# Poor Sleep and Emotion Dysregulation Mediate the Association between Depressive and Premenstrual Symptoms in Young Adult Women

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Conflict of Interest: Jessica Meers declares that she has no conflict of interest. Joanne Bower declares that she has no conflict of interest. Candice Alfano declares that she has no conflict of interest.

ABSTRACT

**Purpose:** A large portion of reproductive-aged women report experiencing distressing premenstrual symptoms. These symptoms can be exacerbated by concurrent mood problems and contribute to long-term depressive risk. However, difficulty sleeping and regulating emotional responses are also associated with the premenstrual phase and represent additional, well-established risk factors for depression. The aim of this study was to investigate whether habitual sleep problems and emotion regulation strategies serve to mediate the relationship between mood and premenstrual symptoms in non-treatment seeking young women.

**Methods:** Participants included 265 adult women between the ages of 18 and 25 who provided retrospective self-reports of depressive symptoms, habitual sleep quality, and premenstrual symptoms for the past month. Trait-based difficulties in regulating emotions were also assessed.

**Results:** Greater depressive symptoms significantly predicted greater premenstrual symptoms and both poor sleep and ineffective emotion regulation were shown to mediate this relationship.

**Conclusions:** Poor sleep may enhance experience of premenstrual symptoms via its well-established impact on physical, cognitive, and/or affective functioning. Similarly, an inability to effectively regulate emotional responses in general may exacerbate experience or perception of somatic and mood symptoms during the premenstrual period, contributing to mood disturbances and risk. Findings require replication in future studies using prospective designs and more diverse samples of women.

**Keywords:** Premenstrual symptoms; depression; sleep; emotion regulation

For many women, the premenstrual phase of the monthly cycle is marked by a range of physical, emotional, and behavioral symptoms that can significantly impact daily functioning. In the U.S., between 70-90% of reproductive age women experience some level of premenstrual discomfort or distress (Mishell 2005). Physical symptoms such as cramping, bloating, body aches, headaches, and breast tenderness are commonly recognized symptoms of the premenstrual period (Biggs and Demuth 2011), but depressive symptoms also occur, with 3-8% of women experiencing symptoms severe enough to fulfill criteria for premenstrual dysphoric disorder (PMDD) (Halbreich et al. 2003). In addition to physical and mood-based changes, sleep disruption and difficulties regulating emotional responses coincide with the premenstrual phase of the monthly cycle (Bowen et al. 2011; Liu et al. 2017; National Sleep Foundation 2007; Wu et al. 2014). However, far fewer studies have examined sleep and emotion regulation in relation to one's premenstrual symptoms, and these symptoms remain poorly understood overall (for review see Sundström-Poromaa & Gignell, 2014). The current study sought to better understand potential relationships among depressive symptoms, habitual poor sleep, and disrupted emotion regulation across the month on symptoms in the premenstrual phase.

Premenstrual symptoms can cause significant impairment, particularly for women already experiencing depressive symptoms (Kornstein et al. 2005). Young adult women with elevated general mood symptoms report greater premenstrual symptoms, including disruptions in affect, increased irritability, pain, and bloating related to their menstrual cycle (Acikgoz et al. 2017). Among women with diagnosed mood disorders, a majority (50-66%) experience premenstrual exacerbation of depressive symptoms (Haley et al. 2013). Depressed women also experience greater somatic symptoms and report more health complaints than their non-depressed counterparts and this is particularly true for those experiencing premenstrual symptoms (Forrester-Knauss et al. 2011; Wilson et al. 1983). Further, those who experience premenstrual worsening of symptoms show longer time to depression recovery (Haley et al.

2013) implying that exacerbation of premenstrual symptoms impacts the course and prognosis of mood disorders.

Clinical and sub-clinical levels of depression are also characterized by ineffective or dysfunctional use of regulatory processes to manage emotional responses. For instance, young adults at risk for depression haven been shown to exhibit disrupted affective reactivity in response to daily stressors (Cohen et al. 2005). Maladaptive coping strategies, such as rumination, suppression, and avoidance are often used in lieu of more adaptive processes, such as reappraisal and problem-solving (Aldao et al. 2010). This may be particularly problematic during the premenstrual period, when enhanced sensitivity to stress and increased difficulty regulating emotion in response to stress are observed (Lusk et al. 2017; Ossewaarde et al. 2013; Petersen et al. 2016; Wu et al. 2016). In fact, there is evidence that women with more dysfunctional trait-level emotion regulatory skills are at increased risk for premenstrual affective symptoms (Sigmon 2009; Welz et al. 2016). Thus, use of maladaptive emotion regulatory strategies may serve to exacerbate the relationship between mood disturbances and premenstrual symptoms.

Another putative yet relatively unexplored contributory factor is sleep. As many as one third of reproductive-aged women report disturbed sleep related to their menstrual cycle (National Sleep Foundation 2007). Those with greater sleep-related complaints across the month report more negative premenstrual symptoms, such as dysmenorrhea (Woosley and Lichstein 2014). Sleep-wake disruption during the premenstrual phase is related to the presence of physical and emotional symptoms (e.g., pain) as well as an increase in core body temperature (Sharkey et al. 2014). Premenstrual sleep therefore tends to be more fragmented and qualitatively poorer (Baker and Driver 2007) even in the absence of objectively identified sleep disruption (Baker et al. 2012).

Although the role of prolonged sleep disruption in relation to the menstrual cycle is less understood, reciprocal links between poor sleep and depressive symptoms are well-established.

