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The association of dysmenorrhea with noncyclic pelvic pain accounting for psychological factors

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Abstract

OBJECTIVE—The factors that underlie pelvic pain are poorly understood. Specifically, the relative influence of dysmenorrhea and psychological factors in the etiology of noncyclic pelvic pain conditions, such as interstitial cystitis and irritable bowel syndrome, is unknown. To further characterize pelvic pain, we compared the frequency of menstrual, somatosensory, and psychological risk factors between women with and without severe noncyclic pelvic pain symptoms.

STUDY DESIGN—A total of 1012 reproductive-aged women completed a 112-item questionnaire with domains including mood, fatigue, physical activity, somatic complaint, and pain. Questionnaire items included existing items for menstrual distress and newly written items derived from qualitative interviews. The relationship of dysmenorrhea and noncyclic pelvic pain complaints (dyspareunia, dyschezia, or dysuria) was modeled using quantile regression.

RESULTS—Among women who menstruate regularly, those with dysmenorrhea had disproportionally more severe noncyclic pelvic pain (54/402, 13%) than women without dysmenorrhea (5/432, 1%; odds ratio, 13; 95% confidence interval, 5–33). In a multivariate-adjusted model, dysmenorrhea ($\beta = .17$), activity capability ($\beta = .17$), somatic complaint ($\beta = .17$), and bodily pain ($\beta = .12$) were the primary predictors of noncyclic pelvic pain. Depression ($\beta = .03$) and anxiety ($\beta = .01$) were not significantly predictive. The presence of dysmenorrhea, somatic complaint, and low activity capability predicted 90% of the cases of women with noncyclic pelvic pain.

CONCLUSION—The association between dysmenorrhea and noncyclic pelvic pain suggests that menstrual pain is an etiological factor in noncyclic pelvic pain, whereas depression and anxiety

may be secondary effects. Longitudinal studies are needed to determine whether dysmenorrhea causally influences development of noncyclic pelvic pain or shares common underlying neural mechanisms.

Keywords

dysmenorrhea; interstitial cystitis; irritable bowel syndrome; pelvic pain

Pelvic pain conditions are common and have significant emotional, functional, and financial impact.¹ Pelvic pain conditions include dysmenorrhea as well as noncyclic forms of pelvic pain, such as interstitial cystitis, irritable bowel syndrome, and dyspareunia. There is a frequent comorbidity of noncyclic pelvic pain with dysmenorrhea.^{2–5} Our research group has recently demonstrated moderate to severe dysmenorrhea is associated with increased bladder pain during clinical laboratory testing in otherwise asymptomatic women.⁶ Thus, the epidemiological and clinical literature supports a hypothesis that repeated uterine inflammation during menses may promote cross-organ pain sensitization, a mechanism by which dysfunction in 1 organ elicits neurogenic inflammation in adjacent organs.^{7–9}

As an alternative, the higher prevalence of depression and anxiety in women with pelvic pain complaints points to alternative mechanisms given a variety of labels—“brain-gut,”¹⁰ neuroendocrine,¹¹ psychosomatic^{12,13}—leading to treatment strategies using psychological management or psychotropic medication. Psychological distress and pain sensitivity are widely considered to be the chief underlying constructs for idiopathic pain disorders.¹⁴ Depression, anxiety, and abnormal circulating cortisol levels correlate with pain sensitivity in women with dysmenorrhea.¹⁵ A meta-analysis cataloging putative pelvic pain risk factors identified both psychological disorders and somatization (the tendency to report multiple unexplainable physical symptoms) as risk factors for noncyclic pelvic pain.¹⁶ In a clinical trial of women diagnosed with pelvic congestion syndrome, simultaneous treatments targeting hormonal and psychological dysfunction provided superior pain relief compared to hormonal therapy alone.¹⁷ However, to streamline treatment for noncyclic pelvic pain it is important to establish whether psychological factors (and other potential predictive factors) are interdependent or independent of reported dysmenorrhea and related menstrual symptoms. We conducted secondary analysis of a data-bank of questionnaire responses obtained for a separate pelvic pain survey project to evaluate the potential relationship among menstrual symptoms, psychological factors (depression, anxiety, somatization), physical activity, bodily pain, and noncyclic pelvic pain (a composite of self-reported urogenital pain symptoms).

