

Article

Delivery and Perinatal Outcomes Associated with Pregnancy-Related Low Back Pain in a Cohort of Physically Active Women

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Abstract

Pregnancy-related low back pain (LBP) is a highly prevalent condition with substantial consequences. However, its relationship with objective perinatal outcomes has been scarcely investigated. The aim of this study was to analyze the association between the presence and intensity of pregnancy-related LBP with delivery and perinatal outcomes. An analysis of a prospective cohort was performed including 147 physically active (PA) pregnant women, defined as those meeting the World Health Organization recommendations for PA (≥ 600 MET·min/week), assessed using the short version of the International PA Questionnaire. Participants were classified according to the presence (64.6%) or absence of LBP during pregnancy. Umbilical artery pH was significantly lower in neonates born to women with LBP compared with those without LBP (7.24 ± 0.10 vs. 7.29 ± 0.06 ; $p < 0.001$). Within the LBP group, higher pain intensity was moderately and inversely correlated with umbilical artery pH (Spearman $\rho = -0.42$, $p = 0.037$) and remained independently associated with lower umbilical artery pH values in multivariable linear regression analysis after adjustment for relevant maternal characteristics and PA. Apgar scores were globally normal, with no clinically relevant differences between groups. No significant differences were observed in other delivery and perinatal outcomes. These findings suggest an association between pregnancy-related LBP and subtle changes in fetal acid-base status at birth but should be interpreted cautiously from a clinical perspective.

Keywords: pregnancy; low back pain; pain intensity; umbilical artery pH; delivery outcomes; perinatal outcomes; physical activity



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1. Introduction

Pregnancy-related low back pain (LBP) is a highly prevalent musculoskeletal complaint during pregnancy, with reported prevalence estimates approaching 80%, although considerable variability exists depending on gestational age, study methodology, and the characteristics of the populations evaluated [1].

Available evidence suggests that the prevalence and severity of pregnancy-related LBP tend to increase progressively throughout gestation, particularly during the later stages of pregnancy [1–3], with the third trimester representing the period of greatest symptom frequency [4,5]. In addition to its high prevalence, pregnancy-related LBP may lead to

substantial functional limitations, negatively affecting mobility, occupational performance, sleep quality, emotional status, and health-related quality of life [4,6–9]. Furthermore, LBP has been associated with increased rates of sick leave and work absenteeism during pregnancy, reinforcing its clinical and socioeconomic impact [9–11].

Although pregnancy-related LBP is highly prevalent and clinically relevant, it continues to be underrecognized and inadequately managed within routine prenatal care settings [5]. Many pregnant women consider LBP to be an expected and inevitable component of pregnancy, which may contribute to reduced healthcare consultation and delayed clinical evaluation or treatment [12]. This perception may also discourage participation in regular physical activity (PA) during gestation, thereby promoting more sedentary lifestyles [12,13]. In turn, physical inactivity during pregnancy has been associated with unfavorable obstetric outcomes, including hypertensive disorders such as gestational hypertension and preeclampsia [14,15], together with higher rates of cesarean delivery [14,16].

International clinical guidelines consistently support the recommendation of regular PA during pregnancy as part of standard prenatal care because of its recognized benefits for both maternal and fetal health, while also identifying exercise as an important strategy for the prevention and conservative treatment of pregnancy-related LBP [17–20]. Consistent with these recommendations, previous studies suggest that exercise interventions focused on core muscle strengthening, postural stabilization, and low-impact modalities, such as Pilates and aquatic exercise, may contribute to reducing the intensity of LBP during pregnancy [21]. Furthermore, findings from both observational and interventional studies support a possible protective role of regular exercise against the development of pregnancy-related LBP during gestation [22,23].

Although the maternal impact of pregnancy-related LBP has been extensively described, evidence regarding its potential association with obstetric and perinatal outcomes remains sparse and inconsistent [24,25]. A limited number of studies have specifically explored the relationship between pregnancy-related LBP and delivery-related outcomes, suggesting possible associations with labor characteristics such as mode of delivery and labor onset; however, findings remain heterogeneous and inconclusive. Some observational studies have reported associations between LBP and selected delivery outcomes, indicating a potential relationship between pain severity and labor characteristics [24,26]. Beyond delivery characteristics, maternal pain and discomfort during pregnancy may influence fetal well-being through biological and behavioral pathways, including activation of the hypothalamic–pituitary–adrenal axis, alterations in cortisol regulation, inflammatory responses, reduced mobility, and sleep disturbances. Collectively, these mechanisms could potentially affect uteroplacental perfusion and fetal adaptation to labor [27]. Supporting this mechanistic framework, maternal stress has been associated with changes in placental cortisol metabolism and alterations in fetal cortisol exposure and metabolic biomarkers, indicating biologically mediated effects of maternal neuroendocrine activation on fetal physiology [28]. Despite these plausible mechanisms, objective perinatal indicators reflecting fetal condition at birth have been scarcely investigated in relation to pregnancy-related LBP. Accordingly, most available studies have primarily focused on maternal symptoms, functional impairment, and quality of life, whereas objective neonatal, perinatal, or biochemical indicators at birth have received comparatively limited attention [25,29]. Little is known about whether pregnancy-related LBP may be associated with subtle alterations in fetal condition at birth [25]. To the best of our knowledge, no previous study has specifically examined the association between pregnancy-related low back pain and umbilical artery pH at birth. In this context, objective perinatal indicators are needed to adequately assess potential fetal effects, and umbilical artery pH is a widely accepted marker of fetal acid–base status and well-being at birth [30–32].

