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Somatic Symptoms in Women with Dysmenorrhea and Noncyclic Pelvic Pain

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Abstract

Purpose—Somatic symptoms are a robust, transdiagnostic risk factor for pain conditions. However, the extent to which somatic symptoms contribute to the manifestation of women's pain syndromes, such as dysmenorrhea and noncyclic pelvic pain (NCP), is unclear due to high rates of co-occurrence. Therefore, the present study investigated the primary hypothesis that somatic symptoms would be elevated in NCP and distinctly influence the relationship between dysmenorrhea and co-occurring NCP.

Methods—A secondary analysis was performed on cross-sectional questionnaire data from 1012 nonpregnant reproductive-aged women. Eligible analyzed participants ($n = 834$) were categorized into four groups: healthy, dysmenorrhea, NCP, and NCP with co-occurring dysmenorrhea (NCP+dysmenorrhea). A parallel mediation analysis was run to evaluate the primary hypothesis that somatic symptoms are the primary factor associated with increased NCP accounting for dysmenorrhea.

Results—The NCP+dysmenorrhea group had higher somatic, anxiety, and depression symptom T-scores (respectively: 61, 61, 60) compared to the healthy controls (46, 51, 51; p 's $< .001$) and the dysmenorrhea group (50, 53, 54; p 's $< .001$). The pain and psychological symptoms were significantly correlated across the entire sample (r 's = .29, -.64, p 's $< .01$). Results from parallel

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Conflicts of Interest:

Rebecca Zuckerman declares that she has no conflict of interest.

Rebecca L. Siltan declares that she has no conflict of interest.

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mediation analysis showed that somatic symptoms were distinctly associated with NCPP +dysmenorrhea.

Conclusions—Women with NCPP+dysmenorrhea have increased psychological and somatic symptoms compared to women with dysmenorrhea alone. Given that NCPP often co-occurs with dysmenorrhea, failure to account for comorbidity in previous studies has likely led to an overestimation of psychological symptoms in dysmenorrhea. Future studies should evaluate whether somatic sensitivity is a modifiable risk factor for NCPP.

Dysmenorrhea is a pain that 25–50% of reproductive-aged women experience during their menses (Ju et al. 2014). Since dysmenorrhea is often treatment resistant (Oladosu et al. 2017), it is a frequent cause of missed school or work (Burnett et al. 2005). The psychosocial effects of pain combined with school absenteeism significantly reduce quality of life for women with menstrual pain (Rencz et al. 2017). Aside from cramping pain, somatic symptoms (i.e., bodily symptoms that cause emotional distress and physical dysfunction, such as faintness, chest pain, nausea, breathing difficulty, numbness, and weakness) have been reported as common extra-uterine symptoms in women with dysmenorrhea (Goldstein-Ferber and Granot 2006; Patel et al. 2006; Kamel et al. 2017), and noncyclic pelvic pain (Latthe et al. 2006; Westling et al. 2013). In fact, prior DSM-III criteria for somatization disorder included dysmenorrhea (APA 1986). Although the recent DSM-5 criteria (APA 2013) discards dysmenorrhea in the revised classification of somatic symptom disorder, the ICD-10 still includes “dysmenorrhea” under F45.8 “Other somatoform disorders.” Since somatic symptoms are associated with worse quality of life in other pain conditions, including back pain (Nickel et al. 2001), and irritable bowel syndrome (Creed et al. 2001), somatic symptoms could also contribute to impaired quality of life in dysmenorrhea.

The psychological mechanisms associated with increased somatic symptoms and menstrual pain may have common underpinnings. Depression (Alonso and Coe 2001; Subasinghe et al. 2016), anxiety (Granot et al. 2001; Tavallaee et al. 2011; Faramarzi and Salmalian 2014), and excess somatic symptoms (Patel et al. 2006) are estimated to be up to three fold higher in women with dysmenorrhea than women without dysmenorrhea (Latthe et al. 2006). The heritability of dysmenorrhea and psychological symptoms have been shown to share genetic variance (Silberg et al. 1987). The significant co-occurrence of menstrual pain and psychological symptoms suggests that menstrual pain expression reflects somatic (Goldstein-Ferber and Granot 2006; Chen et al. 2017a), neurological (Berkley and McAllister 2011), or affective disorders involving brain systems that contribute to pelvic nociceptive gain (Sigmon et al. 2000).