Materials and Methods

The NorthShore University Health-System Institutional Review Board approved all phases of this study. We obtained a waiver of written informed consent due to the anonymous nature of participation.

Data in this study were originally collected as part of the development of a self-report measure of menstrual distress. We followed the strengthening the reporting of observational studies in epidemiology (STROBE) requirements for cross-sectional studies¹⁸ and

guidelines for self-report measures.¹⁹ In the first stage of questionnaire development, 2 of the authors (F.T., J.W.G.) developed a candidate item bank of self-administered questions after reviewing published scales to assess menstrual distress and its associated features. Individual items included in this study (Appendix) were modified from the Menstrual Distress Questionnaire, Calendar of Premenstrual Experiences Inventory, Daily Record of Severity of Problems, and Premenstrual Assessment Form to use our standardized answer scale described below.^{20–23} Answers to questions were anchored on 5-point Likert scales (eg, never, rarely, sometimes, often, always) or 0–10 rating scales (0 = no pain to 10 = worst pain imaginable), and fell into the following domains: dysmenorrhea, depression, anxiety, somatic complaints (as a proxy for somatization), physical functioning (both activity capability and ability), and bodily pain. Additional selected items measuring depression, anxiety, physical functioning, and bodily pain that were judged to relate to the construct of menstrual distress were drawn from scales from the Patient Reported Outcomes Measurement Information System (PROMIS; <http://nihpromis.org/>). An example of a question on depression queried each subject to determine if “I felt worthless” using a 5-point Likert scale.

In the second stage, candidate items were reviewed and modified by 3 gynecologists experienced in the assessment of menstrual distress. In the third stage, women with dysmenorrhea were interviewed on the relevance of the preliminary questionnaire and invited to suggest new questions or modify items where appropriate. The final survey included demographic and menstrual history questions.

We planned to construct a 2-parameter item response theory model to analyze the structure of our item pool. Our prior experience with item response theory^{24,25} and prior published models²⁶ suggested a large sample size would be required. Moreover, we planned to group participants on the basis of their current menstrual phase. Such an analysis plan also requires a large number of subjects per group, so we aimed to recruit approximately 1000 participants to have sufficient subsamples within groups. For the purposes of secondary analyses, we performed a post hoc, random-model power analysis to determine the smallest regression coefficients that would be detectable when examining as many as 10 different predictors (G-power).²⁷ With 1000 subjects, it is possible to ascertain R^2 values >0.02 with 99.9% power ($\alpha = 0.05$). A patient panel company was contracted to acquire our survey data (Toluna USA, Wilton, CT).

Over an 8-day period, 1012 female subjects met quality controls and served as the primary sample for this study. Respondents did not see items about menstruation and menstrual distress until the end of the questionnaire to ensure that these items did not bias their responses to other items. Eligible participants were nonpregnant women 18–45 years of age, had regular periods (21- to 35-day intervals), and completed the survey in a period not <5 minutes or >24 hours.

We constructed the primary dependent variable, noncyclic pelvic pain, by averaging 4 individual questions regarding bladder pain, bowel pain, pain with sexual intercourse, and pain with urination over the last 7 days (0–10 Likert scale). For women who were not sexually active during the time frame, we used the average of the remaining 3 scores to

compute the level of noncyclical pelvic pain. Women with noncyclic pelvic pain who scored 2 SD above the mean score (1.1, SD 1.7) were categorized as having severe noncyclic pelvic pain ($>4.5/10$ on 0–10 scale, hereafter called noncyclic pelvic pain). This is consistent with cut-offs used in a large epidemiological studies of chronic pain ($>5/10$ on 0–10 numeric rating scale)²⁸ as well as the published Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials consensus guidelines²⁹ for studies of chronic pain ($>4/10$ or $>5/10$ on a 0–10 numeric rating scale).

