6 Specific Musculoskeletal Adaptations in Pregnancy: Pelvic Floor, Pelvic Girdle, and Low Back Pain: Implications for Physical Activity and Exercise

Kari Bø, Britt Stuge, and Gunvor Hilde

Contents

K. Bø (✉)
Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway

Department of Obstetrics and Gynecology, Akershus University Hospital, Lørenskog, Norway
e-mail: kari.bo@nih.no

B. Stuge
Division of Orthopaedic Surgery, Oslo University Hospital, Oslo, Norway
e-mail: britt.stuge@medisin.uio.no

G. Hilde
Department of Physiotherapy, OsloMet-Oslo Metropolitan University (Former: Oslo and Akershus University College of Applied Sciences), Oslo, Norway
e-mail: gunvor.Hilde@hioa.no

R. Santos-Rocha (ed.), *Exercise and Sporting Activity During Pregnancy*,
https://doi.org/10.1007/978-3-319-91032-1_6

Abstract

Pregnancy and childbirth bring along several changes to a woman's body, especially to the musculoskeletal system. Pregnancy represents a window of opportunity for the adoption of an active and healthy lifestyle, but it is also a risk period for musculoskeletal disorders, impairments, and other discomforts. This chapter addresses the evidence-based knowledge on the most prevalent pelvic floor muscle dysfunction (urinary incontinence), diastasis recti abdominis, pregnancy-related low back pain, and/or pelvic girdle pain, since these factors are reported to have a negative effect on daily activities. The chapter also provides recommendations for treatment of such disorders and guidance on how to recover functional capacity.

Keywords

Pregnancy · Postpartum · Pelvic floor · Low back pain · Pelvic girdle pain Physical activity · Exercise

6.1 Introduction

Pregnancy and childbirth bring along several changes to a woman's body, especially to the musculoskeletal system. Pregnancy represents a window of opportunity for the adoption of an active and healthy lifestyle, but it is also a risk period for musculoskeletal disorders, impairments, and other discomforts. This chapter addresses the evidence-based knowledge on the most prevalent pelvic floor muscle dysfunction (urinary incontinence), diastasis recti abdominis, pregnancy-related low back pain and/or pelvic girdle pain, since these factors are reported to have a negative effect on daily activities. The chapter also provides recommendations for treatment of such disorders and guidance on how to recover functional capacity.

6.2 The Pelvic Floor

The urethra, bladder, vagina, uterus, and bowel situated within the pelvis are given structural support by pelvic floor structures arranged into a superficial and a deep layer of muscles and connective tissue (ligaments and fascia) [1]. In addition to pelvic organ support, the pelvic floor maintains continence, permitting urination, defecation, intercourse, and vaginal birth [1].

The superficial layer of the pelvic floor includes the perineal muscles (ischiocavernosus, bulbospongiosus, and transversus perinei superficialis), and the deep layer includes the levator ani (LA) muscle [1]. These pelvic floor layers in addition to the urethral and anal sphincter system (external and internal sphincter muscles and vascular elements within the submucosa) play a significant role in maintaining pelvic organ support and continence [1–3].

The pelvic floor muscles (PFM) interact with the supportive ligaments and fasciae in order to maintain support of the pelvic organs and protect the pelvic floor connective tissue from excessive loads [3–5]. The function of this supportive system is illustrated by the "boat in dry dock theory" by Norton [5], where the PFM act as water in the dock floating the boat (pelvic organs) unloading the mooring (ligaments and fasciae) holding the boat in place. If the water is removed (loss of pelvic floor muscle tone), the moorings (pelvic ligaments and fasciae) are placed under excessive strain.

A voluntary PFM contraction can best be described as an inward lift and squeeze around the urethra, vagina, and rectum [6, 7]. During a voluntary PFM contraction, the medial portion of the LA muscle interacts with the endopelvic fasciae and compresses the urethra against adjacent tissues, which creates increased urethral pressure and stabilization of the urethra and bladder neck [2, 3].

The normal baseline activity of the PFM keeps the pelvic openings closed and keeps the pelvic floor elevated in a cranial direction [4, 8]. In situations where abdominal pressure increases, during physical exertions such as coughing, laughing, high-impact activities, etc., a simultaneous well-timed PFM contraction will counteract the increased abdominal pressure by increased structural support and compression of the urethra [2–4, 9]. The PFM is supposed to react automatically when the abdominal pressure increases. The pelvic floor works like a "firm trampoline" giving a quick response when loads are put onto it [10].

Together with the urethral sphincter muscles, the PFM play an important role for maintaining urinary continence [2–4, 9]. The mechanical supportive potential of the PFM is demonstrated by Miller et al. [11]. By perineal ultrasound assessment, they found that a voluntary contraction of the PFM prior to and during a cough (a maneuver called the "Knack") resulted in a significant reduced displacement of the bladder neck [11]. Use of the "Knack" maneuver has also shown to significantly reduce urine loss among women with SUI [12, 13].

6.2.1 Pelvic Floor Dysfunction and Risk Factors

The understanding of the development of pelvic floor muscle dysfunction is far from complete. Rather than a single factor, the most common types of pelvic floor dysfunction (UI, fecal incontinence, and pelvic organ prolapse) probably have a complex list of risk factors [14–16]. Factors that may lead to the development of pelvic floor impairment and dysfunction in women can according to Bump and Norton [14] be classified into the following four categories:

- *Predisposing factors*: e.g., gender, racial, neurologic, anatomic, collagen, muscular, cultural, and environmental.
- *Inciting factors*: e.g., childbirth, nerve damage, muscle damage, radiation, tissue disruption, and radical surgery.
- *Promoting factors*: e.g., constipation, occupation, recreation, obesity, surgery, lung disease, smoking, menstrual cycle, infection, medication, and menopause.
- *Decompensating factors*: e.g., aging, dementia, debility, disease, environment, and medication.

DeLancey et al. [16] integrate factors affecting pelvic floor dysfunction into an "Integrated Lifespan Model," in which pelvic floor function is plotted into three major life phases: (1) development of functional reserve during growth, influenced by predisposing factors, e.g., genetic constitution; (2) amount of injury and potential recovery occurring during and after childbirth; and (3) deterioration occurring with advancing age. Throughout the lifespan a decline of the functional reserve of the pelvic floor may be accelerated by other factors, e.g., obesity and chronic coughing, medications, and dementia.

Knowledge about the various risk factors and their relative importance in relation to type of pelvic floor dysfunction is essential for primary and secondary prevention strategies [14, 16].

6.2.2 Urinary Incontinence

UI has been defined by the International Continence Society as "the complaint of any involuntary leakage of urine" and can further be classified into subtypes with the following definitions [17]:

- Stress urinary incontinence (SUI): "the complaint of involuntary leakage on effort or exertion, or on sneezing or coughing."
- Urge urinary incontinence (UUI): "the complaint of involuntary leakage accompanied by or immediately proceeded by urgency."
- Mixed urinary incontinence (MUI): "the complaint of involuntary leakage associated with urgency and also with exertion, effort, sneezing, or coughing."

6.2.2.1 Prevalence

A systematic literature review by Hunskaar et al. [18], including 36 epidemiological studies from 17 countries, showed a prevalence of any UI within the range 5–69% among the general female population. However, most of these studies showed a UI prevalence within the range 25–45% [18]. A wide range in UI prevalence might be explained by differences in the population studied, definition of UI, type of UI, and assessment of UI [19–21]. According to an updated review on UI prevalence by Milsom et al. [22], recent epidemiological studies report estimates on UI prevalence that are within the prevalence range reported by Hunskaar et al. [18]. The most common type of UI reported by young and middle-aged women is SUI, while older women are more likely to report MUI and UUI [18, 22].

6.2.2.2 Prevalence During Pregnancy

Studies of prevalence of UI during pregnancy have shown period prevalence within the range 32–64% for any type of UI and 40–59% for the combination SUI/MUI [18, 22]. Higher period prevalence has been reported in parous than in nulliparous women [23–27]. The largest prospective population-based study included in the review by Milsom et al. [22] is the study published by Wesnes et al. [27]. This study was part of the Norwegian Mother and Child Cohort Study. Results showed that

prevalence of UI increased from 15% before pregnancy to 48% at gestational week 30 for nulliparous women and from 35% before pregnancy to 67% at gestational week 30 for parous women. SUI was the most common type of UI with figures showing an increase from 9% before pregnancy to 31% at gestational week 30 for nulliparous women and from 24% to 42% for parous women.

