

Low Back Pain and Pelvic Pain During Pregnancy

Prevalence and Risk Factors

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Study Design. Cross-sectional study. Women giving birth at one of two hospitals of northern Sweden from 1 January 2002 until 30 April 2002 were invited to fill in a questionnaire on their obstetric and gynecological history, actual pregnancy, and delivery.

Objective. The aim of this study was to investigate prevalence and risk factors for low back pain and pelvic pain (LBPP) during pregnancy.

Summary of Background Data. Although low back pain and pelvic pain during pregnancy is a most common complication of pregnancy, its etiology is unknown and the pathophysiology is poorly understood.

Methods. The sample was analyzed by calculating the prevalence of LBPP during pregnancy. Univariate and multivariate logistic regression was performed to calculate odds ratio (OR) and its 95% confidence intervals (CI) where applicable. Parametric and nonparametric testing was used to establish differences between groups.

Results. The response rate was 83.2% (N = 891). The prevalence of LBPP during pregnancy was 72%. Most cases reported both anterior and posterior pain. Increasing parity, history of hypermobility, and reported periods of amenorrhea were risk factors for LBPP. Women with LBPP had significantly higher prepregnancy weight, end-pregnancy weight, and prepregnancy and end-pregnancy body mass index. Age at menarche and use of oral contraceptives were not associated with LBPP. Nonrespondents were of the same age and parity as respondents.

Conclusions. A majority of pregnant women report LBPP. Parity, LBPP during a previous pregnancy, body mass index, a history of hypermobility, and amenorrhea are factors influencing the risk of developing LBPP during pregnancy.

Key words: cross-sectional, low back pain, pelvic pain, pregnancy, risk factors, prevalence. *Spine* 2005;30:983–991

back pain and pelvic pain (LBPP) is a common symptom during pregnancy, and the prevalence has been reported to vary from 24% to 90% in different studies.^{2–6} In one third of pregnant women, back pain is a severe problem compromising normal everyday life.⁴ Peripartum pelvic pain interferes with most activities of daily living and with sexual life.^{7,8} Back pain occurs twice as often in women with a history of back pain and women who have been pregnant previously. Younger women tend to have increased risk for back pain.⁹ Occurrence of pelvic pain is associated with twin pregnancy, first pregnancy, larger weight of the fetus, forceps or vacuum extraction, and a flexed position of the woman during childbirth.⁷ Women who experienced pelvic pain during a previous pregnancy report a relapse in 85% during a subsequent pregnancy.⁷ Women experiencing pelvic pain during pregnancy have been found to have normal height and weight and normal weight gain during pregnancy⁷; however, body mass index (BMI) has been reported to be significantly increased among first pregnant women with low back pain.¹⁰ The proportion of sick leave among Swedish pregnant women is high, and back pain is a common cause of sick leave.^{11,12}

Although LBPP during pregnancy is a most common complication of pregnancy, its etiology is unknown and the pathophysiology is poorly understood. Some models propose increased spinal load and decreased stability in the pelvic girdle as major causes.^{13,14} Increases in abdominal diameter, fetal weight, and muscular dysfunction have been found to be associated with LBPP during pregnancy.^{15–17} A general increase in mobility of joints during pregnancy has also been described.^{4,18,19} However, some authors claim that back pain during pregnancy cannot primarily be explained by biomechanical factors.¹⁵ Different attempts to investigate the cause of increased joint mobility during pregnancy have been made; in the 1980s, relaxin was reported to be associated with pelvic pain during pregnancy.²⁰ This association later has both been supported^{21,22} and contradicted.^{19,23,24} Reproductive hormones and procollagen in serum have been found to be associated with pelvic pain during late pregnancy.²⁵ Oral contraception (OC) has also been investigated in relation to LBPP during pregnancy with contradictory findings.^{26–29} OC use has been reported to influence the collagen metabolism.³⁰

Some authors consider LBPP during pregnancy to be a normal condition of pregnancy.^{31,32} The symptoms may vary highly, and individuals are affected to different degrees. Nevertheless, referring to the actual scientific knowledge,^{5–7,16,17,19,33} this condition should be consid-

The lifetime incidence of low back pain has been found to be 66% for 38- to 64-year-old Swedish women.¹ Low

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ered a complication of pregnancy for women with substantial impairment. For a small proportion of the affected individuals, the symptoms will not regress, and instead the condition will progress into chronic LBPP after pregnancy.^{34,35}

The aim of this study was to investigate the prevalence and risk factors for LBPP during pregnancy. The questionnaire used in the study was extensive and investigated many different themes in the obstetric and gynecologic history of the participant, and background factors not reported previously in the literature were inquired.

Materials and Methods

The study was cross-sectional with retrospective data collection. The first date of inclusion was January 1, 2002 (date of delivery) and the last date April 30, 2002. All women who gave birth at the Departments of Obstetrics and Gynaecology at Umeå University Hospital (UUH) or the Sunderby Hospital (SH) in the counties of Västerbotten and Norrbotten in northern Sweden were invited to fill in a questionnaire containing approximately 80 questions. Participation in the study implied competence in the Swedish language. Within approximately 24 hours of the delivery, the women received oral and printed information on the aims of the study from a midwife on duty at the department. Voluntary participation was emphasized. Each woman who gave her oral consent to participate received a questionnaire with a unique number. The questionnaire was usually collected before discharge from hospital; women who had not completed the questionnaire were given a prepaid envelope. Some patients who had given birth at UUH and who had been overlooked in the initial request to participate in the study during their stay at the hospital were contacted by telephone by one of the two authors. They were informed on the phone about the study; and if they agreed to participate, they were sent a questionnaire by post. Missing cases at SH were not telephoned because of lack of personnel.

The women's identification (ID) number, the unique number of the questionnaire, and the date of distribution and date of collection of the questionnaire were registered. If a woman declined to participate, her ID number was recorded for the purpose of analysis of missing data. Women had to be delivered at a gestational age of at least 23 weeks with live or stillbirth to be included in the study.

The study was approved by the Ethics Committee at Umeå University (D No. 01–335) and each participant gave her informed oral consent.