The comorbidity of somatic symptoms and dysmenorrhea is not unique because somatic symptoms frequently co-occur in women with noncyclic pelvic pain (NCPP). Common NCPP conditions that have been shown to be associated with somatic symptoms include Irritable Bowel Syndrome (Koloski et al. 2006) and Bladder Pain Syndrome/Interstitial Cystitis (Warren et al. 2011; Lai et al. 2012). In addition to somatic symptoms, anxiety and depression have also been shown to frequently co-occur in women with NCPP (Lorençatto et al. 2006; Chung et al. 2014a; Chen et al. 2017b). These findings indicate that somatic

symptoms may be a transdiagnostic risk factor that is observed across various pain conditions, including non-pelvic pain conditions such as temporomandibular joint disorder (Fillingim et al. 2013), and lower back pain (Pincus et al. 2002).

However, prior research on the role of somatic symptoms in dysmenorrhea has often failed to account for the comorbidity with NCPP (Goldstein-Ferber and Granot 2006; Patel et al. 2006; Kamel et al. 2017). Many, but not all women with NCPP, have dysmenorrhea (Zondervan et al. 2001; Westling et al. 2013), and only a fraction of women with dysmenorrhea experience NCPP. Additionally, given the high rate of concomitant mood and anxiety symptoms in women with pelvic pain disorders, it is important to account for the variance associated with this frequent co-occurrence. Developing a better understanding regarding the role of excess somatic symptoms in augmenting women's pain experience is critical to improving early intervention strategies. Thus, the primary aim of the present study was to evaluate the hypothesis that somatic symptoms would distinctly influence the relationship between co-occurring NCPP+dysmenorrhea and pain symptoms, relative to women with pure dysmenorrhea. For this purpose, a secondary analysis was conducted to analyze data that were previously published in Westling et al. (2013) to expand upon our previous finding regarding the distinct relationship between somatic symptoms and pelvic pain.

Materials and Methods

Participants

Data from a prior study (Westling et al. 2013) obtained with a web-based panel (Toluna USA, Wilton, CT) were re-analyzed in this study. Participants ($N = 1012$) were eligible for the study if they reported that they were between the ages of 18 and 45, not currently pregnant, and completed the survey within 24 hours, and not less than five minutes. Women who did not report a period in the last three months, and thus did not menstruate regularly enough to report menstrual pain severity, were excluded ($n = 178$), resulting in a final sample of 834 women. Among the 178 excluded, 19 women reported that they were in menopause, 11 women were taking hormones, 59 women reported that they had a hysterectomy, eight women were breast-feeding, 36 endorsed other reasons, and 45 women reported that they did not know why they had not had a period in the last three months.

Self-report variables

Standard demographic profiling questions were used to determine age and race. A multiple-choice question ("What was your family income before taxes? A: Less than \$10,000, B: between \$10,000 and \$29,999, C: between \$30,000 and \$49,999, D: between \$50,000 and \$74,999, E: between \$75,000 and \$99,999, F: more than \$100,000, G: I don't know") was used to determine income level in brackets similar to other studies (Chagan et al. 2005; Conboy et al. 2005). Menstrual pain was quantified using a severity index (White and Wildman 1986). Participants were asked "On average, how painful are your menstrual periods?" and "How painful was your last menstrual period?" Answers to these questions were anchored on a five-point Likert scale (not painful, mild, moderate, severe, very severe). Participants were also asked standard questions with the menstrual distress questionnaire

(MDQ) regarding menstrual characteristics including period heaviness, cycle duration, length of menses, and date of the last menstrual period (Moos 1968). The MDQ is one of the most commonly used questionnaires for studying the relationship between psychological factors and pain throughout the menstrual cycle (Sigmon et al. 2009; Nillni et al. 2013).