6.2.2.3 Prevalence After Childbirth

The estimation of postpartum UI is, according to Milsom et al. [22], challenged by study heterogeneity (study design and method, definition of UI, and sample studied). In their review they therefore chose to summarize data from 22 studies on primiparous women enrolled at larger hospitals serving a defined population. The range of UI prevalence (any type) in primiparous women during the first year postpartum, regardless of delivery mode, was 15–30%. According to Milsom et al. [22], the included studies showed consistently higher UI prevalence in women who delivered vaginally than in women who delivered by caesarean section, with the exception of one study [28].

6.2.3 Etiology and Pathophysiology of Urinary Incontinence

UI also occurs in women who never have gone through pregnancy and childbirth. However, pregnancy and childbirth are considered main etiological factors for the development of UI [29]. Connective tissue, peripheral nerves, and muscular structures are already during pregnancy subjected to hormonal, anatomical, and morphological changes. During vaginal delivery, the abovementioned structures are forcibly stretched and compressed. This may initiate changed tissue properties, which may contribute to altered pelvic floor function and increased risk of UI [29]. The picture of possible causative factors and the pathophysiology of UI are complex, some factors are studied more than others, and the importance of factors associated by the pregnancy itself versus factors associated childbirth is still under discussion [29].

6.2.3.1 Pregnancy

Prospective observational studies have shown increased prevalence of UI from the first trimester to the second and further into the third trimester [26, 30, 31]. One hypothesis of increased prevalence during pregnancy is linked to increased bladder pressure due to the growing uterus and weight of the fetus, and another is linked to hormonal changes altering the viscoelastic properties [26]. In the observational study by Hvidman et al. [26], the authors suggest that UI may not be provoked by the onset of pregnancy but by its progressive concentration of pregnancy hormones which may lead to local tissue changes. They found no association between UI and the birth weight of the child and state less support for the theory linking UI to increased pressure on the bladder caused by weight of the fetus. Studies have shown an association between UI and maternal obesity both during pregnancy [27] and after childbirth [32–34], which could be caused by increased intra-abdominal pressure and increased bladder pressure [35].

The PFM is considered to play a significant role in the continence control system [2–4, 9], and urine loss may be linked to impaired PFM function, e.g., weak PFM. Several observational studies have demonstrated significantly higher PFM strength in continent women than in women having UI [36–43], while some studies did not find such difference [44, 45]. Two of the abovementioned studies were on pregnant women [36, 40, 43]. In addition to significantly higher PFM strength, Mørkved et al. [40] also report a significantly thicker PFM among the continent pregnant women.

UI during pregnancy is transient in some women but may become long-lasting in others. Prospective observational studies have shown that antenatal UI may increase the risk of postpartum UI [34, 46–48].

6.2.3.2 Childbirth

Parity seems to be an increased risk factor for UI [15, 24, 31, 49–51]. In a cross-sectional study of 27,900 women, Rortveit et al. [50] report a relative risk (RR) of UI of 2.2 (95% CI, 1.8–2.6) for primiparous women and 3.3 (95% CI, 2.4–4.4) for grand multiparous women. Altman et al. [48] included 304 primiparous women and followed them 10 years prospectively. They found vaginal delivery to be independently associated with a significant long-term increase in SUI and UUI, regardless of maternal age and number of deliveries. This is supported by Viktrup et al. [34] following 241 primiparous women 12 years after their first delivery.

The protective effects of caesarean section have been and still are much debated. In a systematic review by Press et al. [52], the prevalence of postpartum SUI after caesarean section was compared with vaginal delivery. Based on data from six cross-sectional studies, caesarean section reduced the risk of postpartum SUI from 16% to 10% (OR 0.56; 95% CI, 0.45–0.68), while data from 12 cohort studies gave a reduction from 22% to 10% (OR 0.48; 95% CI, 0.39–0.58). However, risk of severe SUI and UUI did not differ by mode of birth.

6.2.3.3 Bladder Neck and Urethral Hypermobility

Impaired structural support of the urethra may cause increased bladder neck mobility and reduced compression of the urethra which again may lead to UI [3]. Peschers et al. [53] investigated change in bladder neck mobility, during the Valsalva maneuver, from late pregnancy to 6–10 weeks postpartum. They found increased mobility in women who delivered vaginally ($p < 0.001$) but found no such change in women with elective caesarean section ($p = 0.28$). Their findings are supported by Meyer et al. [54] and Dietz et al. [55].

Meyer et al. [56] found significantly higher bladder mobility, during the Valsalva maneuver, in women with SUI (mean parity 2.4, SD 0.8) when compared to nulliparous continent women. However, the association between increased bladder neck mobility and SUI may not solely be explained by vaginal childbirth. King and Freeman [57] followed nulliparous pregnant women with no preexisting UI from gestational week 15–17 to 10–14 weeks postpartum. They found that primiparous women with SUI postpartum had significantly greater antenatal bladder neck mobility than continent counterparts, which could be explained by a

predisposed weak connective tissue, aggravated by pregnancy hormones and collagen remodeling [57, 58].

A study on bladder neck mobility and tissue stiffness was performed by Howard et al. [59]. Results from their study showed that primiparous women with SUI displayed similar bladder neck mobility during a cough and during a Valsalva maneuver ($p = 0.49$), while significantly less mobility was displayed during a cough than during the Valsalva both for continent nulliparous women ($p = 0.001$) and for continent primiparous women (0.002). When controlling for abdominal pressures, their calculations showed that nulliparous women displayed a significantly greater pelvic floor stiffness during a cough than the continent and incontinent primiparous women ($p = 0.001$).

6.2.3.4 Neural Denervation

Neuromuscular impairment is associated with the development of incontinence. Smith et al. [60] found that terminal branches of the pudendal nerve had a delayed conduction both to the striated urethral muscle and to the PFM in women with SUI when compared to continent women. Such denervation seems to be related to parity and vaginal childbirth [61–64]. In a biomechanical study by Lien et al. [65], lengthening of pudendal nerve branches was simulated by using a 3D computer model. The results from this study showed that the inferior rectal branch of the pudendal nerve may exhibit a strain of 35%. Pudendal nerve neuropathy appears to be associated with both a long second stage and high birth weight [63, 66, 67]. Such neural impairment may alter the muscle morphology. In a study by Gilpin et al. [68], biopsy samples from women with SUI showed a significant higher number of muscle fibers with pathological damage when compared to biopsy samples from continent women.

6.2.3.5 Weakening of the Pelvic Floor Muscles

Vaginal delivery is considered as a main risk factor for weakening of the PFM [63, 66, 69–74]. Due to the extensive stretching of muscle fibers and the likelihood of muscle denervation, it is not surprising that vaginal delivery may lead to reduced vaginal resting pressure and impaired PFM strength and endurance and that caesarean section may protect the PFM. A PubMed search gave seven studies [36, 54, 75–79] investigating change in PFM strength from pregnancy to shortly after childbirth in relation to mode of delivery. Except from one study [77], the other six studies showed a significant reduction in PFM strength after vaginal delivery, but no significant decline after caesarean section.

6.2.4 Levator Ani Muscle Defects

Vaginal delivery may stretch and load beyond the physiological properties of the PFM, which may lead to muscle fiber tearing and reduced contractile force. The biomechanical study by Lien et al. [80] showed that muscle fibers, of the most medial part of the LA muscle, might be stretched up to three times their resting

length as the fetal head is crowning. Their findings showing a pronounced stretch and deformation of the medial part of the LA muscle are confirmed by Hoyte et al. [81] and Parente et al. [82].

During recent years, technical advancement within magnetic resonance and ultrasound imaging has enabled diagnosis of defects of the LA muscle [83]. Major defects of the LA muscle are often defined as an abnormal insertion of this muscle toward the pubic bone, visually seen as a complete loss of visible muscle attachment at this specific site either unilaterally or bilaterally [69, 72, 83]. Imaging studies have shown that major LA muscle defects among primiparous women delivering vaginally could appear in 20–36% of the women [69, 84]. The use of forceps [70] and length of the second stage [70, 85] are associated with major LA muscle defects, whereas the importance of fetal head circumference and high fetal birth weight seems to be less clear [84–86].

