toolkit-for-sleep/>. Published 2022. Accessed April 15, 2022.
  79. Schoep ME, Nieboer TE, van der Zanden M, Braat DDM, Nap AW. The impact of menstrual symptoms on everyday life: a survey among 42,879 women. *Am J Obstet Gynecol*. 2019;220(6):569.e1-569.e7. doi:10.1016/j.ajog.2019.02.048
  80. Emran A, Iqbal N, Dar IA. 'Silencing the self' and women's mental health problems: A narrative review. *Asian J Psychiatr*. 2020;53:102197. doi:10.1016/j.ajp.2020.102197
  81. Babapour F, Elyasi F, Yazdani-Charati J, Shahhosseini Z. A comparison between the effects of school-based education programs provided by peer group versus health practitioners on premenstrual syndrome in adolescents: A protocol for a non-masked clinical trial. *Nurs Open*. 2021;8(5):2901-2908. doi:10.1002/nop.2858
  82. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ*. 2021;372:n71. Published 2021 Mar 29. doi:10.1136/bmj.n71



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