Sleep disruption is a core symptom of affective disorders and predicts treatment non-response (Harvey et al. 2013). Similarly, insomnia represents one of the most robust predictors of future depression (Ford and Kamerow 1989). These pervasive relationships are theorized to be rooted in the shared neurobiology of sleep and emotional processing (Yoo et al. 2007) as emotional reactivity to both positive and negative stimuli is amplified when sleep is inadequate (Gujar et al. 2011). Sleep disruption and/or poor sleep quality also predicts significant dysregulation of next day affect (Chue et al. 2018; Kalmbach et al. 2014) and subsequent depressive symptoms in non-depressed women (de Wild-Hartmann et al. 2013). These relationships nonetheless remain unexplored in the context of women's monthly reproductive cycles. The current study sought to address these notable knowledge gaps using a sample of non-treatment seeking young adult women. Based on existing research, we first hypothesized that greater depressive symptoms would predict report of worse premenstrual symptoms. Secondly, we hypothesized that both habitual self-reported poor sleep quality and greater difficulties in regulating emotional responses would interactively mediate the relationship between depressive and premenstrual symptoms. Additionally, in the absence of prior studies, we explored the role of specific premenstrual symptom clusters (i.e., cognitive, physical, affective) within this model.

## METHOD

### Participants

Female undergraduates between the ages of 18 and 25 were recruited from a tier-one research institution in Southeastern Texas to participate in a study on menstrual health. This narrow age range was selected to ensure a homogenous reproductive age range, which is associated with significant menstrual symptoms (Patel et al. 2006). Other inclusion criteria required that women have a naturally occurring menstrual cycle without the use of hormonal contraceptives, were not pregnant or trying to conceive, and the absence of a menstrual-related health disorder (e.g., endometriosis and/or fibroids). Individuals self-reporting a formal diagnosis

of PMDD were not included to avoid inflating estimated relationships of interest (i.e., severe mood and other premenstrual symptoms are required for a PMDD diagnosis).

A total of 526 participants enrolled in the study through the university online undergraduate research panel (SONA). Of these, 182 did not meet the eligibility criteria outlined above, and a further 79 were removed for incomplete responses (details regarding excluded participants are included below). Thus, for the current study, data from 265 participants were included for analysis. Participants were racially and ethnically diverse women (see Table 1) with a mean age of 20.75 years (*SD* = 1.80 years). Participants had a mean menstrual cycle length of 27.80 (*SD* = 4.85) days and menstrual cycle regularity varied from "Usually or always irregular" (*n* = 52; 19.6%) to "Always regular" (*n* = 90; 34.0%). Four participants reported regular use of psychotropic medications. Full participant characteristics are displayed in Table 1.

## Measures

*Screening Questionnaire.* Participants reported demographic information, including age, racial and ethnic background, and health history. Menstrual health questions assessed menstrual history (age of menarche, pregnancy history, and history of menstrual-related health disorders), date of most recent menstrual period, and typical physical/emotional premenstrual symptoms.

*Center for Epidemiologic Studies Depression Scale* (CESD; Radloff, 1977) . This 20-item self-report measure is designed to assess depressive symptoms and was modified to assess symptoms across the previous month. Answers are provided on a 0-3 Likert scale assessing the frequency of symptoms over the past month. Scores are summed and higher scores are indicative of greater depressive severity[1], with scores above 16 considered clinically significant. Reliability in the current sample was high (Cronbach's α = .91).

[1] Total CESD was computed with and without the item addressing sleep.

*Moos Menstrual Distress Questionnaire* (Moos 1968). This 46-item measure is used to measure the experience of 46 symptoms during the premenstrual, menstrual, and intermenstrual phases of the most recent menstrual cycle. Each symptom is assessed on a 4 point Likert scale (no experience of the symptom to present, severe). Eight symptom clusters are assessed, including pain, concentration, behavioral changes, autonomic reactions, water retention, negative affect, arousal, and control. A total premenstrual distress score[2] was computed by summing each of the negative subscales (pain, concentration, behavioral changes, autonomic reactions, water retention, negative affect, and control) and subtracting arousal. Reliability in the current sample was high (Cronbach's α = .96).

*Pittsburgh Sleep Quality Index* (PSQI; Buysse, Reynolds, Mon, Berman & Kupfur, 1989). The PSQI assesses seven dimensions of sleep quality over the previous month to equal one global score of sleep quality. Higher scores are indicative of greater sleep difficulties, with a cut off score greater than 5 used to distinguish "poor" from "good" sleepers. In the current sample, reliability was acceptable (Cronbach's α = .60).

*Difficulties in Emotion Regulation* Scale (DERS; Gratz & Roemer, 2004). This 36-item scale assesses difficulties in engaging with different facets of emotion regulation that individuals regularly engage in. These include non-acceptance of emotional responses, difficulty engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity. Items are measured on a 1 to 5 Likert scale, with higher scores indicating greater difficulties. Full-scale reliability in the current sample was excellent (Cronbach's α = .94).

Procedures

[2] Total MDQ was computed with and without 2 items which addressed depression and naps

Participants were undergraduate students recruited in exchange for course credit at a Tier-one research institution in Southeastern Texas. All participants completed measures online via Qualtrics questionnaire software (www.qualtrics.com). After providing informed consent, individuals completed a 60 minute online survey assessing premenstrual symptoms, emotional responding, typical sleeping patterns, and physical and emotional health. Only the measures outlined above were included for analyses in the current study. Ethical approval was obtained from the University of Houston's Institutional Review Board (IRB). Per the university's IRB guidelines, participants have the right to opt out of answering any question.

## Data Analyses

All analyses were conducted using SPSS version 25 (IBM, 2017) and PROCESS version 3.1 (Hayes 2017). Individuals were removed from the analysis if they reported using hormonal contraceptives ($n$ = 151), did not have periods ($n$ = 3) or reported having been diagnosed with a menstrual-related disorder ($n$ = 24), were outside of the specified age range (18-25 years; $n$ = 4), or did not provide usable data (i.e., selected 'prefer not to answer' for greater than 25% of questions within a measure, or withdrew from the study prior to completing measures of interest). For responses with < 25% missing data, individuals' subscale means were used to estimate values for missing items. All models were run with and without the inclusion of participants who had imputed values.

