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The influence of trauma on the relationship between perceived stress and pre-menstrual symptoms

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1. Introduction and Theoretical Framework

The week preceding menstrual onset, characterized by substantial shifts in reproductive hormone levels, is considered a period particularly vulnerable to affective and physiological distress for most females[1], as they experience at least one premenstrual symptom at some point throughout their reproductive years (American College of Obstetricians and Gynecologists [ACOG], 2001; Grewal et al., 2025). The premenstrual syndrome (PMS) consists of a variety of affective and physical symptoms arising in the week before menstrual onset, namely the late-luteal phase, and remitting within the first four days of menstruation. PMS affects approximately 20–30%, whereas premenstrual dysphoric disorder (PMDD), the most severe manifestation of premenstrual symptomatology, occurs in about 3-8% of the menstruating population (Dennerstein et al., 2012; Reilly et al., 2024a). PMDD is formally recognized in both major diagnostic systems, the American Psychiatric Association's (APA) *Diagnostic and Statistical Manual of Mental Disorders* (5th edition; DSM-5) and the World Health Organization's (WHO) *International Classification of Diseases* (11th revision; ICD-11) underscoring its clinical relevance. While the DSM-5 classifies PMDD as a depressive disorder, the ICD-11 lists it under gynecological conditions with a cross-reference to depressive disorders (APA, 2013; WHO, 2019).

According to DSM-5 criteria, PMDD is characterized by the presence of at least five symptoms during the late luteal phase, drawn from a set of affective, cognitive, behavioral, and physical domains. These include core affective symptoms such as mood lability, irritability, depressed mood, or anxiety, of which at least one must be present, alongside symptoms such as decreased interest, concentration difficulties, fatigue, appetite changes and cravings (potentially coupled with overeating), sleep disturbances, feelings of being overwhelmed, and somatic complaints (e.g., breast tenderness or bloating). Importantly, symptoms must occur cyclically across most menstrual cycles in the past year, and their burden must be associated with impairment in occupational, academic, or social functioning (APA, 2013).

In contrast, PMS, as defined by ACOG (2001), has a substantially lower diagnostic threshold, requiring only one distressing symptom that interferes with daily functioning.

[1] Throughout this thesis, the term "female" is used in a limited biological sense to refer to individuals with reproductive anatomy compatible with a natural menstrual cycle, particularly ovarian function and cyclic hormonal fluctuations. This terminology does not imply that menstrual cycling corresponds to gender identity or that all females menstruate. Although the larger research project aimed to include menstruating individuals regardless of gender identity, non-cis participants are not represented in the present sample.

Importantly, the diagnosis of PMS and PMDD requires careful differential diagnostic assessment, meaning that alternative disorders with overlapping symptoms must be considered and ruled out[2] (Dickerson et al., 2003).

Despite their high prevalence, functional impairment, and clinical relevance, which highlight the need for a better understanding of their etiology and for effective, tailored treatment approaches, premenstrual symptoms historically received little scientific attention before the 1970s (McGregor et al., 2013). Publication activity remained low for several decades and increased markedly only in more recent years, as indicated by an exploratory PubMed search[3].This delayed research focus reflects broader patterns in which gendered health phenomena have historically been underrepresented in biomedical and psychological research and sociocultural norms and stigma that discourage the articulation of menstruation-related experiences and contribute to the trivialization and underestimation of associated symptoms (Hennegan et al., 2021; McGregor et al., 2013). Importantly, these symptoms should not be interpreted as indicators of general emotional instability. Rather, they occur within specific phases of the menstrual cycle and are believed to reflect increased sensitivity to normal cyclical changes in ovarian hormones and neuroactive steroids. They are also influenced by stress-related processes that may be critically affected by trauma exposure and trauma-related changes in physiological and psychological regulation (Bencker, Tran, et al., 2025; Hantsoo et al., 2023; Schweizer-Schubert et al., 2021). Against this background, the present thesis examines whether trauma-related factors influence the association between perceived stress and premenstrual symptom severity. To establish this framework, the following sections first outline menstrual cycle regulation and etiological approaches to PMS/PMDD before turning to stress, trauma, and their potential interaction in premenstrual symptom expression.

1.1. Menstrual Cycle and Hormonal Regulation

The menstrual cycle is a recurrent physiological process preparing the female body for a potential pregnancy and thereby ensures human reproduction (ACOG, 2025). The menstrual cycle is characterized by hormonal fluctuations that regulate internal processes but

[2] Differential diagnoses include affective, personality or eating disorders, anemia, chronic medical conditions, dysmenorrhea, endometriosis, hypothyroidism, perimenopause and substance use disorders. Oral contraceptive use should be considered separately, since PMDD cannot be diagnosed when symptoms are attributable to hormonal contraception (Dickerson et al., 2003).

[3] Search string (May 2026): "premenstrual symptoms" OR "premenstrual syndrome" OR “PMDD” OR “PMS” OR “premenstrual tension syndrome” NOT “progressive multiple sclerosis”

may also cause a range of symptoms in sensitive individuals (Schmalenberger et al., 2021). Throughout their reproductive years, from the first menstrual onset (menarche) to menopause, most females experience the menstrual cycle and its effects.

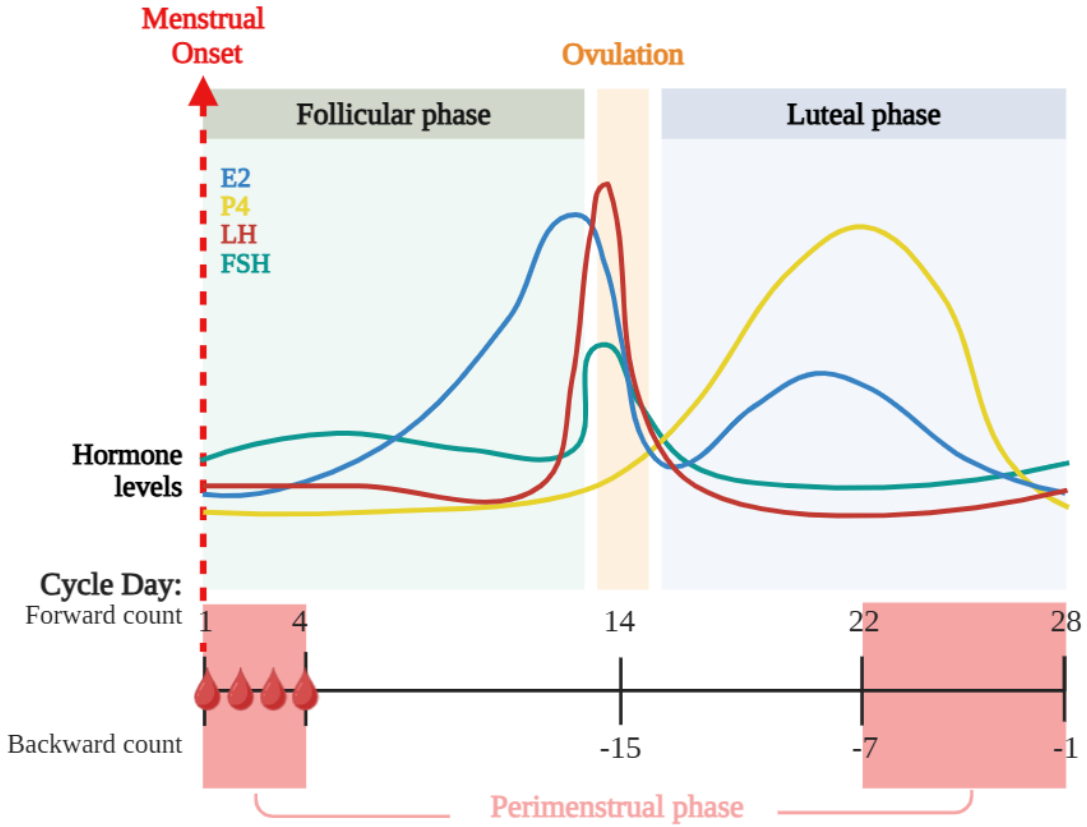
The menstrual cycle is a complex biological rhythm characterized by coordinated fluctuations of the gonadal hormones, particularly estrogens such as estradiol (E2) and progestogens, mainly progesterone (P4) (Schmalenberger et al., 2021). The menstrual cycle comes in a recurring pattern with an average cycle length of approximately 28 days (ACOG, 2025; Stiefel et al., 2020), although both inter- and intraindividual variation are common, ranging from 21 to 37 days (Schmalenberger et al., 2021).

The menstrual cycle ensures reproductive function by enabling ovulation and cyclic endometrial renewal (ACOG, 2025). It is commonly divided into two main phases: the follicular phase, which begins with the onset of menstrual bleeding and extends until ovulation, and the luteal phase, which follows ovulation and lasts until the onset of the next menstruation (Schmalenberger et al., 2021). During the follicular phase, a dominant follicle matures while E2 levels gradually increase, reaching a pre-ovulatory peak, followed by a rapid drop. Ovulation is triggered by a surge in luteinizing hormone (LH), accompanied by a rise in follicle-stimulating hormone (FSH), leading to the release of the oocyte from the ovary (Schmalenberger et al., 2021; Stiefel et al., 2020). The subsequent luteal phase is characterized by a rise in P4 levels, alongside moderate elevations in E2, as the endometrium prepares for potential implantation. If pregnancy does not occur, after the mid-luteal P4 peak both hormone levels decline toward the end of the luteal phase, initiating menstruation, which is the shedding of the endometrium (ACOG, 2025; Schmalenberger et al., 2021) (see Figure 1).

The cyclic hormonal changes are regulated by the hypothalamic–pituitary–gonadal (HPG) axis (Schweizer-Schubert et al., 2021). Gonadotropin-releasing hormone (GnRH) secreted by the hypothalamus stimulates the anterior pituitary to release LH and FSH, which in turn act on the ovaries to regulate E2 and P4 production (Stiefel et al., 2020). These hormones feed back to the central nervous system via negative and positive feedback mechanisms, creating the rhythmic pattern of hormonal fluctuations characteristic of the menstrual cycle (Schweizer-Schubert et al., 2021) (see Figure 2).

Figure 1

Hormone Levels Across the Menstrual Cycle and Definition of the Perimenstrual Phase



Note. E2 = estradiol; FSH = follicle-stimulating hormone; LH = luteinizing hormone; P4 = progesterone. The figure illustrates relative changes in estradiol, progesterone, luteinizing hormone, and follicle-stimulating hormone across a standardized 28-day menstrual cycle. Ovulation occurs around day 14 and separates the follicular and luteal phases. Cycle days are displayed using both forward counting (days since last menstrual onset) and backward counting (days until next menstrual onset). The perimenstrual phase is defined as −7 to +4 days relative to menstrual onset. *Adapted from Clayton (2026)*. Created with https://BioRender.com.

1.2. Etiology, Hormone Sensitivity and Neurobiological Mechanisms

Clinically, PMS and PMDD symptoms are characterized by cyclic recurrence, cluster around the luteal phase and typically remit shortly after the onset of menstruation (ACOG, 2001; APA, 2013; Dickerson et al., 2003; Schmalenberger et al., 2021). This symptom pattern illustrates that menstrual-cycle-related endocrine processes are closely linked to psychological processes.

The luteal timing of these symptoms aligns with the rapid withdrawal of E2 and P4, as well as changes in their neuroactive metabolites, particularly allopregnanolone (ALLO) (Hantsoo & Epperson, 2020). Importantly, absolute hormone levels in individuals with PMS or PMDD are generally within the normal range (Schweizer-Schubert et al., 2021). The hormone sensitivity hypothesis serves as an explanatory basis: Rather than reflecting hormonal abnormalities, these disorders appear to involve an increased sensitivity to otherwise normative hormonal fluctuations, likely mediated by altered central nervous system responses to these hormonal changes (ACOG, 2001; Bencker, Tran, et al., 2025; Dickerson et al., 2003; Peters et al., 2025; Schmidt et al., 2017a; Schweizer-Schubert et al., 2021; Yonkers et al., 2008). Sex steroid hormones exert widespread effects on the central nervous system by modulating brain function and structure (e.g. synaptic plasticity and neurotransmitter systems) (Barth et al., 2023). Accordingly, substantial evidence indicates that fluctuations in E2 and P4 play an important role in brain functioning and mood regulation, as reflected in the markedly increased risk for depressive disorders when females enter their reproductive years, being approximately two times higher than for men, and decreasing again after the perimenopausal transition (Barth et al., 2015; Kundakovic & Rocks, 2022). As mentioned before, the HPG axis is responsible for ovarian hormone secretion through GnRH, LH, and FSH, leading to cyclical fluctuations in E2 and P4 (Stiefel et al., 2020). P4 is postovulatory metabolized to ALLO, a neuroactive steroid that enhances Gamma-aminobutyric acid (GABA)-ergic inhibition acting at the GABA-A receptors, leading to sedative effects comparable to those of benzodiazepines (Hantsoo & Epperson, 2020). Under physiological conditions, E2 tends to reduce GABAergic inhibition and facilitate excitatory neurotransmission, whereas P4 and ALLO potentiate GABAergic inhibition (Barth et al., 2015). This balance is crucial for stable mood regulation and stress damping. In PMS/PMDD, however, this modulatory capacity appears unstable. It has been proposed that GABA-A receptors may not adapt sufficiently to cyclical changes in ALLO levels, such that ALLO no longer reliably exerts its usually calming effects but may instead coincide with dysphoria, anxiety, irritability, and emotional lability in hormone-sensitive individuals (Bencker, Gschwandtner, et al., 2025; Eisenlohr-Moul et al., 2016). Consistent with this interpretation, blocking P4 metabolism to ALLO or blocking ALLO binding at the GABA-A receptor (Schweizer-Schubert et al., 2021), as well as stabilizing ALLO levels, was associated with reduced symptom expression (Lee & Im, 2016).

Supporting evidence for the hormone sensitivity hypothesis comes from treatment studies. Suppression of ovarian steroids using GnRH agonists (Schmidt et al., 1998) as well

as stabilizing ovarian steroid concentrations by a subsequent add-back of E2 and P4 , has been shown to reduce symptoms in many affected individuals by eliminating the dynamic changes in reproductive hormone levels (Schmidt et al., 2017b). Complementing evidence on the effects of P4 in susceptible individuals, a P4 antagonist, which does not affect levels of E2, with its considered (anxiety and neuro-) protective or antidepressant effects (Eisenlohr-Moul et al., 2016; Yonkers et al., 2008), has been proposed as a more targeted pharmacological approach and has confirmatively demonstrated its significant reduction of psychological symptoms of PMDD in a randomized placebo-controlled trial (Comasco et al., 2021). This underscores that symptom expression in PMS/PMDD may be triggered by fluctuating physiological hormone levels (Dickerson et al., 2003; Schweizer-Schubert et al., 2021; Yonkers et al., 2008).