Decreased strength is one of the most common symptoms following muscle tears within sport injuries [87]. Hence, decreased PFM strength in women with major LA muscle defects is expected but has been sparsely investigated. A PubMed search revealed five observational studies [88–92] in which PFM strength in women with and without LA muscle defects was assessed. Results from all five studies showed significantly reduced PFM strength in women with LA muscle defects when compared to women without such defects. Dynamometer was used for assessment of PFM strength in two of these five studies [88, 91], digital palpation in one study [89], transperineal ultrasound in one [90], and manometer in one [92]. These studies did also differ in age and parity of the women included.

Major LA muscle defects have shown a marked effect on hiatal dimensions [93–95] and pelvic organ support [95] which in turn could be explanatory factors for pelvic floor dysfunction. Major defect of the LA muscle has been linked to pelvic organ prolapse in particular [83, 88, 96, 97], while the link between LA muscle defects and UI is debated. Two studies [69, 84] report a significant association between LA muscle defects and SUI in the postpartum period. However, contradictory findings are reported for the link between LA muscle defects and SUI in studies on women with mixed parity and mean age >50 years [86, 98, 99].

6.2.5 Muscle Injury Regeneration

The healing process of a torn muscle has three phases: (1) the destruction phase, (2) the repair phase, and (3) the remodeling phase [87, 100, 101]. In the destruction phase, the rupture is followed by necrosis and formation of a hematoma. In the repair phase, a phagocytosis of necrotized tissue takes place, followed by proliferation of skeletal muscle satellite cells which induce regeneration of myofibrils. Along with this is formation of scar tissue and revascularization of the injured area initiated. During the remodeling phase, a further maturation of the regenerated myofibrils is implemented together with remodeling of the scar tissue, followed by recovery to functional capacity [87, 100, 101].

6.2.5.1 Treatment Principles for Skeletal Muscle Injuries

Recommendations for treatment of muscle injuries and how to recover functional capacity are most often based on theoretical framework from epidemiological studies, clinical practice, and findings from experimental research [102]. Early mobilization is a standard treatment after muscle injury within sports medicine, and training is believed to be important in speeding up tissue healing (repair and remodeling). This approach is supported by experimental studies showing that early mobilization after a muscle injury may facilitate the following: more rapid capillary ingrowths, improved parallel orientation of the regenerating myofibrils, and improved tensile properties [87, 100, 101, 103].

6.3 Diastasis Recti Abdominis

Pregnancy and childbirth bring along several changes to a woman's body, especially to the musculoskeletal system [104]. The most obvious change is related to the growth of the fetus and the stretching of the abdominal muscles, which may influence the mother's posture and balance [104]. Today there is a strong focus on the pregnant woman's appearance, especially through social media. Webpages and apps recommend how women should stay thin and get back into shape and "a flat tummy" at an early stage of the postpartum period. Using the search terms "diastasis recti" and "exercise," 278,000 hits were obtained on Google. In addition, there are easily available advices on how to get rid of what is named "the mum's belly" (e.g., www.mammamage.se, www.breakingmuscle.com, www.befitmom.com, www.babybellybelt.com, tummyzip.com). A systematic review of the scientific literature has found none or very weak evidence behind any of these advices [105].

A strong focus on an area of the body that is naturally changed during pregnancy and after childbirth and that may recover by itself over time maybe a risk factor for development of an unhealthy attitude toward exercise, appearance, body shape, and image, and new mothers may become dissatisfied with their bodies and especially their abdomen. On the other hand, pregnancy and childbirth *are* risk periods for the development of obesity and musculoskeletal complaints such as low back and pelvic girdle pain [106] and pelvic floor dysfunctions including urinary and anal incontinence and pelvic organ prolapse [107]. A possible link between these conditions and injuries and weakness of the abdominal muscles has been postulated [108].

Diastasis recti abdominis (DRA) is defined as an impairment with midline separation of the two rectus abdominis muscles along the linea alba. The condition affects a significant number of women during the antenatal and postnatal period [109]. Prevalence rates (with and without protrusion/hernia) during pregnancy vary between 27% and 100% in the second and third trimesters [110, 111]. Postpartum, the prevalence rates of DRA vary between 30% and 68% [112, 113]. In a longitudinal study of 300 first-time pregnant women at Akershus University Hospital in Norway, Sperstad et al. [114] found that prevalence rates changed from 33% at gestational week 21 to 60%, 45.4%, and 32.6% at 6 weeks, 6 months, and 12 months

postpartum, respectively. However, DRA has been found to be common in middle-aged women [115] and may also be present in men [116]. Whether strong abdominal muscles can prevent or are a risk factor for development of the condition is not known. To date there are no prevalence studies or assessments of this condition among recreational exercisers and elite athletes [104].

DRA is diagnosed by measuring the distance between the median borders of the two rectus abdominis, inter-rectus distance (IRD), and measurement methods in use are palpation with fingerbreadths, caliper, or ultrasound [117]. Palpation is the most commonly used method in clinical practice [118] and has an intra- and inter-tester ICC of 0.7 and 0.5, respectively [119]. However, ultrasound has been found to have the best intra- and inter-tester reliability with ICC > 0.9 [120]. To date there is no consensus on where to measure IRD along the linea alba (frequently used locations are 4.5 cm above the umbilicus, at the umbilicus, and 4.5 cm below the umbilicus) or the cutoff point for diagnosing the condition [117]. A commonly used cutoff point is two fingerbreadths on palpation [117]. Candido et al. [121] have classified severity of the diastasis as mild (2.5–3.5 cm or visible protrusion with diastasis less than 2.5 cm), moderate (3.5–5 cm), and substantial (>5 cm).

The etiology and risk factors for DRA are not clear [122]. Fernandes da Mota et al. [123] found that neither age, BMI, weight gain during pregnancy, hypermobility, birth weight, abdominal circumference at gestational week 35, nor exercise level before and during pregnancy was a risk factor for diastasis 6 months postpartum. This was in agreement with results of Sperstad et al. [114] comparing women with and without diastasis 12 months postpartum. Spitznagle et al. [115] found higher prevalence of DRA in older multiparous women, while Candido et al. [121] did not find any relationship with parity. None of the abovementioned studies found any relationship with mode of delivery (vaginal versus caesarean section) and diastasis.

6.3.1 Consequences of Diastasis Recti Abdominis

It has been postulated that DRA, in addition to being a cosmetic concern for many women, may reduce low back and pelvic stability, cause low back and pelvic girdle pain and be related to pelvic floor dysfunctions such as urinary incontinence, anal incontinence, and pelvic organ prolapse [115, 124]. However, to date there is scant scientific knowledge on this topic. An association between DRA and abdominal muscle strength has not yet been substantiated with strong evidence. In a longitudinal small study following six women from gestational week 14 to 8 weeks postpartum, Gilleard and Brown [125] found that women with IRD >3.5 cm measured with palpation had reduced curl-up "capacity." This was supported by a study following 40 women postpartum, which found that postpartum women had weaker abdominal muscles than a control group [126]. However, at 6 months postpartum, there was no correlation between IRD and reduced abdominal muscle strength.

No strong link between DRA and low back pain has been found. Parker et al. [127] found that women at least 3 months postpartum with DRA had more

abdominal and pelvic pain than women without. However, two other studies did not find any difference in prevalence of low back or pelvic girdle pain in primiparous women 6–12 months postpartum between women with and without DRA [111, 114]. Most of these studies included women with light and moderate diastasis. Hence, it is important to investigate the association between severe diastasis and low back and pelvic girdle pain.

6.4 Pregnancy-Related Low Back Pain and Pelvic Girdle Pain

Pregnancy-related low back pain (LBP) and/or pelvic girdle pain (PGP) is common across many countries, irrespective of socioeconomic factors [128–131], and is reported to have a negative effect on daily activities such as walking, lifting, climbing stairs, lying flat on the back, turning in bed, housework, exercise, employment, leisure, sexual life, hobbies, and personal relationships [132]. Women with LBP and PGP report a significantly lower health-related quality of life than that reported by healthy women, and a major factor affecting their quality of life is found to be lack of physical ability [133, 134]. PGP during pregnancy greatly affects a woman's experience of her pregnancy, her roles in relationships, and her social context [135, 136]. These women are struggling with enduring pain that disturbs most aspects of their lives [135, 137], and the pain is perceived as an unpredictable and potentially disabling condition [138]. Whereas most women recover after delivery, a number of women continue living with disabling PGP. Postpartum PGP may influence women's lives for months and years after delivery. Discouragement, isolation and loneliness may be part of a daily life with pain and limited physical activity [139].