Mediation analyses were conducted in several stages. First, correlations were examined to check for potential covariates and assess overall relationships between variables. Next, if significant associations between predictor, mediator, and outcome variables were detected (Baron and Kenny 1986), PROCESS (model 6) with bootstrapping (10,000 samples) was used to estimate direct (depressive symptoms predicting premenstrual symptoms) and indirect effects of two mediators (sleep quality and emotion dysregulation) within the same model. Finally, if partial or total mediation was detected, premenstrual symptoms were differentiated by type

(physical, affective, cognitive) and further exploratory analyses were conducted to assess the predictive value of depressive symptoms, sleep quality and emotion dysregulation on these different symptom clusters. For all analyses, models were tested using all participants, and also removing participants with any missing values. Similarly, analyses including the full scales for depression and pre-menstrual symptoms were repeated using scale totals where items relating to sleep (CESD, MDQ) and depression (MDQ) were removed.

## RESULTS

### Data cleaning and imputation

A total of 182 responses were removed from analyses because participants did not meet the inclusion criteria (see above). A further 79 participants did not provide data for all measures of interest (*n* = 50) or selected 'prefer not to answer' for more than 25% of one of the questionnaires (*n* = 13). For the CESD, MDQ, and DERS, where individuals had selected 'prefer not to answer' for less than 25% of responses, missing items were imputed using the mean of other subscale items (reversed as appropriate). This resulted in imputation for 3 participants for the MDQ (median 2 items); 6 participants each for the DERS and PSQI, and 5 participants for the CESD (all medians 1 item).

For the PSQI, mean responses were imputed as required for items answered on 0-3 scale (sleep disturbance items). If data from other items (e.g., bed time, wake time, total sleep time) was missing, the full response was removed (*n* = 16). Final analyses were conducted using 265 responses including imputations (248 responses when participants with missing data were removed). The pattern of results did not differ depending on whether participants with imputed values were included. Similarly, the pattern of results remained the same regardless of

whether full scales were used, or overlapping items were removed. Thus the reported analyses include full measures and all participants[3].

Preliminary analyses and bi-variate associations

Analyses were first conducted to identify potential demographic and premenstrual variables associated with the outcome variable (total premenstrual symptoms). No significant associations were detected between age, menstrual frequency, menstrual regularity, current menstrual phase, and premenstrual symptoms. Similarly, no differences in premenstrual symptoms were detected between racial and ethnic groups, or based on whether the participant was currently menstruating. Consequently, all subsequent analyses were conducted without covariates.

Total menstrual symptoms (MDQ total) were positively and moderately associated with depressive symptoms (CESD; $r = .56$, $p < .001$), sleep quality (PSQI total; $r = .46$, $p < .001$), and emotion dysregulation (DERS total; $r = .53$, $p < .001$). Depressive symptoms were also significantly associated with sleep quality ($r = .47$, $p < .001$) and emotion dysregulation ($r = .74$, $p < .001$). Likewise, the proposed mediators (sleep quality and emotion dysregulation) were positively associated ($r = .34$, $p < .001$).

Mediation Analyses

Power analyses (G*Power version 3.1.9.4; Faul, Erdfelder, Buchner, & Lang, 2009) based on correlation coefficients of .30, indicated that a minimum of 145 participants were required for regression analyses of 6 or less predictors. Thus the current study was adequately powered to assess the direct and indirect effects of depressive symptoms on premenstrual symptoms through sleep quality and emotion dysregulation. Higher self-reported depressive

[3] Analyses including only participants with no missing data, and reduced scales (with overlapping items removed) are available in supplemental file.

symptoms significantly predicted greater endorsement of premenstrual symptoms, with the unmediated direct effect accounting for 32.0% of the variance (*b* = 1.02, *se* = .09, 95% CI [.84, 1.20]).

The full mediation model (Figure 1) in which depressive symptoms, sleep quality, emotion dysregulation, and premenstrual symptoms were sequentially predicted was not supported (*b* = .00, *se* = .01, 95% CI [-.03, .02]). This was due to the non-significant pathway between sleep quality and emotion dysregulation (*b* = -.07, *se* = .36, 95% CI [-.78, .64]). In light of this result, subsequent analyses assessed the mediating effects of sleep and emotion dysregulation in separate models (PROCESS model 4).

First, sleep quality was included as a mediator between depression and premenstrual symptoms (Figure 2). The model accounted 36.76% of the variance, and both the mediated direct (*b* = .81, *se* = .10, 95% CI [.61, 1.01]) and overall indirect (*b* = .21, *se* = .06, 95% CI [.08, .33]) effects were significant, suggesting partial mediation of sleep quality on the relationship between depression and premenstrual symptoms. Second, emotion dysregulation also partially mediated the association between depressive symptoms and premenstrual symptoms (overall indirect effect: *b* = .32, *se* = .12, 95% CI [.10, .56]). The direct effect again remained significant (*b* = .69, *se* = .13, 95% CI [.43, .95]), with the mediation model accounting for 34.78% of the variance.

Exploratory Mediation Analyses

Additional analyses assessing the predictive value of depressive symptoms, sleep, and emotion dysregulation on physical, cognitive, and affective premenstrual symptoms were conducted using the pain, concentration, negative affect and arousal MDQ subscales respectively. To account for multiple comparisons, a Bonferroni correction was applied, thus the significance threshold for models was $p < .006$. Significant direct effects were present between

depressive symptoms and premenstrual pain (Figure 2a; $b$ = .12, $se$ = .02, 95% CI [.08, .17], $r^2$ =.11), concentration (Figure 2b; $b$ = .23, $se$ = .02, 95% CI [.18, .27], $r^2$ = .25), negative affect (Figure 2c; $b$ = .28, $se$ = .03, 95% CI [.23, .34], $r^2$ = .29) symptoms. The direct effect between depressive symptoms and premenstrual arousal was non-significant following Bonferroni correction ($b$ = .06, $se$ = .02, $p$ = .02, 95% CI [.01, .10], $r^2$ = .02).

Similar to main mediation analysis, the mediating effects of sleep quality and emotion dysregulation were again assessed in separate models. For pain symptoms, sleep quality but not emotion dysregulation was a significant mediator of the association between depressive symptoms and premenstrual pain. The indirect pathway via sleep quality ($b$ = .05, $se$ = .01, 95% CI [.02, .08]) explained 17.9% of the variance in premenstrual pain symptoms.