Interactions with central nervous system neurotransmitters are indicated: Given that depressed mood is a core symptom in PMDD, serotonergic involvement appears likely. During the luteal phase, when E2 levels decline, women with PMDD often experience low mood, food cravings, impulsivity, and cognitive difficulties, all of which are related to reduced serotonergic activity (Barth et al., 2015; Hantsoo & Epperson, 2015). In line with this, selective serotonin reuptake inhibitors (SSRIs) have shown robust efficacy in PMS/PMDD (Carlini & Deligiannidis, 2020; Shah et al., 2008), suggesting a role of serotonergic dysregulation (ACOG, 2001; Eriksson, 1995). Beyond their serotonergic effects, SSRIs may stabilize GABA-A receptor functioning by improving receptor plasticity (Schweizer-Schubert et al., 2021), thereby potentially reducing dysphoric mood responses to cyclical neurosteroid fluctuations. However, these mechanisms do not fully explain the disorder, as not all patients respond to SSRI treatment (Dickerson et al., 2003).

Genetic factors further contribute to vulnerability, as twin studies suggest a heritable component of PMDD (Kendler et al., 1998). In addition to genetic predisposition and epigenetic influences, other risk factors such as high body mass index (Masho et al., 2005), stress (Bencker, Tran, et al., 2025; Gollenberg et al., 2010), and (especially childhood or adolescent) traumatic experiences (Nayman, Schricker, et al., 2023; Perkonigg et al., 2004) have been associated with increased risk for PMDD.

These risk factors point beyond purely hormonal explanations and suggest that stress-related processes may be central to understanding individual differences in premenstrual symptom severity.

1.3. Stress and Reproductive Endocrine Regulation

As Lazarus (1999) argues, one of the pioneers in cognitive emotion theories, stress is neither a mere stimulus nor solely a physiological reaction, but a relational process between the individual and the environment. What determines whether a situation becomes stressful is not its objective severity, but its perceived significance, whether it involves harm, loss, threat, or challenge. This process of cognitive appraisal entails an evaluation of both situational demands and available coping resources. Stress arises when perceived demands exceed one's coping capacity (Lazarus, 1999). Novelty, unpredictability, threat, and loss of control may be crucial factors in the appraisal of the stressor (Guilliams & Edwards, 2010).

Stress, however, is not merely an individual psychological state but a socially and structurally embedded experience (McEwen, 2007). Importantly, exposure to stressors and traumatic experiences is not evenly distributed across gender, nor does it follow a simple pattern of uniformly higher exposure among women across all event categories. Rather, recent representative data from German-speaking countries indicate gendered patterns in the type, relational context, severity, and consequences of violence exposure (*Bundeskriminalamt*, n.d.): women are particularly affected by sexualized violence, sexual harassment, and stalking, and they report more severe consequences of violence in intimate relationships. These patterns are further shaped by age, with younger people being more frequently affected, as well as by migration background and LGBTQIA+ status. In addition, gendered role expectations and caregiving responsibilities structure everyday stress, as women continue to perform substantially more unpaid care and household work than men (*Zeitverwendung*, n.d.). Thus, gendered socialization processes, role expectations, caregiving demands, and differential exposure to interpersonal and traumatic stressors may shape not only the frequency of stress, but also its appraisal, regulation, and physiological processing in everyday life (Taylor et al., 2000).

At the biological level, stress results in the activation of the hypothalamic-pituitary-adrenal (HPA) axis (Hantsoo et al., 2023; McEwen, 2007). This process begins with the secretion of corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP) from the paraventricular nucleus of the hypothalamus shortly after a stressor occurs (Hantsoo et al., 2023). This cascade stimulates the release of adrenocorticotropic hormones (ACTH) from the pituitary gland, which then travels to the adrenal glands. This ultimately leads to the secretion of corticosteroids such as cortisol, which act on glucocorticoid receptors. This adaptive response prepares the body to cope with stress by ensuring sufficient glucose availability for the muscles and by pausing energy-consuming processes (e.g. within the immune system),

thereby enabling a fight-or-flight reaction (Hassamal, 2023; Lei et al., 2025; McEwen, 2007). The HPA axis follows a rhythmic circadian cycle, making it predictable (Guilliams & Edwards, 2010), and a negative feedback loop is integrated into this system to prevent long-term physiological burden: when cortisol levels become too high, ACTH secretion is reduced, which in turn decreases corticosteroid production and eventually inhibits further CRH release in the hypothalamus (Hantsoo et al., 2023; Schweizer-Schubert et al., 2021) (see Figure 2).

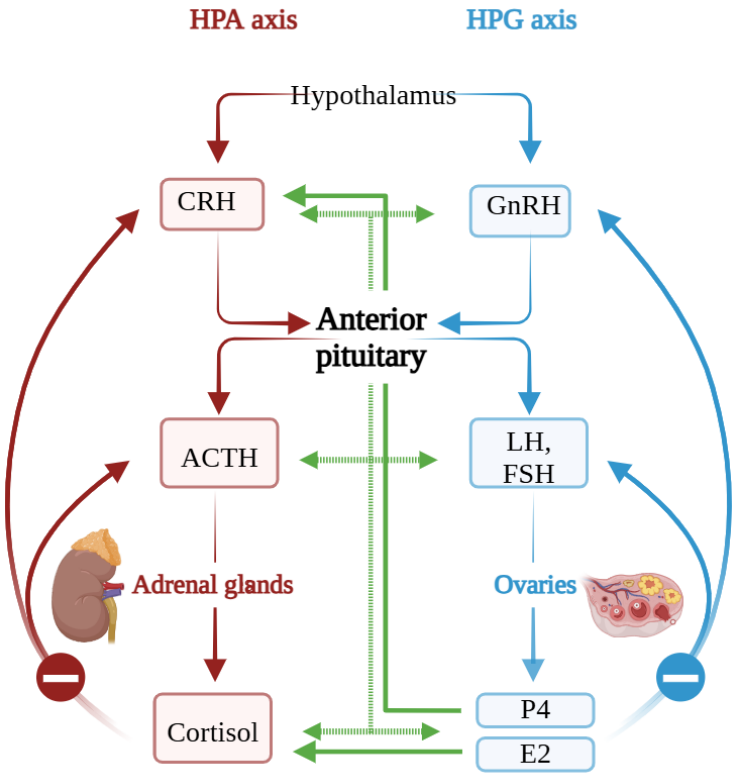
McEwen et al (2015) and McEwen and Akil (2020) reconceptualize stress not merely as a disruption of homeostasis, but as a dynamic process of allostasis, referring to the active regulation through which the brain and body maintain stability. Stress mediators such as glucocorticoids operate in a nonlinear manner and often follow an inverted U-shaped function: moderate and time-limited activation promotes adaptation and effective coping, whereas insufficient activation or prolonged, excessive activation may become maladaptive. When stress becomes chronic, and responses are repeatedly engaged or insufficiently terminated, the cumulative physiological burden is described as allostatic load, which can develop into allostatic overload if regulatory systems lose flexibility. Central to this framework is resilience, understood not as a static trait but as an active process of ongoing neuroplastic adaptation. The brain continuously remodels its structure and function through genomic, nongenomic, and epigenetic mechanisms in response to experience. Recovery from stress-induced changes, therefore, represents not a simple reversal but a recalibration of neural systems. In this framework, whether stress remains adaptive or becomes pathogenic depends not only on the presence of a stressor, but also on its duration, timing (particularly vulnerable during sensitive developmental periods), perceived intensity, individual susceptibility and resilience, and the flexibility of regulatory systems (McEwen et al., 2015; McEwen & Akil, 2020).

To bring it all together, the bidirectional coupling between the HPA and HPG axes (Hantsoo et al., 2023; Hantsoo & Epperson, 2015) (see Figure 2) provides a mechanistic, explanatory framework and suggests a dysregulated HPA-HPG interaction in susceptible individuals (Hantsoo & Epperson, 2020). Under physiological conditions, ovarian steroids modulate stress reactivity: E2 dampens HPA-driven cortisol responses, while P4-derived ALLO enhances GABAergic inhibition and thereby dampens hypothalamic CRH release, resulting in lowered cortisol secretion to re-establish physiological stability (Hantsoo et al., 2023; Hantsoo & Epperson, 2020; Nayman et al., 2024). Conversely, HPA activation suppresses the HPG axis via decreased GnRH signaling, potentially altering cycle dynamics (Eisenlohr-Moul et al., 2016; Schweizer-Schubert et al., 2021). This may be particularly

relevant under prolonged hypercortisolism, which has been associated with reduced availability of ovarian hormones such as E2, which is important for mental stability because of its neuroprotective and anxiolytic properties (Guilliams & Edwards, 2010). Chronic stress may shift neural networks toward hyperexcitability (Hassamal, 2023), further contributing to symptom vulnerability by compromising GABAergic regulation of the HPA axis (Schweizer-Schubert et al., 2021). Rather than representing independent processes, stress and premenstrual symptomatology appear embedded within a coupled neuroendocrine system in which bidirectional feedback loops may shape vulnerability and symptom expression (Hantsoo et al., 2023; Schweizer-Schubert et al., 2021)

Figure 2

HPA and HPG Axis Functioning and Their Interaction



Note. ACTH = adrenocorticotropic hormone; CRH = corticotropin-releasing hormone; E2 = estradiol; FSH = follicle-stimulating hormone; GnRH = gonadotropin-releasing hormone; HPA = hypothalamic-pituitary-adrenal; HPG = hypothalamic-pituitary-gonadal; LH = luteinizing hormone; P4 = progesterone. The hypothalamic-pituitary-adrenal axis is shown in red, and the hypothalamic-pituitary-gonadal axis is shown in blue. Bidirectional regulatory interactions between both systems are indicated by green arrows. Solid green arrows represent direct dampening or inhibitory feedback effects, whereas dashed green arrows

indicate broader or indirect regulatory interactions between the axes. Gonadal steroids, including estradiol and progesterone, and glucocorticoids, including cortisol, exert regulatory effects within and across axes. *Adapted from Schweizer-Schubert et al. (2021)*. Created with https://BioRender.com.

Since stress responsivity may be one underlying, maintaining, and reinforcing mechanism of PMS/PMDD (Hantsoo et al., 2023), its impact may be amplified by heightened hormone sensitivity in susceptible individuals, resulting in a dysregulated HPA-HPA coordination and potentially strengthening premenstrual symptom responses (Eisenlohr-Moul et al., 2016; Hantsoo et al., 2023; Hantsoo & Epperson, 2015; Nayman, Schricker, et al., 2023; Schweizer-Schubert et al., 2021).

This neuroendocrine perspective is supported by findings of altered HPA-axis functioning in affected individuals, including reduced basal cortisol, delayed cortisol awakening responses, and blunted stress reactivity, patterns that have been associated with more severe PMDD (Beddig et al., 2019; Bencker, Tran, et al., 2025; Hou et al., 2019; Nayman et al., 2024). Consistent with this interpretation, research has shown that among women with affective premenstrual symptoms, the degree of dysregulation in HPA responses to a standardized psychosocial stressor predicts greater symptom severity (Eisenlohr-Moul et al., 2016). Furthermore, typical cyclical cortisol shifts, such as late-luteal reductions that may occur in response to rising ALLO levels, appear to be attenuated in premenstrual disorders, suggesting a loss of physiological rhythmicity (Beddig et al., 2019; Nayman et al., 2024). Therefore, altered cortisol levels and diminished cortisol cyclicity across the menstrual cycle have been proposed as potential endocrinological markers of PMDD and may contribute to altered emotion processing and a more severe clinical symptom course (Nayman et al., 2024). However, evidence regarding absolute cortisol abnormalities in PMS/PMDD remains inconsistent (Schweizer-Schubert et al., 2021).

Also suggesting a robust association between stress and the severity of premenstrual symptoms, the recent meta-analysis of Bencker, Tran et al. (2025) reported a significant moderate correlation between perceived stress and premenstrual symptom severity ($r = 0.29$), indicating that higher stress levels are reliably associated with more pronounced symptom expression. Longitudinal data further support this relationship: Women experiencing elevated stress in one cycle were more likely to report an increased number and severity of perimenstrual symptoms in the subsequent cycle, and changes in stress levels were mirrored by changes in symptom severity (Gollenberg et al., 2010).

Importantly, the directionality of this association remains unresolved: Stress may exacerbate premenstrual symptoms, but symptoms themselves may likewise increase stress reactivity (Bencker, Tran, et al., 2025; Gollenberg et al., 2010). Moreover, higher within-person levels of premenstrual symptoms have been shown to predict increased perceived stress and daily rumination, which in turn are associated with further mood deterioration, suggesting a reciprocal reinforcement process that may maintain or amplify symptom severity over time (Nayman, Konstantinow, et al., 2023).

Several factors affect the interplay of HPA and HPG axis, potentially explaining the variability of premenstrual symptom occurrence as well as severity within and between individuals. These include chronic stress exposure, genetic predispositions, and severe stressors (Dickerson et al., 2003; McEwen et al., 2015; McEwen & Akil, 2020; Yonkers et al., 2008). In particular, traumatic experiences represent a form of severe stress that may alter stress responsivity and thereby influence the association between stress and premenstrual symptoms profoundly (Bencker, Tran, et al., 2025).

1.4. Trauma as Stress-Related Vulnerability

Experiences of stress and adversity affect a substantial proportion of females across the lifespan and have been shown to alter physiological stress response, thereby increasing vulnerability to reproductive affective disorders (Hantsoo et al., 2023). ICD-11 defines traumatic events as the

> Exposure to an event or situation (either short- or long-lasting) of an extremely threatening or horrific nature. Such events include, but are not limited to, directly experiencing natural or human-made disasters, combat, serious accidents, torture, sexual violence, terrorism, assault or acute life-threatening illness (e.g., a heart attack); witnessing the threatened or actual injury or death of others in a sudden, unexpected, or violent manner; and learning about the sudden, unexpected or violent death of a loved one. (WHO, 2019, 6B40)

Compared to the relatively strict DSM-5 and ICD-10 criteria, which primarily focus on life-threatening events as a prerequisite for posttraumatic stress disorder (PTSD) diagnosis, ICD-11 adopts a broader formulation that emphasizes the subjective experience of extreme threat (APA, 2013; Meinhardt, 2022; WHO, 2019). This conceptualization highlights that not the occurrence of traumatic events per se, but rather their subjective impact may be critical for subsequent stress and symptom expression. Accordingly, DSM-5 defines PTSD as

a trauma- and stressor-related disorder that develops after exposure to actual or threatened death, serious injury, or sexual violence (APA, 2013). The diagnosis requires at least one intrusion symptom, one avoidance symptom, two symptoms reflecting negative alterations in cognition and mood, and two symptoms of heightened arousal and reactivity. Symptoms must persist for longer than one month, lead to significant impairment, and cannot be explained by substance use or medical conditions (APA, 2013; Pai et al., 2017).