Definitions. *Low back pain or pelvic pain (LBPP) during pregnancy* was defined as "recurrent or continuous pain for more than 1 week from the lumbar spine or pelvis" during actual pregnancy. A woman was considered to have had LBPP during pregnancy if she positively answered a specific question about LBPP with patient-drawn markings of localization of pain on a schematic drawing in the questionnaire (Figure 1). Women with LBPP were requested to report their highest pain score due to LBPP during their pregnancy *before* and *during* delivery on a visual analogue scale (VAS), where 0 denoted "no pain" and 10 denoted "worst thinkable pain." Patients with a maximum of 7 or more on a self-rated pain score (VAS) were considered as having *high pain score LBPP* (hps-LBPP).

In our clinical settings, *emergency caesarean section* denotes

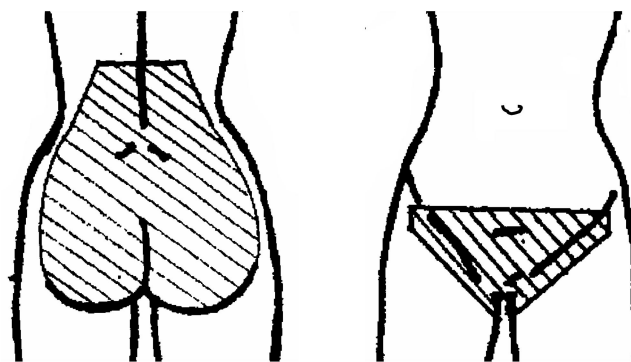


Figure 1. Reported localization of low back pain and pelvic pain during pregnancy.

immediate caesarean section and *acute caesarean section* denotes all other nonelective caesarean sections.

Preterm birth was defined as gestational age < 37 completed weeks (<259 days); *term birth* included gestational age of 37 to 41 completed weeks (259–293 days) and *post-term birth* was defined as a gestational age of ≥ 42 weeks (i.e., ≥294 days). Gestational age was determined by ultrasound in almost all cases.

Prepregnancy weight was defined as reported weight before the actual pregnancy. *End-pregnancy weight* was defined as reported weight before the delivery. *Body mass index (BMI)* was defined as weight (kg)/height (m²).

All women were requested to score their *total experience of the delivery* on a VAS with end-points of 0 and 10 cm, where 0 denoted "very bad" and 10, "very good."

Statistics. The sample was analyzed with calculation of means and standard deviations (SD) for parametric data. Independent-samples *t* test was used to test difference between groups for parametric data. Nonparametric two-independent-samples testing was used to test the difference between groups for nonparametric data. Pearson χ^2 test was used to test the difference between groups for categorical data. The sample was analyzed with calculation of odds ratios (ORs) and their 95% confidence intervals (CIs) by univariate and multivariate logistic regression for LBPP during pregnancy in relation to different background variables. In multivariate logistic regression OR^a denotes an OR with adjustment for place of delivery, maternal age, and parity. OR^b denotes an OR with adjustment for place of delivery, maternal age, parity, and highest educational level while OR^c denotes adjustment for place of delivery, parity, and hypermobility. Bivariate correlation was investigated with Pearson's correlation coefficient. For evaluation of the consistency of the responses in the questionnaire, Cohen's kappa or the intraclass correlation coefficient was calculated for participants answering an identical questionnaire once more.

Results

The total number of women who delivered at UUH and SH was 1,114: 516 women (46.3%) at UUH and 598 women (53.7%) at SH. Participation in the study implied competence in the Swedish language. Non-Swedish-competent women were therefore primarily excluded. Accordingly,

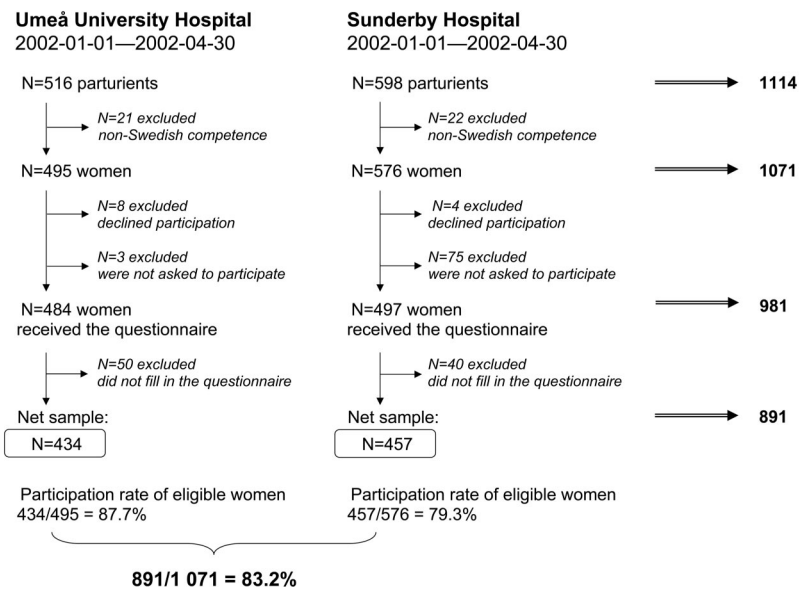


Figure 2. The sample.

the sample of eligible women was 1,071 women and is presented in Figure 2. Nonrespondents were women who either did not receive a questionnaire or who did not fill in a received questionnaire. The net sample consisted of 891 women (Figure 2).

