

**PRE-MENSTRUAL DYSPHORIC DISORDER**

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**ABSTRACT**

Genetic Factors and Clinical Overview of PMS and PMDD: The pathophysiology of premenstrual problems has been linked to genetic susceptibility. In particular, the onset and progression of Premenstrual Dysphoric Disorder (PMDD) have been linked to polymorphisms in the oestrogen receptor alpha (ESR1) gene, such as the A351G variation. The presence of the GG genotype at this polymorphic site may be linked to an increased risk of PMDD, a severe mood disorder that affects women of reproductive age and is marked by significant affective lability during the luteal phase of the menstrual cycle. Variations in the amounts of ovarian steroid hormones during the luteal phase, which happens after ovulation and before menstruation, can cause behavioural, emotional, and physical symptoms in individuals who are at risk. Less than 10% of Reproductive-aged females fit the diagnostic standards for PMDD, a severe type of PMDD characterised

by noticeable mood changes, but 90% of them report having some premenstrual symptoms. The epidemiology and contemporary treatment modalities for PMS and PMDD are described in this study. It comprises a range of physical and psychological symptoms occurring during the luteal phase, typically resolving shortly after menstruation commences.

**KEYWORDS:** PMDD, PMS, SSRIs, SNRIs, GABA, OESTROGEN.

Article Received on  
04 August 2025,

Revised on 25 August 2025,  
Accepted on 15 Sept. 2025

<https://doi.org/10.5281/zenodo.17213351>



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## INTRODUCTION

Pre-menstrual Syndrome (PMS) is a syndrome associated with behavioural changes in women of reproductive age. It encompasses both emotional and physical symptoms during the luteal phase of the menstrual cycle. It is more likely to be seen in 50-80% of women. When PMS gets severe, it results in emotional breakdown, distress, social disturbances, and may require treatment, which leads to Premenstrual Dysphoric Disorder (PMDD). PMS includes somatic complaints, while PMDD is characterised by mental symptoms. The true cause of PMDD is not clear, but it is believed that it occurs due to neurological, hormonal, or psychological factors. Hormonal changes can be caused due to higher oestrogen or progesterone levels. These hormonal changes can be caused by neurotransmitter effects, causing a rise in serotonin, which is the main transmitter of mood regulation. PMDD individuals generally phase serotonin dysfunction leading to mood regulation or depression. Working women's psychological behavior, which impacts both their personal and professional lives, is also impacted by PMDD.

About a week before menstruation begins, women may experience irritability, emotional swings, headaches, anxiety, and depression. In certain women, this manifestation can escalate, causing intense suffering, disrupting family life, resulting in missed work, and tragically, contributing to violence like murder and suicide. When it comes to the body, some may experience swelling, putting on a bit of weight, sore breasts, feeling bloated, and pins and needles feelings. Treating PMDD is mainly about managing the symptoms. So, that involves taking pain relievers, medication to ease anxiety, and hypnotics for insomnia.

<b>SOMATIC (PHYSICAL) SYMPTOMS</b>	<b>AFFECTIVE AND COGNITIVE (PSYCHOLOGICAL/BEHAVIORAL) SYMPTOMS</b>
Abdominal distension	Irritability, anger, and dysregulation
Myalgia	Generalized anxiety
Mastalgia	Hyperphagia (increased appetite)
Dysmenorrhea	Altered sexual desire (libido fluctuations)
Lethargy	Impaired cognitive function (difficulty concentrating)
Cephalalgia	Depressive affect (low mood)
Nausea	Perceived loss of self-regulation

## CLINICAL MANIFESTATIONS

A variety of symptoms that negatively impact a woman's general health and quality of life are indicative of PMDD. These symptoms can be minimal and sometimes severe, affecting

normal life conditions. The mild symptoms include anxiety, stress, anger, depression, confusion, irritability, poor concentration, forgetfulness, crying easily, hypersomnia, and insomnia, among which the more common are anger, irritability. The physical symptoms include increased appetite, food cravings, muscle pain, breast tenderness, fatigue, acne, joint pain, bloating of the abdomen, headache, and swelling of the extremities. Above mentioned symptoms are mostly associated with 1 or 2 weeks before bleeding or 2 days before bleeding in PMDD women.