The association between depressive symptoms and premenstrual concentration were significantly mediated by sleep quality (indirect effect: $b$ = .06, $se$ = .02, 95% CI [.02, .09]) accounting for 31.1% of the variance in premenstrual concentration. Emotion dysregulation also significantly mediated this association (indirect effect: $b$ = .08, $se$ = .03, 95% CI [.02, .15]) accounting for 28.2% of symptom variance.

Similarly, in models assessing associations between depressive symptoms and premenstrual negative affect, both sleep quality (indirect effect: $b$ = .04, $se$ = .02, 95% CI [.01, .07]) and emotion dysregulation (indirect effect: $b$ = .12, $se$ = .03, 95% CI [.05, .18]) were significant mediators, explaining 30.6% (sleep quality) and 32.9% (emotion dysregulation) of the variance in premenstrual negative affect.

## DISCUSSION

Despite the well-established independent relationships that exist between depression, poor sleep, and emotion dysregulation, few studies have explored these problems in relation to the premenstrual period. The primary aim of the current study was to investigate the role of

sleep and emotion dysregulation as potential contributors to the relationship between depressive symptoms and premenstrual symptomatology. Based on prior work, we hypothesized that habitual poor sleep quality and greater difficulties in regulating emotional responses would interactively mediate the relationship between depressive symptoms and premenstrual symptoms. Indeed, analyses identified a direct pathway from depressive to premenstrual symptoms and revealed sleep and emotion dysregulation to exert mediational effects. However, contrary to hypotheses, sleep did not predict emotion dysregulation. Thus, poor sleep and emotion dysregulation appear to represent co-occurring rather than interactive factors in the context of mood disturbances and elevated premenstrual symptoms.

The association between depression and disrupted sleep is well established (Ford and Kamerow 1989). Similarly, the rise and fall of progesterone during the premenstrual phase is known to contribute to sleep disruption (Sharkey et al. 2014). Poor sleep is also independently linked with increased perception of and sensitivity to pain (Larson and Carter 2016), impairments in cognitive functioning (Killgore 2010), and disrupted affect (Kalmbach et al. 2014); symptoms that, when occurring together at the end of the menstrual cycle, form the basis of premenstrual symptomatology. It is perhaps unsurprising then that we found poorer sleep quality to predict greater total premenstrual symptoms and symptomatology across physical, cognitive and emotional domains specifically. However, the current study is, to our knowledge, the first to suggest that habitual sleep disruption may contribute to the worsening of premenstrual symptoms in those with elevated depressive symptoms.

The significant pathway between emotion dysregulation and premenstrual symptoms suggests that those with greater difficulties accessing and applying adaptive strategies to manage their experiences of negative emotions (including perhaps, the cognitive and affective symptoms of the late menstrual cycle), are more susceptible to, or at least more sensitive to these changes. Emotional changes occurring during this menstrual phase are in part biological

(Sundstrom-Poromaa 2018) but impairments in the ability to regulate these emotional experiences could lead to enhanced negative effects. As emotion dysregulation is also a core feature of depression (Aldao et al. 2010), it may be that those with greater depressive symptoms have greater difficulty refocusing their attention away from and/or reframing their negative cognitions in relation to their physical, cognitive, and emotional symptoms, leading to greater premenstrual problems.

We expected that sleep quality would be related to emotion dysregulation as this relationships has been consistently found in other studies (Palmer and Alfano 2017). The fact that this was not born out in our data is surprising but may reflect the need for multi-dimensional measures of sleep and emotional functioning. Indeed, many of the associations between sleep and emotional outcomes have focused on sleep duration specifically (e.g. Watling et al. 2017), rather than a subjective measure of habitual sleep quality as used in the current study. Notably, assessment of sleep quality relies on subjective perception of the overall restfulness of one's sleep and is informed by numerous aspects of sleep and wake (Ohayon et al. 2017). Additionally, when considering the interaction between sleep and emotional responding in relation to the menstrual cycle, the use of longitudinal designs that can capture day-to-day variability in responding (e.g., EMA) may be beneficial in better understanding these nuanced relationships.

Limitations and considerations

Our current work should be considered in light of several limitations. First, our sample was found to have relatively high depression scores. Although not entirely unusual for the age range utilized (Radloff 1991), this may be related to the time of year when data collection was conducted, which coincided with final exams for many students and may have artificially inflated scores. Additionally there is potential for selection bias, as those who participated in the study may have chosen to do so because they experience elevated premenstrual, mood and/or sleep-

related problems. However, there was significant spread in CESD scores across the sample, and reporting of depressive symptoms was based on the past month (rather than a week, which is typical for many self-report scales). Similarly, average sleep disturbance was in the clinical range – and notably higher than reported population norms. This is likely reflective of our sample of undergraduates, who are at greater risk of insufficient sleep than the general population (Owens et al. 2017). Inherent in this demographic are variable sleep patterns and 'social jetlag' (Forquer et al. 2008). Findings from the current study await replication in more heterogeneous, community-based samples and with objectively measured sleep variables, such as actigraphy. Finally, inherent in any cross-sectional study is the inability to make causal inferences regarding associations. Indeed, it is plausible that greater experience of monthly premenstrual symptoms increases overall depression through dysregulated emotional responding and poor sleep. The current study nonetheless provides a useful basis from which to design longitudinal and experimental research to test the causal directions of these associations.

**Conclusions.** The finding that habitually poor sleep quality and emotion dysregulation influence the relationship between depressive symptoms and premenstrual symptomatology has important clinical implications. Specifically, because regulation of sleep and emotion are two modifiable processes, they may prove to be useful treatment targets for menstrual distress and depression. If premenstrual distress in those seeking treatment for depression can be mitigated, to some degree, via interventions targeting sleep or emotion, overall outcomes may be enhanced and treatment effects may be more durable. Future studies should therefore specifically consider these potential mechanisms.

Compliance with Ethical Standards: The authors have no funding and no conflicts of interest to report.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study.