Therefore, the aim of this study was to examine the association between pregnancy-related LBP and objective perinatal outcomes in PA women, with a particular focus on umbilical artery pH at birth as an indicator of fetal acid–base status. Secondary objectives included the evaluation of other immediate perinatal outcomes, such as Apgar scores at 1 and 5 min, onset and mode of delivery, gestational age at birth, birth weight, and the need for neonatal resuscitation.

2. Results

2.1. Study Population and Prevalence of Pregnancy-Related LBP

The final cohort included 147 women with low-risk pregnancies who fulfilled the WHO criteria for being PA during gestation. Mean PA during pregnancy was 1896.7 ± 1012.1 MET·min/week (range: 660.0–6597.0 MET·min/week), with percentile values of P25 = 1229.5, P50 = 1746.0, and P75 = 2390.0 MET·min/week, reflecting activity levels substantially above current recommendations. The mean maternal age was 34.1 ± 4.0 years. Most participants were primiparous (66.7%), had completed university education (92.5%), and were employed outside the home (97.3%). Among employed women, full-time employment predominated, with a mean daily working duration of 7.7 h. Sedentary occupational posture was the most frequently reported work pattern (55.1%), followed by mixed postures (30.6%) and predominantly standing positions (14.3%).

Pregnancy-related LBP during gestation was reported by 95 women (64.6%), whereas 52 participants (35.4%) reported no LBP. Baseline maternal characteristics are presented in Table 1. Women without LBP were slightly older than those reporting pregnancy-related LBP (35.2 ± 3.6 vs. 33.5 ± 4.2 years; $p = 0.011$). No statistically significant between-group differences were identified regarding prepregnancy BMI, parity, or PA levels during pregnancy.

Table 1. Maternal baseline characteristics according to the presence of pregnancy-related low back pain.

Variable	LBP (n = 95)	No LBP (n = 52)	p Value
Maternal age (years)	33.5 ± 4.2	35.2 ± 3.6	0.011
Prepregnancy BMI (kg/m^2)	23.4 ± 3.5	23.2 ± 3.4	0.736
Primiparous, n (%)	52 (54.7)	25 (48.1)	0.548
Physical activity (MET·min/week)	1897.8 ± 952.8	1894.8 ± 1112.1	0.987

BMI: body mass index; MET: metabolic equivalent of task.

2.2. Obstetric and Perinatal Outcomes According to the Presence of Pregnancy-Related LBP

Obstetric and perinatal outcomes according to the presence of pregnancy-related LBP are presented in Table 2. Mean gestational age at delivery was similar in women with and without LBP (39.7 ± 1.1 vs. 39.8 ± 1.0 weeks). Birth weight did not differ significantly between groups. Apgar scores at both 1 and 5 min were high in the two groups. The Apgar score at 1 min was slightly lower in the LBP group, although this difference did not reach statistical significance ($p = 0.054$). No difference was observed in the Apgar score at 5 min. Umbilical artery pH differed significantly between groups, with lower mean values observed in neonates born to women with LBP compared with those born to women without LBP (7.24 ± 0.10 vs. 7.29 ± 0.06 ; $p < 0.001$). Although the observed differences were modest, mean umbilical artery pH values were statistically significantly lower in neonates born to women with LBP and were located at the lower threshold of normality, overlapping with values commonly described as mild fetal preacidosis, whereas

values in the non-LBP group remained within the normal range. The need for neonatal resuscitation and admission to the neonatal unit was infrequent in the overall sample and did not differ significantly according to maternal LBP status. Sensitivity analyses using non-parametric tests (Mann–Whitney U) yielded results consistent with those obtained using parametric methods.

Table 2. Obstetric and perinatal outcomes according to the presence of pregnancy-related low back pain.