The prevalence rates of pregnancy-related LBP and PGP vary depending on the criteria used and mode of reporting, but are estimated to be about 50% during pregnancy [140]. Whereas LBP is usually defined as pain between the 12th rib and the gluteal fold, PGP is defined as pain experienced between the posterior iliac crest and the gluteal fold, particularly in the vicinity of the sacroiliac joints [106]. PGP generally arises in relation to pregnancy and is defined as pain in the pelvic musculoskeletal system that does not derive from gynecological and urological disorders. A diagnosis of PGP can be reached after the exclusion of lumbar causes, and the pain or functional impairments in relation to PGP must be reproducible by specific clinical tests [106]. Although similar and overlapping features may be ascribed to LBP and to PGP, it is argued that a distinction should be made [106, 140]. PGP has more impact on pain intensity and disability than LBP [141, 142]. Whereas the normal progression of LBP during pregnancy peaks between 12 and 30 weeks [143, 144], PGP increases progressively with advancing pregnancy [145]. Most probably about 20–25% of all pregnant women who suffer from PGP sufficiently seriously require medical help [106, 146]. Though the majority of women with PGP recover spontaneously soon after delivery, 3–7% report having serious problems from persistent PGP years after delivery [140, 147]. In the only study among elite athletes, 12.6% reported retrospectively that they experienced PGP 6 weeks postpartum and 9.7%

experienced LBP. The prevalence increased to 19.4% for PGP and 29% for LBP at the time of completing the questionnaire 0–17 years after delivery [148].

The etiology and pathogenesis of PGP are unclear and probably multifactorial. Possible underlying causes include hormonal and biomechanical aspects, inadequate motor control, and stress on ligament structures [149]. PGP often occurs during the early stages of pregnancy [151], and the symptoms typically regress shortly after delivery [147]. A possible association between serum relaxin levels and PGP is debated [147, 150, 151]. The exact movements that occur in the pelvic joints have been traced [152, 153]. Recently, it was shown that the movement in the sacroiliac joints is small and almost undetectable by precise radiostereometric analysis [154]. Even though small, any increased motion in the pelvic joints may diminish the efficiency of load transfer and increase the shear forces across the joints. Increased shear forces has been suggested to be one factor for pain in women with PGP [106]. The self-locking mechanism of the sacroiliac joints with the principles of form and force closure, based on a theoretical model from anatomical and biomechanical studies, was introduced in 1997 by Snijders and co-workers [155]. Failure of the self-locking mechanism and load transfer through the pelvis has been suspected in patients with sacroiliac pain [156, 157], and asymmetric laxity of the sacroiliac joints has been shown to correlate with moderate to severe levels of symptoms in subjects with postpartum PGP [158].

The sacrospinous ligament and superficial sacroiliac joint structures, such as the long dorsal sacroiliac ligament, are a potential source of pain in PGP [159–162]. An impaired load transfer during activities may result in overload of the ligaments of the pelvis and hence have an influence on PGP [163, 164]. Frequent or sustained pain-provoking postures might influence the pelvic ligaments and in turn link to other symptoms. Changes in spinal curvature and posture may be caused by pregnancy. Both increased lumbar lordosis [165] and a tendency for lumbar kyphosis or a flattening of the lumbar spine is reported to be prevalent during pregnancy [166, 167].

There is some evidence that PGP is related to an altered pelvic mechanism and/or motor control [168]. PGP disorders have been associated with an alteration in the strategy for lumbopelvic stabilization with excessive as well as insufficient motor activation of the lumbopelvic and surrounding musculature [169]. Impaired motor control patterns may be a possible mechanism for ongoing pain and disability in patients with persistent PGP [170]. Attention has been paid to motor control of local muscles, especially the transverse abdominals [170, 171]. Also the pelvic floor muscles are considered to be an important part of the local muscle system, and Stuge and co-workers found significantly smaller levator hiatus area in women with PGP than in controls both at rest, during voluntary contraction, and during automatic contraction [172, 173].

Whereas the role of muscle function in LBP in the general population is debated, an association between reduced muscle function and the development of LBP and/or PGP in pregnant women is reported [174]. Indications exist that pregnant women with gluteus medius weakness are more likely to have LBP than those without this weakness [175]. In pregnant women with LBP and/or PGP, both lower levels of trunk muscle endurance and hip extension muscle strength [176] and increased

muscle activity during the active straight leg raise test are reported [177]. Consequently, an association between muscle dysfunction and LBP and/or PGP during and after pregnancy may exist.

References

1. Healey CH, Borley NR, Mundy A. True pelvis, pelvic floor and perineum. In: Standring S, Ellis H, Healy JC, Johnson D, Williams A, editors. Gray's anatomy: the anatomical basis of clinical practice. Edinburgh: Elsevier; 2005. p. 1357–71.
2. Delancey JO. Structural aspects of the extrinsic continence mechanism. Obstet Gynecol. 1988;72(3 Pt 1):296–301.
3. Delancey JO. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol. 1994;170(6):1713–20.
4. Ashton-Miller JA, Delancey JO. Functional anatomy of the female pelvic floor. Ann N Y Acad Sci. 2007;1101:266–96.
5. Norton PA. Pelvic floor disorders: the role of fascia and ligaments. Clin Obstet Gynecol. 1993;36(4):926–38.
6. Kegel AH. Stress incontinence and genital relaxation; a nonsurgical method of increasing the tone of sphincters and their supporting structures. Ciba Clin Symp. 1952;4(2):35–51.
7. Bø K, Lilleas F, Talseth T, Hedland H. Dynamic MRI of the pelvic floor muscles in an upright sitting position. Neurourol Urodyn. 2001;20(2):167–74.
8. Taverner D, Smiddy FG. An electromyographic study of the normal function of the external anal sphincter and pelvic diaphragm. Dis Colon Rectum. 1959;2(2):153–60.
9. Ashton-Miller JA, Howard D, Delancey JO. The functional anatomy of the female pelvic floor and stress continence control system. Scand J Urol Nephrol Suppl. 2001;207:1–7.
10. Bø K, Aschehoug A. Pelvic floor and exercise science: strength training. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-based physical therapy for the pelvic floor. Edinburgh: Elsevier; 2007. p. 119–32.
11. Miller JM, Perucchini D, Carchidi LT, De Lancey JO, Ashton-Miller J. Pelvic floor muscle contraction during a cough and decreased vesical neck mobility. Obstet Gynecol. 2001;97(2):255–60.
12. Miller JM, Ashton-Miller JA, DeLancey JO. A pelvic muscle precontraction can reduce cough-related urine loss in selected women with mild SUI. J Am Geriatr Soc. 1998;46(7):870–4.
13. Miller JM, Sampselle C, Ashton-Miller J, Hong GR, Delancey JO. Clarification and confirmation of the Knack maneuver: the effect of volitional pelvic floor muscle contraction to preempt expected stress incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(6):773–82.
14. Bump RC, Norton PA. Epidemiology and natural history of pelvic floor dysfunction. Obstet Gynecol Clin N Am. 1998;25(4):723–46.
15. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. BJOG. 2000;107(12):1460–70.
16. DeLancey JO, Kane LL, Miller JM, Patel DA, Tumbarello JA. Graphic integration of causal factors of pelvic floor disorders: an integrated life span model. Am J Obstet Gynecol. 2008;199(6):610–5.
17. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, et al. The standardisation of terminology in lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. Urology. 2003;61(1):37–49.
18. Hunskaar S, Burgio K, Clark AL, Lapitan MC, Nelson R, Sillén U, et al. Epidemiology of urinary incontinence (UI) and faecal incontinence (FI) and pelvic organ prolapse (POP). In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence (3rd international consultation on incontinence 26–29 Jun 2004). 3rd ed. Plymouth: Health Publication Ltd; 2005. p. 255–312.