NCPP was evaluated by calculating the maximum bladder pain, bowel pain, and urination pain over the past seven days using a 0–10 numerical rating scale as previously described (Westling et al. 2013). The maximum was chosen because it reflects the most disturbing symptom of NCPP for each participant and many patients have difficulty determining the site of origin in visceral pain (Brumovsky and Gebhart 2010).

Somatic symptoms were calculated by averaging responses to somatic subscales from existing menstrual inventories including cold sweats, confusion, dizziness (Moos 1968), bloating (Endicott et al. 2006), nausea, and upset stomach (Mortola et al. 1990). Similar questions appear in standard somatic symptom questionnaires such as the Pennebaker Inventory of Limbic Languidness (Pennebaker 2012), and the Brief Symptom Inventory (Derogatis and Melisaratos 1983). Participants were asked how often in the last seven days they were bothered by these symptoms on a Likert scale (never, rarely, sometimes, often, always). The derivation of this scale is reported in Westling et al. (2013; $\alpha = .85$). The average score was transformed to a T-score to facilitate comparison to anxiety and depression symptomology.

NIH Patient-Reported Outcomes Measurement Information System (PROMIS) short forms were used to measure anxiety and depression symptoms (Cella et al. 2007). The PROMIS anxiety questionnaire (eight items, $\alpha = .93$) and depression questionnaire (10 items, $\alpha = .96$) were implemented to measure anxiety and depression respectively.

Group criteria

Group criteria for the four groups (healthy controls, dysmenorrhea, NCPP, and NCPP +dysmenorrhea) were implemented as follows: 1) healthy controls ($n = 386$) were defined a priori as women with regular menses who reported minimal menstrual or bodily pain (i.e., < 6 on a 0–10 scale for bladder, urination, or bowel pain). 2) Women with dysmenorrhea ($n = 285$) reported moderate to very severe menstrual pain and regular menses but without NCPP pain. 3) Women with NCPP pain ($n = 32$) reported bodily pain >5 on a 0–10 scale for bladder, urination, or bowel pain without moderate menstrual pain. This threshold is consistent with other large epidemiological studies of chronic pain (>5 on 0–10 numeric rating scale) as well as the consensus guidelines for chronic pain studies published by the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (Breivik et al. 2006; Dworkin et al. 2012). 4) Women with comorbid NCPP and dysmenorrhea ($n = 131$) reported bodily pain >5 on a 0–10 scale for bladder, urination, or bowel pain along with moderate menstrual pain.

Statistics

Analyses were run in SPSS 24. The parallel mediation model was calculated using the PROCESS Macro for SPSS (version 2.16) with 5000 bootstrap samples with bias corrected confidence intervals (Hayes 2013). The following variables were used for the parallel

mediation analysis, X = group (0 = dysmenorrhea, 1 = NCPP+dysmenorrhea), M = psychological symptoms ($M1$ = somatic symptoms, $M2$ = anxiety, $M3$ = depression), and Y = pelvic pain. Menstrual pain was included as a covariate in the model.

Power analysis

Published odds ratios of somatic symptoms, depression, and anxiety from a meta-analysis on dysmenorrhea (Latthe et al. 2006) suggest a Cohen's $f = .25$ or $r = .24$. Based on these estimated effect sizes, power analysis for a four group ANOVA ($\alpha = .05$, $1-\beta = .8$) in G-power (Faul et al. 2007) suggested a total sample size of 180 participants would be required. A power analysis for regression with up to five different predictors ($\alpha = .05$, $1-\beta = .8$) suggested that a total sample size of 256 would be needed.