Dysmenorrhea subtypes were generated based on the response to the question, “On average, how painful are your menstrual periods?” Women who rated their average menstrual pain levels as moderate, severe, or very severe were considered to have dysmenorrhea. This level of severity was chosen based on prior research showing that comparable levels of menstrual pain are sufficient to predict impact on daily activity³⁰ and bladder sensitivity.⁶ Women with no or mild menstrual pain served as the comparison group. Menstrual phase was calculated based on the reported last menstrual period in regularly cycling women not on hormonal contraception (menstrual phase: days 1–5, follicular phase: days 6–14, luteal phase: days 15–35).

The internal consistency of constructs were assessed with Cronbach α (noncyclic pelvic pain: 0.88, depression: 0.95, anxiety: 0.93, somatic complaint: 0.85, activity capability: 0.95, activity interference: 0.85, bodily pain: 0.81). Whereas questions on activity capability asked whether subjects were capable of certain activities, questions on activity interference focused on whether health issues or pain actually limited activity. The somatic complaint category, which we use as a surrogate for somatization tendency, included questions on general bodily health complaints without reference to pain, unlike the category for bodily pain (Appendix). All variables in the study were transformed to a percentile score to adjust skewed distributions, permit comparisons between parameters, and increase interpretability.³¹

We compared demographic data (age, ethnicity, education, income) and pelvic pain variables using nonparametric statistics when appropriate. Other variables analyzed included (Appendix) menstrual history (6 items), dysmenorrhea (1–5 Likert scale), anxiety (8 items), depression (9 items), activity capability (10 items), activity interference (6 items), somatic complaint (average of 7 items), bodily pain (5 items), and general health (1–5 Likert scale). We used a type I error rate of $\alpha = .05$ for all statistical tests.

Spearman correlation tables were generated to evaluate the nonparametric associations among dysmenorrhea, menstrual function, psychological contributing factors, and noncyclic pelvic pain. We performed quantile regression on hypothesized factors on noncyclic pelvic pain because it is less sensitive to distribution assumptions that would not have been met with linear regression.³² Quantile regression allows for the ability to adjust for confounding as well the selection of relevant quantiles to evaluate regression parameters. Quantile regression is similar to ordinary least squares regression, but can evaluate model coefficients at relevant specific response quantiles rather than just the mean outcome. Model coefficients were calculated at the median quantile as well as the identified cutoff for severe noncyclic pelvic pain, the 93rd percentile (2 SD above the mean). Whereas co-efficients at

the median quartile imply factors associated with above average risk, coefficients at the 93rd percentile are more representative of factors responsible for severe pelvic pain. Thus, while both sets of coefficients are reported in tables, the coefficients at the 93rd percentile are most relevant and primarily discussed in the text. The significance of coefficients was determined through bootstrapping using the default settings for the R package quantreg. All analyses were performed with R.³³

Results

Seventy-one subjects met the criteria for noncyclic pelvic pain. The remaining 941 participants served as controls for the purposes of our analyses. This community-based sample was diverse with regard to socioeconomic and racial status: 55% of the subjects participating in this survey had below the median household US income (\$51,413) and our cohort included 17% racial minorities. There were no significant differences in menstrual phase at the time of participation, income, or education between subjects with and without noncyclic pelvic pain (Table 1). Factors known to be associated with elevated menstrual pain were significantly increased in women with noncyclic pelvic pain, including minority racial status, menstrual flow heaviness, depression, anxiety, somatic complaint, and bodily pain ($P < .01$). Most notably, those with noncyclic pelvic pain also had significantly worse menstrual pain than control subjects ($P < .001$). Out of the 59 women with noncyclic pelvic pain who regularly menstruated, 54 had moderate to severe menstrual pain (odds ratio [OR], 13.3; 95% confidence interval, 5.2–33.5).