Women with PMDD often exhibit comorbidities, including both somatic and psychiatric disorders, highlighting the complex and multidimensional nature of the condition. This condition should not be neglected and taken into immediate action.

## **ETIOLOGY**

Major criteria created to clarify the causes of PMDD are discussed as

### **Psychosocial proposition**

According to the psychosocial thesis, PMDD or PMS is the conscious manifestation of an individual's unconscious conflict over parenting and gentility. According to a psychotherapist, premenstrual physical changes served as a reminder to people that they were not pregnant, and therefore, a social path. Demonstrating this thesis through logical proof is veritably delicate.

### **Cognitive and social literacy thesis**

The cognitive-social literacy model proposes that menarche may serve as a psychologically distressing milestone for individuals predisposed to Premenstrual Dysphoric Disorder (PMDD). Moreover, such individuals may have encountered adverse or atypical psychosocial experiences that contribute to the intensification of premenstrual symptomatology.

Accordingly, these people produce maladaptive adaptation techniques (e.g., avoidance of temperament nonappearance from school or work, and gorging) in a bid to dwindle the prompt stretch. The prompt lessening drive acts as a fortification, driving to the customary reprise of suggestion amid the premenstrual period.

### **Sociocultural thesis**

According to social and cultural theories, PMDD is a manifestation of the conflict between the double corridor society's expectations for personal growth and parenting advice. It is

hypothesised that PMDD is a social manifestation of people's dissatisfaction with traditional social routes.

### **Past traumatic occasion**

Uncomfortable, sporadic, and preexisting anxiety, fear, and risk factors for PMDD progression. The introductory instrument is obscure, making help examination necessary.

### **Cigarette smoking**

Current smoking status is strongly linked to moderate-to-severe types of premenstrual syndrome (PMS), with smokers at a greater risk than non-smokers. The chance of developing PMS increases with cigarette usage, and this elevated risk is also seen in ex-smokers. Additionally, women who started smoking throughout adolescence have a considerably greater chance of developing Premenstrual Dysphoric Disorder (PMDD).

### **The following variables may increase susceptibility to PMDD**

- A positive family history of mood disorders, such as bipolar illness or severe depressive disorder
- Recurrent premenstrual dysphoria
- Premenstrual affective lability or mood disturbances
- Personal history of sexual trauma or abuse
- Experiences of relational trauma associated with domestic or partner violence

### **PATHOPHYSIOLOGY**

Recent studies indicate that Individuals meeting Standardized symptom-based criteria for identifying Premenstrual Dysphoric Disorder (PMDD) exhibit normal patterns of gonadal hormone secretion. However, they demonstrate an increased neurobiological sensitivity to the cyclical fluctuations of reproductive hormones, particularly estrogen and progesterone, Characterized by marked affective, cognitive, and somatic manifestations occurring in the premenstrual (luteal) phase.

### **The function of sex steroids**

This theory is disputed by other expert groups, who contend that several symptoms may begin before the peri-ovulatory period and initial luteal phase preceding the decline in progesterone levels. Additional research supported this finding by demonstrating that

gonadotropin-releasing hormone treatment stopped the hormonal cycle and that symptoms were replicated by progesterone exposure even in cases where hormone levels were stable.

### **Oestrogen**

An alternative hypothesis proposes that the post-ovulatory rise in progesterone, the pre-ovulatory surge in estradiol, or a combination of both hormonal changes may contribute to the onset of symptoms in Premenstrual Dysphoric Disorder (PMDD). However, a key limitation of this theory is its inability to account for the variability in symptom onset, which may occur immediately following ovulation in some individuals, while in others, symptoms are restricted to the late luteal phase. Furthermore, evidence suggests that estrogen, like progesterone, is capable of inducing PMS-like symptoms. Notably, the estrogenic component of hormone replacement therapy has been reported to exacerbate progesterone-induced dysphoria. Premenstrual mastalgia symptoms have been found to be lessened by the use of an oestrogen antagonist during the luteal phase of the menstrual cycle. The administration of exogenous estrogen late in the menstrual cycle, during the secretory phase, has been shown to provoke or worsen symptoms of premenstrual breast pain. These findings underscore the complex interplay between the roles of estrogen and progesterone in the underlying mechanisms of PMDD.