19. Thom D. Variation in estimates of urinary incontinence prevalence in the community: effects of differences in definition, population characteristics, and study type. J Am Geriatr Soc. 1998;46(4):473–80.
20. Botlero R, Urquhart DM, Davis SR, Bell RJ. Prevalence and incidence of urinary incontinence in women: review of the literature and investigation of methodological issues. Int J Urol. 2008;15(3):230–4.
21. Minassian VA, Stewart WF, Wood GC. Urinary incontinence in women: variation in prevalence estimates and risk factors. Obstet Gynecol. 2008;111(2 Pt 1):324–31.
22. Milsom I, Altman D, Lapitan MC, Nelson R, Sillén U, Thom D. Epidemiology of urinary (UI) and faecal (FI) incontinence and pelvic organ prolapse (POP). In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence: 4th international consultation on incontinence. 4th ed. Paris: Health Publication Ltd; 2009. p. 35–111.
23. Dimpfl T, Hesse U, Schussler B. Incidence and cause of postpartum urinary stress incontinence. Eur J Obstet Gynecol Reprod Biol. 1992;43(1):29–33.
24. Marshall K, Thompson KA, Walsh DM, Baxter GD. Incidence of urinary incontinence and constipation during pregnancy and postpartum: survey of current findings at the Rotunda Lying-In Hospital. Br J Obstet Gynaecol. 1998;105(4):400–2.
25. Mørkved S, Bo K. Prevalence of urinary incontinence during pregnancy and postpartum. Int Urogynecol J Pelvic Floor Dysfunct. 1999;10(6):394–8.
26. Hvidman L, Hvidman L, Foldspang A, Mommsen S, Bugge NJ. Correlates of urinary incontinence in pregnancy. Int Urogynecol J Pelvic Floor Dysfunct. 2002;13(5):278–83.
27. Wesnes SL, Rortveit G, Bo K, Hunskaar S. Urinary incontinence during pregnancy. Obstet Gynecol. 2007;109(4):922–8.
28. Borello-France D, Burgio KL, Richter HE, Zyczynski H, Fitzgerald MP, Whitehead W, et al. Fecal and urinary incontinence in primiparous women. Obstet Gynecol. 2006;108(4):863–72.
29. Koelbl H, Nitti V, Baessler K, Salvatore S, Sultan A, Yamaguchi O. Pathophysiology of urinary incontinence, faecal incontinence and pelvic organ prolapse. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence: 4th international consultation on incontinence. 4th ed. Paris: Helarh Publicationa Ltd; 2009. p. 255–330.
30. Thorp JM Jr, Norton PA, Wall LL, Kuller JA, Eucker B, Wells E. Urinary incontinence in pregnancy and the puerperium: a prospective study. Am J Obstet Gynecol. 1999;181(2):266–73.
31. Burgio KL, Locher JL, Zyczynski H, Hardin JM, Singh K. Urinary incontinence during pregnancy in a racially mixed sample: characteristics and predisposing factors. Int Urogynecol J Pelvic Floor Dysfunct. 1996;7(2):69–73.
32. Wilson PD, Herbison RM, Herbison GP. Obstetric practice and the prevalence of urinary incontinence three months after delivery. Br J Obstet Gynaecol. 1996;103(2):154–61.
33. Rasmussen KL, Krue S, Johansson LE, Knudsen HJ, Agger AO. Obesity as a predictor of postpartum urinary symptoms. Acta Obstet Gynecol Scand. 1997;76(4):359–62.
34. Viktrup L, Rortveit G, Lose G. Risk of stress urinary incontinence twelve years after the first pregnancy and delivery. Obstet Gynecol. 2006;108(2):248–54.
35. Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med. 1997;241(1):71–9.
36. Sampselle CM. Changes in pelvic muscle strength and stress urinary incontinence associated with childbirth. J Obstet Gynecol Neonatal Nurs. 1990;19(5):371–7.
37. Hahn I, Milsom I, Ohlsson BL, Ekelund P, Uhlemann C, Fall M. Comparative assessment of pelvic floor function using vaginal cones, vaginal digital palpation and vaginal pressure measurements. Gynecol Obstet Investig. 1996;41(4):269–74.
38. Samuelsson E, Victor A, Svardsudd K. Determinants of urinary incontinence in a population of young and middle-aged women. Acta Obstet Gynecol Scand. 2000;79(3):208–15.
39. Morin M, Bourbonnais D, Gravel D, Dumoulin C, Lemieux MC. Pelvic floor muscle function in continent and stress urinary incontinent women using dynamometric measurements. Neurourol Urodyn. 2004;23(7):668–74.
40. Mørkved S, Salvesen KA, Bo K, Eik-Nes S. Pelvic floor muscle strength and thickness in continent and incontinent nulliparous pregnant women. Int Urogynecol J Pelvic Floor Dysfunct. 2004;15(6):384–9.

41. Thompson JA, O'Sullivan PB, Briffa NK, Neumann P. Assessment of voluntary pelvic floor muscle contraction in continent and incontinent women using transperineal ultrasound, manual muscle testing and vaginal squeeze pressure measurements. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17(6):624–30.
42. Shishido K, Peng Q, Jones R, Omata S, Constantinou CE. Influence of pelvic floor muscle contraction on the profile of vaginal closure pressure in continent and stress urinary incontinent women. J Urol. 2008;179(5):1917–22.
43. Hilde G, Staer-Jensen J, Ellstrom EM, Braekken IH, Bo K. Continence and pelvic floor status in nulliparous women at midterm pregnancy. Int Urogynecol J. 2012;23(9):1257–63.
44. Bø K, Stien R, Kulseng-Hanssen S, Kristofferson M. Clinical and urodynamic assessment of nulliparous young women with and without stress incontinence symptoms: a case-control study. Obstet Gynecol. 1994;84(6):1028–32.
45. Sartore A, Pregazzi R, Bortoli P, Grimaldi E, Ricci G, Guaschino S. Assessment of pelvic floor muscle function after vaginal delivery. Clinical value of different tests. J Reprod Med. 2003;48(3):171–4.
46. Hvidman L, Foldspang A, Mommsen S, Nielsen JB. Postpartum urinary incontinence. Acta Obstet Gynecol Scand. 2003;82(6):556–63.
47. Foldspang A, Hvidman L, Mommsen S, Nielsen JB. Risk of postpartum urinary incontinence associated with pregnancy and mode of delivery. Acta Obstet Gynecol Scand. 2004;83(10):923–7.
48. Altman D, Ekstrom A, Gustafsson C, Lopez A, Falconer C, Zetterstrom J. Risk of urinary incontinence after childbirth: a 10-year prospective cohort study. Obstet Gynecol. 2006;108(4):873–8.
49. Groutz A, Gordon D, Keidar R, Lessing JB, Wolman I, David MP, et al. Stress urinary incontinence: prevalence among nulliparous compared with primiparous and grand multiparous premenopausal women. Neurourol Urodyn. 1999;18(5):419–25.
50. Rortveit G, Hannestad YS, Daltveit AK, Hunskaar S. Age- and type-dependent effects of parity on urinary incontinence: the Norwegian EPINCONT study. Obstet Gynecol. 2001;98(6):1004–10.
51. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. Obstet Gynecol. 2006;107(6):1253–60.
52. Press JZ, Klein MC, Kaczorowski J, Liston RM, von DP. Does cesarean section reduce postpartum urinary incontinence? A systematic review. Birth. 2007;34(3):228–37.
53. Peschers U, Schaer G, Anthuber C, Delancey JO, Schuessler B. Changes in vesical neck mobility following vaginal delivery. Obstet Gynecol. 1996;88(6):1001–6.
54. Meyer S, Schreyer A, De GP, Hohlfeld P. The effects of birth on urinary continence mechanisms and other pelvic-floor characteristics. Obstet Gynecol. 1998;92(4 Pt 1):613–8.
55. Dietz HP, Clarke B, Vancaillie TG. Vaginal childbirth and bladder neck mobility. Aust NZ J Obstet Gynaecol. 2002;42(5):522–5.
56. Meyer S, De GP, Schreyer A, Caccia G. The assessment of bladder neck position and mobility in continent nullipara, mulitpara, forceps-delivered and incontinent women using perineal ultrasound: a future office procedure? Int Urogynecol J Pelvic Floor Dysfunct. 1996;7(3):138–46.
57. King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? Br J Obstet Gynaecol. 1998;105(12):1300–7.
58. Lavin JM, Smith ARB, Anderson J, Grant M, Buckley H, Critchley H, et al. The effect of the first pregnancy on the connective tissue of the rectus sheath. Neurourol Urodynam. 1997;16:381–2.
59. Howard D, Miller JM, DeLancey JO, Ashton-Miller JA. Differential effects of cough, valsalva, and continence status on vesical neck movement. Obstet Gynecol. 2000;95(4):535–40.
60. Smith AR, Hosker GL, Warrell DW. The role of pudendal nerve damage in the aetiology of genuine stress incontinence in women. Br J Obstet Gynaecol. 1989;96(1):29–32.
61. Snooks SJ, Setchell M, Swash M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet. 1984;2(8402):546–50.