To ensure that noncyclic pelvic pain did not merely reflect referred organ pain flaring during coincident menstruation at time of survey, the effect of menstrual phase was also controlled for in additional analysis. There was no statistical difference between noncyclic pelvic pain and pain-free groups in the proportion of regularly menstruating subjects responding during the menstrual phase ($P = .9$). Similarly, there was no statistically significant difference in noncyclic pelvic pain between subjects categorized with noncyclic pelvic pain during and outside of the menstrual phase ($P = .9$). The lack of significant relationship between noncyclic pelvic pain and menstrual phase indicates that noncyclic pelvic pain investigated in this study was not contemporaneous with menstrual pain.

The relationship between menstrual health variables (regularity, heaviness, period recency) and noncyclic pelvic pain was evaluated. Spearman correlations (Table 2) and quantile regression (Table 3) supported the hypothesis that dysmenorrhea was a principal factor correlated with noncyclic pelvic pain at the median and at critical thresholds ($P < .0001$). An overall pattern of increased pain within noncyclic pelvic pain constructs including the 2 general descriptor variables (pelvic pain and abdominal pain) was observed as a function of menstrual pain severity ($P < .001$) (Figure 1). Thus, dysmenorrhea was a strong indicator of both the likelihood and severity of noncyclic pelvic pain.

To estimate the unique contribution of dysmenorrhea to noncyclic pelvic pain, Spearman correlation (Table 4) and quantile regression (Table 5) were performed on all hypothesized risk factors. In the multivariate model, at the critical threshold for noncyclic pelvic pain the only consistent contributing factors were somatic complaints ($\beta = 0.17$), bodily pain ($\beta =$

0.12), and dysmenorrhea ($\beta = 0.17$). Although psychological factors initially appeared to have stronger correlations to noncyclic pelvic pain than dysmenorrhea (Table 4), neither depression ($\beta = 0.03$) nor anxiety ($\beta = 0.01$) were significant in the adjusted multivariate model.

To evaluate whether somatic complaint and bodily pain responses stemmed from a pathologic process as opposed to being the result of age or physical health-mediated processes, we evaluated age, overall self-reported general health, and physical functioning as confounders. Age and general overall health were inversely associated with noncyclic pelvic pain (Tables 4 and 5). Thus, subjects with noncyclic pelvic pain displayed features typical of idiopathic pain disorders rather than age-related health decline.

Application of model coefficients (Figure 2) demonstrates that levels of menstrual pain, total somatic complaints, and activity capability predict 44% of the variance in magnitude of noncyclic pelvic pain. Within the upper quartile of our model, noncyclic pelvic pain occurred in a predictable cluster. Although bootstrapping statistics confirmed the reliability of the model (Table 5), it could be reduced further: simultaneous menstrual pain, somatic complaint, and activity capability (reverse coded) in the upper quartile correctly predicted 90% of the subjects with noncyclic pelvic pain with a 5% false-positive rate and 4% false-negative rate.

Comment

Our findings indicate that menstrual pain, even after controlling for depression and anxiety, is associated with the severity of noncyclic pelvic pain. Additionally, self-reported pain complaints in other systems are associated with the magnitude of noncyclic pelvic pain. Thus, our findings provide strong support for involvement of neurophysiological end-organ dysfunction, such as dysmenorrhea, as opposed to psychological dysfunction, in noncyclical pelvic pain.