Data Sets From UUH and SH

The overall prevalence of LBPP was 71.7% ($N = 639$), with no significant difference ($P = 0.480$) in the prevalence of LBPP between UUH and SH (Table 1) in non-parametric test. Distribution of educational level, mater-

Table 1. Prevalence of Specified Variables

Variable	UUH and SH		UUH		SH	
	No.	(%)	No.	(%)	No.	(%)
No. of participants	891	(100.0)	434	(48.7)	457	(51.3)
Prevalence of LBPP	639	(71.7)	316	(72.8)	323	(70.7)
Prevalence of hps-LBPP	207	(23.2)	114	(26.3)	93	(20.4)
Highest educational level	882	(99.0)	430	(99.1)	452	(98.9)
9-yr compulsory schooling	51	(5.8)	22	(5.1)	29	(6.4)
Folk high school	7	(0.8)	4	(0.9)	3	(0.7)
Senior high school	424	(48.1)	190	(44.2)	234	(51.8)
University	400	(45.4)	214	(49.8)	186	(41.2)
Maternal age	891	(100.0)	434	(100.0)	457	(100.0)
≤19 yr	14	(1.6)	7	(1.6)	7	(1.5)
20–24 yr	112	(12.6)	49	(11.3)	63	(13.8)
25–29 yr	334	(37.5)	164	(37.8)	170	(37.2)
30–34 yr	293	(32.9)	146	(33.6)	147	(32.2)
35–39 yr	116	(13.0)	57	(13.1)	59	(12.9)
≥40 yr	22	(2.5)	11	(2.5)	11	(2.4)
Parity	891	(100.0)	434	(100.0)	457	(100.0)
1	375	(42.1)	182	(41.9)	193	(42.2)
2	313	(35.1)	152	(35.0)	161	(35.2)
3	143	(16.0)	72	(16.6)	71	(15.5)
≥4	60	(6.7)	28	(6.5)	32	(7.0)
Gestational age	890	(99.9)	434	(100.0)	456	(99.8)
<37 wk	69	(7.8)	40	(9.2)	29	(6.4)
37–41 wk	758	(85.2)	357	(82.3)	401	(87.9)
≥42 wk	63	(7.1)	37	(8.5)	26	(5.7)
Mode of delivery	891	(100.0)	434	(100.0)	457	(100.0)
Vaginal delivery	657	(73.7)	301	(69.4)	356	(77.9)
Vacuum extraction	62	(7.0)	39	(9.0)	23	(5.0)
Forceps	4	(0.4)	—	—	4	(0.9)
Caesarean section, elective	82	(9.2)	40	(9.2)	42	(9.2)
Caesarean section, acute	70	(7.9)	45	(10.4)	25	(5.5)
Caesarean section, emergency	16	(1.8)	9	(2.1)	7	(1.5)
Caesarean section, total	168	(18.9)	94	(21.7)	74	(16.2)

UUH = Umeå University Hospital; SH = Sunderby Hospital; LBPP = low back pain and pelvic pain during pregnancy; hps-LBPP = high pain score LBPP.

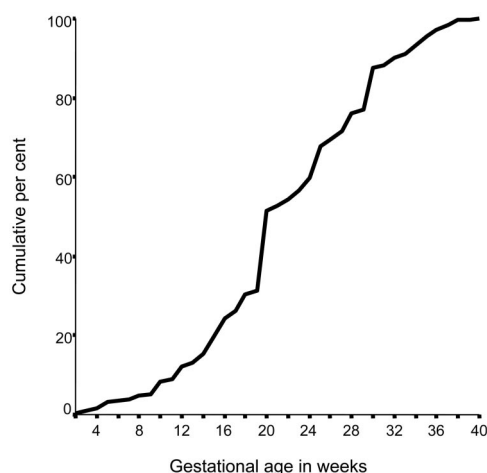


Figure 3. Week of gestation at first appearance of low back pain and pelvic pain.

nal age, parity, gestational age, and mode of delivery is presented in Table 1. There was no difference in age between women delivered in UUH (mean age, 30.12 years, SD, 4.90 years) and SH (mean age, 29.89 years; SD, 4.90 years) in *t* test ($P = 0.478$), and this was also the case for parity ($P = 0.614$; *t* test) when comparing UUH (mean parity, 1.88; SD, 0.94) and SH (mean parity, 1.92; SD, 1.08). Gestational age in 887 women (99.6%) was determined by routine ultrasound. Mean gestational age in days was 277.2 (SD, 16.0) at UUH and 277.6 (SD, 12.9) at SH ($P = 0.693$; *t* test). Mean birth weight at UUH was 3,514 g (SD, 686 g) and at SH, 3,582 g (SD, 624 g) ($P = 0.118$; *t* test). Highest educational level ($P = 0.163$) and mode of delivery ($P = 0.542$) did not differ between women delivered at UUH and SH in Pearson χ^2 test.

LBPP During Pregnancy

Anterior pelvic pain alone was reported by 12.1% ($N = 77$) and posterior pain alone was noted by 28.0% ($N = 179$) of the total number of women reporting LBPP ($N = 639$). Combined anterior and posterior pain was

reported by 59.8% ($N = 382$). Therefore, posterior low back pain or pelvic pain was experienced by a total of 87.8% of women and anterior pelvic pain by a total of 71.8% of the women with LBPP. *T* test showed that maternal mean age did not differ significantly for women with and without LBPP. First appearance of pain symptoms of LBPP was reported from the gestational age of 1 week until 39 weeks of gestation, with a mean gestational age at the start of LBPP of 22.1 week (SD, 7.9 weeks). The distribution of first appearance of pain symptoms is presented in Figure 3 ($N = 551$). The reported mean value and SD of the highest pain score due to LBPP during pregnancy was 5.8 and 2.2, respectively ($N = 593$). The corresponding mean value and SD for highest pain score (due to LBPP) during their delivery for women with LBPP was 5.4 and 3.8 ($N = 529$). *Total experience of delivery* did not differ between women with and women without LBPP during pregnancy (Table 2). Reported highest pain score of LBPP during pregnancy presented a moderate inverse correlation to gestational age at first appearance of pain symptoms ($r^2 = -0.27$). Women who reported previous LBPP in life reported first appearance of LBPP during a pregnancy in 50%. Mean age at first appearance of LBPP was reported to be 22.8 years of age (SD, 5.7 years; range, 10–39 years). Women with LBPP had a mean gestational age of 277.7 days (SD, 14.0 days), and the corresponding mean gestational age for women without LBPP was 276.9 (SD, 15.8; $P = 0.717$, *t* test). The prevalence of LBPP in mothers giving preterm birth, term birth, and post-term birth was 72.5%, 71.9%, and 68.3%, respectively. Prevalences of LBPP during pregnancy in relation to parity were 72.5%, 85.4%, 87.2%, and 87.5% for parity 1 to 4, respectively. The risk for recurrence of LBPP in women who reported LBPP in a previous pregnancy was 94.6% for women in their second parity and 89.3% for women in their third parity. Women reporting LBPP during a previous pregnancy reported total recovery from LBPP after that pregnancy (before start of the actual pregnancy) in 58.3%,