62. Smith AR, Hosker GL, Warrell DW. The role of partial denervation of the pelvic floor in the aetiology of genitourinary prolapse and stress incontinence of urine. A neurophysiological study. Br J Obstet Gynaecol. 1989;96(1):24–8.
63. Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol. 1990;97(9):770–9.
64. Snooks SJ, Swash M, Mathers SE, Henry MM. Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. Br J Surg. 1990;77(12):1358–60.
65. Lien KC, Morgan DM, Delancey JO, Ashton-Miller JA. Pudendal nerve stretch during vaginal birth: a 3D computer simulation. Am J Obstet Gynecol. 2005;192(5):1669–76.
66. Snooks SJ, Swash M, Henry MM, Setchell M. Risk factors in childbirth causing damage to the pelvic floor innervation. Int J Color Dis. 1986;1(1):20–4.
67. Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labour: prospective study before and after childbirth. Br J Obstet Gynaecol. 1994;101(1):22–8.
68. Gilpin SA, Gosling JA, Smith AR, Warrell DW. The pathogenesis of genitourinary prolapse and stress incontinence of urine. A histological and histochemical study. Br J Obstet Gynaecol. 1989;96(1):15–23.
69. Delancey JO, Kearney R, Chou Q, Speights S, Binno S. The appearance of levator ani muscle abnormalities in magnetic resonance images after vaginal delivery. Obstet Gynecol. 2003;101(1):46–53.
70. Kearney R, Miller JM, Ashton-Miller JA, Delancey JO. Obstetric factors associated with levator ani muscle injury after vaginal birth. Obstet Gynecol. 2006;107(1):144–9.
71. Dietz HP. Pelvic floor trauma following vaginal delivery. Curr Opin Obstet Gynecol. 2006;18(5):528–37.
72. DeLancey JO, Ashton-Miller JA. Measurement of pelvic floor muscle function and strength and pelvic organ prolapse: MRI of intact and injured female pelvic floor muscles. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-based physical therapy for the pelvic floor. Edinburgh: Churchill Livingstone Elsevier; 2007. p. 93–105.
73. Ashton-Miller JA, Delancey JO. On the biomechanics of vaginal birth and common sequelae. Annu Rev Biomed Eng. 2009;11:163–76.
74. Turner CE, Young JM, Solomon MJ, Ludlow J, Benness C. Incidence and etiology of pelvic floor dysfunction and mode of delivery: an overview. Dis Colon Rectum. 2009;52(6):1186–95.
75. Peschers UM, Schaer GN, Delancey JO, Schuessler B. Levator ani function before and after childbirth. Br J Obstet Gynaecol. 1997;104(9):1004–8.
76. Botelho S, Riccetto C, Herrmann V, Pereira LC, Amorim C, Palma P. Impact of delivery mode on electromyographic activity of pelvic floor: comparative prospective study. Neurourol Urodyn. 2010;29(7):1258–61.
77. Caroci AS, Riesco ML, Sousa WS, Cotrim AC, Sena EM, Rocha NL, et al. Analysis of pelvic floor musculature function during pregnancy and postpartum: a cohort study: (a prospective cohort study to assess the PFMS by perineometry and digital vaginal palpation during pregnancy and following vaginal or caesarean childbirth). J Clin Nurs. 2010;19(17–18):2424–33.
78. Sigurdardottir T, Steingrimsdottir T, Arnason A, Bo K. Pelvic floor muscle function before and after first childbirth. Int Urogynecol J. 2011;22(12):1497–503.
79. Hilde G, Staer-Jensen J, Siafarikas F, Engh ME, Braekken IH, Bo K. Impact of childbirth and mode of delivery on vaginal resting pressure and on pelvic floor muscle strength and endurance. Am J Obstet Gynecol. 2013;208(1):50–7.
80. Lien KC, Mooney B, Delancey JO, Ashton-Miller JA. Levator ani muscle stretch induced by simulated vaginal birth. Obstet Gynecol. 2004;103(1):31–40.
81. Hoyte L, Damaser MS, Warfield SK, Chukkapalli G, Majumdar A, Choi DJ, et al. Quantity and distribution of levator ani stretch during simulated vaginal childbirth. Am J Obstet Gynecol. 2008;199(2):198–5.
82. Parente MP, Jorge RM, Mascarenhas T, Fernandes AA, Martins JA. Deformation of the pelvic floor muscles during a vaginal delivery. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(1):65–71.

83. Dietz HP. Quantification of major morphological abnormalities of the levator ani. Ultrasound Obstet Gynecol. 2007;29(3):329–34.
84. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. Obstet Gynecol. 2005;106(4):707–12.
85. Valsky DV, Lipschuetz M, Bord A, Eldar I, Messing B, Hochner-Celnikier D, et al. Fetal head circumference and length of second stage of labor are risk factors for levator ani muscle injury, diagnosed by 3-dimensional transperineal ultrasound in primiparous women. Am J Obstet Gynecol. 2009;201(1):91–7.
86. Krofta L, Otcenasek M, Kasikova E, Feyereisl J. Pubococcygeus-puborectalis trauma after forceps delivery: evaluation of the levator ani muscle with 3D/4D ultrasound. Int Urogynecol J Pelvic Floor Dysfunct. 2009;20(10):1175–81.
87. Jarvinen TA, Jarvinen TL, Kaariainen M, Aarimaa V, Vaittinen S, Kalimo H, et al. Muscle injuries: optimising recovery. Best Pract Res Clin Rheumatol. 2007;21(2):317–31.
88. Delancey JO, Morgan DM, Fenner DE, Kearney R, Guire K, Miller JM, et al. Comparison of levator ani muscle defects and function in women with and without pelvic organ prolapse. Obstet Gynecol. 2007;109(2 Pt 1):295–302.
89. Dietz HP, Shek C. Levator avulsion and grading of pelvic floor muscle strength. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(5):633–6.
90. Steensma AB, Konstantinovic ML, Burger CW, de RD, Timmerman D, Deprest J. Prevalence of major levator abnormalities in symptomatic patients with an underactive pelvic floor contraction. Int Urogynecol J. 2010;21(7):861–7.
91. Brincat CA, Delancey JO, Miller JM. Urethral closure pressures among primiparous women with and without levator ani muscle defects. Int Urogynecol J. 2011;22(12):1491–5.
92. Hilde G, Staer-Jensen J, Siafarikas F, Gjestland K, Ellstrom EM, Bo K. How well can pelvic floor muscles with major defects contract? A cross-sectional comparative study 6 weeks after delivery using transperineal 3D/4D ultrasound and manometer. BJOG. 2013;120(11):1423–9.
93. Shek KL, Dietz HP. The effect of childbirth on hiatal dimensions. Obstet Gynecol. 2009;113(6):1272–8.
94. Majida M, Braekken IH, Bo K, Engh ME. Levator hiatus dimensions and pelvic floor function in women with and without major defects of the pubovisceral muscle. Int Urogynecol J. 2012;23(6):707–14.
95. Morgan DM, Larson K, Lewicky-Gaupp C, Fenner DE, Delancey JO. Vaginal support as determined by levator ani defect status 6 weeks after primary surgery for pelvic organ prolapse. Int J Gynaecol Obstet. 2011;114(2):141–4.
96. Dietz HP, Steensma AB. The prevalence of major abnormalities of the levator ani in urogynaecological patients. BJOG. 2006;113(2):225–30.
97. Dietz HP, Simpson JM. Levator trauma is associated with pelvic organ prolapse. BJOG. 2008;115(8):979–84.
98. Tunn R, Goldammer K, Neymeyer J, Gauruder-Burmester A, Hamm B, Beyersdorff D. MRI morphology of the levator ani muscle, endopelvic fascia, and urethra in women with stress urinary incontinence. Eur J Obstet Gynecol Reprod Biol. 2006;126(2):239–45.
99. Dietz HP, Kirby A, Shek KL, Bedwell PJ. Does avulsion of the puborectalis muscle affect bladder function? Int Urogynecol J Pelvic Floor Dysfunct. 2009;20(8):967–72.
100. Bodine-Fowler S. Skeletal muscle regeneration after injury: an overview. J Voice. 1994;8(1):53–62.
101. Jarvinen TA, Jarvinen TL, Kaariainen M, Kalimo H, Jarvinen M. Muscle injuries: biology and treatment. Am J Sports Med. 2005;33(5):745–64.
102. Orchard JW, Best TM, Mueller-Wohlfahrt HW, Hunter G, Hamilton BH, Webborn N, et al. The early management of muscle strains in the elite athlete: best practice in a world with a limited evidence basis. Br J Sports Med. 2008;42(3):158–9.
103. R L. Physical therapy and related interventions. In: PM T, editor. Skeletal muscle damage and repair. Champaign, IL: Human Kinetics; 2007. p. 219–30.
104. Bø K, Artal R, Barakat R, Brown W, Davies GA, Dooley M, et al. Exercise and pregnancy in recreational and elite athletes: 2016 evidence summary from the IOC expert group meeting,