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480 Table 1. Demographic information

| | Mean (SD) / % (n) | Median (IQR) |
|---|---|---|
| Age | 20.75 (1.80) | 21.00 (3.00) |
| Race | | |
| White | 34.7 (92) | N/A |
| African-American | 13.6 (36) | N/A |
| Asian | 29.4 (78) | N/A |
| Mixed Race | 5.3 (14) | N/A |
| Other | 7.5 (20) | N/A |
| Prefer not to specify | 7.2 (19) | N/A |
| Ethnicity | | |
| Non-Hispanic | 52.1 (138) | N/A |
| Hispanic | 41.1 (109) | N/A |
| Mixed | 1.9 (5) | N/A |
| Other | 2.3 (6) | N/A |
| Prefer not to specify | 2.6 (7) | N/A |
| Menstrual Cycle Regularity | | |
| Usually or always irregular | 19.6 (52) | N/A |
| Somewhat regular | 46.0 (122) | N/A |
| Always regular | 34.0 (90) | N/A |
| Unknown/Prefer not to answer | 0.4 (1) | N/A |
| Moos Menstrual Distress Questionnaire | | |
| Total | 62.38 (20.10) | 58.00 (23.00) |
| Pain | 13.60 (4.09) | 13.00 (7.00) |
| Concentration | 11.97 (5.02) | 10.00 (6.00) |

| | | |
|---|---|---|
| Behavioral Change | 10.26 (3.73) | 10.00 (6.00) |
| Automatic Reactions | 5.72 (2.55) | 5.00 (2.00) |
| Water Retention | 6.12 (2.30) | 5.00 (3.00) |
| Negative Affect | 15.69 (5.88) | 15.00 (9.00) |
| Arousal | 8.54 (3.57) | 7.00 (6.00) |
| Control | 7.57 (3.16) | 6.00 (1.00) |
| Center for Epidemiologic Studies Depression Scale Total (clinical cut off: 16 or above) | 17.67 (11.17) | 16.00 (11.17) |
| Difficulties in Emotion Dysregulation Scale Total | 83.93 (23.36) | 84.00 (35.00) |
| Pittsburgh Sleep Quality Index Total (scores >5 indicate poor sleep quality) | 6.36 (3.05) | 6.00 (4.00) |

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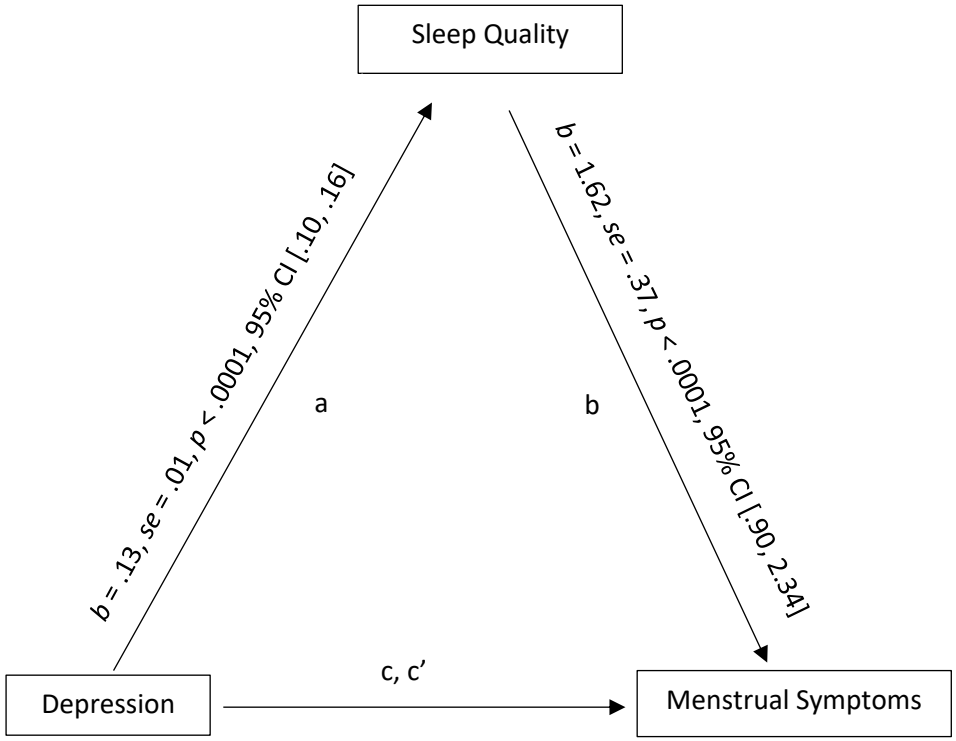
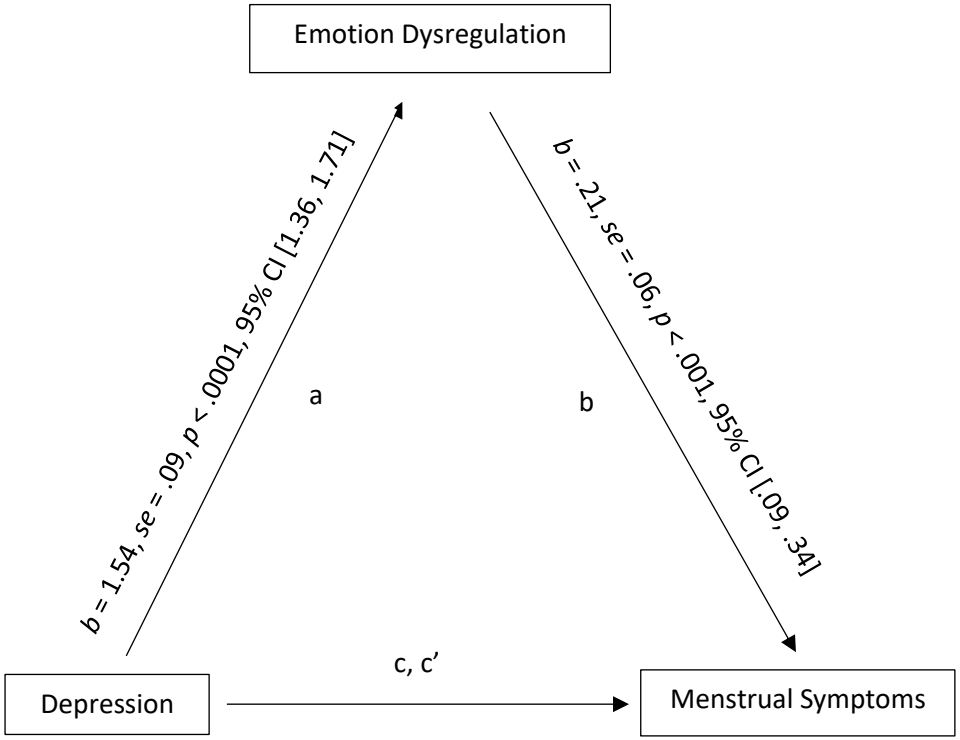
Figure 1. Direct and indirect pathways for primary mediation analyses (*n* = 265).