Table 2. Test for Difference Between Groups for Specified Variables Between Women With and Women Without LBPP Using the Independent-Samples *t* Test and the Pearson's χ^2 Test Where Applicable

Variable	No.	T Test, <i>P</i>	Pearson's χ^2 Test, <i>P</i>	LBPP			No LBPP		
				No. (%)	Mean	SD	No. (%)	Mean	SD
Mean maternal age	891	0.237	0.101	639 (71.7)	29.88	4.80	252 (28.3)	30.31	5.15
Total experience of delivery	864	0.241		624	7.87	2.21	240	8.06	1.88
Mean age at menarche	873	0.340		628	12.7	1.35	245	12.8	1.35
History of menstruations	878			632 (100.0)			246 (100.0)		
Mainly regular	709			499 (79.0)			210 (85.4)		
Mainly irregular	80			58 (9.2)			22 (8.9)		
Mainly regular*	46			39 (6.2)			7 (2.8)		
Mainly irregular†	39			33 (5.2)			6 (2.4)		
Other bleeding pattern	4			3 (0.5)			1 (0.4)		

LBPP = low back pain and pelvic pain.

*Mainly regular menstruations with one or more periods of amenorrhea.

†Mainly irregular menstruations with one or more periods of amenorrhea.

Table 3. Odds Ratio (OR) and Its 95% Confidence Interval (CI) for LBPP for Specified Variables Calculated by the Univariate and Multivariate Logistic Regression

Variable	Crude OR	CI 95%	OR*	CI 95%*	OR†	CI 95%†	LBPP	No LBPP
Parity								
1	1.00	—	1.00	—	1.00	—	244	129
2	1.78	1.26–2.49	1.78	1.26–2.50	2.02	1.40–2.90	241	68
3	1.92	1.22–3.01	1.91	1.21–3.01	2.65	1.60–4.39	112	29
4–8	1.14	0.63–2.06	1.15	0.64–2.06	1.51	0.79–2.89	41	18
Maternal age	Crude OR	CI 95%	OR*	CI 95%*	OR‡	CI 95%‡	LBPP	No LBPP
≤24 yr	1.00	—	1.00	—	1.00	—	89	35
25–29 yr	1.41	0.89–2.24	1.40	0.88–2.23	1.23	0.74–2.02	258	74
30–34 yr	0.87	0.55–1.37	0.86	0.54–1.36	0.62	0.36–1.07	198	93
≥35 yr	0.89	0.52–1.51	0.88	0.52–1.49	0.67	0.36–1.23	93	42
Highest educational level	Crude OR	CI 95%	OR*	CI 95%*	OR§	CI 95%§	LBPP	No LBPP
9-yr compulsory schooling	1.00	—	1.00	—	1.00	—	42	9
Folk high school	1.28	0.13–12.02	1.26	0.13–11.85	1.70	0.17–16.64	6	1
Senior high school	0.60	0.28–1.27	0.60	0.28–1.27	0.64	0.29–1.39	312	112
University	0.49	0.23–1.04	0.48	0.22–1.03	0.59	0.26–1.32	278	122

Note: Values included in analyses are specified.

*Adjusted for place of delivery (*i.e.*, hospital).

†Adjusted for maternal age and highest educational level.

‡Adjusted for parity and highest educational level.

§Adjusted for parity and maternal age.

66.4%, and 70.7% of cases (women in their second, third and fourth parity, respectively).

Parity and Educational Level

Increasing parity was associated with increasing risk for LBPP. The association was strengthened when adjusting for maternal age and educational level (Table 3). The association was further pronounced when selecting the group of hps-LBPP (Table 4). Estimates of risk for LBPP and hps-LBPP indicated an inverse association between LBPP and hps-LBPP and increasing educational level (Tables 3 and 4).

Maternal Weight, Body Mass Index, and Birth Weight of the Child

Women developing LBPP weighed significantly more and had a significantly higher BMI (Table 5). Prepregnancy and end-pregnancy weight and BMI were highly correlated ($r^2 = 0.90$ and $r^2 = 0.89$, respectively) for women with LBPP, and this was also the case for non-LBPP women ($r^2 = 0.89$ and $r^2 = 0.86$, respectively). Women with BMI ≥ 30 had an increased risk for LBPP

(crude OR [COR], 1.87; 95% CI, 1.18–2.94, and OR^b, 1.96; 95% CI, 1.22–3.16) in comparison with women with BMI < 25 . Women with BMI ≥ 30 had an even higher risk of hps-LBPP (COR, 3.07; 95% CI, 1.65–5.69 and OR^b = 3.69, 95% CI: 1.88–7.23).

Mean birth weight was 3,549 g (SD, 656 g; N = 891 g), with a range of 952 to 5,820 g. Women with LBPP (mean birth weight, 3,573 g; SD, 649 g; range, 952–5,620 g) had an almost significantly higher birth weight ($P = 0.080$, t test) than women without LBPP (mean birth weight, 3,488 g; SD, 669 g; range, 1,088–5,820 g). Birth weight of 4,000 g or more was associated with increased risk for LBPP (COR, 1.45; 95% CI, 1.06–2.25), however, not remaining significant when adjusting for parity, place of delivery, highest educational level, and maternal BMI (OR, 1.42; 95% CI, 0.94–2.14).

History of Hypermobility

A total of 150 women (17.3%) reported that they were diagnosed as having hypermobile joints (150 of 869). Seventy-seven (8.8%) reported at least one person

Table 4. Odds Ratio (OR) and Its 95% Confidence Interval (CI) for hps-LBPP for Specified Variables in Univariate and Multivariate Logistic Regression

Variable	Crude OR	CI 95%	OR*	CI 95%*
Parity				
1	1.00	—	1.00	—
2	2.28	1.47–3.54	2.51	1.57–4.01
3	2.88	1.66–4.99	4.34	2.28–8.25
4–8	1.92	0.94–3.92	2.88	1.26–6.55
Highest educational level	Crude OR	CI 95%	OR†	CI 95%†
9-yr compulsory schooling	1.00	—	1.00	—
Folk high school	0.53	0.03–9.50	1.48	0.77–2.85
Senior high school	0.51	0.21–1.20	0.65	0.31–1.33
University	0.35	0.14–0.83	0.45	0.19–1.06

*Adjusted for maternal age, highest educational level, and place of delivery (*i.e.*, hospital).