Lausanne. Part 1-exercise in women planning pregnancy and those who are pregnant. Br J Sports Med. 2016;50(10):571–89. https://doi.org/10.1136/bjsports-2016-096218.
105. Benjamin DR, van der Water ATM, Peiris CL. Effects of exercise on diastasis of the rectus abdominis muscle in the antenatal and postnatal periods: a systematic review. Physiotherapy. 2014;100:1–8.
106. Vleeming A, Albert HB, Ostgaard HC, et al. European guidelines for the diagnosis and treatment of pelvic girdle pain. Eur Spine J. 2008;17:794–819.
107. Koelbl H, Igawa TY, Salvatore S, et al. Pathophysiology of urinary incontinence, faecal incontinence and pelvic organ prolapse. In: Abrams P, Cardozo L, Khouy S, Wein A, editors. Incontinence. 5th ed: Committee 4. Health publication Ltd; 2013. p. 261–359.
108. Lee D, Hodges PW. Behavior of the Linea Alba During a Curl-up task in diastasis rectus abdominis. An observational study. JOSP. 2016;46:580–9.
109. Venes D, Taber CW. Taber's cyclopedic medical dictionary. 20th ed. Philadelphia, PA: Davis; 2005.
110. Boissonnault JS, Blaschak MJ. Incidence of diastasis recti abdominis during the childbearing year. Phys Ther. 1988;68(7):1082–6.
111. Mota PGF, Pascoal AG, Carita AI, Bo K. The immediate effects on inter-rectus distance of abdominal crunch and drawing in exercises during pregnancy and the postpartum period. JOSP. 2015;45:781–8.
112. Rett MT, Braga MD, Bernardes NO, Andrade SC. Prevalence of diastasis of the rectus abdominis muscles immediately postpartum: comparison between primiparae and multiparae. Rev Bras Fisioter. 2009;13(4):275–80.
113. Turan V, Colluoglu C, Turkyilmaz E, Korucuoglu U. Prevalence of diastasis recti abdominis in the population of young multiparous adults in Turkey. Ginekol Pol. 2011;82(11):817–82.
114. Sperstad JB, Tennfjord MT, Hilde G, Engh ME, Bø K. Diastasis recti abdominis and risk of low back and pelvic girdle pain. Br J Sports Med. 2016;50:1092–6.
115. Spitznagle TM, Leong FC, Van Dillen LR. Prevalence of diastasis recti abdominis in a urogynecological patient population. Int Urogynecol J. 2007;18:321–8.
116. Lockwood T. Rectus muscle diastasis in males: primary indication for endoscopically assisted abdominoplasty. Plast Reconstr Surg. 1998;101:1685–91. discussion 1684–92
117. van de Water AT, Benjamin DR. Measurement methods to assess diastasis of the rectus abdominis muscle (DRAM): a systematic review of their measurement properties and meta-analytic reliability generalisation. Man Ther. 2016;21:41–53.
118. Keeler J, Albrecht M, Eberhardt L, Horn L, Donelly C, Lowe D. Diastasis recti abdominis: a survey of women's health specialists for current physical therapy clinical practice for postpartum women. J Womens Health Phys Ther. 2012;36:131–42.
119. Mota P, Pascoal AG, Sancho F, Carita AI, Bo K. Reliability of the inter-rectus distance measured by palpation. Comparison of palpation and ultrasound measurements. Man Ther. 2013;18(4):294–8.
120. Mota P, Pascoal AG, Sancho F, Bø K. Test-retest and intrarater reliability of 2-dimensional ultrasound measurements of distance between rectus abdominis in women. J Orthop Sports Phys Ther. 2012;42(11):940–6.
121. Candido G, Lo T, Janssen PA. Risk factors for diastasis of the recti abdominis. J Assoc Chart Physiother Women's Health. 2005;97:49–54.
122. Lo T, Candido G, Janssen P. Diastasis of the Recti abdominis in pregnancy: risk factors and treatment. Physiother Can. 1999;51:32.
123. Fernandes da Mota PG, Pascoal AG, Carita AI, Bo K. Prevalence and risk factors of diastasis recti abdominis from late pregnancy to 6 months postpartum, and relationship with lumbopelvic pain. Man Ther. 2015;20(1):200–5.
124. Lee DG, Lee LJ, McLaughlin L. Stability, continence and breathing: The role of fascia following pregnancy and delivery. J Bodywork Movement Ther. 2008;12:333–48.
125. Gilleard WL, Brown JM. Structure and function of the abdominal muscles in primigravid subjects during pregnancy and the immediate postbirth period. Phys Ther. 1996;76:750–62.