Against this background, traumatic experiences represent a particularly relevant class of stressors that may exert lasting effects on neurobiological and psychological functioning (Ehlert, 2013). Adverse childhood experiences, such as childhood maltreatment, are especially relevant because they occur during sensitive developmental periods in which stress-regulatory systems are still highly plastic (McEwen et al., 2015; McEwen & Akil, 2020). Such experiences may shape stress responsivity across the life course, partly via epigenetic modifications, thereby narrowing adaptive flexibility and increasing vulnerability to affective and stress-related disorders. Accordingly, adverse childhood experiences have been linked to long-term alterations in HPA axis responsivity, including changes in cortisol secretion, impaired negative feedback regulation, as well as altered inflammatory marker levels (Guilliams & Edwards, 2010; Hantsoo et al., 2023; Teicher et al., 2022). Early-life adversity has also been associated with epigenetic modifications affecting the glucocorticoid receptor and its function, potentially contributing to increased stress-sensitive HPA axis reactivity (Hassamal, 2023).

As the meta-analysis by Schumacher et al. (2019) indicates, PTSD is associated with alterations in HPA axis regulation, including lower basal cortisol levels compared to controls. Trauma-related conditions such as PTSD are therefore conceptualized as stress-related disorders characterized by dysregulated stress responsivity (McEwen & Akil, 2020).

Notably, more severe traumatic experiences have been associated with HPA hypoarousal, whereas less severe adversities may be linked to heightened HPA reactivity (Hantsoo et al., 2023), reflecting on stage-dependent dynamics from an initially heightened to a blunted stress response under sustained activation.

Taken together, these findings suggest that trauma may contribute to persistent and impairing alterations in HPA axis functioning, which may constitute fertile ground for the pathogenesis of affective disorders (Blecker et al., 2025; Hassamal, 2023). Importantly, trauma exposure per se does not imply a specific or uniform alteration of HPA axis functioning. The observed heterogeneity in cortisol patterns (Schumacher et al., 2019) suggests that exposure to traumatic events and the development of trauma-related

psychopathology such as PTSD should be analytically distinguished (an approach that is reflected in the methodological design of the present study, see also H2a and H2b).

This distinction is important because trauma exposure captures whether potentially traumatic events have occurred, whereas PTSD symptoms reflect the extent to which such experiences are associated with persistent trauma-related stress responses, including heightened threat appraisal, hyperarousal, alterations in stress-system functioning, and difficulties in affect regulation (Ehlers & Clark, 2000). Thus, traumatic experiences may increase vulnerability to symptoms not only through exposure itself, but also through the individual's response to trauma.

1.5. Trauma, Stress and Premenstrual Symptoms

Growing evidence from primary studies suggests that traumatic experiences represent an important risk factor for PMS and PMDD, with PTSD symptom severity appearing to further amplify this risk in a graded manner. For instance, in a prospective community cohort, exposure to traumatic events was associated with more than fourfold higher odds of developing PMDD during follow-up (Perkonigg et al., 2004). Similarly, longitudinal evidence from a 14-year follow-up study demonstrated that trauma exposure was associated with an elevated risk of PMS, with risk increasing proportionally to the number of PTSD symptoms reported (Jung et al., 2019). Consistent findings have been reported in cross-sectional research showing higher odds of PMDD among women with trauma histories, with the odds being about four times higher in those with PTSD (Pilver et al., 2011). Interestingly, this association may also operate in the opposite direction, as women with PMDD may be particularly susceptible to developing PTSD following traumatic events, potentially because they perceive stressful situations as more threatening, unpredictable, and difficult to cope with than individuals without PMDD (Pilver et al., 2011). Such altered stress appraisal processes may further amplify stress reactivity and contribute to symptom exacerbation across the menstrual cycle.

Supporting these epidemiological findings, evidence syntheses indicate a robust association between traumatic experiences and menstrual-related mood disorders (MRMDs), including PMS and PMDD. Two meta-analyses found that exposure to physical or sexual violence during childhood or adolescence nearly doubles the risk of developing MRMDs in adulthood (Islas-Preciado et al., 2021), and that individuals who experienced trauma had 2.5 times higher odds of PMS (Bencker, Tran, et al., 2025). In line with these findings, a recent

systematic review summarized primary evidence indicating that women with PMDD are approximately twice as likely to report traumatic exposure compared to women without PMDD (Grewal et al., 2025).

Importantly, current research indicates that trauma may influence not only the occurrence of premenstrual symptoms but also their severity. In particular, trauma exposure appears to be a stronger predictor of symptom severity than of symptom occurrence itself (Bencker, Tran, et al., 2025; Islas-Preciado et al., 2021; Schweizer-Schubert et al., 2021). In line with these findings, greater exposure to childhood adversity has been associated with a higher number and greater severity of PMDD symptoms: In particular, experiences such as sexual abuse during childhood or adolescence, as well as emotional neglect, have repeatedly been linked to more severe PMS symptomatology (Hantsoo et al., 2023). Further evidence from studies specifically examining childhood adversity supports this pattern. For example, higher levels of childhood adversity have been associated with stronger increases in negative affect and stress appraisal as well as greater decreases in positive affect from the follicular phase to the late luteal phase in women with PMDD (Nayman, Schricker, et al., 2023). These findings indicate that early adverse experiences may contribute to more pronounced mood deterioration across the menstrual cycle. However, it remains unclear whether the association between trauma and (severity of) premenstrual symptoms is primarily driven by trauma exposure itself or by the development of PTSD following trauma (Pilver et al., 2011).

Beyond these epidemiological and clinical associations, emerging etiological research suggests that trauma may contribute to individual differences in hormonal sensitivity underlying MRMD. Eisenlohr-Moul et al. (2016) demonstrated that histories of abuse predicted stronger associations between normal cyclical elevations of ovarian steroids and the emergence of symptoms. Specifically, women reporting physical abuse showed stronger symptom responses to increases in P4, including elevated depression, mood instability, rejection sensitivity, and interpersonal conflict. Sexual abuse, in contrast, was associated with stronger anxiety responses to E2 fluctuations. These findings suggest that trauma-related vulnerabilities may contribute to individual differences in hormone sensitivity among women with MRMD (Eisenlohr-Moul et al., 2016). At the neurobiological level, this may converge with GABAergic mechanisms involved in both stress regulation and hormonal sensitivity, with impaired sensitivity to cyclical ALLO fluctuations proposed as a key mechanism underlying premenstrual symptoms (Schweizer-Schubert et al., 2021). Since ALLO modulates GABA-A receptor functioning and thereby contributes to the inhibitory regulation of stress responses, trauma-related alterations in stress regulation may lower the threshold for

affective dysregulation during hormonally sensitive cycle phases (Bencker, Tran, et al., 2025; Hantsoo & Epperson, 2020; Schweizer-Schubert et al., 2021).

The findings outlined above suggest that the relationship between trauma, stress, and premenstrual symptoms cannot be understood through a single explanatory mechanism but rather through the interaction of multiple biological and psychosocial processes: Traumatic experiences and PTSD may lead to long-term alterations in stress response systems, disrupting the regulatory loop between the HPA and HPG axes involving altered sensitivity to ALLO, potentially leading to emotional lability (Hantsoo & Epperson, 2020; Schweizer-Schubert et al., 2021), and increasing stress sensitivity (Bencker, Tran, et al., 2025; Blecker et al., 2025; Hantsoo et al., 2023; Hassamal, 2023; Teicher et al., 2022). Importantly, such dysregulation may involve both heightened and blunted HPA activity, suggesting that trauma-related stress vulnerability cannot be reduced to a single cortisol pattern (Hantsoo et al., 2023; Schumacher et al., 2019). The expression of mental health problems becomes more likely when stress sensitivity is higher, which reflects the essence of the stress sensitization hypothesis (Blecker et al., 2025). This implies that dysregulated stress responsivity, intensified by trauma, may thereby increase not only vulnerability to premenstrual disorders, as it has already been shown in depressive disorders (Schweizer-Schubert et al., 2021), but also the severity of symptoms.

Overall, these findings point toward a dynamic framework: At the physiological level, interactions between the HPA and HPG axes influence how the body responds to stress, trauma, and cyclical hormonal changes. At the psychological level, trauma-related alterations in stress appraisal and coping may increase perceived stress and emotional reactivity. Rather than operating independently, these levels may mutually reinforce one another, forming a triangular stress–trauma–hormone framework in which trauma-related stress sensitization amplifies the impact of cyclical hormonal fluctuations in susceptible individuals.

1.6. Research Gap, Research Question, and Hypothesis

Despite the growing body of research on menstrual and reproductive health, important gaps remain.

The necessity of a unified approach becomes evident when considering that prevalence rates for premenstrual symptoms and related disorders vary considerably across studies. Estimates suggest that approximately 80–90% experience at least one premenstrual symptom (ACOG, 2001; Grewal et al., 2025). For PMS, reported prevalence rates range from about 5–10% in some studies (ACOG, 2001; Dickerson et al., 2003), to 13-19% (Schweizer-

Schubert et al., 2021), around 20% (Hantsoo et al., 2023), and even up to 40-60% (Gollenberg et al., 2010). Prevalence estimates for PMDD also differ, typically ranging from approximately 1% to 8% (Eisenlohr-Moul et al., 2016; Grewal et al., 2025; Hantsoo et al., 2023; Kundakovic & Rocks, 2022; Reilly et al., 2024b; Schweizer-Schubert et al., 2021).

These discrepancies likely reflect methodological and quality differences across studies. One important source of variation is that PMS has long been defined inconsistently, from broad descriptions to more narrowly specified clinical symptom patterns. Accordingly, studies differ in their diagnostic criteria and, additionally, in their assessment strategies, particularly with regard to retrospective and prospective symptom assessment (Bencker, Tran, et al., 2025; Dennerstein et al., 2012). Retrospective assessment refers to participants reporting symptoms from previous cycles based on memory, whereas prospective assessment involves repeated symptom ratings throughout the menstrual cycle, often on a daily basis. This distinction is crucial because retrospective reports may lead participants to overestimate symptoms and do not necessarily correspond to prospectively recorded symptom patterns (Eisenlohr-Moul et al., 2017). Therefore, valid diagnosis requires prospective daily symptom ratings, which are better suited to capturing the cyclical symptom pattern required for PMS/PMDD diagnosis. Beyond diagnosis, such cycle-sensitive assessment is also important for understanding symptom development, maintenance, and intensification, as interactions between person-level factors, such as trauma, and time-varying biochemical processes across menstrual cycle phases should be considered (Eisenlohr-Moul et al., 2016). Recognizing these challenges, recent efforts in reproductive mental health research aim to improve methodological consistency and diagnostic standardization. For instance, initiatives such as the Carolina Premenstrual Assessment Scoring System (C-PASS by Eisenlohr-Moul et al., 2017) have been developed to facilitate standardized prospective diagnosis, and methodological recommendations have been proposed to guide rigorous research on the menstrual cycle (Schmalenberger et al., 2021).

Despite the emergence of standardized tools and high prevalences, reliable research on reproductive mood disorders remains comparatively limited, as accurate assessment is resource and time-demanding, requiring validation of menstrual cycle phases and prospective symptom tracking across at least two menstrual cycles to confirm the cyclical pattern of symptoms (Bencker, Tran, et al., 2025; Schmalenberger et al., 2021). These considerations informed the design and measurement strategy of the present study.

Moreover, previous studies and review literature have examined associations between stress and premenstrual symptoms (Bencker, Tran, et al., 2025; Gollenberg et al., 2010),

between premenstrual symptoms, PMS/PMDD and trauma (Bencker, Tran, et al., 2025; Eisenlohr-Moul et al., 2016; Islas-Preciado et al., 2021; Pilver et al., 2011), and have further linked trauma to alterations in stress responsivity and HPA-axis functioning (Hantsoo et al., 2023; Schweizer-Schubert et al., 2021). However, the interplay between all three constructs has not yet been systematically investigated within a unified framework. In addition, when trauma was included as a variable, studies typically assessed either trauma exposure or PTSD symptomatology rather than considering both dimensions simultaneously. This distinction is crucial to advance the understanding of the association between stress, trauma and premenstrual symptoms.

To address these limitations and extend previous research, the present study adopts a triangular perspective and a longitudinal, cycle-sensitive design across two menstrual cycles, using daily assessments to capture fluctuations in stress and premenstrual symptoms while accounting for between-person differences in trauma exposure and PTSD symptomatology. To align the analysis with the cyclical diagnostic definition of PMS/PMDD, the study additionally distinguishes between the perimenstrual and non-perimenstrual phases and uses validated cycle phase estimation via ovulation tests, as described in more detail in the Methods section.

Altogether, the present study aims to address an important research gap by integrating stress, trauma exposure, trauma-related reactivity in the form of PTSD symptomatology, and premenstrual symptoms within a cycle-sensitive framework, ultimately seeking to contribute to a more precise understanding of underlying mechanisms and informing more targeted, phase-sensitive treatment approaches.

Against this background, the central research question of the present study is:

Is the association between perceived stress and premenstrual symptom severity stronger in individuals with greater trauma exposure and higher levels of PTSD symptomatology compared to those with little or no trauma exposure and PTSD symptomatology? And is this association particularly pronounced during the perimenstrual phase?

Based on the theoretical considerations outlined above, the following hypotheses are proposed:

(H1) Daily perceived stress positively predicts daily premenstrual symptom severity, such that individuals are expected to report higher premenstrual symptom severity on days

when their perceived stress is higher than their own average stress level. This association is expected to be stronger during the perimenstrual phase than during non-perimenstrual days.

(H2a) The positive within-person association between daily perceived stress and daily premenstrual symptom severity is moderated by trauma exposure, such that individuals with greater trauma exposure show a stronger stress-symptom association. This moderating effect is expected to be particularly pronounced during the perimenstrual phase.

(H2b) The positive within-person association between daily perceived stress and daily premenstrual symptom severity is moderated by PTSD symptomatology, such that individuals with higher levels of PTSD symptoms show a stronger stress-symptom association. This moderating effect is expected to be particularly pronounced during the perimenstrual phase.

2. Methods

The present study is a secondary analysis of Bencker's (2025) larger research project investigating the relationship between stress and the premenstrual symptoms, namely "Interactions in premenstrual symptoms and stress across the menstrual cycle" (ISSAC). The ISSAC study is a longitudinal, observational study employing a prospective, ecologically valid experience sampling design, in which regularly menstruating individuals provide daily self-report measures (ecological momentary assessment; EMA study) and saliva samples over two menstrual cycles, with hormonally validated cycle phase recording via ovulation tests. Data was taken from the ISSAC data pool, in consequence, same strengths and limitations concerning the study design are given. Seven other master students, Lea Maria Beinbauer, Laura Christina Stasch, Nicola Notz, Carlotta June Petrusch, Anette von Specht, Theresa Steidl and Chiara Hitchcock, elaborated their master theses around the ISSAC project as well.