†Adjusted for maternal age, parity, and place of delivery.

Table 5. Test of Difference With Independent-Samples *t* Test Between Women With and Women Without LBPP in Relation to Specified Variables and Distribution of Body Mass Index (BMI)

Weight	No.	Minimum Weight (kg)	Maximum Weight (kg)	Mean Weight (kg)	SD	<i>t</i> Test, <i>P</i>
Pre-pregnancy weight, no LBPP	244	44	110	64.7	11.0	0.001
Pre-pregnancy weight, LBPP	623	44	133	68.0	13.5	
End-pregnancy weight, no LBPP	245	56	126	79.3	11.8	<0.001
End-pregnancy weight, LBPP	619	46	138	83.3	14.2	
BMI	No.	Minimum BMI (kg/m ²)	Maximum BMI (kg/m ²)	Mean BMI (kg/m ²)	SD	<i>t</i> Test, <i>P</i>
Pre-pregnancy BMI, no LBPP	242	17.21	37.42	23.30	3.56	
Pre-pregnancy BMI, LBPP	623	16.65	44.96	24.57	4.57	<0.001
End-pregnancy BMI, no LBPP	243	21.30	42.10	28.56	3.74	
End-pregnancy BMI, LBPP	619	19.15	46.85	30.10	4.77	<0.001
End-pregnancy BMI, divided into groups	No.	%				
< 25	121	14.1				
25–29	378	43.9				
≥30	363	42.0				

among their parents and siblings as being hypermobile (77 of 872). Diagnosed hypermobility was more common in women reporting LBPP ($P = 0.012$) and the risk for LBPP was increased when the woman was diagnosed hypermobile (COR, 1.74; 95% CI, 1.12–2.70; and OR^b, 1.79; 95% CI, 1.14–2.80). The risk of hps-LBPP was even higher (COR, 2.65; 95% CI, 1.61–4.36; and OR^b, 2.66; 95% CI, 1.58–4.46) if hypermobility was diagnosed. Women diagnosed with hypermobility and/or with a history of hypermobility in the family (19.9%) presented also an increased risk for LBPP (COR, 2.07; 95% CI, 1.35–3.18; and OR^b, 2.12; 95% CI, 1.37–3.28).

Family History of LBPP During Pregnancy

All women were asked whether their own mother had had a history of LBPP during one or more of her pregnancies. The response alternatives were “yes” (14.2%), “no” (26.2%), and “don’t know” (59.6%), with 2.5% being missing responses. The women answering “yes” and “no” (39.4% of all women) were included in the analyses ($N = 351$). Women reporting a history of low back pain or pelvic pain during pregnancy in the mother had an increased risk for LBPP (COR, 2.11; 95% CI, 1.21–3.65; and OR^c, 2.01; 95% CI, 1.14–3.54). Hps-LBPP demonstrated the same risk level (COR, 2.26; 95% CI, 1.21–4.21; and OR^c, 2.00; 95% CI, 1.03–3.86). Thirty-nine percent ($N = 342$) of the women reported having at least one sister with one or more births. If the sister had a history of LBPP during pregnancy, the risk for LBPP was increased for the woman (COR, 2.75; 95% CI, 1.42–5.31; and OR^c, 2.92; 95% CI, 1.45–5.86).

History of OC Use

Mean age at starting to take OCs was 17.5 years (SD, 2.8 years; range, 10–32 years) and differed between women with and women without LBPP, as shown in the *t* test (mean age 17.4 years; SD, 2.8 years *vs.* 17.8 years; SD, 2.6 years, $P = 0.028$). Oral contraceptive (OC) use was reported by 90.1% ($N = 794$ of 881), while 9.9% ($N = 87$) had never used OCs. The mean user time of mainly combined OCs was 6.6 years (SD, 3.6 years; range, 0.1–20.0 years, $N = 525$), while the corresponding user time

for mainly mini-pills was 5.1 years (SD, 4.5 years; range, 0.1–22.0 years, $N = 99$). The mean user time of OCs did not differ between women with and women without LBPP (6.0 years, SD, 3.4 years *vs.* 6.0 years; SD, 3.8 years; $P = 0.813$, *t* test). Use, at some point in their lives, of mainly combined OCs or mini-pills did not influence the women’s risk for LBPP in relation to women with no previous use of OCs (COR, 0.78; 95% CI, 0.46–1.34; and COR, 0.68; 95% CI, 0.35–1.31, respectively). Corresponding estimates were similar for hps-LBPP.

Menarche and History of Menstrual Pattern

Mean age at menarche did not differ significantly for women with and women without LBPP, as calculated in the *t* test (Table 2). The pattern of menstrual bleedings is shown in Table 2. All women reporting experience of one or more periods of amenorrhea, irrespective of whether their bleeding pattern, was mainly regular or irregular, had an increased risk for LBPP compared with women with mainly regular menstruations (COR, 2.33; 95% CI, 1.26–4.30; and OR^b, 2.37; 95% CI, 1.27–4.42). The corresponding estimates for hps-LBPP was even higher (COR, 2.80; 95% CI, 1.39–5.60; and OR^b, 2.95; 95% CI, 1.41–6.14). Women with irregular menstruations had no increased risk for LBPP in comparison with women with regular menstruations. Women treated for irregular menstruations, anovulation, and amenorrhea presented no association with LBPP.