Results

Participant Demographics

Eligible participants had an average age of 34.6 (SD 7.7) years, with 16% of minority race/ethnicity, and 10% self-identifying as Hispanic. Participants reported a median income between \$30,000 and \$49,999. Participants in the healthy control, dysmenorrhea, NCPP, NCPP + dysmenorrhea groups had comparable distributions in these demographic parameters (Table 1).

Group Differences in Pain and Psychological Symptoms

To evaluate differences in psychological and pain symptoms among the four groups, a series of five one-way ANOVAs were conducted (Table 2). All ANOVAs showed significant omnibus between-group differences across menstrual pain, pelvic pain, somatic, anxiety, and depression symptoms (p 's < .001). Bonferroni-corrected t-tests were run to probe group differences.

Menstrual symptoms—Consistent with our classification approach, the healthy control group reported less menstrual pain compared to the dysmenorrhea and NCPP+dysmenorrhea groups (p 's < .001) but not the NCPP group ($p > .05$; Table 2). Notably, menstrual pain was significantly worse in NCPP compared to all of the other groups (p 's < .001). Women with dysmenorrhea and NCPP + dysmenorrhea also had longer periods than healthy controls and the NCPP group (p 's < .001).

Pelvic pain symptoms—The healthy control group reported fewer pelvic pain symptoms compared to the other three clinical groups (p 's < .001; Table 2). The dysmenorrhea group had significantly less pelvic pain compared to the NCPP and NCPP+dysmenorrhea groups (p 's < .001). The NCPP+dysmenorrhea group had increased pelvic pain symptoms compared to the NCPP group ($p = .002$), indicating that similar to menstrual pain, pelvic pain is also amplified for individuals with co-occurring dysmenorrhea and NCPP.

Somatic symptoms—The healthy control group reported less somatic symptoms compared to the other three clinical groups (p 's < .001; Table 2). The NCPP and NCPP +dysmenorrhea had significantly elevated somatic symptoms compared to the dysmenorrhea

group (p 's < .001). Overall, these results suggest that NCPP, more so than dysmenorrhea, is related to clinically elevated somatic symptoms given that the NCPP+dysmenorrhea group had a mean T-score that was 1 SD higher than the population average (Table 2).

Anxiety and depression symptoms—Group differences in anxiety and depression symptoms were similar to those observed for somatic symptoms. The healthy control group reported fewer anxiety and depression symptoms compared to the other three clinical groups (p 's < .001). The NCPP and NCPP+dysmenorrhea groups had significantly elevated anxiety and depression symptoms compared to the dysmenorrhea group (p 's < .005). Since the NCPP+dysmenorrhea group had mean T-scores that were 1 SD higher than the general population (Table 2), these results imply that NCPP, more so than dysmenorrhea, is related to clinically elevated anxiety and depression symptoms.

Dimensional Analyses

Correlations among pain and psychological symptoms—To establish the relationship between factors contributing to dysmenorrhea and NCPP, correlations among psychological and pain symptoms were calculated across all participants. Menstrual pain, pelvic pain, somatic symptoms, anxiety symptoms and depression symptoms were all significantly associated with each other (p 's < .01; Table 3).

Parallel mediation analysis—A parallel mediation analysis (Figure 1) was conducted to evaluate whether somatic symptoms were a distinct contributory factor for pelvic pain, particularly for women with co-occurring NCPP and dysmenorrhea, compared to women with dysmenorrhea alone. The model examined the effects of group (dysmenorrhea vs. NCPP+dysmenorrhea) mediated by somatic, anxiety and depression symptoms on pelvic pain. Menstrual pain was included as a covariate in the model.