2.1. Study Procedure

For recruiting participants flyers were spread in different locations in Vienna and social media posts were made, both including a QR-Code or link to a short screening questionnaire, which would check for eligibility for the ISSAC study. To be included, criteria

such as age between 21 and 45 years, regular menstrual cycles (21-35 days, not varying more than 4 days over the last three cycles), good German language skills and a BMI between 17 and 34.99 must be given. Exclusion criteria are the following: pregnancy or breastfeeding, high alcohol or illegal drug use, psychotropic medication, a lifetime diagnosis of substance-induced disorders, bipolar disorder or schizophrenia, use of hormonal contraceptives (within the past three months), severe somatic or gynecological disorders affecting the menstrual cycle, shift work and regularly waking up later than 11:00 a.m.

If participants were found to be eligible, they would receive an invitation to the lab for a baseline assessment. The timing was important and has been calculated based on the last four menstrual onsets, so that they would come in four to ten days after the first day of menstrual bleeding. Throughout the appointment participants receive important information about the study as well as study materials (tubes for saliva collection, ovulation testing kits, manuals, a study overview and, if needed, a study smartphone), the study app “movisensXS” (Movisens GmbH, Karlsruhe, Germany) was coupled, participants gave informed consent and filled out a baseline questionnaire (SoSci Survey GmbH, Munich, Germany). This questionnaire includes the International Trauma Exposure Measure, (ITEM; Meinhardt, 2022) and the PTSD Checklist for DSM-5 (PCL-5; Krüger-Gottschalk et al., 2017), which are moderator variables of the present research question. EMA period begins four days before the earliest calculated date of ovulation and ends on day 10 days after the onset of the second menstrual bleeding. During the first study phase participants have to conduct five questionnaires and three saliva samples per day. Whereas there is only the evening assessment during the second study phase. All questionnaires were answered in “movisensXS”. For the present research question, only the evening questionnaire (which is equal in study phase 1 and 2), that participants filled out throughout the whole study length, is relevant. It includes a visual analogue scale from zero to 100 to measure perceived stress and the Premenstrual Symptom Diary (PSD; Janda et al., 2017).

Throughout the EMA study phase, participants were in constant contact with someone from the study team, who provided information, instructions (e.g., when the ovulation test should be conducted), and assistance with any questions or concerns.

At the end of the study period, participants were invited five to eight days prior to the calculated upcoming menses to fill out the follow-up questionnaire on chronic stress and emotion regulation (and to bring back the materials and saliva samples). If their compliance was 90% or more, they received total compensation of 120 euros (if less, an accordingly reduced amount).

2.2. Measurements

Premenstrual Symptoms Measurement

Premenstrual symptomatology, the outcome variable in the statistical model, was assessed using the PSD (Janda et al., 2017), a prospective symptom diary based on DSM-5 criteria covering the full menstrual cycle over 2 consecutive cycles. Participants rated a series of emotional, behavioral, and somatic symptoms every evening. Daily premenstrual symptom severity was calculated as the daily mean across items 1–28.

The PSD assesses premenstrual symptoms using the first 27 items, which were categorized into core (items 1–11, including affective lability, irritability, depressed mood, and anxiety), secondary psychological and behavioral (items 12–19), and somatic symptoms (items 20–27), building a symptom-intensity score (SI-score) and adds three items about the impact those symptoms have on their daily life for an interference score (INT-score). The latter wasn't included in the present research, as the focus is on symptom intensity.

The scale was translated into German, with one minor modification: item 19 ("Feeling out of control or overwhelmed") was split into two separate items in the German version to assess "feeling less in control" (item 19: "Gefühl, weniger Kontrolle zu haben") and "feeling overwhelmed" (item 20: "Gefühl, überfordert zu sein") as distinct experiences and resulting in a total of 28 items.

Another modification concerned the response format: whereas the original PSD uses a 4-point Likert scale ranging from 0 ("not true at all") to 3 ("absolutely true"), the translated German version employs a 6-point Likert scale from 0, meaning "not at all" ("gar nicht") to 5, which would be "extremely" ("extrem").

Psychometric analyses indicated moderate to good internal consistency (Cronbach's $\alpha = .83–.96$). Evidence for convergent validity was provided by significant associations with a retrospective screening measure, the Pain Disability Index, and the German PMS-Impact Questionnaire, whereas discriminant validity was reflected in weak correlations with the Big Five Inventory-10 (Janda et al., 2017).

For the present research question, the PSD served as the primary instrument for assessing premenstrual symptomatology. It was chosen as a standardized procedure in PMDD diagnostics. Its daily ratings enable the identification of cyclic symptom patterns, including symptom peaks before menstruation and remission after the onset of menses, in line with DSM-5 criteria for PMDD.

Menstrual Cycle Measurement and Cycle Phases of Interest

Before onboarding on the study, potential participants had to report the last three menstrual cycles, which is the last three dates of menstrual onsets to verify that variations between the cycle lengths are not too high. This is important for later-cycle-phase determination.

Throughout the EMA period of the study, participants were asked to inform the study team when menstruation began and to report it in the study application (movisensXS). The same applies to ovulation tests (urinary LH detection), which were used to verify ovulation and improve precision in cycle phase estimation.

Menstrual cycle phases were determined using a combined forward- and backward-counting approach in line with the standardized recommendations by Schmalenberger et al. (2021). Forward counting anchors cycle day +1 at the onset of menstrual bleeding and defines subsequent days relative to this reference point (e.g., day +1, +2). Backward counting anchors cycle days relative to the next menstrual onset (day -1 refers to the day before menstrual onset), allowing days in the late luteal and perimenstrual phase to be defined retrospectively (e.g., day −1 to −7). This combined approach reduces misclassification that may arise from interindividual variability in cycle length.

As the present paper focuses on PMS and PMDD, it is important to refer to the relevant symptom time window. The gold standard for diagnostics of PMDD is the C-PASS (Eisenlohr-Moul et al., 2017): The premenstrual week (day -7 to -1) is contrasted with the postmenstrual week (days +4 to +10), as symptom remission after menstrual onset is a defining characteristic of PMDD. To ensure an evidence-based operationalization, this approach was integrated but adapted to avoid statistical power loss, as restricting analyses to the premenstrual and postmenstrual windows would have excluded a substantial proportion of observations (i.e., all data that wouldn't lie within day -7 to -1 or +4 to +10). To address this, dummy coding was used to define a *perimenstrual phase* (days −7 to +4) (see also Figure 1), allowing the inclusion of the full dataset while retaining sensitivity to the PMS/PMDD-relevant period. This approach reflects the fact that although the premenstrual week represents the core window of symptom vulnerability, complete symptom remission may extend into the first days of menstruation.

Stress Measurement

Daily perceived stress was assessed using a single-item measure derived from the German version of the Perceived Stress Scale (PSS; Cohen et al., 1983; Klein et al., 2016).

The original PSS is a validated and widely used instrument assessing the degree to which situations in one's life are appraised as stressful. In its original form, the scale refers to stress experienced over the past month and demonstrates good psychometric properties, including high internal consistency (Cronbach's $\alpha \approx .84$).

In contrast to the original version, which assesses stress retrospectively on a 5-point Likert scale from 0 ("never") to 4 ("very often"), the present study employed an adapted single-item version to capture daily perceived stress. Participants rated their stress experienced on the same day during the evening assessment before going to bed by answering the question "Wie gestresst haben Sie sich heute gefühlt?" ("How stressed did you feel today?"). Responses were given on a visual analogue scale ranging from 0 ("überhaupt nicht" / "not at all") to 100 ("sehr stark" / "very much"), with higher values indicating higher levels of perceived stress.

This variable was decomposed into a within-person (daily deviation from each participant's mean) and a between-person component (person-specific mean stress). This approach allows us to assess daily fluctuations to see whether a person feels more stressed relative to their usual level (state-level) while controlling for the general stress level of a person, comparing the means between the individuals (trait level).

Trauma Measurements

There are two different measurements related to trauma used in this study and assessed within the baseline questionnaire. Because trauma exposure and trauma-related reactivity represent distinct constructs, this distinction is crucial for the present research question and was therefore examined in two separate hypotheses: H2a is looking at the exposure to trauma (*trauma load)*, whereas H2b wants to know about the moderating effect the reactivity of the experienced trauma has (*PTSD symptom severity*).

Trauma Load

Lifetime exposure to potentially traumatic events was assessed with the translated German version of the International Trauma Exposure Measure (ITEM; Hyland et al., 2021; German version by Meinhardt, 2022). The ITEM is a structured checklist developed to operationalize the ICD-11 definition of trauma, which includes both events involving extreme threat to life or physical integrity and events involving severe psychological or emotional threat. The measure consists of 21 discrete trauma categories (e.g., physical assault, sexual violence, severe accidents, neglect, humiliation, chronic bullying), plus one open category for unlisted events. For each event type, participants indicate whether they experienced it and

specify the developmental period in which it occurred (childhood, adolescence, adulthood), allowing the instrument to capture timing of exposure, a factor known to influence symptom trajectories (Blecker et al., 2025). The ITEM also includes follow-up questions for the event rated as “worst”, representing the index trauma, documenting its earliest occurrence, approximate frequency, and predominant emotional response.

In this study, the variable was derived from the total number of trauma types experienced across the lifespan, described in the following as “trauma load”.

PTSD Symptom Severity

To measure the reactivity one shows to the exposure of traumatic events, the PTSD Checklist for DSM-5 (PCL-5) is a self-report questionnaire with 20 items measuring the severity of PTSD symptoms as defined by DSM-5, and its German version was used for this research project (Krüger-Gottschalk et al., 2017). Symptom severity is rated with reference to the past month on a 5-point Likert scale ranging from 0 (“not at all”/”überhaupt nicht”) to 4 (“extremely”/”sehr stark”). The items range from intrusive memories or dreams, avoidance, to feelings of fear or physical reactions, covering all DSM-5 symptoms of PTSD.

PTSD symptom severity was operationalized using the PCL-5 total score. For this purpose, all 20 items assessed at baseline were summed, resulting in a possible score range from 0 to 80. Higher scores indicate greater PTSD symptom severity. Missing item values were excluded from the sum calculation.

The questionnaire has demonstrated good internal consistency (Cronbach’s $\alpha = .95$) and high test–retest reliability ($r = .91$) in clinical samples. Convergent validity was supported by a strong correlation with the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; $r = .77$).

2.3. Statistical Procedure

All statistical analyses were conducted using R for Windows (version 4.5.2) (R Core Team, 2026). Data preprocessing, management, and visualization were performed using the *tidyverse* suite of packages, while multilevel analyses were estimated using the *lme4* and *lmerTest* packages.

EMA datasets from study phase 1 and study phase 2 were imported and merged into a single longitudinal dataset. To ensure consistency across datasets, participant identifiers were converted to a uniform format, and records originating from multiple device IDs (e.g., due to

technically necessary replacement of smartphones) were harmonized to a single participant code.

In addition, a subject identification log containing participant-level information (e.g., EMA start and end dates, menstrual onset dates, ovulation test dates, and study completion status movisensXS and Soscisurvey participant codes), was imported and cleaned. Participants who did not yet complete the EMA period or stopped the study prematurely were excluded at this stage. Date variables originating from different data formats were converted into standardized date objects to allow accurate temporal alignment across datasets. After merging EMA data with the subject identification log, the final analytic dataset comprised all participants who completed both study phases by mid-December 2025. EMA entries falling outside the individual study period defined by EMA start and end dates were excluded.

Analyses were restricted to the baseline questionnaire, which provided the trauma-related measures, and to the daily evening EMA assessment, as this assessment was administered consistently across both study phases and contained the repeated daily measures relevant to the present research question. Premenstrual symptom severity was operationalized as the mean across PSD items 1–28 for each participant and evening assessment, resulting in a repeated daily outcome measure. Daily stress was then decomposed into within-person stress and person-mean stress to distinguish day-to-day deviations from participants' average stress levels.

To account for evening assessments completed shortly after midnight, a corrected study date variable was created. If an evening assessment was completed between 00:00 and 06:00 am, the assessment was assigned to the preceding calendar day to ensure correct alignment with daily symptom and stress reports. Based on this corrected date, a study day variable was computed for each participant, representing the number of days since the individual's EMA start.

Menstrual cycle information was derived using recorded dates of menstrual onset and, where available, ovulation test results. For each daily observation, cycle day and cycle number were determined relative to the corresponding menstrual onset. To account for interindividual variability in cycle length, menstrual phase assignment was based on a combined forward- and backward-counting approach (Schmalenberger et al., 2021).

Forward counting indexed the number of days since the most recent menstrual onset, while backward counting indexed the number of days until the next menstrual onset. These complementary indices allowed cycle phases to be defined relative to both past and upcoming menstrual bleeding, thereby increasing temporal precision. In addition, days corresponding to

positive luteinizing hormone (LH) tests were identified when available and used to refine phase classification.

Baseline measures of trauma exposure (from ITEM) and PTSD symptoms (from PCL-5) were merged into the EMA dataset at the participant level. Trauma exposure was operationalized as cumulative trauma load, defined as the total number of different trauma categories endorsed. PTSD symptom severity was computed as the total score of the PCL-5 and mean-centered prior to inclusion in interaction models to facilitate interpretation.

Given the hierarchical structure of the data, with repeated daily observations (on stress and premenstrual symptoms) nested within individuals with differences in their trauma load and PTSD symptoms, hypotheses were tested using a multi-level model (MLM). MLM allows random intercepts for participants to account for individual differences in overall symptom levels as well as random slopes accounting for interindividual differences in stress reactivity. An unconditional null model was estimated first to compute the intraclass correlation coefficient (ICC), quantifying the proportion of variance in daily premenstrual symptoms attributable to between-person differences.

To test the primary hypothesis (H1), daily premenstrual symptom severity was regressed on within-person stress and between-person stress simultaneously. This approach allowed the effects of daily stress fluctuations to be examined independently of stable individual differences in stress. To assess whether individuals differed in their stress reactivity, a random slope for within-person stress was added and model fit was compared using likelihood ratio tests.

Cycle sensitivity was examined by including the perimenstrual phase dummy.

Prior to testing the secondary hypotheses (H2a and H2b), the correlation between trauma load and PTSD symptom severity was examined to rule out substantial redundancy between the variables.

Moderation hypotheses were tested by extending the stress model to include interaction terms. First, trauma load and PTSD symptom severity were examined separately as moderators of the within-person stress–symptom association. Second, cycle sensitivity was examined by including the perimenstrual phase dummy and its interaction with daily stress. Finally, three-way interaction models tested whether stress reactivity during the perimenstrual phase differed as a function of trauma exposure or PTSD symptom severity.