Variable	LBP (n = 95)	No LBP (n = 52)	p Value
Gestational age at delivery (weeks)	39.7 ± 1.1	39.8 ± 1.0	0.784
Birth weight (g)	3300.8 ± 516.0	3269.3 ± 430.2	0.711
Apgar score at 1 min	8.65 ± 1.04	8.88 ± 0.37	0.054
Apgar score at 5 min	9.57 ± 0.85	9.65 ± 0.51	0.321
Umbilical artery pH	7.24 ± 0.10	7.29 ± 0.06	<0.001
Neonatal resuscitation, n (%)	8 (8.4)	6 (11.5)	0.57
Neonatal unit admission, n (%)	6 (6.3)	6 (11.5)	0.35

2.3. Onset of Labor and Mode of Delivery

The onset of labor and mode of delivery according to LBP status are shown in Table 3. Spontaneous onset of labor was the most frequent pattern in both groups. The distribution of spontaneous labor, induced labor, and elective cesarean section was similar in women with and without LBP. Likewise, no significant differences were observed in the mode of delivery. The proportions of normal vaginal delivery, operative vaginal delivery, and cesarean delivery during labor, were not different between groups.

Table 3. Onset of labor and mode of delivery according to the presence of pregnancy-related low back pain.

Variable	LBP (n = 95)	No LBP (n = 52)	p Value
Onset of labor			0.642
Spontaneous, n (%)	56 (58.9)	27 (51.9)	
Induced, n (%)	31 (32.6)	21 (40.4)	
Elective cesarean, n (%)	8 (8.4)	4 (7.7)	
Mode of delivery			0.865
Normal vaginal delivery, n (%)	58 (61.0)	34 (65.4)	
Operative vaginal delivery, n (%)	11 (11.6)	5 (9.6)	
Cesarean delivery during labor, n (%)	26 (27.4)	13 (25.0)	

2.4. Associations Between Lumbar Pain Intensity, Physical Activity Level, and Neonatal Outcomes in Women with Pregnancy-Related LBP

Spearman correlation analyses restricted to women with pregnancy-related LBP (n = 95) are summarized in Table 4. Maximum lumbar pain intensity was moderately and inversely correlated with umbilical artery pH, indicating lower pH values with increasing pain intensity. In addition, weaker inverse correlations were observed between maximum pain intensity and Apgar scores at 1 and 5 min. Mean lumbar pain intensity was also inversely correlated with umbilical artery pH and Apgar score at 1 min, whereas

its association with Apgar score at 5 min did not reach statistical significance. In contrast, PA level showed positive but non-significant correlations with umbilical artery pH and was not significantly associated with Apgar scores. These correlations are reported for descriptive completeness and should not be interpreted as evidence of a statistically significant association. Overall, both maximum and mean lumbar pain intensity were inversely associated with umbilical artery pH and early neonatal indicators, whereas PA level was not significantly associated with neonatal outcomes.

Table 4. Correlations between lumbar pain intensity, physical activity, and neonatal outcomes in women with pregnancy-related low back pain.

Variable	Umbilical Artery pH (ρ)	Apgar Score 1 min (ρ)	Apgar Score 5 min (ρ)
Maximum lumbar pain (VAS)	−0.42 *	−0.31 *	−0.29 *
Mean lumbar pain (VAS)	−0.36 *	−0.27 *	−0.25
Physical activity (MET·min/week)	0.34	0.20	0.18

Spearman correlation coefficient (ρ); VAS: visual analog scale; MET: metabolic equivalent of task; * $p < 0.05$.

2.5. Multivariable Linear Regression Analysis of Umbilical Artery pH

To further examine whether the association between LBP intensity and umbilical artery pH was independent of maternal characteristics and PA level, a multivariable linear regression analysis was performed (Table 5). Maximum LBP intensity, assessed using the VAS, remained independently associated with lower umbilical artery pH values after adjustment for PA during pregnancy, maternal age, prepregnancy BMI, and parity. Specifically, each one-point increase in maximum LBP intensity was associated with a decrease of 0.022 units in umbilical artery pH ($\beta = -0.022$; 95% CI: −0.043 to −0.001; $p = 0.042$). In contrast, PA level, maternal age, prepregnancy BMI, and primiparity were not significantly associated with umbilical artery pH in the adjusted model.

Table 5. Multivariable linear regression model for umbilical artery pH.

Predictor	β Coefficient	95% Confidence Interval	p Value
Maximum lumbar pain intensity (VAS)	−0.022	−0.043 to −0.001	0.042
Physical activity (per 500 MET·min/week)	0.016	−0.020 to 0.052	0.378
Maternal age (years)	−0.008	−0.019 to 0.004	0.189
Prepregnancy BMI (kg/m^2)	−0.002	−0.013 to 0.010	0.779
Primiparous (yes vs. no)	−0.001	−0.114 to 0.111	0.979

VAS: Visual Analog Scale; MET: metabolic equivalent of task; BMI: body mass index.