### **Testosterone**

When Testosterone therapy starts, transgender men show improvements in interpersonal relationships, sociability, and reduced depression and anxiety.

Role of Central Neurotransmitters

### **Serotonin**

One major neurotransmitter that has been shown to play a role in mood and behaviour regulation is serotonin. Through their effects on serotonergic transmission, sex steroids may have an impact on behavior. This theory is validated by three proofs. First, serotonin-releasing drugs and other pharmacological treatments that elevate serotonin concentrations, such as SSRIs, reduce premenstrual symptoms. Secondly, contrary to the first statement, a decrease in serotonergic transmission caused by serotonin-receptor antagonist medication or a tryptophan-free diet may result in PMS/PMDD symptoms. Their follicular serotonergic reactivity is also higher than the luteal phase, which is different from what is seen in women who do not have PMS or PMD.

Mood and Behaviour regulations are controlled by serotonin, the central neurotransmitter. Serotonergic transmission gets affected by exerting effects of sex steroids on behaviour.

### **Gamma-amino-butyric acid**

The action of progesterone metabolites, which exert their effects by positively modulating the GABA-A receptor at allosteric binding sites has been associated with altered sensitivity of the receptor complex in symptomatic women as compared to asymptomatic individuals. Gama-aminobutyric acid (GABA) is the central nervous systems main inhibitory neurotransmitter. Its action of slowing down neural signals is likely a key aspect of how the brains neurobiology operates premenstrual dysphoric disorder (PMDD), according to neuroimaging research. Given the deep functional connections between GABAergic and serotonergic neurones, the GABAergic system's involvement is consistent with the serotonin hypothesis. Furthermore, it has been demonstrated that serotonin reuptake inhibitors (SRIs) affect the enzymatic pathways that produce neuroactive progesterone metabolites, which in turn affect GABA-A receptor activity indirectly.

### **Glutamate**

There is an excitatory neurotransmitter called Glutamate present in the body during the menstrual cycle. There is a fluctuation that is cyclic it occurring in women (symptomatic and asymptomatic), with symptomatic women who can heighten sensitivity to these cyclical changes.

### **Beta Endorphins**

Throughout the follicular and luteal stages of the menstrual cycle, those with PMDD show lower levels of beta-endorphins and cortisol. Due to this, it results in abnormality in the hypothalamic-pituitary-gonadal axis in PMDD, causing mood disorders.

## **EPIDEMIOLOGY**

All individuals assigned female at birth who are within their reproductive years are vulnerable to experiencing premenstrual symptoms, which fluctuate throughout the luteal phase, from menarche until menopause. Approximately 3 to 8% are diagnosed with Premenstrual Dysphoric Disorder (PMDD), which is a serious disorder where intense, debilitating symptoms lead to a substantial decrease in quality of life.

Research shows that roughly half of all women experience symptoms that are both mild and temporary. This lack of clear, persistent symptoms often makes it difficult to get a formal diagnosis for a specific menstrual condition. According to Halbreich et al., the average American woman undergoes approximately 481 menstrual cycles throughout her lifetime. For those with PMDD, the severe symptoms can last an average of 6.4 days per cycle. This number is even more significant when considering that they also have to account for periods of pregnancy and postpartum. These findings underscore the critical importance of addressing PMS and PMDD within women's healthcare due to their profound impact on daily functioning and long-term well-being.

## DIAGNOSIS

A diagnosis of Premenstrual Dysphoric Disorder is supported when multiple characteristic symptoms emerge during the final week of the luteal phase and remit completely within several days following the onset of menstruation. Notably, at least one of these must involve a prominent affective disturbance.