Fixed-effect estimates, confidence intervals, and significance values were extracted from the final models and used for reporting and visualization. To facilitate the interpretation and comparability of effect sizes, the outcome variable and all focal predictors (within-person

stress, trauma load, and PTSD symptom severity) were standardized prior to analysis. The between-person stress variable was retained in its original metric to preserve the within–between decomposition of stress. Corresponding models with unstandardized variables are reported in the Appendix.

3. Results

3.1. Participants

A total of 188 individuals completed the screening questionnaire by mid-December 2025. The most frequent exclusion criteria (of those listed in section 3.1) were irregular menstrual cycles or cycle length, gynecological disorders, intake of psychotropic medication and excessive alcohol consumption.

The final sample comprised 44 participants who completed the EMA phase by the end of January 2026 and all identified as women[4]. Participants' mean age was 27.89 years (*SD* = 5.10), ranging from 22 to 43 years. The reported mean menstrual cycle length was 28.18 days (*SD* = 2.78), corresponding to average cycle lengths described by ACOG (2025), thereby supporting the plausibility of the sample with regard to menstrual cycle characteristics (see also Table 1).

3.2. Descriptive Statistics

Descriptive statistics are presented in Table 1. Across all observations (*n* = 2315), the mean level of premenstrual symptom severity was moderate (*M* = 0.60, *SD* = 0.55; range = 0.00–3.93), while perceived stress showed substantial variability (*M* = 38.10, *SD* = 22.79; range= 0.00–100.00), indicating considerable day-to-day fluctuations in stress levels.

Decomposition of the variables into within- and between-person components revealed that a substantial proportion of variability occurred at the within-person level. For perceived stress, within-person variability (*SD* = 19.55) exceeded between-person variability (*SD* = 11.70), suggesting that daily fluctuations in stress were more pronounced than stable differences between individuals. A similar pattern was observed for premenstrual symptoms, with within-person variability (*SD* = 0.46) exceeding between-person variability (*SD* = 0.30).

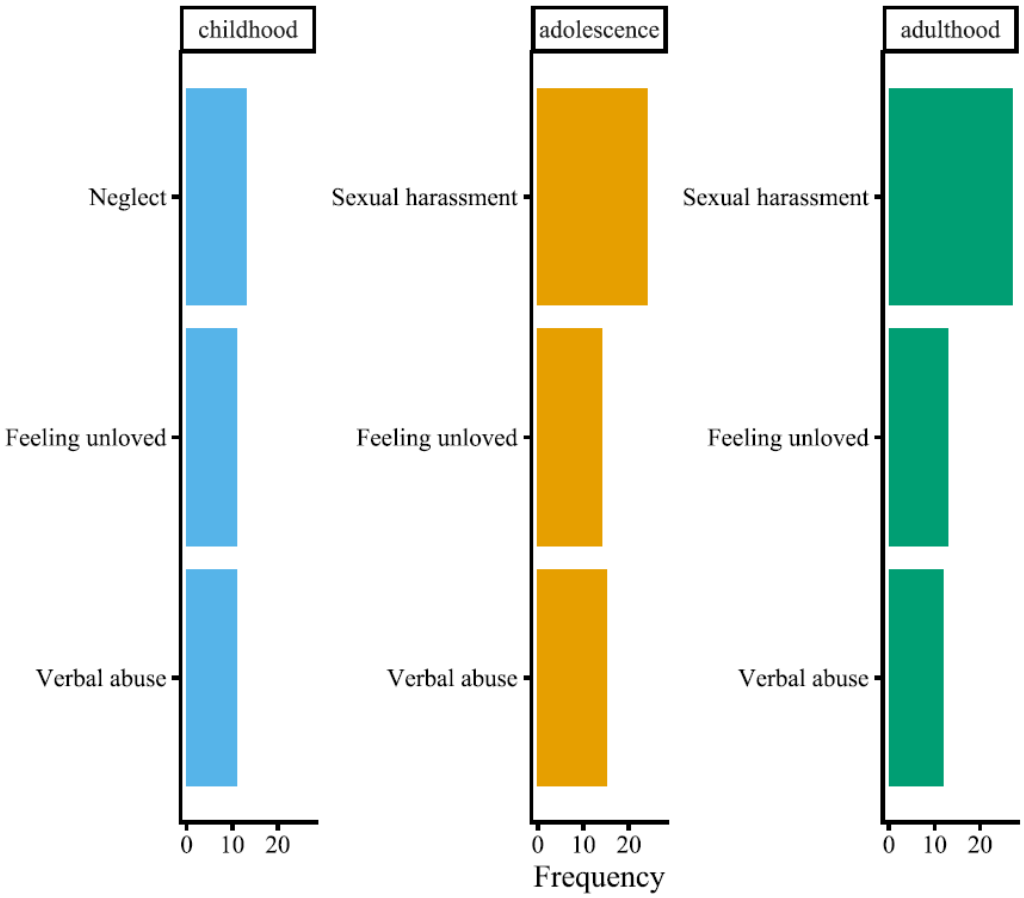
These findings indicate that both stress and symptom severity vary considerably within individuals across days, supporting the use of multilevel modeling to disentangle within- and between-person effects.

[4] In this study, participants were asked "Which gender do you identify with?" with the response options "female", "male", "non-binary", "other", "prefer not to answer".

Participants reported on average a moderate trauma load (M = 3.63, SD = 2.46; range = 0.00-10.00), indicating substantial heterogeneity in exposure to different types of traumatic events. Descriptive analyses of trauma exposure indicated both continuity and phase-specific patterns across the lifespan. Emotional forms of adversity, such as feeling unloved and verbal abuse, were among the most frequently reported experiences across all life periods. In contrast, neglect was particularly prominent in childhood, whereas sexual harassment emerged as the most frequently reported event during adolescence and adulthood (see Figure 3). Overall, approximately 75% of participants reported having experienced sexual harassment at least once across the lifespan (see Figure 4).

Figure 3

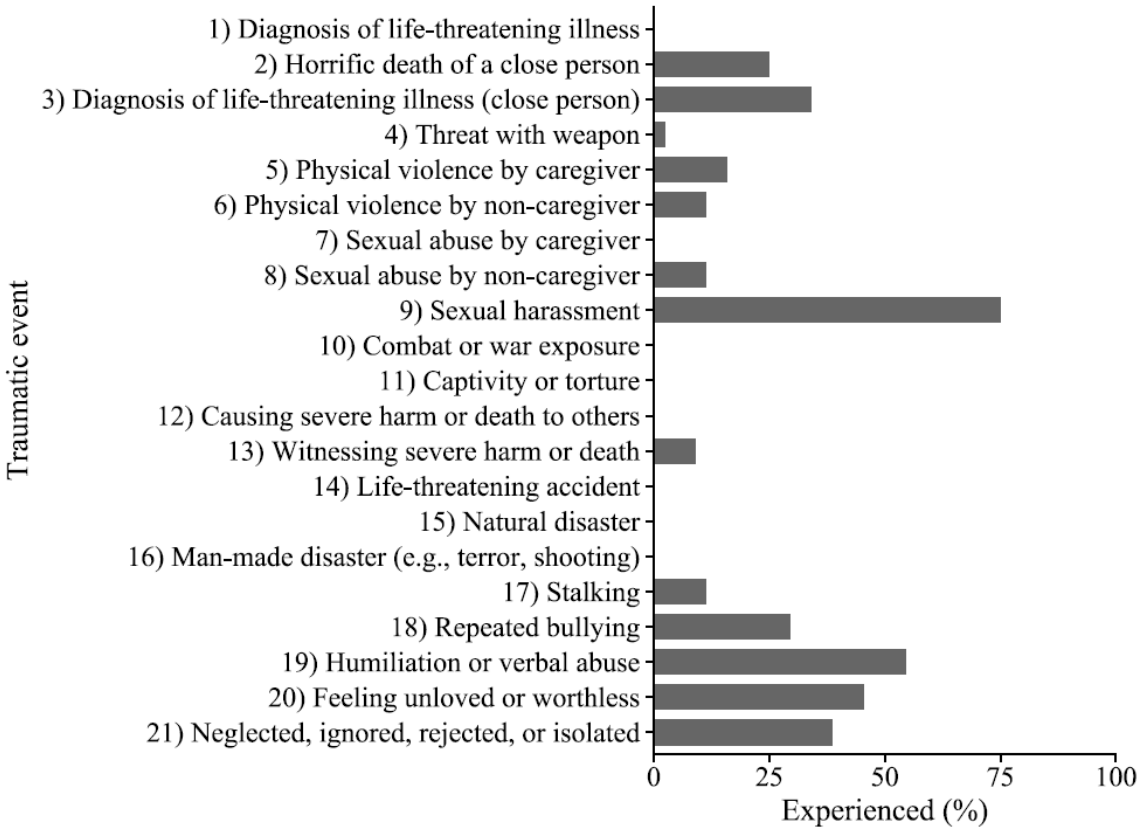
Frequency of Most Common Adverse Events Across Childhood, Adolescence, and Adulthood



Note. The figure displays the frequency of the three most commonly reported adverse events within each developmental period (childhood, adolescence, adulthood). Frequencies represent the number of participants reporting each event.

Figure 4

Prevalence of Traumatic Events Across the Lifetime



Note. The figure displays the percentage of participants reporting each traumatic event across their lifetime.

PTSD symptom severity also showed considerable variability (M = 33.68, SD = 12.02; range = 20.00-63.00).

Descriptive statistics by menstrual cycle phase further indicated elevated premenstrual symptom levels during the perimenstrual phase (M = 0.83, SD = 0.63; range = 0.00-3.93) compared to the non-perimenstrual phase (M = 0.46, SD = 0.43; range = 0.00-2.50). Perceived stress was also slightly higher during the perimenstrual phase (M = 39.56, SD = 23.46; range= 0.00-100.00) compared to the non-perimenstrual phase (M = 37.18, SD = 22.31; range= 0.00-100.00).

Together, these descriptive findings provide initial support for both substantial within-person variability and cycle-related increases in symptom severity, which are further examined in the multilevel analyses.

Table 1

Descriptive Statistics

Variable	*M*	*SD*	Min	Max
Age	27.89	5.10	22.00	43.00
Cycle length	28.18	2.78	23.67	36.00
PSD overall	0.60	0.55	0.00	3.93
PSS overall	38.10	22.79	0.00	100.00
PSD within-person	-0.00	0.46		
PSD between-person	0.60	0.30		
PSS within-person	0.00	19.55		
PSS between-person	38.12	11.70		
PSD perimenstrual phase	0.83	0.63	0.00	3.93
PSD not premenstrual phase	0.46	0.43	0.00	2.50
PSS perimenstrual phase	39.56	23.46	0.00	100
PSD not premenstrual phase	37.18	22.31	0.00	100
Trauma load	3.63	2.46	0.00	10.00
PCL total	33.68	12.02	20.00	63.00

Note. *M* = mean; *SD* = standard deviation; Min = minimum; Max = maximum; PSD = Premenstrual Symptom Diary score, referring to premenstrual symptom severity; PSS = Perceived Stress Scale; PTSD Checklist score, referring to PTSD symptom severity. Within-person variables represent deviations from each participant's own mean. Between-person variables represent participants' mean levels across observations. Perimenstrual phase was dummy coded with 0 = observations outside the perimenstrual phase, 1 = perimenstrual phase).

3.3. Intraclass Correlation

An unconditional null model with random intercepts for participants was estimated to quantify the proportion of variance in daily premenstrual symptom severity attributable to between-person differences. The intraclass correlation coefficient (ICC) was 0.29, indicating that 29% of the variance in premenstrual symptoms was located at the between-person level,

while the remaining variance reflected within-person fluctuations, thus justifying the use of MLM.

3.4. Model Specification and Comparison

Model comparison using likelihood ratio tests indicated that the random slope model provided a significantly better fit to the data than the random intercept model, $\chi^2(2) = 138.77$, $p < .001$, accompanied by substantial reductions in AIC and BIC. The random slope model was therefore retained for all subsequent analyses.

Model assumptions were evaluated using standard diagnostic procedures. Homoscedasticity and linearity were assessed by visual inspection of residuals versus fitted values and scale–location plots. Residuals were centered around zero with no pronounced systematic patterns, although a mild increase in residual variance at higher fitted values was observed (see Figure A1 in the Additional Results section). Normality of residuals was examined using Q–Q plots, which indicated minor deviations from normality in the upper tails (see Figure A2 in the Additional Results section). Such deviations are common in longitudinal data with bounded outcome variables and were not considered to substantially affect model estimates. Additional diagnostics were conducted using the "check_model()" function from the *performance* package. Overall, model assumptions were deemed sufficiently met for the present analyses.

3.5. Primary Hypothesis (H1): Association of Stress and Premenstrual Symptoms

In the final random slope model, within-person stress was a significant positive predictor of daily premenstrual symptom severity ($b = 0.346$, $SE = 0.039$, $t = 8.91$, $p < .001$), indicating that on days when participants experienced higher stress than usual, they also reported more severe premenstrual symptoms.

Between-person stress was also positively associated with symptom severity ($b = 0.022$, $SE = 0.006$, $t = 3.97$, $p < .001$), reflecting higher overall symptom levels among individuals with higher average stress.

The cycle-sensitive model showed that the perimenstrual phase was associated with higher symptom severity ($b = 0.614$, $SE = 0.029$, $t = 21.32$, $p < .001$).

Furthermore, in the model including cycle phase, within-person stress was a significant positive predictor of premenstrual symptom severity outside the perimenstrual phase ($b = 0.295$, $SE = 0.037$, $t = 7.99$, $p < .001$). This indicates that on days with higher-than-usual stress, participants reported higher symptom severity even outside the perimenstrual phase. In addition, the interaction between within-person stress and

perimenstrual phase was statistically significant ($b = 0.101$, $SE = 0.029$, $t = 3.41$, $p < .001$), indicating that this positive association was even stronger during the perimenstrual phase.

Results are presented in Table 2 and illustrated in Figure 5.