Regressions showed that NCPP+dysmenorrhea was a significant predictor of more somatic (path a_1 , $R^2 = .26$, $p < .01$), anxiety (path a_2 , $R^2 = .16$, $p < .01$), and depression (path a_3 , $R^2 = .11$, $p < .01$) symptoms compared to dysmenorrhea alone. Menstrual pain, the covariate in the parallel mediation model, was significantly associated with psychopathology symptoms (p 's < .01). With regard to pelvic pain symptoms, the full regression model was significant ($p < .001$) and it accounted for 70.8% of the variance. Group ($\beta = 2.7$) was a significant predictor of pelvic pain ($p < .001$), indicating that the NCPP + dysmenorrhea group experienced significantly higher levels of pelvic pain, independent of variance associated with psychological symptoms and menstrual pain. Thus, the direct effect between group and pelvic pain was significant. Additionally, somatic symptoms significantly predicted pelvic pain ($p < .001$). Depression, anxiety, and menstrual pain were not significant independent predictors of pelvic pain. Results showed that somatic symptoms, but not anxiety or depression symptoms, mediated the relationship between group and pelvic pain. The indirect effect for somatic symptoms (.543) was significantly different than zero as indicated by a 95% CI (.348 – .785) that was above zero. The indirect effects for anxiety (.069) and depression (–.021) were not significantly different than zero (95% CI_{anxiety} = –.067 – .215; 95% CI_{depression} = –.132 – .085). These results show that somatic symptoms, beyond what

was observed in women with dysmenorrhea, were the primary factor associated with co-occurring dysmenorrhea and NCPP.

Discussion

The present study shows that somatic symptom sensitivity influences the relationship between cyclic and noncyclic pelvic pain disorders and pain symptoms. As predicted, somatic symptoms, but not depression or anxiety symptoms, were associated with pelvic pain symptoms for women with co-occurring dysmenorrhea and NCPP, compared to women with dysmenorrhea only. Since somatic symptoms were particularly elevated in women with NCPP, and given that somatic symptoms distinctly mediated the relationship between co-occurring NCPP+dysmenorrhea and increased pelvic pain symptoms, somatic symptoms are likely to be an important contributing factor to NCPP.

Findings from the present study were consistent with those from prior studies (Zondervan et al. 2001; Altman et al. 2006; Chung et al. 2014b) showing that women with dysmenorrhea reported more NCPP than women without dysmenorrhea. We further extend the implications of this association, by also showing a smaller degree of association between somatic symptoms and isolated menstrual pain than prior studies. Both a large population based study (Patel et al. 2006) and a meta-analysis (Latthe et al. 2006) suggested up to three-fold more somatization compared to healthy controls. As we predicted, concomitant NCPP may have inflated prior estimates of the magnitude of the relationship between somatic symptoms and menstrual pain. While we observed increased somatic symptoms in women with isolated dysmenorrhea compared to healthy controls, the symptom severity was significantly lower compared to women with NCPP and co-occurring dysmenorrhea and NCPP. Related, our correlation coefficients for anxiety/menstrual pain and depression/menstrual pain were lower than those observed in a different study (Alonso and Coe 2001) that did not clearly distinguish NCPP components. Indeed, our correlation coefficients between anxiety or depression and NCPP were higher than those observed for menstrual pain. Therefore, our findings suggest the possibility that the strong attribution of somatic symptoms and psychological factors to dysmenorrhea in prior studies was due to unassessed NCPP.

The finding that somatic symptoms mediate the relationship between pain syndrome group and the level of pelvic pain reported suggests that somatic symptoms may be a critical risk factor for the transition from dysmenorrhea to NCPP. In other words, it is possible that women who have a high level of somatic symptoms in addition to menstrual pain are more likely to develop NCPP in the future, while those with menstrual pain alone and fewer somatic symptoms are not. Prior longitudinal studies have also shown that somatic symptoms are a risk factor for other chronic pain conditions. In the ongoing landmark study of incident temporomandibular joint disorder (TMD), heightened somatic symptoms convey a four-fold higher risk of developing TMD within two years, indicative of a critical risk factor in this comprehensive, deeply phenotyped cohort (Fillingim et al. 2013). Similar results have been observed for functional abdominal pain (Mulvaney et al. 2006), back pain (Jegan et al. 2017), musculoskeletal pain (Palmer et al. 2005), and chronic pain, in general (McBeth et al. 2001). As the present study was cross-sectional, longitudinal studies are

warranted to further evaluate temporal precedence regarding causal risk factors associated with NCPP.