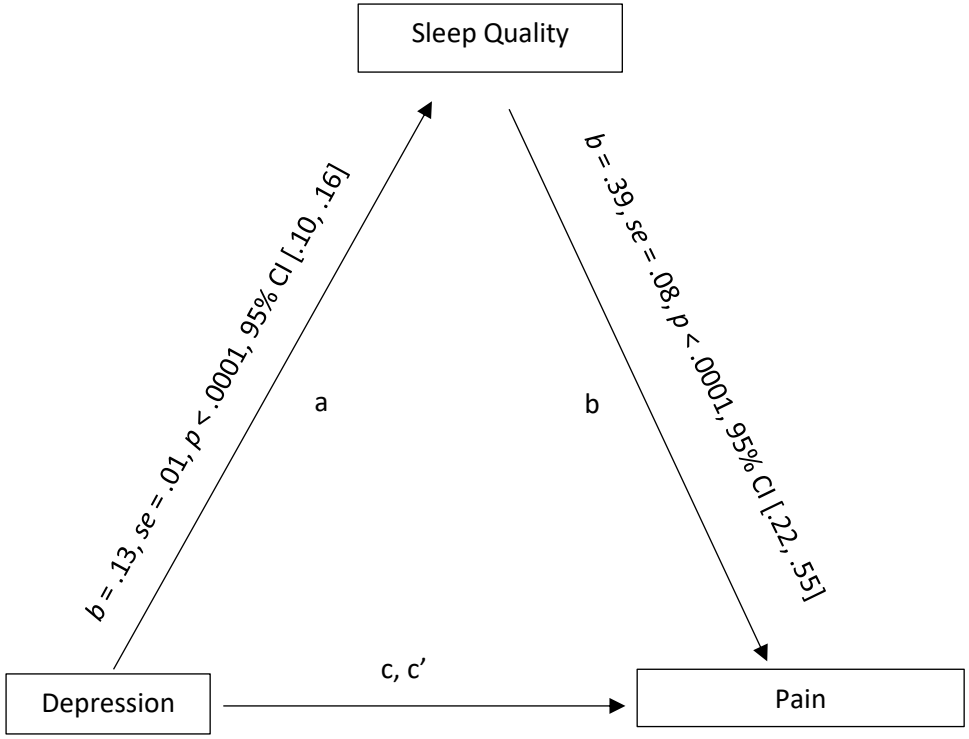

Direct Effect (c): *b* = 1.02, *se* = .09, *p* < .0001, 95% CI [.84, 1.20]
Mediated Direct Effect (c'): *b* = .81, *se* = .10, *p* < .0001, 95% CI [.61, 1.01]
Indirect Effect (ab): *b* = .21, *se* = .06, 95% CI [.08, .33]



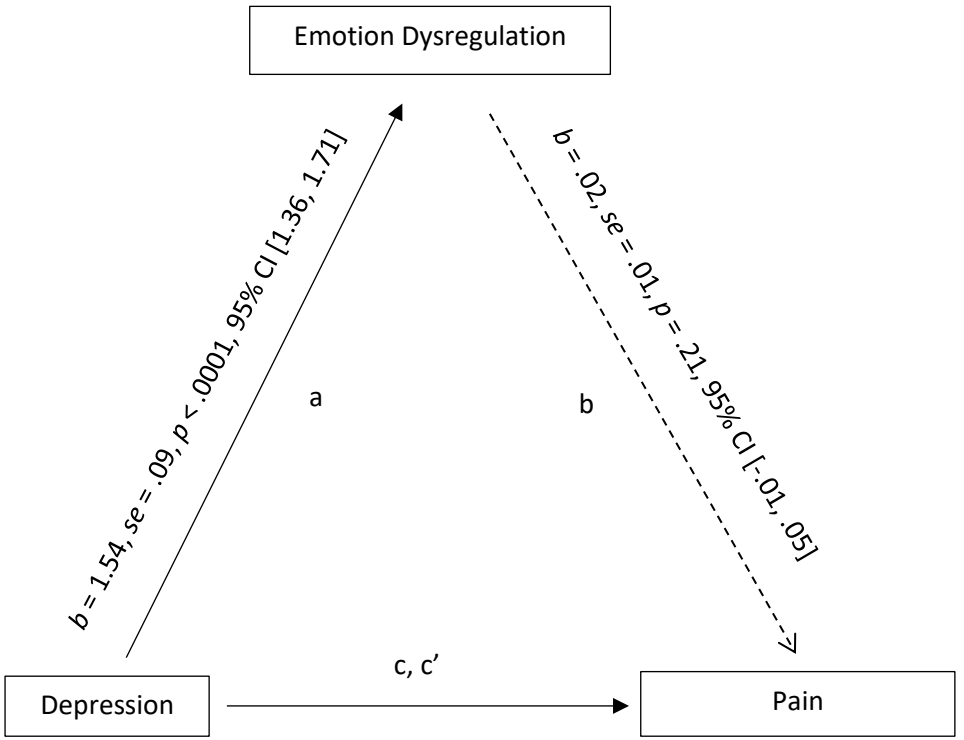

Direct Effect (c): *b* = 1.02, *se* = .09, *p* < .0001, 95% CI [.84, 1.20]
Mediated Direct Effect (c'): *b* = .69, *se* = .13, *p* < .0001, 95% CI [.43, .95]
Indirect Effect (ab): *b* = .32, *se* = .12, 95% CI [.10, .56]

Figure 2a. Direct and indirect pathways for exploratory mediation analyses (*n* = 265): Pre-Menstrual Pain.