Table 2

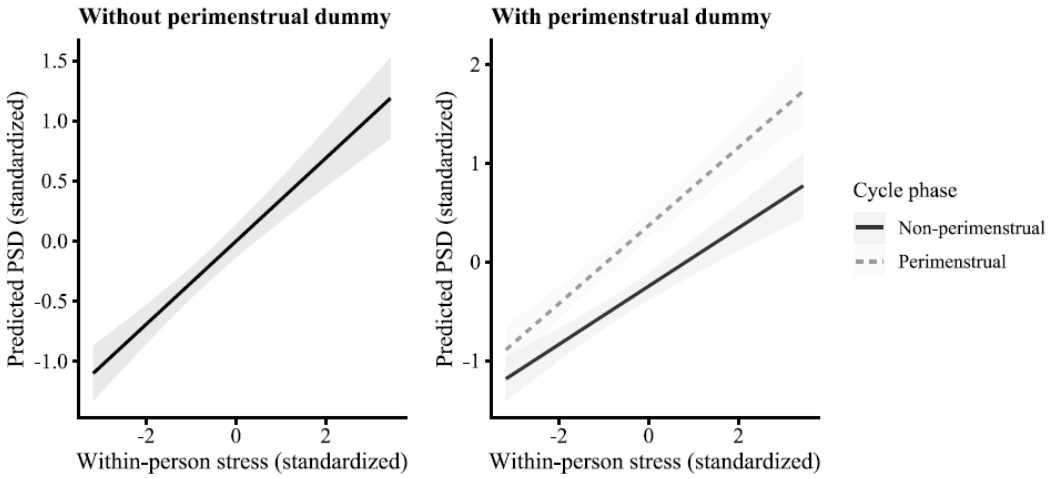
Standardized Multilevel Model Predicting Premenstrual Symptom Severity From Stress, Without and With Adjustment for Perimenstrual Phase

	Without perimenstrual dummy				**With perimenstrual dummy**			
Predictor	***b***	***SE***	***t***	***p***	***b***	***SE***	***t***	***p***
Intercept	-0.847	0.224	-3.78	< .001***	-1.074	0.225	-4.78	< .001
Within-person stress (standardized)	0.346	0.039	8.91	< .001***	0.295	0.037	7.99	< .001***
Between-person stress	0.022	0.006	3.97	< .001***	0.022	0.006	3.89	< .001***
Perimenstrual phase					0.614	0.029	21.32	< .001***
Stress × Perimenstrual phase					0.101	0.029	3.41	< .001***

Note. b = regression coefficient; SE = standard error; t = t statistic; p = p value. The outcome and focal predictors were standardized. Within-person stress represents person-mean-centered daily deviations. The perimenstrual phase variable was dummy coded (0 = outside the perimenstrual phase, 1 = perimenstrual phase). Models include random intercepts and random slopes for within-person stress at the participant level. *$p < .05$, **$p < .01$, ***$p < .001$

Figure 5

Predicted Association Between Within-Person Stress and Premenstrual Symptom Severity (Standardized Models)



Note. PSD = Premenstrual Symptom Diary score, referring to premenstrual symptom severity. Predicted premenstrual symptom severity is shown as a function of within-person stress. The left panel displays the model without adjustment for perimenstrual phase, whereas the right panel displays the model including perimenstrual phase, with separate lines for non-perimenstrual and perimenstrual observations. Shaded areas represent 95% confidence intervals. Continuous predictors were standardized and centered as appropriate. Predictions are based on fixed effects, with between-person stress held constant at its sample mean.

3.6. Secondary Hypothesis (H2): Moderators

Preliminary Analysis for secondary hypotheses showed that Trauma load and PTSD symptoms were strongly positively correlated ($r = .66$, $t = 5.6572$, $p < .001$), indicating substantial overlap but not redundancy between the constructs.

3.6.1. (H2a): Trauma Exposure as a Moderator

To test whether cumulative trauma exposure moderated the association between daily stress and premenstrual symptom severity, trauma load and its interaction with within-person stress were added to the final random slope model.

In the model without cycle adjustment, the interaction between within-person stress and trauma load was not statistically significant ($b = 0.018$, $SE = 0.040$, $t = 0.45$, $p = .655$), indicating that trauma load did not moderate the association between daily stress and premenstrual symptoms.

In the cycle-adjusted model, the three-way interaction between within-person stress, trauma load, and perimenstrual phase was also not significant (b = -0.011, SE = 0.029, t = -0.38, p = .703). Thus, trauma exposure did not further intensify the stress–symptom association during the perimenstrual phase.

Results are presented in Table 3 and illustrated in Figure 6.

Table 3

Standardized Multilevel Model Testing Trauma Load as a Moderator of the Stress–Symptom Association, Without and With Adjustment for Perimenstrual Phase

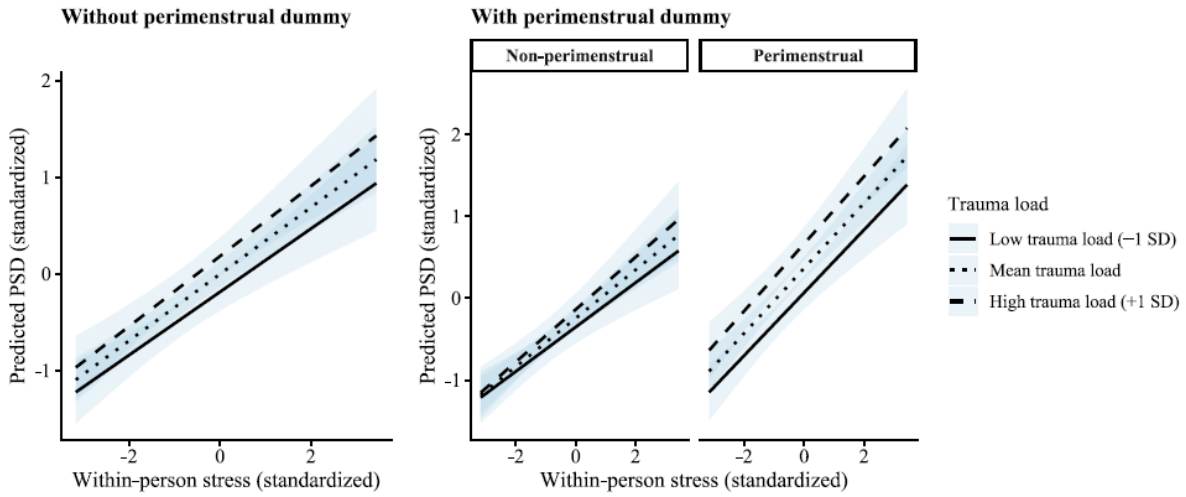
	Without perimenstrual dummy				**With perimenstrual dummy**			
Predictor	***b***	***SE***	***t***	***p***	***b***	***SE***	***t***	***p***
Intercept	-0.618	0.227	-2.72	0.009**	-0.871	0.231	-3.78	< .001***
Within-person stress (standardized)	0.344	0.039	8.77	< .001***	0.295	0.037	7.98	< .001***
Between-person stress	0.016	0.006	2.84	0.007**	0.016	0.006	2.84	0.007**
Trauma load (standardized)	0.185	0.072	2.55	0.015*	0.107	0.074	1.46	0.152
Perimenstrual phase					0.612	0.029	21.47	< .001***
Stress × Trauma load	0.018	0.040	0.45	0.655	0.025	0.037	0.66	0.511
Stress × Perimenstrual phase					0.101	0.029	3.47	< .001***
Trauma load × Perimenstrual phase					0.192	0.029	6.69	< .001***
Stress × Trauma load × Perimenstrual phase					-0.011	0.029	-0.38	0.703

Note. b = regression coefficient; SE = standard error; t = t statistic; p = p value. The outcome and focal predictors were standardized. Within-person stress represents person-mean-centered

daily deviations. The perimenstrual phase variable was dummy coded (0 = outside the perimenstrual phase, 1 = perimenstrual phase). Models include random intercepts and random slopes for within-person stress at the participant level. *$p < .05$, **$p < .01$, ***$p < .001$

Figure 6

Trauma Load as a Moderator of the Stress-Symptom Association (Standardized Models)



Note. PSD = Premenstrual Symptom Diary score, referring to premenstrual symptom severity; *SD* = standard deviation. Predicted premenstrual symptom severity as a function of within-person stress are shown for low (−1 *SD*), mean, and high (+1 *SD*) levels of trauma load. The left panel displays the model without adjustment for perimenstrual phase; the right panel displays the model including perimenstrual phase dummy, with separate panels for non-perimenstrual and perimenstrual observations. Shaded areas represent 95% confidence intervals. Continuous predictors were centered (and standardized where indicated). Predictions are based on fixed effects, with between-person stress held constant at its sample mean.

3.6.2. (H2b): PTSD Symptoms as a Moderator

To examine whether posttraumatic stress symptom severity moderated the association between daily stress and premenstrual symptom severity, PTSD symptoms (PCL-5, mean-centered) and their interaction with within-person stress were added to the final random slope model.

In the model without cycle adjustment, the interaction between within-person stress and PTSD symptoms was statistically significant ($b = 0.080$, $SE = 0.038$, $t = 2.09$, $p = .053$).

This indicates that higher PTSD symptom severity was associated with a stronger within-person stress–symptom association.

In the cycle-adjusted model, the three-way interaction between within-person stress, PTSD symptoms, and perimenstrual phase was also significant ($b = 0.067$, $SE = 0.032$, $t = 2.11$, $p = .035$). This indicates that the increase in stress-related symptom severity during the perimenstrual phase was more pronounced among individuals with higher levels of PTSD symptoms.

Results are presented in Table 4 and illustrated in Figure 7.

Table 4

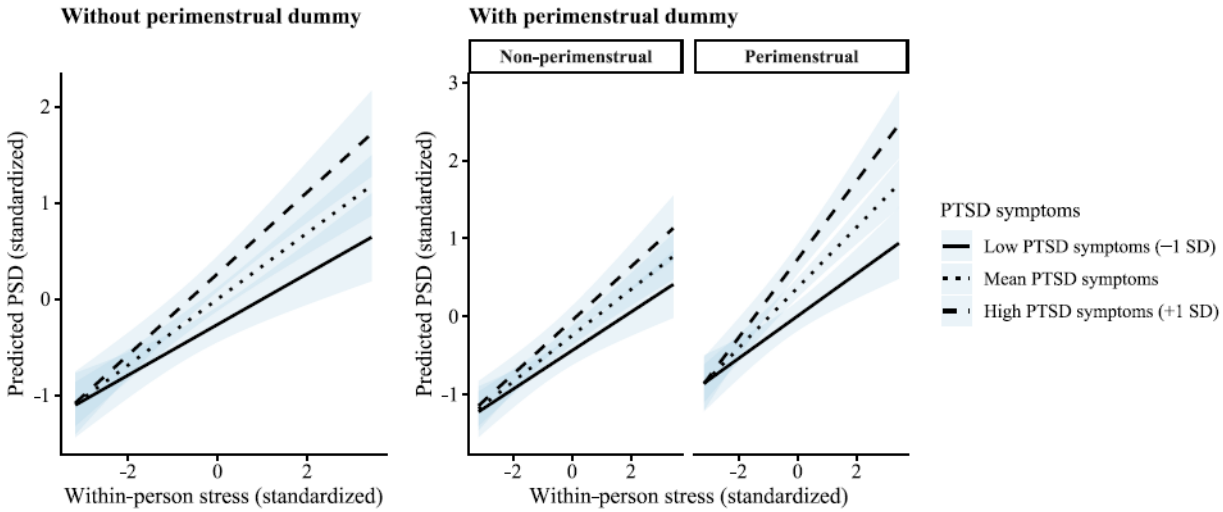
Standardized Multilevel Model Testing PTSD Symptom Severity as a Moderator of the Stress–Symptom Association, Without and With Adjustment for Perimenstrual Phase

	Without perimenstrual dummy				**With perimenstrual dummy**			
Predictor	***b***	***SE***	***t***	***p***	***b***	***SE***	***t***	***p***
Intercept	-0.531	0.227	-2.34	0.024*	-0.775	0.229	-3.38	0.002**
Within-person stress (standardized)	0.343	0.038	9.14	< .001***	0.296	0.035	8.48	< .001***
Between-person stress	0.014	0.006	2.42	0.020*	0.014	0.006	2.40	0.021*
PTSD symptom severity (standardized)	0.264	0.070	3.78	< .001***	0.193	0.071	2.73	0.009**
Perimenstrual phase					0.614	0.029	21.50	< .001***
Stress × PTSD symptoms	0.080	0.038	2.09	0.043*	0.049	0.036	1.37	0.177
Stress × Perimenstrual phase					0.091	0.030	3.08	0.002**
PTSD symptoms × Perimenstrual phase					0.171	0.029	5.91	< .001***
Stress × PTSD symptoms × Perimenstrual phase					0.067	0.032	2.11	0.035*

Note. *b* = regression coefficient; *SE* = standard error; *t* = t statistic; *p* = p value. The outcome and focal predictors were standardized. Within-person stress represents person-mean-centered daily deviations. The perimenstrual phase variable was dummy coded (0 = outside the perimenstrual phase, 1 = perimenstrual phase). Models include random intercepts and random slopes for within-person stress at the participant level. *$p < .05$, **$p < .01$, ***$p < .001$

Figure 7

PTSD Symptom Severity as a Moderator of the Stress-Symptom Association (Standardized Models)



Note. PSD = Premenstrual Symptom Diary score, referring to premenstrual symptom severity; *SD* = standard deviation. Predicted values of premenstrual symptom severity (PSD) as a function of within-person stress are shown for low (−1 *SD*), mean, and high (+1 *SD*) levels of PTSD symptom severity. The left panel displays the model without adjustment for perimenstrual phase; the right panel displays the model including perimenstrual phase dummy, with separate panels for non-perimenstrual and perimenstrual observations. Shaded areas represent 95% confidence intervals. Continuous predictors were centered (and standardized where indicated). Predictions are based on fixed effects, with between-person stress held constant at its sample mean.

4. Discussion

The present study investigated whether trauma exposure and trauma-related reactivity in the form of posttraumatic stress symptoms moderate the association between perceived stress and premenstrual symptom severity across the menstrual cycle. This approach moves beyond dyadic associations and instead tests a cycle-sensitive triangular model of stress, trauma, and premenstrual symptoms, thereby contributing to a more differentiated understanding of the underlying mechanisms and informing more targeted, cycle-phase–sensitive treatment approaches.

Using EMA data across two menstrual cycles and multilevel modelling, the study aimed to capture within-person fluctuations in stress and symptom expression while accounting for between-person differences in trauma history and posttraumatic stress symptoms.

Summary of the Main Findings

Several key findings emerged. First, there was a significant positive association of daily perceived stress with daily premenstrual symptom severity both at the within-person and between-person level. On days when participants experienced higher stress than usual, they reported more severe premenstrual symptoms. In addition, individuals with generally higher levels of stress reported overall higher symptom severity.

Second, trauma exposure itself did not moderate the association between daily stress and premenstrual symptoms significantly. In contrast, posttraumatic stress symptom severity significantly strengthened the association between daily stress and symptom severity. Individuals with higher PTSD symptoms showed a stronger coupling between daily stress and premenstrual symptoms.

Third, clear cycle-related differences in symptom expression were observed. Premenstrual symptoms were significantly elevated during the perimenstrual phase compared to the remainder of the cycle.

Fourth, the association between daily stress and premenstrual symptoms was significantly stronger during the perimenstrual phase, indicating increased stress sensitivity during this hormonally vulnerable period.