126. Liaw LJ, Hsu MJ, Liao CF, Liu MF, Hsu AT. The relationships between inter-recti distance measured by ultrasound imaging and abdominal muscle function in postpartum women: a 6-month follow-up study. J Orthopaed Sports Phys Ther. 2011;41(6):435–43.
127. Parker MA, Millar LA, Dugan SA. Diastasis rectus abdominis and lumbo-pelvic pain and dysfunction-are they related? J Women's Health Phys Ther. 2009;33:15–22.
128. Bjorklund K, Bergstrom S. Is pelvic pain in pregnancy a welfare complaint? Acta Obstet Gynecol Scand. 2000;79:24–30.
129. Charpentier K, Leboucher J, Lawani M, et al. Back pain during pregnancy and living conditions: a comparison between Beninese and Canadian women. Ann Phys Rehabil Med. 2012;55:148–59.
130. Mousavi SJ, Parnianpour M, Vleeming A. Pregnancy related pelvic girdle pain and low back pain in an Iranian population. Spine (Phila Pa 1976). 2007;32:E100–4.
131. Gutke A, Boissonnault J, Brook G, et al. The severity and impact of pelvic girdle pain and low-back pain in pregnancy: a multinational study. J Womens Health (Larchmt). 2017;27(4):510–7.
132. Wormslev M, Juul AM, Marques B, et al. Clinical examination of pelvic insufficiency during pregnancy. An evaluation of the interobserver variation, the relation between clinical signs and pain and the relation between clinical signs and physical disability. ScandJRheumatol. 1994;23:96–102.
133. Olsson C, Nilsson-Wikmar L. Health-related quality of life and physical ability among pregnant women with and without back pain in late pregnancy. Acta Obstet Gynecol Scand. 2004;83:351–7.
134. Stuge B, Veierød MB, Lærum E, et al. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy. A two-year follow-up of a randomized clinical trial. Spine. 2004;29:E197–203.
135. Persson M, Winkvist A, Dahlgren L, et al. Struggling with daily life and enduring pain: a qualitative study of the experiences of pregnant women living with pelvic girdle pain. BMC Pregnancy Childbirth. 2013;13:111.
136. Pierce H, Homer CS, Dahlen HG, et al. Pregnancy-related lumbopelvic pain: listening to Australian women. Nurs Res Pract. 2012;2012:387428.
137. Gutke A, Bullington J, Lund M, et al. Adaptation to a changed body. Experiences of living with long-term pelvic girdle pain after childbirth. Disabil Rehabil. 2017;2017:1–7.
138. Fredriksen EH, Moland KM, Sundby J. Listen to your body. A qualitative text analysis of internet discussions related to pregnancy health and pelvic girdle pain in pregnancy. Patient Educ Couns. 2008;73:294–9.
139. Engeset J, Stuge B, Fegran L. Pelvic girdle pain affects the whole life—a qualitative interview study in Norway on women's experiences with pelvic girdle pain after delivery. BMC Res Notes. 2014;7:686.
140. Wu WH, Meijer OG, Uegaki K, et al. Pregnancy-related pelvic girdle pain (PPP), I: terminology, clinical presentation, and prevalence. Eur Spine J. 2004;13:575–89.
141. Ostgaard HC, Zetherstrom G, Roos-Hansson E, et al. Reduction of back and posterior pelvic pain in pregnancy. Spine. 1994;19:894–900.
142. Gutke A, Ostgaard HC, Oberg B. Pelvic girdle pain and lumbar pain in pregnancy: a cohort study of the consequences in terms of health and functioning. Spine. 2006;31:E149–55.
143. Robinson HS, Mengshoel AM, Bjelland EK, et al. Pelvic girdle pain, clinical tests and disability in late pregnancy. Man Ther. 2010;15:280–5.
144. Kristiansson P, Svardsudd K, von Schoultz B. Back pain during pregnancy: a prospective study. Spine. 1996;21:702–9.
145. Ostgaard HC, Andersson GB. Previous back pain and risk of developing back pain in a future pregnancy. Spine. 1991;16:432–6.
146. Ostgaard HC, Andersson GB, Karlsson K. Prevalence of back pain in pregnancy. Spine. 1991;16:549–52.
147. Bjelland EK, Stuge B, Engdahl B, et al. The effect of emotional distress on persistent pelvic girdle pain after delivery: a longitudinal population study. BJOG. 2013;120:32–40.

148. Bø K, Backe-Hansen KL. Do elite athletes experience low back, pelvic girdle and pelvic floor complaints during and after pregnancy? Scand J Med Sci Sports. 2007;17:480–7.
149. O'Sullivan PB, Beales DJ. Diagnosis and classification of pelvic girdle pain disorders, part 2: illustration of the utility of a classification system via case studies. Man Ther. 2007;12:e1–12.
150. Aldabe D, Ribeiro DC, Milosavljevic S, et al. Pregnancy-related pelvic girdle pain and its relationship with relaxin levels during pregnancy: a systematic review. Eur Spine J. 2012;21:1769–76.
151. Nielsen LL. Clinical findings, pain descriptions and physical complaints reported by women with post-natal pregnancy-related pelvic girdle pain. Acta Obstet Gynecol Scand. 2010;89:1187–91.
152. Goode A, Hegedus EJ, Sizer P, et al. Three-dimensional movements of the sacroiliac joint: a systematic review of the literature and assessment of clinical utility. J Man Manip Ther. 2008;16:25–38.
153. 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.
154. Kibsgard TJ, Roise O, Sturesson B, et al. Radiosteriometric analysis of movement in the sacroiliac joint during a single-leg stance in patients with long-lasting pelvic girdle pain. Clin Biomech (Bristol., Avon.). 2014;29:406–11.
155. Snijders CJ, Vleeming A, Stoeckart R, et al. Biomechanics of the interface between spine and pelvis in different postures. In: Vleeming A, Mooney V, Dorman T, et al., editors. Movement, stability & low back pain. The essential role of the pelvis: Churchill Livingstone; 1997. p. 103–14.
156. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine. 2003;28:1593–600.
157. Hungerford B, Gilleard W, Lee D. Altered patterns of pelvic bone motion determined in subjects with posterior pelvic pain using skin markers. Clin Biomech. 2004;19:456–64.
158. Damen L, Buyruk HM, Guler-Uysal F, et al. Pelvic pain during pregnancy is associated with asymmetric laxity of the sacroiliac joints. Acta Obstet Gynecol Scand. 2001;80:1019–24.
159. Palsson TS, Graven-Nielsen T. Experimental pelvic pain facilitates pain provocation tests and causes regional hyperalgesia. Pain. 2012;153:2233–40.
160. Palsson TS, Hirata RP, Graven-Nielsen T. Experimental pelvic pain impairs the performance during the active straight leg raise test and causes excessive muscle stabilization. Clin J Pain. 2014;31(7):642–51.
161. Torstensson T, Lindgren A, Kristiansson P. Corticosteroid injection treatment to the ischiadic spine reduced pain in women with long-lasting sacral low back pain with onset during pregnancy: a randomized, double blind, controlled trial. Spine (Phila Pa 1976). 2009;34:2254–8.
162. Torstensson T, Lindgren A, Kristiansson P. Improved function in women with persistent pregnancy-related pelvic pain after a single corticosteroid injection to the ischiadic spine: a randomized double-blind controlled trial. Physiother Theory Pract. 2013;29:371–8.
163. Eichenseer PH, Sybert DR, Cotton JR. A finite element analysis of sacroiliac joint ligaments in response to different loading conditions. Spine (Phila Pa 1976). 2011;36:E1446–52.
164. Snijders CJ, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. 1. Biomechanics of self-bracing of the sacroiliac joints and its significance for treatment and exercise. Clin Biomech. 1993;8:285–94.
165. Franklin ME, Conner-Kerr T. An analysis of posture and back pain in the first and third trimesters of pregnancy. J Orthop Sports Phys Ther. 1998;28:133–8.
166. Moore K, Dumas GA, Reid JG. Postural changes associated with pregnancy and their relationship with low-back pain. Clin Biomech. 1990;5:169–74.
167. Okanishi N, Kito N, Akiyama M, et al. Spinal curvature and characteristics of postural change in pregnant women. Acta Obstet Gynecol Scand. 2012;91:856–61.
168. Aldabe D, Milosavljevic S, Bussey MD. Is pregnancy related pelvic girdle pain associated with altered kinematic, kinetic and motor control of the pelvis? A systematic review. Eur Spine J. 2012;21:1777–87.

169. Beales DJ, O'Sullivan PB, Briffa NK. Motor control patterns during an active straight leg raise in chronic pelvic girdle pain subjects. Spine. 2009;34:861–70.
170. Richardson CA, Snijders CJ, Hides JA, et al. The relation between the transversus abdominis muscles, sacroiliac joint mechanics, and low back pain. Spine. 2002;27:399–405.
171. Stuge B, Morkved S, Dahl HH, et al. Abdominal and pelvic floor muscle function in women with and without long lasting pelvic girdle pain. Man Ther. 2006;11:287–96.
172. Stuge B, Saetre K, Braekken IH. The association between pelvic floor muscle function and pelvic girdle pain—a matched case control 3D ultrasound study. Man Ther. 2012;17:150–6.
173. Stuge B, Saetre K, Ingeborg HB. The automatic pelvic floor muscle response to the active straight leg raise in cases with pelvic girdle pain and matched controls. Man Ther. 2013 18(4):327-332.
174. Dumas GA, Leger A, Plamondon A, et al. Fatigability of back extensor muscles and low back pain during pregnancy. Clin Biomech (Bristol, Avon). 2010;25:1–5.
175. Bewyer KJ, Bewyer DC, Messenger D, et al. Pilot data: association between gluteus medius weakness and low back pain during pregnancy. Iowa Orthop J. 2009;29:97–9.
176. Gutke A, Ostgaard HC, Oberg B. Association between muscle function and low back pain in relation to pregnancy. J Rehabil Med. 2008;40:304–11.
177. de GM, Pool-Goudzwaard AL, Spoor CW, et al. The active straight leg raising test (ASLR) in pregnant women: differences in muscle activity and force between patients and healthy subjects. Man Ther. 2008;13:68–74.

Further Reading

Fitzgerald CM, Segal NA, editors. Musculoskeletal health in pregnancy and postpartum. Cham: Springer; 2015.