3. Discussion

In this cohort of PA pregnant women, pregnancy-related LBP was common and associated with a modest but statistically significant reduction in umbilical artery pH at birth, whereas most conventional obstetric and neonatal outcomes remained comparable between groups. Neonates born to women with LBP showed lower mean arterial pH values compared with those born to women without LBP, with values approaching the lower limit of the physiological range and partially overlapping with thresholds commonly described as mild fetal preacidosis. Apgar scores at 1 and 5 min were similar between

groups, and no significant differences were observed in gestational age at delivery, birth weight, onset of labor, mode of delivery, need for neonatal resuscitation, or neonatal unit admission. Within women with LBP, higher maximum and mean lumbar pain intensity were inversely correlated with umbilical artery pH and early neonatal clinical status. Importantly, this association persisted after adjustment for PA level and key maternal characteristics in multivariable linear regression analysis, supporting the robustness of the observed relationship. In contrast, PA level showed no statistically significant associations with neonatal outcomes. These findings indicate a subtle association between maternal LBP severity and neonatal acid–base status at birth, without clinically overt adverse perinatal outcomes.

Therefore, the principal contribution of the present study is the identification of a modest but statistically significant association between pregnancy-related LBP severity and umbilical artery pH, while other conventional perinatal outcomes remained largely unchanged.

Literature examining the influence of pregnancy-related LBP on obstetric and perinatal outcomes remains limited and heterogeneous. A cross-sectional prospective study by Fruscalzo et al. reported a higher rate of cesarean section among women with LBP compared with those without LBP, suggesting a potential association between pain and increased obstetric intervention, although no differences in operative vaginal deliveries were observed [24]. In contrast to these findings, our cohort, composed exclusively of PA women, did not show differences in labor onset or mode of delivery according to LBP status. This discrepancy may reflect differences in study populations and clinical contexts, including baseline PA level, pain severity and functional impact, obstetric management protocols, and overall maternal health profile. PA, as a marker of better functional capacity and physiological reserve, may partly act as a modifying factor in the relationship between pregnancy-related LBP and obstetric interventions, potentially attenuating the need for operative delivery in otherwise low-risk women.

In line with this interpretation, Brown et al. reported that higher levels of musculoskeletal pain during the third trimester, particularly LBP and pelvic pain, were associated with increased rates of cesarean section, assisted delivery, and longer labor duration. The authors suggested that greater pain severity and functional limitation might contribute to delivery complications, potentially through mechanisms such as altered fetal positioning or reduced maternal mobility during late pregnancy [26].

Regarding neonatal outcomes, our results for conventional parameters are consistent with earlier observational research indicating that pregnancy-related LBP does not substantially affect standard perinatal metrics. Ostgaard et al. reported no significant differences in Apgar scores, birth weight, or neonatal length in relation to the presence of LBP during pregnancy, supporting the notion that clinically meaningful differences in classic neonatal outcomes are not consistently observed in this population [33]. In line with these findings, Apgar scores in our cohort were similar between women with and without LBP, with only a borderline reduction at 1 min that resolved by 5 min. Although Apgar scores are frequently categorized for clinical interpretation, nearly all neonates in our cohort had values within the normal range (7–10), with minimal variability and no clinically relevant depressed scores. Therefore, continuous analysis was considered appropriate to preserve statistical information.

A novel contribution of the present work is the integration of umbilical artery pH as an objective marker of fetal acid–base balance at birth within the analysis of the association between pregnancy-related LBP and delivery outcomes in PA women. Umbilical cord blood gas analysis, particularly arterial pH, is widely used as an indicator of intrapartum fetal status and has recognized prognostic value for adverse neonatal outcomes at more

severe thresholds. In clinical and research settings, an arterial pH < 7.0 is commonly used to define severe fetal acidemia, often in combination with base deficit, and has been associated with increased neonatal risk [34]. In this context, a large systematic review and meta-analysis by Malin et al. demonstrated a strong association between low umbilical cord pH and adverse perinatal and long-term outcomes, including increased neonatal mortality and neurological morbidity [31]. Although mean pH values in both groups in our study remained within the normal range (>7.20), neonates born to women with pregnancy-related LBP exhibited significantly lower umbilical artery pH values, approaching the lower limit of normality. Together with the observed inverse relationship between pain intensity and pH, as well as the dose–response pattern identified, these findings suggest that greater maternal pain severity may be linked to subtle physiological shifts in intrapartum fetal adaptation, even in the absence of clinically overt neonatal compromise. However, the absolute differences observed were modest and should be interpreted cautiously from a clinical perspective. Potential biological mechanisms underlying these associations may involve neuroendocrine and stress-related pathways. Pregnancy-related LBP has been associated with functional limitation, sleep disturbances, and psychosocial stress. These factors can activate the hypothalamic–pituitary–adrenal axis and sympathetic responses. In turn, this activation may affect uteroplacental perfusion and fetal tolerance to intrapartum stress. These mechanisms are consistent with broader perinatal research linking maternal stress exposures to neonatal biochemical markers, though direct evidence specifically connecting LBP with cord gas alterations remains sparse and warrants further investigation [29,35]. These proposed pathways are speculative and were not directly evaluated in the present study, which was not designed to assess neuroendocrine or placental biomarkers. Therefore, these mechanisms should not be interpreted as evidence of causality but rather as biologically plausible hypotheses requiring further investigation. Future research incorporating objective biomarkers (e.g., cortisol levels or placental function indicators) is needed to clarify potential causal pathways.