Finally, PTSD symptoms further amplified this cycle-sensitive stress reactivity: individuals with higher PTSD symptoms showed particularly strong stress-related symptom increases during the perimenstrual phase.

Stress-Symptoms-Association

The observed association between daily perceived stress and premenstrual symptom severity is consistent with previous research highlighting the role of stress in the development and maintenance of premenstrual symptoms. Meta-analytic findings indicate a moderate association between perceived stress and premenstrual symptom severity (Bencker, Tran, et al., 2025), and longitudinal studies have shown that increases in stress predict increases in symptom severity across menstrual cycles (Gollenberg et al., 2010).

Importantly, the present study extends this literature by demonstrating that stress–symptom associations operate not only between individuals but also within individuals across days. This supports the notion that stress functions as a dynamic trigger of symptom fluctuations rather than merely reflecting stable vulnerability factors.

Crucially, this association was not uniform across the menstrual cycle but was significantly stronger during the perimenstrual phase, indicating cycle-sensitive stress reactivity. This finding aligns with theoretical and empirical models suggesting that hormonal changes during the late luteal phase, particularly declining levels of E2, P4, and associated neurosteroid (such as ALLO) alterations, are linked to increased affective reactivity and stress sensitivity (Bencker, Tran, et al., 2025; Hantsoo & Epperson, 2020; Schweizer-Schubert et al., 2021).

These findings also align with ambulatory assessment research demonstrating phase-specific effects of cognitive processes in PMDD, showing that momentary cognitive states such as rumination predict mood changes particularly during the late luteal phase (Nayman et al., 2024).

Taken together, these findings highlight the interactions among hormonal, cognitive, and stress responses across the menstrual cycle, supporting a model in which vulnerability to stress is both person- and phase-specific. Accordingly, the present results support a cycle-sensitive stress model, in which everyday stress exerts stronger effects during hormonally vulnerable windows.

Trauma Exposure vs. Trauma Reactivity

A central contribution of the present study lies in the differentiation between trauma exposure and trauma-related psychological reactivity. While cumulative trauma exposure did not moderate the association between daily stress and premenstrual symptoms, PTSD symptom severity significantly amplified this relationship, suggesting that posttraumatic reactivity is critical rather than traumatic exposure alone, which appears to be insufficient for explaining individual differences in premenstrual symptom expression.

This finding refines existing models linking trauma and premenstrual disorders. Previous research has often treated trauma exposure as a general vulnerability factor (Eisenlohr-Moul et al., 2016; Grewal et al., 2025; Islas-Preciado et al., 2021; Perkonigg et al., 2004). However, the present results indicate that trauma may only translate into heightened symptom vulnerability when it results in persistent alterations in stress responsivity systems.

Within a stress sensitization framework, PTSD symptoms can be understood as reflecting lowered thresholds for stress reactivity, involving heightened emotional and physiological responses to everyday stressors. Building on the neuroendocrine mechanisms outlined above, this may involve dysregulation of the HPA axis and its interaction with the HPG axis (Hantsoo & Epperson, 2020), as well as altered neurosteroid modulation of GABAergic inhibitory circuits (Eisenlohr-Moul et al., 2016; Schweizer-Schubert et al., 2021). In susceptible individuals, increased sensitivity to cyclical neurosteroid fluctuations may amplify responses to stressors during the luteal phase, resulting in a stronger coupling between momentary stress and affective symptoms.

This interpretation is consistent with dimensional models such as Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle (DASH-MC), which conceptualize hormone sensitivity as dynamic and shaped by interacting contextual vulnerabilities (Peters et al., 2025). From this perspective, PTSD symptomatology may reflect a vulnerability profile characterized by heightened sensitivity to stress and cyclical hormonal change. Importantly, the present findings suggest that this stress sensitization is context-dependent, with particularly strong stress–symptom coupling during the perimenstrual phase, indicating an interaction between trauma-related stress reactivity and hormonally driven changes in emotional regulation.

Taken together, these findings support a model in which trauma-related reactivity, rather than trauma exposure per se, acts as a key mechanism linking stress processes to premenstrual symptom expression. This shifts the focus from static risk factors to dynamic vulnerability mechanisms and aligns with dimensional frameworks such as DASH-MC, which emphasize interacting, partially distinct sensitivity pathways (Peters et al., 2025).

Clinical Implications

The present findings have several important implications for the assessment and treatment of premenstrual disorders.

First, the observed cycle-sensitive stress reactivity indicates that psychological interventions for premenstrual symptoms may benefit from explicitly incorporating menstrual cycle dynamics. The stronger association between stress and symptom severity during the perimenstrual phase suggests that this period represents not only a window of increased vulnerability but also a potential window for targeted intervention. Accordingly, interventions focusing on stress regulation, rumination, or emotion regulation strategies during the late luteal phase may help attenuate symptom exacerbation.

This perspective is supported by recent research emphasizing the relevance of momentary cognitive processes in premenstrual mood dynamics (Nayman et al., 2024). In this context, accessible stress-reduction approaches (Gollenberg et al., 2010) may represent a particularly relevant element of psychotherapy, while also being valuable for individuals who do not have access to formal psychotherapy. Building on this work, digital mental health approaches using ecological momentary assessment data may further enable the development of just-in-time adaptive interventions that deliver tailored support during periods of elevated stress or symptom worsening.

This phase-specific vulnerability is also reflected in pharmacological treatment approaches. For PMDD and severe PMS, SSRIs can be administered continuously or intermittently during the luteal phase, with evidence suggesting that intermittent dosing may be a viable treatment strategy for some individuals (ACOG, 2023; Reilly et al., 2023). Such approaches may be clinically relevant because they reduce continuous medication exposure and may thereby be preferable for individuals concerned about side effects or long-term antidepressant use (Reilly et al., 2023).

Second, the findings highlight the importance of distinguishing between trauma exposure and trauma-related symptomatology in clinical contexts. The observation that PTSD symptoms, but not trauma exposure, moderated the stress–symptom association suggests that assessing trauma history alone may be insufficient; rather, clinicians should evaluate current trauma-related symptoms, as these more directly inform vulnerability to stress-related symptom exacerbation. This is consistent with previous research emphasizing the importance of screening for both trauma and PTSD in individuals with severe premenstrual symptoms, as comorbidity between these conditions may complicate diagnosis and treatment (Pilver et al.,

2011). Failure to recognize such comorbidity may obscure symptom dynamics and hinder effective treatment planning. Accordingly, the findings underscore the need for trauma-informed approaches to treatment. Interventions may benefit from addressing not only stress management but also trauma-related cognitive and affective processes, such as heightened threat sensitivity, maladaptive coping, or trauma-related core beliefs, in order to reduce stress sensitivity and mitigate symptom exacerbation.

Third, the findings highlight the importance of interdisciplinary collaboration between gynecology and mental health care. Although hormonal treatments and SSRIs are commonly used in the management of PMS and PMDD, the empirical evidence regarding their effectiveness remains heterogeneous, reflecting an incomplete understanding of the underlying neurobiological mechanisms and eventual genetic effects (Bencker, Tran, et al., 2025; Schweizer-Schubert et al., 2021; Yonkers et al., 2008). This uncertainty is particularly critical given that current models suggest that premenstrual disorders are not driven by abnormal hormone levels, but by altered sensitivity to normative hormonal fluctuations (ACOG, 2001; Bencker, Tran, et al., 2025; Dickerson et al., 2003; Eisenlohr-Moul et al., 2016; Schweizer-Schubert et al., 2021; Yonkers et al., 2008), which makes the use of hormonal treatments such as P4 supplementation appear theoretically ambiguous. While such treatments are being prescribed in clinical practice, which was earlier the case for one of our participants in the study as well, there is evidence suggesting that heightened P4 levels may be associated with dysphoric mood responses, particularly in individuals with trauma histories (Eisenlohr-Moul et al., 2016). This raises the possibility that certain pharmacological approaches may not only be ineffective for some patients but could even be counterproductive if underlying hormone sensitivities are not adequately considered.

These inconsistencies highlight the need for more precise diagnostic approaches and targeted treatment research. The DASH-MC framework offers a valuable extension in this regard, as it conceptualizes premenstrual symptomatology as arising from distinct, potentially overlapping dimensions of hormone sensitivity rather than a single, uniform mechanism (Peters et al., 2025). Potentially, these sensitivity types may be differently impacted by trauma, which remains to be examined. From this perspective, variability in treatment response, both for hormonal interventions and SSRIs, may reflect underlying heterogeneity in sensitivity profiles rather than inconsistent treatment efficacy per se.

Accordingly, effective treatment may require a more individualized and mechanism-informed approach that integrates endocrine, psychological, and contextual factors. Current clinical guidelines already emphasize the importance of multimodal treatment strategies

combining pharmacological, psychological, and lifestyle-based interventions (ACOG, 2023). Building on this, the present findings suggest that treatment planning should additionally incorporate cycle-sensitive and trauma-informed perspectives. Strengthening interdisciplinary collaboration between gynecology and mental health care may therefore not only improve symptom management but also facilitate the development of more precise, patient-centered interventions that account for the interaction between hormonal dynamics, stress processes, and trauma-related vulnerability.

Finally, from a broader public health perspective, the present findings underscore that premenstrual symptoms should not be trivialized as purely hormonal phenomena or dismissed as an inevitable aspect of "female nature." Rather, individuals with a female reproductive system experience cyclical endocrine processes that are fundamental to human reproduction but may, in interaction with psychosocial factors, increase vulnerability to affective and somatic symptom expression.

This perspective challenges societal stigma surrounding menstruation and premenstrual experiences that either perpetuate assumptions about female emotionality (such as earlier notions of "hysteria"), pathologize reproductive biology or dismiss associated suffering as normative and therefore clinically irrelevant. Historically, many conditions related to female reproductive health, including endometriosis, dysmenorrhea, and reproductive mood disorders, have been neglected or overlooked, with diagnoses often delayed and health systems remaining insufficiently responsive, despite substantial impacts on quality of life (Langer et al., 2015; Nnoaham et al., 2011; Samulowitz et al., 2018).

Importantly, these biological processes do not operate in isolation but are embedded within broader psychosocial contexts. Exposure to sexual abuse in childhood or adolescence has been associated with nearly twice the risk of MRMDs in adulthood compared to non-exposed individuals. At the same time, girls are substantially more likely to experience sexual violence than boys (Islas-Preciado et al., 2021). Thus, societal conditions that shape the gendered distribution of exposure to adversity may indirectly contribute to later vulnerability in reproductive mental health. This perspective is also reflected in the descriptive pattern of trauma exposure observed in the present sample. Across life periods, emotional forms of adversity such as feeling unloved and verbal abuse were consistently among the most frequently reported experiences, while phase-specific patterns also emerged, with neglect being particularly prominent in childhood and sexual harassment in adolescence and adulthood. Notably, approximately 75% of participants reported experiences of sexual harassment across the lifespan. Although the present sample is limited to individuals with a

female reproductive system and does not include a male comparison group, the observed pattern is consistent with previous reported gendered patterns of trauma exposure (Islas-Preciado et al., 2021).

From this perspective, addressing premenstrual symptomatology is not solely a matter of individual treatment but also of societal and clinical awareness and responsibility. Failing to acknowledge premenstrual symptoms as a legitimate health concern risks perpetuating avoidable suffering. A more differentiated understanding of reproductive mental health and the interacting effects of stress and trauma is therefore essential for more effective prevention, diagnosis, and intervention strategies.

Strengths and Limitations

From a methodological perspective, several strengths of the present study should be highlighted. First, the use of a prospective EMA design across two menstrual cycles allowed the investigation of stress and premenstrual symptoms in daily life while reducing retrospective recall bias.

Second, hormonally informed cycle tracking using LH kits increased cycle phase precision, addressing calls for more fine-grained and physiologically valid menstrual cycle research (Bencker, Tran, et al., 2025; Peters et al., 2025; Schmalenberger et al., 2021).

Third, the multilevel modelling approach enabled the simultaneous examination of within-person and between-person effects, allowing for a clear distinction between dynamic stress processes and stable individual differences.

Fourth, the study directly compared trauma exposure and PTSD symptoms, thereby differentiating between traumatic experiences and trauma-related reactivity.

Finally, the dimensional sample, not restricted to clinical diagnoses, allowed for the investigation of premenstrual symptom dynamics across a broader range of symptom severity.

Despite these strengths, several limitations should be considered when interpreting the findings.

First, the sample size was relatively small, which may limit statistical power and the generalizability of the findings. This is particularly relevant when interpreting the non-significant moderating effect of trauma exposure. Previous epidemiological research has shown that both trauma exposure and PTSD are associated with PMDD, with a graded relationship indicating stronger effects for trauma accompanied by PTSD compared to trauma exposure alone (Pilver et al., 2011). While this pattern is broadly consistent with the present

findings, the lack of a significant effect of trauma exposure in the current study may partly reflect limited statistical power. It is therefore possible that trauma exposure per se exerts a weaker, yet still meaningful, effect that could not be detected in the present sample.

Second, although saliva samples were collected multiple times per day, cortisol analyses were not yet available at the time of the present analysis. As a result, the study could not directly examine biological stress markers and instead relied on subjective stress measures. This is particularly relevant given that the theoretical model underlying the present study explicitly assumes altered HPA-axis functioning. Future analyses of the saliva data may therefore substantially enrich the interpretation of the present findings.

Third, although the study employed prospective symptom tracking across two cycles, it cannot determine causal directionality. It remains unclear whether stress increases symptoms or whether symptoms increase stress.

Lastly, a further limitation concerns sample composition: non-cis individuals with female reproductive organs were not represented in the present sample. This reflects a broader gap in literature and limits the generalizability of the findings.

Future research should further differentiate between potentially distinct hormonal sensitivity profiles such as luteal-onset, perimenstrual-onset, or periovulatory sensitivity as proposed by the DASH-MC framework (Peters et al., 2025) and should investigate whether trauma-related stress sensitization is associated with particular symptom timing patterns or with broader vulnerability across different forms of hormone sensitivity.

Combining EMA with endocrine biomarkers such as cortisol or neurosteroid measures would provide valuable insights into the biological mechanisms underlying stress-related premenstrual symptom expression. In addition, future treatment studies are needed to clarify whether the present findings can inform targeted, cycle-phase–sensitive interventions and whether trauma-related symptomatology moderates the effectiveness of existing pharmacological or psychotherapeutic treatment approaches.

Conclusion

The present study provides evidence that daily perceived stress is closely linked to fluctuations in premenstrual symptom severity and that this association is shaped by both menstrual cycle dynamics and trauma-related stress reactivity.

Crucially, the findings demonstrate that trauma-related psychological reactivity, rather than trauma exposure per se, moderates the stress–symptom association, thereby shaping individual differences and suggesting that it is the ongoing stress sensitivity associated with

PTSD symptomatology that amplifies symptom expression, particularly during the hormonally vulnerable late luteal phase of the menstrual cycle.

