Gunvor Hilde

Pelvic floor muscle function in pregnancy and after childbirth and the effect of postpartum pelvic floor muscle training on urinary incontinence in women with and without major defects of the levator ani muscle

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Du høge fjell

Du høge fjell
ti sjog og eld,
du blåe hø
og foss og sjø,
du fuglemyr
og blomster-eventyr,
du kvite tind
og ville vind
som mot me syng,
du brone lyng
du grøne li -
du stille tona mi,
med brus tå æ
frå fonn og bræ
ti vårljos natt, -
på fangade dine
e me sjøl og mine
ska finne att!

av Ragnar Solberg

Contents

Summary

Background: Urinary incontinence (UI) is highly prevalent in the female population and strongly associated with pregnancy and childbirth. The pelvic floor muscles (PFM) play an important role in pelvic organ support and for staying continent. Vaginal delivery is considered the most established risk factor for weakening of the PFM. Women having their first child should be offered supervised PFM training (PFMT) during pregnancy, and PFMT is first-line treatment of UI (Grade A recommendations). However, in populations of postpartum women with and without UI (mixed population), the current evidence on efficacy of PFMT on UI prevalence is not clear. Further, the effect of PFMT in women with major levator ani (LA) muscle defects is unknown.

Aims: Paper I: To investigate nulliparous pregnant women's knowledge and practice of PFMT, assess their ability to contract the PFM correctly and their PFM function (vaginal resting pressure, PFM strength, and PFM endurance), and to further compare PFM function in continent women versus women with UI. Paper II: To study the impact of childbirth and mode of delivery on PFM function (same variables as in Paper I), and further to compare PFM function in continent women versus women with UI. Paper III: To assess whether primiparous women with major LA muscle defect are able to contract the PFM, and further to compare PFM function (same variables as in Paper I-II) in women with and without major LA muscle defects. Paper IV: To evaluate the effect of postpartum PFMT on UI prevalence in primiparous women, with stratified analyses on women with and without major LA muscle defects.

Methods: Paper I was a cross-sectional study of 300 nulliparuos pregnant women at mid-pregnancy (gestational week 18-22). Paper II was a prospective cohort study, following 277 nulliparous pregnant women from mid-pregnancy to six weeks after delivery. Paper III was a cross-sectional study six weeks after vaginal delivery of the 175 the primiparous women included in the RCT. Paper IV was an assessor-blinded RCT including 175 primiparous women, stratified on major LA muscle defects. All participants were taught to contract the PFM correctly. The control participants received no further intervention, whereas the training participant received weekly supervised PFMT and performed daily home training. Data on knowledge about and practicing of PFMT was collected through a questionnaire. UI was assessed by the International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form (ICIQ UI SF), ability to contract the PFM by digital palpation, and PFM function (vaginal resting pressure, PFM strength, and PFM endurance) by a vaginal balloon connected to a pressure transducer (manometer). Major LA muscle defects were diagnosed by transperineal 3D/4D ultrasound.

Main results: Paper I: Of the 300 nulliparous pregnant women had 89% heard about PFMT at mid-pregnancy, 35% of them performed PFMT ≥ once a week, and 15% ≥ three times per week. Thirty-five % reported UI, of whom 48% performed PFMT ≥ once a week. Continent women had significantly higher PFM strength and endurance than women with UI, mean difference was 6.6 cmH_2O for PFM strength ($p=0.003$) and 41.5 cmH_2Osec for endurance ($p=0.010$). Paper II: All PFM measurements changed significantly ($p<0.001$), both in the group with normal vaginal delivery (n=193) and in the group with instrumental assisted vaginal delivery (vacuum/forceps, n=45): Vaginal resting pressure was reduced by 29% and 30%, PFM strength by 54% and 66%, and endurance by 53% and 65%, respectively. In the group of women with emergency caesarean section (n=29), only vaginal resting pressure changed significantly from pregnancy to after childbirth (10% reduction, $p=0.003$). Urinary continent women at both clinical visits (mid-pregnancy and six weeks after delivery) had significantly higher PFM strength and endurance than incontinent counterparts being incontinent at both points in time ($p<0.05$). Paper III: 4% of the 175 women were unable to contract their PFM six weeks after delivery. Women with major LA muscle defects (n=55) had 47% lower PFM strength and 47% lower endurance when compared with women without major LA muscle defects (n=120). Mean difference was 7.5 cmH_2O for PFM strength ($p <0.001$) and 51.2 cmH_2Osec for endurance ($p<0.001$). No difference was found regarding vaginal resting pressure ($p=0.670$). Paper IV: The prevalence of UI post-intervention (6 months postpartum) was 34.5 % in the training group and 38.6 % in the control group. The relative risk analysis gave a non-significant effect for PFMT on UI prevalence, RR of 0.89 (95% CI: 0.60 to 1.32). Stratified analysis of women with and without major LA muscle defects gave respectively a RR of 0.89 (95% CI: 0.51 to 1.56) and 0.90 (95% CI: 0.53 to 1.52).