PA in our cohort, defined according to recognized guideline thresholds, did not exhibit statistically significant correlations with neonatal outcomes, suggesting that in an already active population, general PA level may be less influential on immediate perinatal acid–base status than pain severity per se. This is consistent with the multifactorial nature of pregnancy-related LBP [29] and underscores the complexity of disentangling behavioral and physiological factors in this context. Collectively, our results do not suggest an association between pregnancy-related LBP in PA, low-risk women and increased obstetric interventions or major adverse neonatal outcomes. Nevertheless, the observed association between higher pain intensity and lower umbilical artery pH, while remaining within normal limits, suggests that maternal pain severity may be linked to subtle alterations in intrapartum fetal acid–base status. However, these differences remained relatively small and were not accompanied by clinically overt compromise or deterioration in conventional neonatal outcomes, although they underscore the importance of systematic assessment and appropriate management of LBP during pregnancy.

A major strength of the present study is the inclusion of umbilical artery pH as an objective perinatal outcome, providing a direct and clinically relevant indicator of fetal acid–base status at birth [30,32]. To our knowledge, this is the first study to specifically examine the association between pregnancy-related LBP and umbilical artery pH, addressing a relevant gap in the literature, as previous studies have primarily focused on maternal symptoms or conventional neonatal outcomes without incorporating biochemical markers of fetal well-being. The study is further strengthened by the use of objective delivery and perinatal outcomes to evaluate their association with pregnancy-related LBP, including umbilical artery pH, Apgar scores, gestational age at delivery, birth weight, onset of

labor, mode of delivery, and neonatal resuscitation requirements. The incorporation of these standardized and clinically meaningful endpoints allows the relationship between maternal LBP and delivery and perinatal outcomes to be examined beyond subjective symptom reporting, enhancing the robustness of the findings and reducing the influence of measurement subjectivity on the main outcomes.

A further strength of the present study lies in the relatively homogeneous profile of the cohort, composed exclusively of PA women with low-risk pregnancies who fulfilled WHO criteria based on standardized MET·min/week thresholds [36]. This strategy reduced potential confounding associated with sedentary lifestyle, maternal comorbidities, and obstetric risk factors, thereby facilitating a more specific evaluation of the association between pregnancy-related LBP and perinatal outcomes. Moreover, the prospective design, together with standardized first-trimester recruitment and objective assessment of delivery and perinatal outcomes at birth, reinforces the temporal consistency of the observed associations. The detailed characterization of pregnancy-related LBP, including assessment of pain intensity and positional variability, also allowed exploration of potential dose–response relationships with neonatal outcomes, supporting the biological plausibility of the findings [27,28,35]. Finally, the integration of perinatal biochemical markers with clinical neonatal outcomes provides a more comprehensive evaluation of fetal condition at birth, contributing novel and clinically informative evidence to the existing literature on pregnancy-related LBP.

Nevertheless, several limitations should be acknowledged when interpreting the findings of the present study. Although participant recruitment and outcome collection were conducted prospectively, information regarding pregnancy-related LBP was obtained retrospectively through a structured postpartum telephone interview. Consequently, self-reported variables related to pain characteristics, including onset, frequency, and positional intensity, may have been influenced by recall bias [37,38]. In contrast, obstetric and perinatal outcomes were extracted directly from electronic medical records and therefore were not affected by participant recall.

To reduce potential recall bias in pain assessment, the postpartum interview was performed within the first month after delivery, thereby limiting the interval between symptom occurrence and retrospective reporting [37,38]. Participants had previously been informed at recruitment that pregnancy-related outcomes would be reassessed postpartum and had already completed the assessment instruments during the first-trimester face-to-face visit. In addition, women received a printed 0–10 VAS at recruitment and were instructed to retain the scale for reference during the telephone interview, which may have improved consistency in pain reporting. Nevertheless, although the same numerical scale was used in both assessments, the different modes of administration (face-to-face versus telephone-based assessment) could have introduced minor measurement variability.