Our cross-sectional study is consistent with findings from prior epidemiological studies, but also allows adjustment of confounding variables. The rate of comorbid dysmenorrhea in subjects with severe noncyclic pelvic pain identified in our study, 54 of 59 (91%), is slightly higher than in 2 other epidemiological studies reporting 81%⁵ and 83%.⁴ However, these other studies included subjects with milder levels of chronic pelvic pain. Although the role of depression and anxiety were not systematically investigated in prior studies of the role of dysmenorrhea in pelvic pain, subjects' reported mood dysfunction was comorbid with these conditions.⁵

Other studies have identified relationships among chronic pelvic pain, depression, anxiety, and somatization, but neglected to examine the role of dysmenorrhea. In a metaanalysis, depression and anxiety were modestly positive risk factors for chronic pelvic pain (OR, 2.3–2.7).¹⁶ Psychosomatic symptoms (somatization) was reported to be one of the highest risk factors for chronic pelvic pain (OR, 8.0; 95% confidence interval, 5.2–12.4).¹⁶ Our study shows that even after adjusting for depression and anxiety, high levels of psychosomatic complaint are significantly associated with pelvic pain. Somatization has been shown to correlate with depression and anxiety in a prior study.³⁴ In another study, 27% of women

with chronic pelvic pain report bothersome medically unexplained symptoms involving different organ systems.³⁵ Notably, psychosomatic disorders like somatization are difficult to reverse, so available treatments focus on enhancing coping rather than actually reducing symptoms.³⁶

In contrast to targeting psychosomatic disorders, studies suggest dysmenorrhea is an exploitable target of treatments to prevent or reverse pelvic pain. As an example, women with dysmenorrhea and irritable bowel syndrome who were given hormonal treatment to improve menstrual distress reported that their bowel symptoms and abdominal pain sensitivity also improved.⁷ Likewise, oral contraceptive pills have been shown to reduce bladder pain in interstitial cystitis.³⁷ How such treatment could work may be hormonally mediated reduction of elevated levels of pronociceptive mediators (ie, cytokines) within the inflamed uterus and resultant reversal of sensitization of neural pathways that promote the inflammation of the adjacent bladder and/or bowel.³⁸ An attractive hypothesis needing further study is that by blocking or reducing repetitive adverse menstrual periods, cross-organ sensitization could be reduced. Attending to episodic pain conditions such as dysmenorrhea as a prophylactic strategy is supported by work in headache prevention. Acute, repetitive headaches are a modifiable risk factor in the development of chronic migraine headaches.³⁹ Application of nonsteroidal antiinflammatory drugs during episodic migraine can substantially reduce (OR, 0.31) the likelihood of transformation into chronic migraine.⁴⁰ Although it is possible that the mechanisms underlying chronic pelvic pain exacerbate dysmenorrhea, dysmenorrhea is more genetically stable than chronic pelvic pain^{41,42} and typically precedes the onset of chronic pelvic pain.^{43,44}

Our findings must be interpreted with several caveats that are inherent to cross-sectional research. A causal link cannot be inferred between somatic sensitivity and future noncyclic pain risk. There was no physician exam of somatic complaints for unrelated disease processes, such as nausea due to gastroesophageal reflux, although surveys are typically used to identify nonspecific functional somatic disorders.^{35,45} In addition, our subjects were asked about current levels of pain rather than a history of chronic pain. Therefore, the outcome variable was cautiously labeled as noncyclic pelvic pain without explicit reference to chronicity. Noncyclic pelvic pain and chronic pelvic pain share many of the same complaints including pain during bowel movements, pain with urination, pain with sexual intercourse, and pain with a full bladder. However, future clinical studies are needed confirm the pain constructs used here.

Another potential limitation of our study is that it did not involve clinically structured interviews. The questions used in our study were drawn from several National Institutes of Health (NIH) PROMIS item banks that demonstrate strong correlations to legacy scales and can gauge severity across a wide spectrum of symptom severity.⁴⁶ While we economized by not using the full item bank, the NIH PROMIS short forms for anxiety and depression were included, which have been shown to give scores highly correlated to the full item bank score.⁴⁶

Strengths of this study include the sample size, the diversity of the sample and the use of patients and physicians to develop the initial item banks for menstrual distress. Specifically,

the evaluation of key potential psychological confounders of the relationship between menstrual distress and reported noncyclic pelvic pain provide some insight on interactions unavailable in prior studies.