A limitation of our study is that it was based on self-report survey data rather than clinically structured interviews. However, the questions used in our study were drawn from several National Institutes of Health PROMIS item banks that demonstrate strong correlations to legacy scales and can gauge severity across a wide spectrum of symptom severity (Cella et al. 2007). Strengths of this study include the large sample size and the diversity of the sample, with similar demographics across groups. Additionally, focus groups and qualitative analysis of input from patients and physicians were utilized to develop the initial item banks for menstrual distress, increasing validity (Westling et al. 2013).

Conclusions

Elevated somatic symptoms are associated with worse pain, particularly in women with NCPP. It has been previously observed that 27% of patients with bladder pain syndrome had multiple comorbid symptoms indicative of somatization disorder (Lai et al. 2012). This is consistent with our present findings and reinforces the importance of identifying somatic symptoms as a primary risk factor in pelvic pain to adequately direct treatment. One possibility is that in NCPP, neural circuitry involved in somatic symptoms undergoes changes promoting sensory amplification that can worsen the experience of bodily discomfort, menstrual pain, and non-menstrual pain simultaneously (Rief and Barsky 2005). Therefore, somatic symptoms and the process involved in widespread sensory amplification in the context of NCPP may represent a primary constellation of symptoms to target to alleviate physical and psychological discomfort. Psychological interventions such as cognitive behavioral therapy, mindfulness based stress reduction, as well as psychotropic medications have been shown to improve somatic symptoms (Fishbain et al. 1998; Kroenke and Swindle 2000; Lakhan and Schofield 2013). Optimistically, longitudinal studies that aim to modify critical risk factors, such as somatic symptoms, could be implemented to identify effective intervention strategies to prevent progression to chronic pelvic pain.

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Figure 1.

Parallel Mediation Model for Pelvic Pain. Statistical diagram of parallel multiple mediator model (with three mediators: somatization, anxiety, and depression, and menstrual pain as a covariate). Significant pathways are red. The diagram shows A) the total effect of group (NCPD+dysmenorrhea contrasted to dysmenorrhea only) on Pelvic Pain and B) the direct effect and causal pathways associating group with pelvic pain. The NCPD+dysmenorrhea group had significantly higher scores on all psychopathology and pain variables. Independent of the influence of the mediators, group was directly associated with pelvic pain. Somatic symptoms mediated the relationship between group and pelvic pain. Beyond the significant effects of group and somatic symptoms, anxiety, depression, and menstrual pain were not independent predictors of pelvic pain.

Table 1

Participant Demographics

	Healthy	Dysmenorrhea	NCPP	NCPP+Dys.	P value
Number of Participants	386	285	32	131	
Mean Age (years) [SD]	35.2 [7.1]	34.1 [7.6]	31.1 [7.1]	35.0 [7.8]	.080 ^a
Race					
White	315 (82%)	234 (82%)	19 (59%)	98 (25%)	.156 ^b
American Indian	3 (1%)	0 (0%)	1 (3%)	2 (1%)	
Asian	23 (6%)	17 (6%)	5 (16%)	9 (2%)	
Black/African American	33 (9%)	20 (7%)	2 (6%)	14 (4%)	
Other	8 (2%)	9 (2%)	4 (9%)	5 (1%)	
Multiracial	4 (1%)	7 (2%)	2 (6%)	3 (1%)	
Ethnicity					
Hispanic	37 (10%)	31 (11%)	4 (13%)	18 (14%)	.999 ^b
Income					
Less than \$10,000	35 (9%)	18 (6%)	0 (0%)	13 (10%)	.999 ^b
\$10,000 to \$29,999	70 (18%)	72 (25%)	5 (16%)	35 (27%)	
\$30,000 to \$49,999	94 (24%)	63 (22%)	8 (25%)	28 (21%)	
\$50,000 to \$74,999	82 (21%)	61 (21%)	12 (38%)	24 (18%)	
\$75,000 to \$99,999	39 (10%)	33 (12%)	3 (9%)	12 (9%)	
\$100,000 or more	48 (12%)	31 (11%)	2 (6%)	11 (8%)	
"I don't know" ^c	18 (5%)	7 (2%)	2 (6%)	8 (6%)	
Education					
Less than 12th grade	6 (2%)	5 (2%)	0 (0%)	8 (6%)	.255 ^b
High School/GED	91 (24%)	57 (20%)	9 (28%)	30 (23%)	
Some College	102 (26%)	97 (34%)	8 (25%)	45 (34%)	
Bachelors/Associates	150 (39%)	96 (34%)	13 (41%)	40 (31%)	
Graduate Degree	37 (10%)	30 (11%)	2 (6%)	8 (6%)	