Dashed line indicates non-significant pathways.

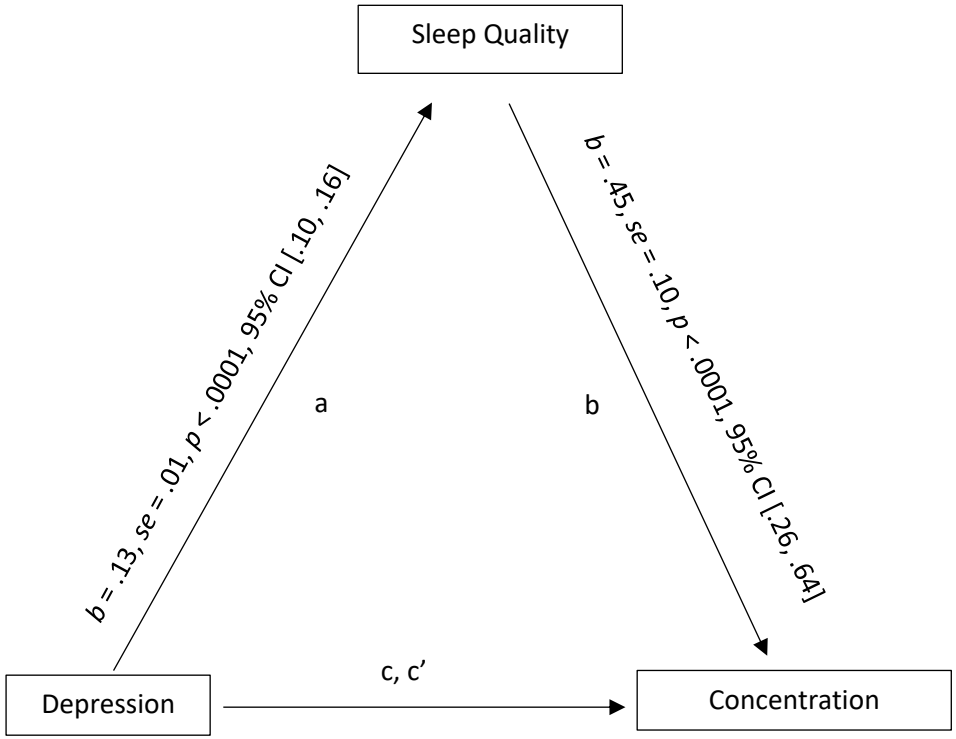



Direct Effect (c): *b* = .12, *se* = .02, *p* < .0001, 95% CI [.08, .17]
Mediated Direct Effect (c'): *b* = .07, *se* = .02, *p* = .0015, 95% CI [.03, .12]
Indirect Effect (ab): *b* = .05, *se* = .01, 95% CI [.02, .08]

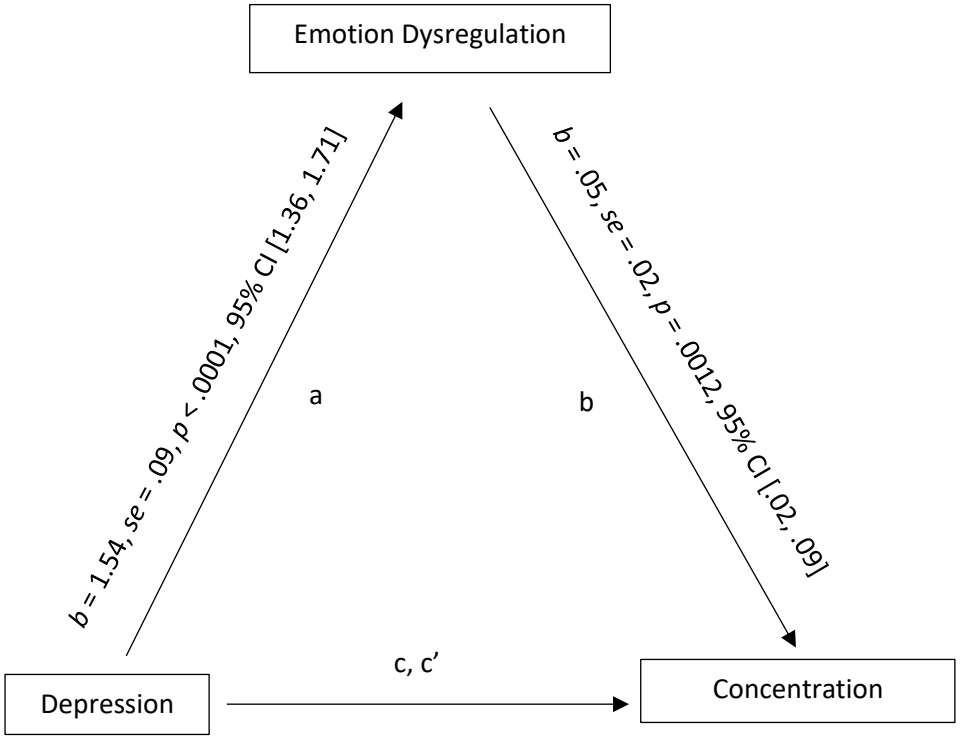



Direct Effect (c): *b* = .12, *se* = .02, *p* < .0001, 95% CI [.08, .17]
Mediated Direct Effect (c'): *b* = .09, *se* = .03, *p* = .003, 95% CI [.03, .16]
Indirect Effect (ab): *b* = .03, *se* = .03, 95% CI [-.02, .08]

Figure 2b. Direct and indirect pathways for exploratory mediation analyses (*n* = 265): Pre-Menstrual Concentration.