The findings of this study should heighten awareness of practicing physicians that patients exhibiting high degrees of somatic sensitivity and dysmenorrhea may be at risk of developing symptoms of chronic pelvic pain. Future prospective studies should clinically evaluate the relationship between dysmenorrhea and chronic pelvic pain as a first step before establishing the value of prophylactic strategies (eg, nonsteroidal antiinflammatory drugs and oral contraceptive pills) to reduce risk of developing chronic pelvic pain.

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FIGURE 1. Relationship of pelvic pain to dysmenorrhea

Box and whiskers plots indicate median, upper/lower decile, and upper/lower quartile for noncyclic pelvic pain constructs in subjects categorized with differing levels of menstrual pain (indicated by color and y axis). Related, but less specific constructs (abdominal, pelvic, and overall pain) also were correlated to severity of dysmenorrhea.

FIGURE 2. Binned scatter plot of combined model of hypothesized factors involved in noncyclic pelvic pain

Number of subjects with given noncyclic pelvic pain percentile score (x axis) is shown by diameter of each circle. Output of model based on amount of somatic complaint, activity capability, bodily pain, and dysmenorrhea is shown on y axis. Cluster of subjects has no noncyclic pelvic pain (left) and is predicted to have below median noncyclic pelvic pain. Above median levels of noncyclic pelvic pain, linear correlation between actual noncyclic pelvic pain and predicted noncyclic pelvic pain is shown by line generated by locally weighted scatter plot smoothed regression.

TABLE 1

Subject demographics categorized by noncyclic pelvic pain status

Variable	Severe noncyclic pelvic pain n = 71	Healthy n = 941	Significance
Age, y	38 (28–44)	36 (29–42)	$P = .63$
Average length of menses, d	5 (4–6)	5 (4–6)	$P = .99$
Average menstrual pain ^a	68 (68–90)	34 (34–68)	$P < .001$
Regular menstrual cycles, %	66.2	72.8	$P = .23$
General health ^a	38 (9–38)	38 (38–75)	$P = .001$
Depression ^a	83 (72–90)	48 (27–72)	$P < .001$
Anxiety ^a	87 (72–93)	48 (23–72)	$P < .001$
Somatic complaint ^a	92 (84–97)	47 (20–71)	$P < .001$
Bodily pain ^a	87 (77–94)	45 (26–70)	$P < .001$
Menstrual phase during survey administration, %			$P = .67$
Follicular	12.7	12.5	
Luteal	18.3	22.0	
Menses	12.7	11.4	
Missed period (>35 d)	11.3	9.4	
Hormone-regulated cycles	11.3	17.5	
Irregular, not applicable	33.8	27.2	
Menstrual heaviness, %			$P = .01$
Extremely light	3.4	5.0	
Light	23.7	24.7	
Moderate	37.3	52.8	
Heavy	28.8	13.4	
Very heavy	6.8	4.1	
Race, %			$P = .01$
American Indian	4.2	0.6	
Asian	9.9	5.3	

Variable	Severe noncyclic pelvic pain	Healthy	Significance
	n = 71	n = 941	
Black	11.3	7.7	
Native Hawaiian/Pacific Islander	0.0	0.2	
White	71.8	83.7	
Other	2.8	2.4	
Highest education level, %			<i>P</i> = .09
<High school	7.0	2.2	
High school (or GED)	29.6	23.9	
Some college	29.6	30.8	
Bachelor or associate degree	26.8	33.9	
Graduate or professional degree	7.0	9.1	
Annual income, %			<i>P</i> = .92
<\$10,000	8.5	8.3	
\$10,000–29,999	26.8	23.0	
\$30,000–49,999	16.9	23.7	
\$50,000–74,999	23.9	20.9	
\$75,000–99,999	9.9	9.6	
\$100,000	9.9	10.4	
Do not know	4.2	4.1	

Subjects were categorized as having noncyclic pelvic pain or as control. Data are presented as medians (with 25th and 75th percentiles) or distribution percentages. To determine significance, Wilcoxon rank sum tests were performed on scored variables and Pearson χ^2 tests were performed on distributions. All subjects were included in this analysis (n = 1012).