Notes: Significance (p-value) after Bonferroni corrections in demographics across groups are were evaluated with an ANOVA as designated by ^a or X² as designated by ^b. Some participants selected an option stating "I don't know" in response to a question asking their level of income. ^c

Table 2

Group Characteristics of Menstrual, Pelvic, and Psychological Variables

	Healthy Controls	Dysmenorrhea	NCPP	NCPP + Dys.
Number of Participants	386	285	32	131
Menstrual Pain				
Not Painful	113 (29%)		6 (19%)	
Mild	273 (61%)		26 (81%)	
Moderate		203 (71%)		64 (49%)
Severe		62 (22%)		52 (40%)
Very Severe		20 (7%)		15 (11%)
Period Heaviness				
Extremely Light (use just one pad or tampon a day)	28 (7%)	6 (2%)	4 (13%)	3(2%)
Light (2–3 pads or tampons per day)	133 (34%)	41 (14%)	6 (19%)	25(19%)
Moderate (4–6 pads or tampons per day)	186 (48%)	175 (61%)	18 (56%)	52(40%)
Heavy (7–10 pads or tampons per day)	37 (10%)	43 (15%)	4(13%)	37(28%)
Very Heavy (more than 10 pads or tampons per day)	2 (1%)	20 (7%)	0 (0%)	14(11%)
Days Since Period Start	14.9 [11.7]	17.6 [14.7]	19.1 [18.2]	18.5 [15.6]
Duration of Menstrual Bleeding	4.8 [1.5]	5.3 [1.5] *	4.8 [1.6]	5.4 [1.7] *
Pelvic Pain				
Bladder Pain	0.4	0.9 *	4.9 [^]	5.4 [^]
Urination Pain	0.1	0.3	1.5 [^]	2.3 [^]
Bowel Pain	0.4	0.8 *	3.6 [^]	4.4 [^]
Average Pelvic Pain	0.3	.7 *	3.4 [^]	4.0 [^]
Psychopathology				
Somatic Symptoms (T-score)	46.0 [7.1]	49.5 [8.7] *	58.4 [10.2] [^]	60.8 [10.9] [^]
Anxiety (T-score)	50.6 [8.5]	53.1 [8.7] *	58.5 [7.9] [^]	60.8 [8.7] [^]
Depression (T-score)	50.9 [8.8]	53.6 [9.2] *	57.0 [6.6] *	59.7 [8.5] [^]

Notes: Participant counts (% within each group) or mean [SD] values shown above.

* $p < .05$ compared to healthy controls (Bonferroni corrected two-tailed t-test).[^] $p < .05$ compared to dysmenorrhea (Bonferroni corrected two-tailed t-test).

Table 3

Correlations Among Pain and Psychological Variables.

	2.	3.	4.	5.
1. Menstrual Pain	.375	.353	.288	.273
2. Pelvic Pain		.624	.463	.396
3. Somatic Symptoms			.729	.661
4. Anxiety				.837
5. Depression				

All shown correlations were significant (p 's < .01)

Regression coefficients, standard error, and model summary information for the parallel mediator model depicted in Figure 3.

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