- Marked liability must be present, like mood swings, suddenly feeling low, etc.
- Increase in interpersonal conflicts, anger, and irritation.
- Anxiety, tension
- Self-doubt thoughts, feelings of hopelessness, feeling unacceptable
- Showing a lack of interest in day-to-day activities like work, friends, school, and hobbies
- Food cravings or heavy eating
- Lack of concentration
- Appetite disturbances
- Hypersomnia or insomnia
- Exhaustion and weakness, Lack of energy
- It also includes physical symptoms like sore breasts, bloating, muscle pain and joint pain, inflammation, and weight gain.

## TREATMENT

PMDD treatment is mainly used for psychiatric and physical symptoms. The treatments mainly suppress the body's hormonal activity by affecting the brain's concentration of key neurotransmitters, including dopamine, serotonin, and norepinephrine. It also suppresses ovulation. For primary treatment, SSRIs are used.



Therapeutic strategies for Premenstrual Dysphoric Disorder (PMDD) often focus on two primary neuroendocrine targets: (1) suppression of hypothalamic–pituitary–ovarian (HPO) axis activity to stabilize fluctuations in gonadal hormone levels, and (2) modulation of central serotonergic neurotransmission. To disrupt the hormonal signals of the Hypothalamic–Pituitary–Ovarian (HPO) axis, doctors sometimes use medications known as gonadotropin-releasing hormone (GnRH) analogs, transdermal estradiol, and combined oral contraceptives (COCs), which function to attenuate cyclical ovarian hormone variation. Simultaneously, pharmacological interventions—such as selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs)—enhance serotonergic activity and have shown clinical effectiveness in reducing the severity of PMDD symptoms. Given the episodic and cyclical nature of PMDD, treatment approaches must account for the periodicity of symptom recurrence. Both continuous and intermittent (luteal phase-only) treatment regimens are employed, each with distinct implications for patient adherence, tolerability, and long-term safety. The optimal therapeutic approach may vary depending on individual symptom patterns, response to treatment, and risk–benefit considerations over prolonged use.

## 1. Pharmacological

### • Selective Serotonin Reuptake Inhibitors (SSRIs)

Selective serotonin reuptake inhibitors (SSRIs) are widely recognized as the primary pharmacological treatment for Premenstrual Dysphoric Disorder (PMDD), supported by substantial evidence from randomized controlled trials. These medications can be prescribed either continuously throughout the entire menstrual cycle or intermittently, specifically targeting the luteal phase. All dosages have demonstrated efficacy in ameliorating affective symptoms, with dose titration guided by patient tolerability. Higher doses may be necessary for the management of somatic symptoms.

Bupropion (Wellbutrin), a norepinephrine-dopamine reuptake inhibitor (NDRI), has not demonstrated efficacy in the treatment of PMS or PMDD. An open-label trial of sertraline in PMDD reported increased peripheral allopregnanolone concentrations, though the magnitude of change varied depending on baseline levels. It is postulated that SSRIs may influence neurosteroidogenesis, specifically the synthesis of allopregnanolone, which modulates GABA-A receptor activity. Serotonin-norepinephrine reuptake inhibitors (SNRIs), such as venlafaxine, have been used off-label in patients with predominantly psychological PMDD symptoms. Clinical improvement is typically observed within 3–4 weeks and persists across



subsequent menstrual cycles. SNRIs are considered second-line agents when SSRIs are contraindicated or not tolerated; however, further placebo-controlled studies are required to establish their efficacy. Continuous or luteal-phase administration of SSRIs, including sertraline and fluoxetine, has also shown effectiveness in the treatment of severe premenstrual syndrome (PMS).