GED, General Educational Development.

^aConverted percentile score (0–100).

TABLE 2

Correlations of menstrual factors and noncyclic pelvic pain

Variable	1	2	3	4	5
Noncyclic pelvic pain (1)		.0001 ^a	.0011 ^a	.0001 ^a	.57 ^a
Dysmenorrhea (2)	0.37		.0001 ^a	.11 ^a	.13 ^a
Period heaviness (3)	0.11	0.35		.90 ^a	.10 ^a
Period regularity (4)	−0.13	−0.05	0		.0006 ^a
Recent period (5)	0.02	−0.05	0.06	0.12	

Spearman correlation table shows correlation coefficients in lower left diagonal.

^aUpper right diagonal indicates statistical significance.

TABLE 3

Quantile regression of menstrual factors and noncyclic pelvic pain

Variable	β_{median}	<i>P</i> value	β_{crit}	<i>P</i> value
Dysmenorrhea	0.65	.0001	0.3	.0001
Period heaviness	−30.02	.66	−0.05	.04
Period regularity	−0.3	.0003	−0.06	.33
Recent period	0.1	.15	0.04	.17

Coefficients were determined for median levels of noncyclic pelvic pain (β_{median}) and 93rd percentile, cutoff for noncyclic pelvic pain (β_{crit}). Significance was evaluated with bootstrapping.

TABLE 4

Correlations of hypothesized risk factors and noncyclic pelvic pain

Variable	1	2	3	4	5	6	7	8	9	10
Noncyclic pelvic pain (1)		.0001	.0001	.0001	.0001	.0001	.0001	.0001	.0001	.075
Somatic complaint (2)	0.58		.0001	.0001	.0001	.0001	.0001	.0001	.0001	.39
Bodily pain (3)	0.53	0.68		.0001	.0001	.0001	.0001	.0001	.0001	.0001
Activity capability (4)	-0.5	-0.52	-0.61		.0001	.0001	.0001	.0001	.0001	.0001
Anxiety (5)	0.46	0.72	0.59	-0.47		.0001	.0001	.0001	.0001	.66
Depression (6)	0.4	0.66	0.52	-0.41	0.83		.0001	.0001	.0001	.95
Dysmenorrhea (7)	0.37	0.33	0.37	-0.28	0.28	0.26		.0001	.0001	.59
Activity interference (8)	0.34	0.57	0.46	-0.27	0.63	0.63	0.23		.0001	.54
General health (9)	-0.27	-0.33	-0.31	0.44	-0.32	-0.3	-0.17	-0.22		.0014
Age (10)	-0.06	0.03	0.13	-0.14	0.01	0	-0.02	-0.02	-0.1	

Spearman correlation table is shown with correlation coefficients (lower left diagonal) and statistical significance (upper right diagonal).

TABLE 5

Quantile regression of hypothesized risk factors and noncyclic pelvic pain

Variable	β_{median}	<i>P</i> value	β_{crit}	<i>P</i> value
Somatic complaint	0.42	.0001	0.17	.002
Bodily pain	0.17	.01	0.12	.03
Activity capability	−0.28	.9	−0.17	.0001
Anxiety	0.04	.66	0.01	.9
Depression	−0.07	.17	0.03	.69
Dysmenorrhea	0.12	.002	0.17	.0001
Activity interference	0.01	.9	0.02	.71
General health	−0.02	.57	0.03	.4
Age	−0.11	.0001	−0.12	.0001

Shown are model parameters for median levels of noncyclic pelvic pain (β_{median}) and cutoff for noncyclic pelvic pain (β_{crit}).