In addition, women experiencing more complex or stressful childbirth experiences may potentially recall or report pregnancy-related pain differently, which could contribute to differential recall bias. Although previous studies suggest that maternal recall of pregnancy-related events may demonstrate acceptable validity and reproducibility, the possibility of memory-related inaccuracies cannot be completely excluded, even when participants are informed during pregnancy that this information will subsequently be collected [37,38]. Furthermore, the development and full validation of reliable, pregnancy-specific instruments for assessing PA during gestation remain necessary. Moreover, PA was assessed globally for pregnancy rather than longitudinally across trimesters; therefore, temporal changes in activity patterns during gestation could not be evaluated.

Furthermore, the ad hoc questionnaire used to characterize pregnancy-related LBP did not undergo formal psychometric validation. Although the instrument was specif-

ically developed to capture clinically relevant and pregnancy-specific pain characteristics, the absence of formal validation procedures may limit comparability with studies employing standardized instruments and could reduce the reproducibility of certain descriptive findings.

Umbilical artery pH may be influenced by intrapartum factors such as labor characteristics, fetal distress, mode or urgency of delivery. In our cohort, detailed intrapartum variables such as duration of labor stages, epidural analgesia use, or meconium-stained amniotic fluid were not systematically included in the regression model. However, no statistically significant differences were observed between women with and without LBP regarding labor onset (spontaneous vs. induced), elective cesarean rates, or mode of delivery (vaginal, instrumental, or intrapartum cesarean). These similarities reduce the likelihood of major imbalance in key delivery characteristics between groups. Nevertheless, residual confounding due to unmeasured intrapartum factors cannot be excluded. Therefore, the observed associations should be interpreted cautiously, particularly regarding potential intrapartum influences on umbilical artery pH values. Moreover, other potential confounders, such as gestational age at LBP onset, use of analgesics, or maternal comorbidities, were not systematically included in the regression model. Some of these variables were explored in the original cohort analysis [5] but were not the primary focus of the present study; therefore, their potential influence on the observed associations cannot be excluded.

The study design exclusively included women who met the recommended PA criteria during pregnancy, which limits the ability to evaluate the relative contribution of regular exercise to the occurrence and characteristics of pregnancy-related LBP. Consequently, the associations observed between LBP and delivery and perinatal outcomes must be interpreted strictly from an observational perspective, without inferring protective or modifying effects attributable to PA compared with sedentary populations.

This study represents a secondary analysis of a previously published prospective cohort [5], and the sample size was determined by the available cohort composition. Although the number of participants is moderate, the cohort has been previously peer-reviewed and characterized in detail, supporting the internal consistency of the findings. Nevertheless, the results should be interpreted as exploratory and confirmed in larger studies.

Additionally, although the analyses were adjusted for relevant maternal characteristics, women without LBP were slightly older than those with LBP, and residual confounding cannot be entirely excluded.

Finally, the relatively homogeneous nature of the study sample should also be recognized as a limitation affecting the external validity of the findings. Most participants had a high educational level (92.5% held a university degree), all fulfilled recommended PA criteria during pregnancy, and recruitment was conducted in a private university hospital setting. These characteristics may have contributed to selection bias and may limit the applicability of the results to populations with different educational, socioeconomic, or PA profiles. Nevertheless, this homogeneity also enhanced internal consistency and reduced potential confounding related to sedentary behavior, obstetric risk, and clinical variability. Therefore, although the relative homogeneity of the cohort strengthens internal validity, caution is warranted when extrapolating these findings to broader and more heterogeneous populations. Taken together, these limitations should be considered when interpreting the findings, particularly in terms of external validity and causal inference.

4. Materials and Methods

4.1. Study Design and Setting

The study was conducted at La Moraleja University Hospital (Madrid, Spain). The present investigation represents a secondary analysis of a previously published prospective cohort of PA pregnant women ($n = 147$), in which the incidence and clinical characteristics of pregnancy-related LBP had been previously evaluated [5]. The cohort included women with low-risk pregnancies attending a specialized first-trimester obstetric clinic, where regular physical exercise throughout pregnancy was routinely recommended. Recruitment and baseline assessment were performed between 11 and 13 + 6 weeks of gestation.

The overall study period extended from October 2023 to June 2025 and consisted of two consecutive phases: a first phase from October 2023 to September 2024, followed by a second phase completed in June 2025. Obstetric and perinatal outcomes were collected through a systematic review of electronic medical records. The variables evaluated included gestational age at delivery, mode of delivery, neonatal birth weight, Apgar scores at 1 and 5 min, umbilical artery pH, and admission to the neonatal unit when applicable. As these data were obtained directly from clinical records, they were not influenced by recall bias.

Participant recruitment and the assessment of obstetric and perinatal outcomes were performed prospectively through routine antenatal follow-up and review of medical records. In contrast, information regarding pregnancy-related LBP characteristics and PA during gestation was obtained postpartum using a structured self-report interview, thereby incorporating a retrospective component into exposure assessment. Consequently, the study presents an ambispective component, being prospective for obstetric and perinatal outcomes and retrospective for self-reported exposure variables, which may have introduced recall bias in self-reported variables but not in objectively recorded obstetric and perinatal outcomes.