### **Estrogen-progestin pill**

There is a wealth of evidence supporting the effectiveness of combination oral contraceptives (COCs) in treating menstrual cycle somatic symptoms, such as menorrhagia, dysmenorrhea, and gastrointestinal abnormalities. Nevertheless, there has been conflicting evidence regarding how COCs impact emotional premenstrual symptoms. The availability of various hormone combinations, as well as variations in dosage, usage, and timing, further complicates the data. Researchers reviewed four moderate-quality clinical trials involving Participants who recorded their symptoms using the DRSP during continuous administration of an oral combined hormonal contraceptive preparation comprising levonorgestrel 90 µg (a progestin) and ethinyl estradiol 20 µg (a synthetic estrogen), typically administered in a continuous or extended-cycle regimen. This formulation is designed to inhibit ovulation, alter the endometrial lining, and increase cervical mucus viscosity, thereby providing effective pregnancy prevention and potential menstrual cycle regulation. Despite significant discrepancies in the results, there was a noticeable improvement in both physical and depressive symptoms (from 30% to 59%).

- **Anxiolytics**

Benzodiazepines have been used therapeutically to treat PMDD for a long time; however, the research evaluating their effectiveness is limited and clinically outdated. Alprazolam was not shown to provide any advantages above a placebo in two investigations, including women with PMS.<sup>[82,83]</sup>

Overall, the evidence supporting the use of benzodiazepines as an adjuvant treatment for PMDD is minimal, and they should only be evaluated in refractory instances, even if they could offer therapeutic value during symptomatic episodes in patients absent of concurrent illnesses or follicular phase manifestations.

### Non-pharmacological methods

- **Exercise:** It enhances characteristics through an increase in endogenous morphinomimetic peptides levels. PMS symptoms in women can be reduced with sedentary exercise. One study suggests that aerobic exercise can be beneficial.
- **Cognitive Behavior Therapy:** This form of psychotherapy aims to modify maladaptive cognitions, emotions, and behaviors. This treatment is considered a potential option for managing PMS because it has a proven track record of effectively treating other emotional and physical symptoms, such as anxiety and chronic pain.
- **Dietary Modification:** It is thought that consuming more complex proteins or carbs ("slow-burning fuels") raises tryptophan availability, which in turn raises serotonin levels. A meta-analysis has shown that vitamin B6 has certain advantages over a placebo. By having a dopaminergic impact, chaperberry, also known as *Vitex agnus-castus*, a phytotherapeutic agent, has shown moderate effectiveness in alleviating symptoms of Premenstrual Syndrome (PMS), particularly mastalgia and mood-related symptoms, with evidence supported by randomized controlled trials. Its mechanism is thought to involve dopaminergic modulation of prolactin secretion.
- **Stress Management:** Breathing exercises, yoga, meditation, and relaxation.

### Comorbidities and Complications

Significant difficulties from PMDD might affect daily functioning, relationships, and mental health. If left untreated, PMDD can exacerbate pre-existing Mental health disorders, including anxiety and major depressive symptoms, that can escalate to suicidal thoughts or attempts in extreme instances. Other frequent side effects include relationship stress, difficulties at work and school, and a general decline in quality of life.

**Mental Wellness:** Exacerbation of pre-existing conditions: Depression, anxiety, and other mental health illnesses can be made worse by PMDD.

**Increasing risk of suicide:** Suicidal thoughts and attempts are linked to severe PMDD.

**Relationship strain:** Relationships with partners, family, and friends might suffer as a result of irritability, mood swings, and emotional instability.

**Work and Academic Performance:** Difficulty functioning: Symptoms of PMDD may make it difficult to focus, be productive, or fulfill obligations at work or school.

**Physical well-being:** Debilitating physical symptoms: Although PMDD is predominantly a mood disease, it can also result in debilitating Somatic manifestations, including arthralgia, abdominal bloating, mastalgia, and headaches.

**Increased Healthcare Utilisation:** additional medication and doctor visits. Women with PMDD may need to take medication to control their symptoms and seek out additional medical treatment.

**Effect on Life Quality:** Diminished general well-being: A woman's capacity to enjoy hobbies, interact with others, and carry out everyday tasks can all be adversely affected by PMDD, which may significantly diminish her overall quality of life.

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