Data Collection Routines

At distribution and collection of the questionnaires, the distribution and collection dates were continuously registered. The following information is from UUH, which had the most complete registered data on dates of distribution and collection of questionnaires. Most women at UUH received the questionnaire during the date of delivery ($N = 313$; 72.6%, total registered number = 431) and the mean time of distribution of the questionnaire was 1.2 days after the date of delivery (SD, 4.2 days; range, 0–53 days). Most women filled in and returned the questionnaire during their stay at the hospital (70.5% of participants handed in the questionnaire

within 5 days of the date of delivery) and the mean time of return of the questionnaire was 9.6 days (SD, 22.9 days; range, 0–275 days) after date of delivery. The mean time of the women keeping the questionnaire was 8.6 days (SD, 22.5 days; range, 0–274 days). The time calculations above are slightly overestimated since some questionnaires were sent by post (assigned date of distribution was equal date of sending the questionnaire) and received by post (assigned date of collection was equal to the date of postal delivery).

Validity of the Data and Nonrespondents

The content of the questionnaire was validated. Participants were asked to fill in the questionnaire a second time. Twenty-nine women were asked (by telephone) and reacted positively to the request to redo the questionnaire. A second questionnaire was sent by post within approximately 2 to 3 weeks of the collection of the primary questionnaire. Twenty-five of these women filled in an identical questionnaire. The women reported LBPP in 60% of cases ($N = 15$). There was total agreement between the first and the second set of answers to questions on the woman's birth year, date of delivery, birth weight, method of delivery, amenorrhea, contraceptives (specific question), highest educational level, and LBPP ($N = 25$). There was no difference in reported age at start of OC use and age at menarche. Cohen's kappa was 0.89 (95% CI, 0.69–1.00) for pattern of menstrual bleedings ($N = 25$). The intraclass correlation coefficient (consistency definition) was 0.89 (95% CI, 0.77–0.96) for *total experience of delivery* (VAS). Gestational age at the first symptom of LBPP differed between the primary and the second questionnaire (21.8 weeks *vs.* 19.6 weeks).

Eventually, 180 parturients did not participate in the study, 61 of whom delivered at UUH (33.9%) and 119 (66.1%), at SH (Figure 2). The nonrespondent group was compared with the group of respondents using *t* test or nonparametric test. There was no difference between nonrespondents and respondents with regard to the mean number of pregnancies (2.4; SD, 1.5; *vs.* 2.4, SD, 1.4), parity (1.9; SD, 1.2; *vs.* 1.9; SD, 1.0) and maternal age (30.0 years; SD, 5.5 years; *vs.* 30.0 years; SD, 4.9 years) in *t* tests. Gestational age in days differed significantly (271.2 days; SD, 21.6 days; *vs.* 277.4 days; SD, 14.5 days; $P < 0.001$, *t* test), with an increased number of preterm births (11.7% *vs.* 7.8%) for nonrespondents. The distribution of method of delivery did not differ significantly ($P = 0.776$, vaginal delivery nonrespondents *vs.* respondents 73.7% *vs.* 71.7%, caesarean section 18.9% *vs.* 19.4%) in Pearson χ^2 test. Emergency caesarean section was more frequent among nonrespondents than among respondents (2.8% *vs.* 1.8%). In 36.1% ($N = 65$) of the records of nonrespondents, there was a note on probable symptoms or diagnosis of LBPP.

Time of return of the questionnaire was not associated with prevalence of LBPP. Women responding within 5 days of delivery reported a prevalence of LBPP of 73.6%,

and women responding after 6 days or more had a prevalence of LBPP of 67.2% (Pearson χ^2 test, $P = 0.198$).

Discussion

The high prevalence of LBPP during pregnancy suggests that LBPP is a major public health issue. Previous studies have reported different prevalences.^{2–5} One of the explanations may be the lack of a uniform classification for LBPP during pregnancy, as discussed below. Other explanatory factors may be the level of representativity in different studies and a possible time-dependent cohort effect.

UUH is the primary referral unit for SH and other local hospitals in the counties of Västerbotten and Norrbotten. Five delivery units are available in the area, including UUH and SH. The total population of the counties of Västerbotten and Norrbotten reached 254,818 (127,814 females) and 254,733 (125,844 females), respectively, on December 31, 2001.³⁶ During the study period, a total of 912 and 776 women delivered in the counties of Västerbotten and Norrbotten, respectively (personal message, Centre of Epidemiology at the National Board of Health and Welfare, Sweden). Accordingly, the proportion of women delivered at UUH and SH in relation to the total number of women delivered in the two counties was 56.6% (516 of 912) and 77.1% (598 of 776). Neither mode of delivery nor educational attainment differed significantly between UUH and SH. In multivariate logistic regression, place of delivery (*i.e.*, hospital) was used as a possible confounder; however, the estimates were unaffected. We consider our participating women to be representative of the women in the counties of Västerbotten and Norrbotten and most probably also of Swedish women as a whole.

There is a lack of a uniform classification of low back pain and pelvic pain during pregnancy. Low back pain is commonly defined as pain referred to the area between the twelfth rib and the gluteal folds.³⁷ This anatomic area also includes the sacroiliac joints, which probably can be considered as a separate functional entity in the pathophysiology of pain in the pelvis.¹⁵ Other terms that are used are posterior pelvic pain,¹⁵ pelvic pain,^{20,38} pelvic girdle relaxation,²³ and pelvic joint instability.³⁹ Different attempts at subclassification of pelvic pain during pregnancy have been presented previously.^{5,6} In the present study, we used an original pain schedule based on the clinical experience of the authors in their clinical work with patients with LBPP. Evidently, absence of a uniform definition of LBPP will create difficulties in comparing results of different studies, if not in comprehending them. However, we consider our model to be suitable for a questionnaire asking for subject-reported perception of symptoms. Obviously, women may have understood other pain symptoms to have originated from LBPP, when they had in fact had other causes. For example, true ischiadic pain (not related to the pregnancy) and pain from an inguinal hernia can be located in the

same areas as LBPP. However, both conditions are fairly rare during pregnancy⁴; and where women overestimated the prevalence of LBPP, the strength of associations was probably diminished.

Methodologic Considerations

One of the aims was to study the prevalence of LBPP during the pregnancy; therefore, the end point must evidently be the delivery. In this type of study, the individual's recall ability is of major significance and recall bias is therefore a problem, especially when the woman has been asked to recall events during a fairly long period (*i.e.*, the pregnancy, previous obstetric, and gynecologic histories, and other background factors). However, we have made efforts to limit the drawback of recall bias on issues of the pregnancy through almost immediate distribution of the questionnaire (for most of the women) and also through a fairly short mean time of collection after distribution.