Before inclusion, all participants received verbal and written information describing the objectives and procedures of the study and subsequently provided written informed consent. Participation was entirely voluntary, and all women were informed that they could withdraw their consent at any stage of the investigation. The study protocol received approval from the Research Ethics Committee of La Princesa University Hospital (Madrid, Spain) (approval No. 5365).

4.2. Study Population

4.2.1. Inclusion Criteria

Eligible participants were pregnant women aged 18 years or older with a singleton, low-risk pregnancy showing normal physiological progression. Inclusion additionally required regular engagement in PA during the six months preceding pregnancy, at the time of recruitment, and throughout gestation, as well as attendance at the standardized first-trimester visit for ultrasound assessment and routine obstetric follow-up.

PA during pregnancy was classified according to the World Health Organization (WHO) Guidelines on Physical Activity and Sedentary Behavior for pregnant women [36]. These recommendations establish a minimum of 150 min per week of moderate-intensity aerobic PA, or an equivalent combination of moderate and vigorous-intensity activity, corresponding to at least 600 metabolic equivalent of task minutes per week (MET·min/week). PA levels were assessed both at study inclusion through face-to-face evaluation and postpartum by telephone interview using the short-form of the International Physical Activity Questionnaire (IPAQ) [39]. The IPAQ has been widely applied in pregnant populations and has demonstrated good test-retest reliability [40], in addition to being validated in the Spanish non-pregnant population [41]. During the postpartum telephone interview, participants reported PA corresponding to a typical week during pregnancy, using an

adapted recall framework instead of the conventional IPAQ “last 7 days” format. Likewise, at recruitment during the first trimester, women reported PA levels corresponding to a typical week during pregnancy and during the six months preceding conception. Participants had previously completed the IPAQ during the face-to-face first-trimester visit and were informed at recruitment that gestational PA would be reassessed postpartum, which facilitated participant familiarity with the questionnaire structure and PA reporting.

4.2.2. Exclusion Criteria

Participants were not eligible for inclusion if, at the time of recruitment, they presented any obstetric or pregnancy-related pathology, or any acute or chronic medical condition, including musculoskeletal, cardiovascular, neurological, or other diseases, that could interfere with or contraindicate the safe practice of physical exercise during pregnancy.

Women who did not comply with the WHO recommendations for PA either during pregnancy or during the six months preceding conception were likewise excluded from the analysis. In particular, participants reporting less than 150 min per week of moderate-intensity PA, equivalent to <600 MET·min/week, were not eligible for inclusion. Therefore, the final cohort was composed exclusively of PA pregnant women.

4.3. Instruments

At the time of recruitment, participants received a comprehensive written information dossier on PA during pregnancy. This material summarized the evidence-based benefits of exercise for maternal, fetal, neonatal, and infant health and provided practical guidance regarding the recommended duration, frequency, intensity, and types of PA. It also included information on absolute and relative contraindications to exercise, warning signs requiring cessation of activity, activities to be avoided during pregnancy, and strategies aimed at preventing or alleviating LBP during gestation.

Questionnaires

Data collection was performed using a specifically developed ad hoc questionnaire. During the first-trimester face-to-face interview conducted at recruitment, information regarding participant characteristics was obtained, including sociodemographic data, relevant medical and obstetric history, lifestyle variables, and anthropometric measurements. Because no validated international or Spanish instrument was considered sufficiently comprehensive to capture the clinical and contextual variables relevant to the present investigation, a tailored questionnaire was designed for this study. The questionnaire content was reviewed by obstetric specialists and researchers experienced in questionnaire design to ensure adequate clinical relevance and conceptual validity. Formal pilot validation procedures were not performed before its implementation.

The ad hoc questionnaire was developed to obtain a detailed clinical description of pregnancy-related LBP and its perceived impact during gestation. Information was collected regarding pain characteristics, including onset, duration, irradiation, aggravating factors, perceived triggers and associated functional limitations affecting activities such as work, sleep, and walking. Additional variables included emotional impact, healthcare utilization, and self-perceived effectiveness of management strategies. The instrument was intended for descriptive and exploratory purposes and was designed to capture the clinical and contextual experience of pregnancy-related LBP rather than to establish diagnostic classifications or standardized disability scores. Consequently, specific disability instruments such as the Oswestry Disability Index [42] were not used. Although widely applied in chronic LBP populations [43], such scales may be less appropriate for assessing pregnancy-specific pain characteristics, temporal variability, contextual triggers and obstetric-related functional limitations relevant to the objectives of the present investigation.

During the same first-trimester interview, PA performed during the six months preceding conception was assessed using the short-form IPAQ. These data were collected to characterize habitual pre-pregnancy activity levels and to examine potential differences between pre-pregnancy and gestational PA patterns, as well as their possible relationship with the presence and intensity of pregnancy-related LBP.