These results contribute to a growing body of literature highlighting the complex interaction between stress physiology (HPA axis), trauma-related vulnerability, and hormonal dynamics (HPG axis) in the development and maintenance of premenstrual symptoms. Importantly, they extend existing research by shifting the focus from static risk factors (e.g., trauma exposure) to dynamic, process-based mechanisms of vulnerability. In this sense, the present study contributes to a broader understanding of the etiology, worsening, and maintenance of premenstrual symptoms. Future research should further investigate the so far proposed mechanisms and conduct randomized controlled trials to evaluate targeted treatment approaches.

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6. Tables

7. Figures

8. Abbreviations

ACOG	American College of Obstetricians and Gynecologists
ACTH	Adrenocorticotropic Hormone
ALLO	Allopregnanolone
APA	American Psychiatric Association
AVP	Arginine Vasopressin
CRH	Corticotropin-Releasing Hormone
DASH-MC	Dimensional Affective Sensitivity to Hormones across the Menstrual Cycle
DSM-5	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
E2	Estrogen
EMA	Ecological Momentary Assessment
FSH	Follicle-Stimulating Hormone
GABA	Gamma-Aminobutyric Acid
GnRH	Gonadotropin-Releasing Hormone
HPA	Hypothalamic–Pituitary–Adrenal
HPG	Hypothalamic–Pituitary–Gonadal
ICD-11	International Classification of Diseases, 11th Revision
ITEM	International Trauma Exposure Measure
ISSAC	Interactions in Premenstrual Symptoms and Stress Across the Menstrual Cycle
LH	Luteinizing Hormone
MLM	Multi-Level Model
MRMD	Menstrual-Related Mood Disorder
P4	Progesterone
PCL-5	PTSD Checklist for DSM-5
PMDD	Premenstrual Dysphoric Disorder
PMS	Premenstrual Syndrome
PSD	Premenstrual Symptom Diary
PTSD	Posttraumatic Stress Disorder
SSRIs	Selective Serotonin Reuptake Inhibitors
WHO	World Health Organization

9. Appendix

9.1. Abstract

Associations between perceived stress and premenstrual symptoms are well established, yet the underlying mechanisms remain unclear. Trauma has been identified as a risk factor, but it remains unclear whether trauma exposure itself or trauma-related symptomatology drives this relationship. The present study investigated whether trauma exposure and posttraumatic stress disorder (PTSD) symptoms differentially moderate the association between daily stress and premenstrual symptoms.

Data were drawn from a larger-scale longitudinal ecological momentary assessment study (ISSAC), in which 44 regularly menstruating women (*M_age* = 27.89; *SD* = 5.10) provided daily self-reports of stress and premenstrual symptoms across two menstrual cycles. Trauma exposure and PTSD symptoms were assessed at baseline. Menstrual cycle phase was hormonally validated using ovulation tests. Cycle-sensitive multilevel models were used to examine stress effects on premenstrual symptoms and their moderation by trauma exposure and PTSD symptoms, contrasting perimenstrual with non-perimenstrual days.

Higher daily stress was associated with increased premenstrual symptoms, with the stress–symptom relationship being stronger during the perimenstrual phase than during non-perimenstrual days. Trauma exposure did not moderate this association. In contrast, PTSD symptom severity significantly strengthened the stress–symptom relationship. These findings suggest that trauma-related symptomatology, rather than trauma exposure per se, indicates altered stress responsivity underlying premenstrual symptom severity. Such alterations may make susceptible individuals particularly vulnerable to symptom expression, particularly during the hormonally vulnerable perimenstrual phase. This shifts the focus from static risk factors to dynamic mechanisms of vulnerability, informing more precise, cycle-sensitive diagnostic and intervention approaches. Future research should examine the effectiveness of existing pharmacological and psychotherapeutic treatments for premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD), with particular attention to the moderating role of trauma-related symptomatology.

9.2. Zusammenfassung

Der Zusammenhang zwischen wahrgenommenem Stress und prämenstruellen Symptomen ist gut belegt, während die zugrunde liegenden Mechanismen weiterhin unklar bleiben. Trauma gilt zwar als Risikofaktor, doch ist bislang nicht geklärt, ob bereits die Exposition oder erst traumaassoziierte Symptomatik die Kopplung von Stress und prämenstrueller Symptomatik verstärkt. Die vorliegende Studie geht dieser Frage nach, indem sie untersucht, inwieweit Traumaerfahrungen und posttraumatische Belastungssymptome (PTBS) den Zusammenhang zwischen täglichem Stress und prämenstrueller Symptomschwere moderieren.

Die Analyse basiert auf Daten einer größeren longitudinalen Ecological-Momentary-Assessment-Studie (ISSAC), in der 44 regelmäßig menstruierende Frauen (*M_Alter* = 27.89; *SD* = 5.10) über zwei Menstruationszyklen hinweg tägliche Angaben zu Stress und Symptomatik machten. Traumaexposition und PTBS-Symptome wurden zu Studienbeginn erhoben; die Zyklusphase wurde hormonell mittels Ovulationstests validiert. Zur Auswertung kamen zyklussensitive Multilevel Modelle zum Einsatz, in denen die perimenstruelle Phase und nicht-perimenstruelle Tage miteinander verglichen wurden.

Die Ergebnisse zeigen einen robusten positiven Zusammenhang zwischen täglichem Stress und prämenstrueller Symptomschwere, wobei dieser Zusammenhang in der perimenstruellen Phase stärker ausgeprägt war als an nicht-perimenstruellen Tagen. Während sich für die Traumaexposition kein moderierender Effekt nachweisen ließ, verstärkte die Ausprägung der PTBS-Symptomatik den Zusammenhang signifikant.

Damit sprechen die Befunde dafür, dass nicht die Erfahrung eines Traumas an sich, sondern erst die daraus resultierende Symptomatik auf eine veränderte Stressreaktivität hinweisen könnte. Diese veränderte Stressregulation könnte Betroffene insbesondere in der hormonell vulnerablen perimenstruellen Phase anfälliger für eine Zunahme prämenstrueller Symptome machen. Der Fokus verschiebt sich damit von statischen Risikofaktoren hin zu dynamischen Vulnerabilitätsmechanismen. Daraus ergeben sich Ansatzpunkte für präzisere, zyklussensitive diagnostische und therapeutische Strategien sowie für zukünftige Forschung zur Frage, inwieweit traumaassoziierte Symptomatik die Wirksamkeit bestehender pharmakologischer und psychotherapeutischer Interventionen beim prämenstruellen Syndrom (PMS) oder der prämenstruellen dysphorischen Störung (PMDS) beeinflusst.

9.3. Additional Results

Table A1

Unstandardized Multilevel Model Predicting Premenstrual Symptom Severity From Stress, Without and With Adjustment for Perimenstrual Phase

	Without perimenstrual dummy				**With perimenstrual dummy**			
Predictor	***b***	***SE***	***t***	***p***	***b***	***SE***	***t***	***p***
Intercept	0.137	0.123	1.11	0.272	0.013	0.124	0.10	0.918
Within-person stress	0.010	0.001	8.91	< .001***	0.008	0.001	7.99	< .001***
Between-person stress	0.012	0.003	3.97	< .001***	0.012	0.003	3.89	< .001***
Perimenstrual phase					0.338	0.016	21.32	< .001***
Stress × Perimenstrual phase					0.003	0.001	3.41	< .001***

Note. *b* = regression coefficient; *SE* = standard error; *t* = t statistic; *p* = p value. Within-person stress represents person-mean centered daily deviations. The perimenstrual phase variable was dummy coded (0 = outside the perimenstrual phase, 1 = perimenstrual phase). Models include random intercepts and random slopes for within-person stress at the participant level.
*$p < .05$, **$p < .01$, ***$p < .001$

Table A2

Unstandardized Multilevel Model Testing Trauma Load as a Moderator of the Stress–Symptom Association, Without and With Adjustment for Perimenstrual Phase

	Without perimenstrual dummy				**With perimenstrual dummy**			
Predictor	***b***	***SE***	***t***	***p***	***b***	***SE***	***t***	***p***
Intercept	0.114	0.119	0.96	0.343	0.037	0.120	0.31	0.758
Within-person stress	0.009	0.002	4.51	< .001***	0.007	0.002	3.90	< .001***
Between-person stress	0.009	0.003	2.84	0.007**	0.009	0.003	2.84	0.007**
Trauma load	0.041	0.016	2.55	0.015*	0.024	0.016	1.46	0.152
Perimenstrual phase					0.181	0.028	6.44	< .001***
Stress × Trauma load	0.000	0.000	0.45	0.655	0.000	0.000	0.66	0.511
Stress × Perimenstrual phase					0.003	0.001	2.25	0.024*
Trauma load × Perimenstrual phase					0.043	0.006	6.69	< .001***
Stress × Trauma load × Perimenstrual phase					0.000	0.000	-0.38	0.703

Note. *b* = regression coefficient; *SE* = standard error; *t* = t statistic; *p* = p value. Within-person stress represents person-mean-centered daily deviations. The perimenstrual phase variable was dummy coded (0 = outside the perimenstrual phase, 1 = perimenstrual phase). Models include random intercepts and random slopes for within-person stress at the participant level.
*$p < .05$, **$p < .01$, ***$p < .001$

Table A3

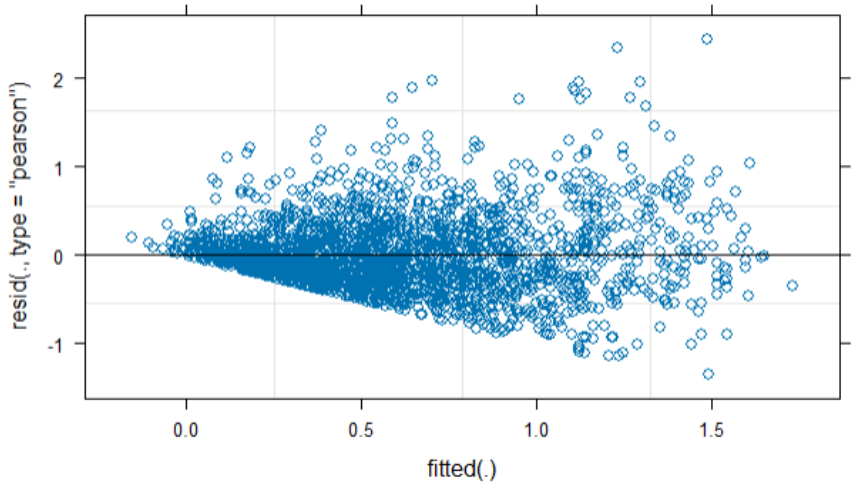
Unstandardized Multilevel Model Testing PTSD Symptom Severity as a Moderator of the Stress–Symptom Association, Without and With Adjustment for Perimenstrual Phase

	Without perimenstrual dummy				With perimenstrual dummy			
Predictor	***b***	***SE***	***t***	***p***	***b***	***SE***	***t***	***p***
Intercept	0.312	0.125	2.49	0.017*	0.177	0.126	1.40	0.168
Within-person stress	0.010	0.001	9.14	< .001***	0.008	0.001	8.48	< .001***
Between-person stress	0.008	0.003	2.42	0.020*	0.008	0.003	2.40	0.021*
PTSD symptom severity (centered)	0.012	0.003	3.78	< .001***	0.009	0.003	2.73	0.009**
Perimenstrual phase					0.338	0.016	21.50	< .001***
Stress × PTSD symptoms	0.000	0.000	2.09	0.043*	0.000	0.000	1.37	0.177
Stress × Perimenstrual phase					0.003	0.001	3.08	0.002**
PTSD symptoms × Perimenstrual phase					0.008	0.001	5.91	< .001***
Stress × PTSD symptoms × Perimenstrual phase					0.000	0.000	2.11	0.035*

Note. *b* = regression coefficient; *SE* = standard error; *t* = t statistic; *p* = p value. Within-person stress represents person-mean-centered daily deviations. The perimenstrual phase variable was dummy coded (0 = outside the perimenstrual phase, 1 = perimenstrual phase). Models include random intercepts and random slopes for within-person stress at the participant level. *$p < .05$, **$p < .01$, ***$p < .001$

Figure A1

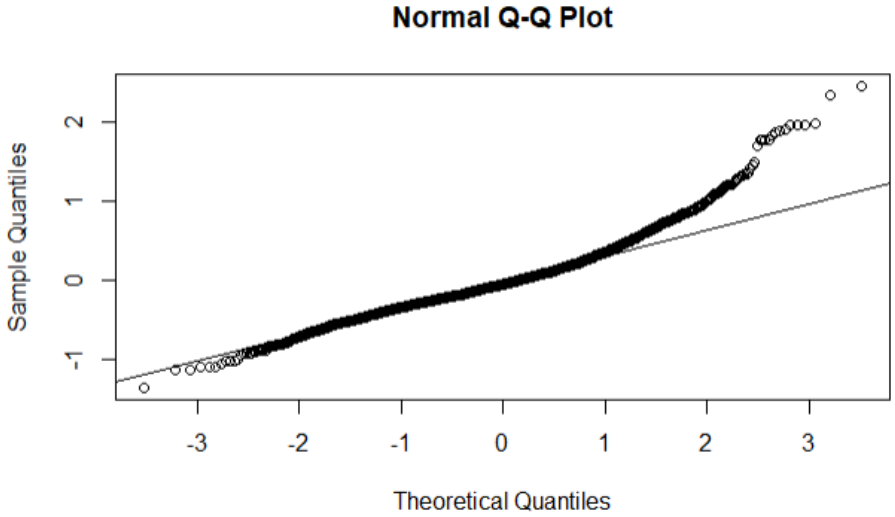
Residuals Versus Fitted Values Plot for the Multilevel Model



Note. Pearson residuals plotted against fitted values from the multilevel model predicting premenstrual symptom severity.

Figure A2

Normal Q–Q Plot of Residuals



Note. The reference line represents the expected distribution under normality.

9.4. Statement of Authorship and Use of AI Tools

I hereby declare that I have written my master's thesis titled "The Influence of Trauma on the Relationship Between Perceived Stress and Premenstrual Symptoms" independently and that I have used only the sources, materials, and aids indicated. I further declare that I used artificial intelligence tools in the preparation of this thesis. OpenAI's ChatGPT (GPT-5.5 Thinking) was used to support the development of R code, assist in the interpretation of statistical results, translate self-written passages into English, support the paraphrasing and reformulation of selected text passages, and improve language clarity, grammar, and academic style. All outputs generated by the AI tool were critically reviewed, adapted where necessary, and incorporated under my own responsibility. I take full responsibility for the selection, evaluation, and use of all AI-assisted results included in this thesis.