The nonrespondents were of the same age and had the same experience of number of pregnancies and births and the same delivery methods. Preterm births and emergency caesarean section were more frequent among nonrespondents, and these experiences may have negatively influenced the interest of the women in participating owing to a strained situation. However, we do not consider these differences to be a major source of bias since the literature does not report specific associations with LBPP. More important was that LBPP was probably less prevalent among nonrespondents than among respondents; and since the questionnaire focused on LBPP, women with LBPP may have been more inclined to participate in the study, although all women were encouraged to participate. Consequently, the prevalence of LBPP in the study has most probably been overestimated. If we presume that nonrespondents had a nonprevalence of LBPP, then we could estimate the prevalence at 59.7% (639 of 1,071). Based on the assumption that the prevalence of LBPP for nonrespondents was 36.1% ($N = 65$), we calculated that the total prevalence among eligible women would have been 65.7% (704 of 1,071). The true prevalence of LBPP probably ranges from 59.7% to 71.7%.

History of previous LBPP during pregnancy was associated with recurrence of LBPP at the same risk level as previously shown.⁷ Parity was a risk factor for LBPP, confirming results of previous studies.^{10,34,40} Young women have been found to have more pain than older women⁵; however, in our study maternal age was not associated with level of pain or with prevalence of LBPP during pregnancy. Prolonged pregnancy has previously been demonstrated to be significantly associated with LBPP⁴¹; however, gestational age *per se* was not related to LBPP, and the prevalence of LBPP at post-term was lower (68.3%) than the prevalence of LBPP at term. However, our results may have been confounded by induction of women with pronounced LBPP, which would have lowered the prevalence at post-term.

Women developing LBPP weighed significantly more and had a significantly higher BMI, which supports some previous results¹⁰ but contradicts others.⁷ The prevalence of hypermobility among women with pelvic pain has previously been estimated to be 12%.⁸ In our study, the prevalence of reported diagnosed hypermobility was 17.3%. Diagnosed hypermobility or a family history of hypermobility was associated with an increased risk for LBPP. These results indicate the importance of hypermobility as a contributing factor to LBPP during pregnancy.

A family history of LBPP was associated with an increased risk for LBPP. This association may correspond to inheritance or/and lifestyle-dependent factors. However, recall bias may be an important confounder, and these results must be interpreted with caution since some analyses only included a minority of respondents.

Studies on OC use and back pain have diverged. No association has been found among female athletes,²⁶ and short-term users of OC have been reported to have an increased risk for persistent pain after delivery compared with long-term users.²⁸ By contrast, significantly more cases of back pain among OC users have been reported elsewhere.²⁹ Our results did not support an association between use of OC and risk for LBPP during pregnancy.

Mean age at menarche did not differ between women with and women without LBPP. A history of amenorrhea was associated with an increased risk for LBPP during pregnancy. To our knowledge, this association has not previously been investigated. Menstrual patterns are closely related to the function of the ovary and serum levels of estrogens and gestagens. Dysfunctional ovaries previously in life may result in changed receptor-level responses that may be evoked during pregnancy and may result in LBPP. Progesterone but not estradiol has been found to correlate to the incidence of pelvic pain.²⁵

This study has further investigated other background and outcome factors in relation to LBPP, such as sick leave, perceived health, psychosocial situation, sexual life, treatment of LBPP, and history of physical leisure activities, and these results will be presented in coming publications. Women with LBPP in this sample have further been followed up at 6 months and 12 months postpartum, and these results will also be reported in the future.

Conclusion

A majority of pregnant women report low back pain and pelvic pain. Parity, previous low back pain and pelvic pain, BMI, a history of hypermobility and amenorrhea are factors influencing the risk of developing LBPP. The high prevalence of LBPP during pregnancy makes LBPP a major public health issue, and efforts should be made to promote causal studies with the future aim of prevention and therapy of LBPP. Different methods such as histologic examinations, immunology characterizations, and investigation of distribution of different hormone-receptors in connective tissue and muscle tissue may be

possible attempts in discriminating deviating patterns in women with LBPP during pregnancy.

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■ Key Points

- The high prevalence of low back pain and pelvic pain during pregnancy (LBPP) makes it a major public health issue.
- Body mass index is a significant determinant for LBPP during pregnancy.
- Parity, previous low back pain and pelvic pain, and a history of hypermobility and amenorrhea are factors influencing the risk of LBPP during pregnancy.