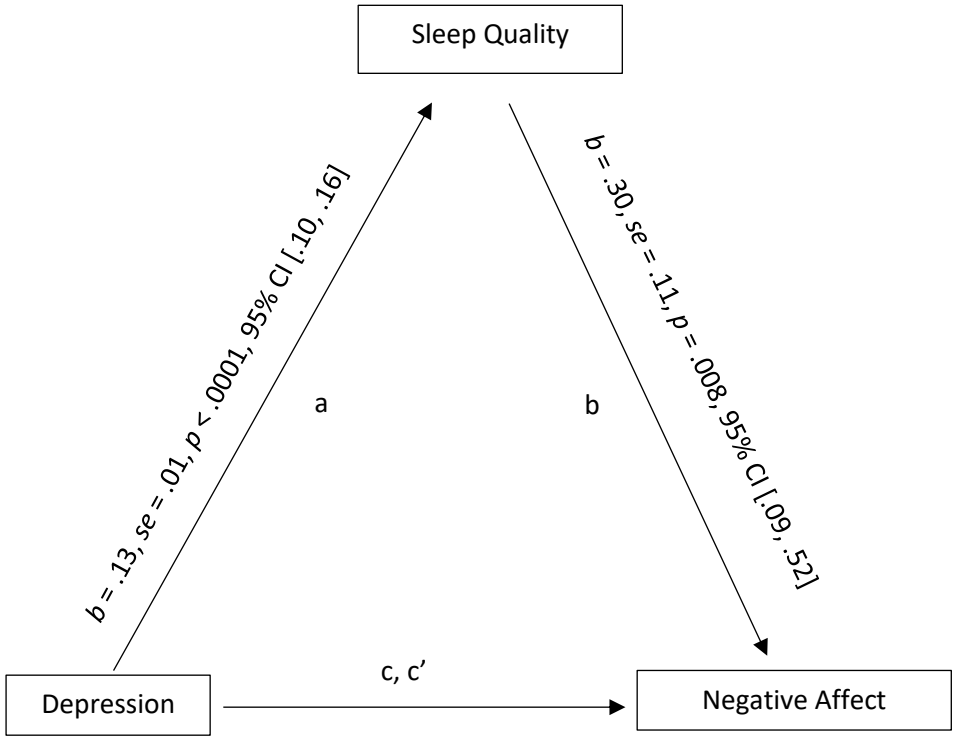



Direct Effect (c): *b* = .23, *se* = .02, *p* < .0001, 95% CI [.18, .27]
Mediated Direct Effect (c'): *b* = .17, *se* = .03, *p* < .0001, 95% CI [.12, .22]
Indirect Effect (ab): *b* = .06, *se* = .02, 95% CI [.02, .09]

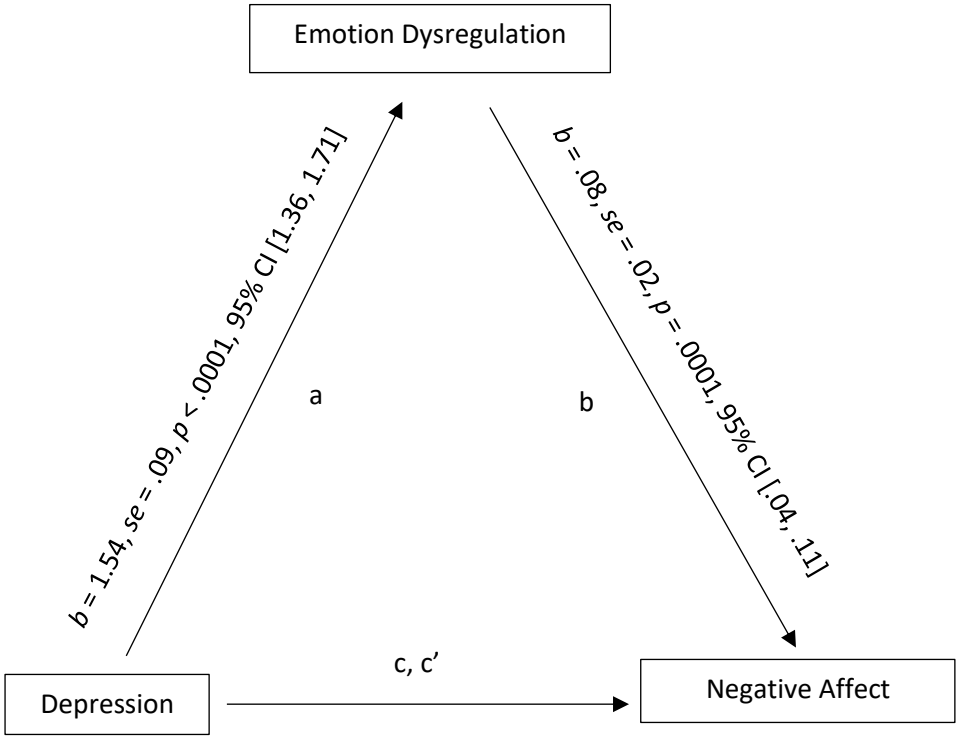



Direct Effect (c): *b* = .23, *se* = .02, *p* < .0001, 95% CI [.18, .27]
Mediated Direct Effect (c'): *b* = .14, *se* = .03, *p* = .0001, 95% CI [.07, .21]
Indirect Effect (ab): *b* = .08, *se* = .03, 95% CI [.02, .15]

Figure 2c. Direct and indirect pathways for exploratory mediation analyses (*n* = 265): Pre-Menstrual Negative Affect.




Direct Effect (c): *b* = .28, *se* = .03, *p* < .0001, 95% CI [.23, .34]
Mediated Direct Effect (c'): *b* = .24, *se* = .03, *p* < .0001, 95% CI [.18, .30]
Indirect Effect (ab): *b* = .04. *se* = .02, 95% CI [.01, .07]




Direct Effect (c): *b* = .28, *se* = .03, *p* < .0001, 95% CI [.23, .34]
Mediated Direct Effect (c'): *b* = .17, *se* = .04, *p* < .0001, 95% CI [.09, .24]
Indirect Effect (ab): *b* = .12, *se* = .03, 95% CI [.05, .18]