Following delivery, participants completed a structured postpartum telephone interview. PA or exercise performed during pregnancy was reassessed using the short-form IPAQ. Information regarding pregnancy-related LBP was also obtained through a structured questionnaire addressing pain location, frequency, duration, clinical characteristics, aggravating factors, management approaches, and perceived effectiveness of interventions. Because recommendations and therapeutic advice for LBP were not standardized among healthcare professionals, these management-related variables were collected for descriptive purposes only and were not incorporated into the analytical models. Participants additionally reported their perceived usefulness of the information dossier and whether it had influenced their motivation to remain PA during pregnancy; however, these findings were considered ancillary to the primary objectives of the present investigation and are therefore not presented in detail.

Pain severity was quantified using a 0–10 Visual Analog Scale (VAS), with higher scores indicating greater pain intensity. Assessments were recorded for four body positions: sitting, standing, supine, and lateral decubitus. During the first-trimester face-to-face interview, participants received the VAS in printed numerical format and were asked to retain the scale for use during the postpartum telephone assessment, thereby maintaining consistency between evaluations. Although data collection was performed through different modes of interaction, the format of the scale remained unchanged across both assessments. Pregnancy-related LBP was defined as pain localized between the twelfth rib and the gluteal fold, with or without irradiation to the lower extremities.

4.4. Sample Size

Given the nature of this study as a secondary analysis of an existing cohort [5], the available sample size ($n = 147$) was determined by the cohort composition, which has been previously described. The sample size is consistent with the original cohort and allows for the detection of clinically relevant differences in the primary outcome.

4.5. Statistical Analysis

Distributional properties of continuous variables were evaluated by visual inspection of histograms and Q–Q plots. Variables showing approximate normal distribution were analyzed using parametric methods (independent-samples Student's t -test), whereas non-normally distributed or ordinal variables were analyzed using non-parametric tests. Data are presented as mean $\pm$ standard deviation (SD) for continuous variables and as number (percentage) for categorical variables. Baseline maternal characteristics, together with obstetric and perinatal outcomes, were compared between women with and without pregnancy-related LBP. Group comparisons for continuous variables were performed using the independent-samples Student's t -test, while categorical variables were analyzed using the Chi-square (χ^2) test or Fisher's exact test when appropriate. Because some variables deviated from strict normality assumptions, sensitivity analyses using the Mann–Whitney U test were additionally conducted for the principal between-group comparisons, yielding findings consistent with the primary analyses. Among women with pregnancy-related LBP, associations between pain intensity (VAS), PA level (MET·min/week), and neonatal outcomes (umbilical artery pH and Apgar scores at 1 and 5 min) were evaluated using Spearman's rank correlation coefficient (ρ), given the non-normal distribution of

pain intensity and PA variables together with the ordinal nature of Apgar scores. To determine whether LBP intensity was independently associated with umbilical artery pH, a multivariable linear regression analysis was performed, with umbilical artery pH entered as a continuous dependent variable. Maximum LBP intensity (VAS) was included as the main independent variable. The model was adjusted for PA during pregnancy (entered per 500 MET·min/week), maternal age, prepregnancy body mass index (BMI), and parity according to clinical relevance. Regression assumptions were assessed through visual inspection of residual plots. Regression coefficients (β), 95% confidence intervals (95% CI), and corresponding p values were reported. With the available sample ($n = 147$; 95 women with LBP and 52 without LBP) and an observed pooled standard deviation of approximately 0.09 for umbilical artery pH, a post hoc power calculation indicated that the study had approximately 80% power (two-sided $\alpha = 0.05$) to detect a minimum absolute difference of approximately 0.04–0.05 pH units between groups. All statistical analyses were performed using R software (version 4.3.2). Statistical significance was established at $p < 0.05$.

5. Conclusions

In this cohort of PA, low-risk pregnant women, pregnancy-related LBP was common and associated with a small but statistically significant reduction in umbilical artery pH at birth. Greater pain intensity was independently associated with lower umbilical artery pH values, supporting a dose–response relationship, whereas PA level within this active sample was not associated with adverse perinatal outcomes. No clinically relevant differences were observed in Apgar scores, birth weight, gestational age at delivery, onset or mode of labor, or neonatal care needs. These findings suggest that greater maternal musculoskeletal pain severity may be associated with subtle intrapartum changes in fetal acid–base status, although the observed differences were modest and not accompanied by clinically overt adverse perinatal outcomes, and underscore the importance of systematic assessment and appropriate management of LBP during pregnancy. Further studies in more heterogeneous populations, including less PA women, are warranted to clarify generalizability and underlying mechanisms. Accordingly, extrapolation of the present findings to broader obstetric populations should be performed with caution.

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