References

1. Svensson HO, Andersson GB, Hagstad A, et al. The relationship of low-back pain to pregnancy and gynecologic factors. *Spine* 1990;15:371–5.
2. Berg G, Hammar M, Moller-Nielsen J, et al. Low back pain during pregnancy. *Obstet Gynecol* 1988;71:71–5.
3. Fast A, Shapiro D, Ducommun EJ, et al. Low-back pain in pregnancy. *Spine* 1987;12:368–71.
4. Ostgaard HC, Andersson GB, Karlsson K. Prevalence of back pain in pregnancy. *Spine* 1991;16:549–52.
5. Kristiansson P, Svardsudd K, von Schoultz B. Back pain during pregnancy: a prospective study. *Spine* 1996;21:702–9.
6. Albert H, Godskesen M, Westergaard J. Prognosis in four syndromes of pregnancy-related pelvic pain. *Acta Obstet Gynecol Scand* 2001;80:505–10.
7. Mens JM, Vleeming A, Stoockart R, et al. Understanding peripartum pelvic pain: implications of a patient survey. *Spine* 1996;21:1363–9; discussion 1369–70.
8. Hansen A, Jensen DV, Wormslev M, et al. Pregnancy associated pelvic pain: II. Symptoms and clinical findings. *Ugeskr Laeger* 2000;162:4813–7.
9. Ostgaard HC, Andersson GB. Previous back pain and risk of developing back pain in a future pregnancy. *Spine* 1991;16:432–6.
10. Orvieto R, Achiron A, Ben-Rafael Z, et al. Low-back pain of pregnancy. *Acta Obstet Gynecol Scand* 1994;73:209–14.
11. National Swedish Social Insurance Board. *Gravida kvinnors situation* [in Swedish]. (The situation of pregnant women) [Report No. 2003:7]. Stockholm: National Swedish Social Insurance Board, 2003.
12. Sydsjo G, Sydsjo A. Newly delivered women's evaluation of personal health status and attitudes towards sickness absence and social benefits. *Acta Obstet Gynecol Scand* 2002;81:104–11.
13. Brynhildsen J. *Low back pain in women in relation to different exposures to female sex hormones* [Doctoral thesis]. Linköping: Linköping University, 1998.
14. Paul JA, van Dijk FJ, Frings-Dresen MH. Work load and musculoskeletal complaints during pregnancy. *Scand J Work Environ Health* 1994;20:153–9.
15. Ostgaard HC, Andersson GB, Schultz AB, et al. Influence of some biomechanical factors on low-back pain in pregnancy. *Spine* 1993;18:61–5.
16. Mens JM, Vleeming A, Snijders CJ, et al. The active straight leg raising test and mobility of the pelvic joints. *Eur Spine J* 1999;8:468–74.
17. Sihvonen T, Huttunen M, Makkonen M, et al. Functional changes in back muscle activity correlate with pain intensity and prediction of low back pain during pregnancy. *Arch Phys Med Rehabil* 1998;79:1210–2.
18. Ostgaard HC. *Back pain and pregnancy* [Doctoral thesis]. Gothenburg: Gothenburg University, 1991.
19. Schauburger CW, Rooney BL, Goldsmith L, et al. Peripheral joint laxity increases in pregnancy but does not correlate with serum relaxin levels. *Am J Obstet Gynecol* 1996;174:667–71.
20. MacLennan AH, Nicolson R, Green RC, et al. Serum relaxin and pelvic pain of pregnancy. *Lancet* 1986;2:243–5.
21. Kristiansson P, Svardsudd K, von Schoultz B. Serum relaxin, symphyseal pain, and back pain during pregnancy. *Am J Obstet Gynecol* 1996;175:1342–7.
22. Kristiansson P, Nilsson-Wikmar L, von Schoultz B, et al. Back pain in in-vitro fertilized and spontaneous pregnancies. *Hum Reprod* 1998;13:3233–8.
23. Hansen A, Jensen DV, Larsen E, et al. Relaxin is not related to symptom-giving pelvic girdle relaxation in pregnant women. *Acta Obstet Gynecol Scand* 1996;75:245–9.
24. Petersen LK, Larsen E, Hansen A. Unmeasurable relaxin concentrations in repeated pregnancies after embryo transfer. *Eur J Obstet Gynecol Reprod Biol* 1996;65:255–7.
25. Kristiansson P, Svardsudd K, von Schoultz B. Reproductive hormones and aminoterminal propeptide of type III procollagen in serum as early markers of pelvic pain during late pregnancy. *Am J Obstet Gynecol* 1999;180(1 Pt 1):128–34.
26. Brynhildsen J, Lennartsson H, Klemetz M, et al. Oral contraceptive use among female elite athletes and age-matched controls and its relation to low back pain. *Acta Obstet Gynecol Scand* 1997;76:873–8.
27. Brynhildsen JO, Hammar J, Hammar ML. Does the menstrual cycle and use of oral contraceptives influence the risk of low back pain? A prospective study among female soccer players. *Scand J Med Sci Sports* 1997;7:348–53.
28. Bjorklund K, Nordstrom ML, Odland V. Combined oral contraceptives do not increase the risk of back and pelvic pain during pregnancy or after delivery. *Acta Obstet Gynecol Scand* 2000;79:979–83.
29. Wreje U, Isacson D, Aberg H. Oral contraceptives and back pain in women in a Swedish community. *Int J Epidemiol* 1997;26:71–4.
30. Wreje U, Brynhildsen J, Aberg H, et al. Collagen metabolism markers as a reflection of bone and soft tissue turnover during the menstrual cycle and oral contraceptive use. *Contraception* 2000;61:265–70.
31. Sydsjo A, Sydsjo G, Wijma B. Increase in sick leave rates caused by back pain among pregnant Swedish women after amelioration of social benefits: a paradox. *Spine* 1998;23:1986–90.
32. Carlson HL, Carlson NL, Pasternak BA, et al. Understanding and managing the back pain of pregnancy. *Curr Womens Health Rep* 2003;3:65–71.
33. Pool-Goudzwaard AL, Vleeming A, Stoockart R, et al. Insufficient lumbopelvic stability: a clinical, anatomical and biomechanical approach to 'a-specific' low back pain. *Man Ther* 1998;3:12–20.
34. Larsen EC, Wilken-Jensen C, Hansen A, et al. Symptom-giving pelvic girdle relaxation in pregnancy: I. Prevalence and risk factors. *Acta Obstet Gynecol Scand* 1999;78:105–10.
35. Ostgaard HC, Zetherstrom G, Roos-Hansson E. Back pain in relation to pregnancy: a 6-year follow-up. *Spine* 1997;22:2945–50.
36. The population in Sweden, in the counties and in the communes divided in sex and age-groups at 31 st December of 2002. In: *Statistical Yearbook of Sweden 2003* (In Swedish). Statistics Sweden. Stockholm, 2002.
37. Biering-Sørensen F. A prospective study of low back pain in a general population: I. Occurrence, recurrence and aetiology. *Scand J Rehabil Med* 1983;15:71–9.
38. Endresen EH. Pelvic pain and low back pain in pregnant women: an epidemiological study. *Scand J Rheumatol* 1995;24:135–41.
39. Saugstad LF. Is persistent pelvic pain and pelvic joint instability associated with early menarche and with oral contraceptives? *Eur J Obstet Gynecol Reprod Biol* 1991;41:203–6.
40. Harreby M, Kjer J, Hesselsoe G, et al. Epidemiological aspects and risk factors for low back pain in 38-year-old men and women: a 25-year prospective cohort study of 640 school children. *Eur Spine J* 1996;5:312–8.
41. Saugstad LF. Persistent pelvic pain and pelvic joint instability. *Eur J Obstet Gynecol Reprod Biol* 1991;41:197–201.