Conclusions: Most nulliparous pregnant women knew about PFMT, but few performed PFMT. Pronounced reductions in vaginal resting pressure, PFM strength and endurance were found after vaginal delivery, whereas only vaginal resting pressure changed after caesarean section. Women with major LA muscle defects had weaker PFM than women without such defects, however most women were able to contract their PFM. The postpartum PFMT intervention did not decrease UI prevalence six months after delivery in primiparous women, and the stratified analysis on women with and without major LA muscle defects showed similar non-significant results.

Key words: Levator ani muscle; Mode of delivery; Pelvic floor muscle strength and endurance; Pregnancy and Childbirth; Urinary incontinence; Vaginal delivery; Vaginal resting pressure.

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Oslo, February 2014.
Gunvor Hilde

List of papers

This dissertation is based on the following original research papers; they are referred to in the text by their Roman numerals.

I. Hilde G, Staer-Jensen J, Ellström EM, Braekken IH, Bø K. Continence and pelvic floor status in nulliparous women at midterm pregnancy. Int Urogynecol J 2012; 23(9):1257-1263.

II. Hilde G, Staer-Jensen J, Siafarikas F, Engh ME, Braekken IH, Bø 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.e1-7.

III. Hilde G, Staer-Jensen J, Siafarikas F, Gjestland K, Ellström EM, Bø 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):1423-1429.

IV. Hilde G, Staer-Jensen J, Siafarikas F, Ellström EM, Bø K. Postpartum pelvic floor muscle training and urinary incontinence: a randomized controlled trial. Obstet Gynecol 2013; 122(6):1231-1238.

Abbreviations

3D	Three dimensional
4D	Four dimensional
BMI	Body mass index
CI	Confidence interval
LA	Levator ani
MUI	Mixed urinary incontinence
MRI	Magnetic resonance imaging
PFM	Pelvic floor muscles
PFMT	Pelvic floor muscle training
RCT	Randomised controlled trial
RR	Relative risk
SD	Standard deviation
SUI	Stress urinary incontinence
UI	Urinary incontinence
UUI	urinary urge incontinence

Note regarding references in background chapter:

The four papers included in this dissertation are based on a cohort study and a randomised controlled trial. The references in the background chapter (pp: 2-21) reflect our knowledge of the field before project start (January 2010), and therefore include literature published before January 2010. Literature published during the project period is included when discussing Paper I-IV in the general discussion chapter (pp: 40-53).

Introduction

Pregnancy and childbirth are associated with happiness and a positive life change for most women. Unfortunately, pregnancy and childbirth can also be considered as risk periods for development of pelvic floor dysfunction [1].

Around 4.2 births take place every second [2]. Approximately 134 million births take place every year worldwide, 10 million of them in developed countries [3]. Data from the Medical Birth Registry of Norway [4] shows that 61884 deliveries took place in Norway during 2009 with 62994 babies being born; 83% of the babies were delivered vaginally, 17.0% by caesarean section. 42.6% of the women giving birth that year were primiparous [4].

Advancements in obstetric care during the 20th century have reduced the maternal and infant mortality rate dramatically allowing increased focus on reduction of morbidity in conjunction with pregnancy and childbirth such as pelvic floor dysfunction [5].

Urinary incontinence (UI), faecal incontinence and pelvic organ prolapse are highly prevalent in the female population [1;6] and are among the most common conditions seen in clinical gynaecological practice [7]. These conditions may significantly impact the physical, psychological and social well-being of those women affected [8;9]. UI is shown to be a barrier for physical activity and exercise, [10-12], and may inhibit women from lifelong participation in regular physical activity [11;12], which is important for maintaining health and fitness [13]. A population based study conducted in Portland; Oregon showed an 11% lifetime risk of undergoing surgery for UI or pelvic organ prolapse [14].

Background

Pelvic floor anatomy

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) [15]. In addition to pelvic organ support, the pelvic floor maintains continence, permit urination, defecation, intercourse and vaginal birth [15].

Pelvic floor muscles

The superficial layer of the pelvic floor includes the perinal muscles (ischiocavernosus, bulbospongiosus and transversus perinea superficialis), and the deep layer includes the levator ani (LA) muscle [15]. 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 [15-17].

The levator ani muscle

The LA muscle is the largest pelvic floor muscle (PFM), innervated by the inferior branches of the pudendal nerve [18;19]. This muscle can be subdivided into: the iliococcygeus, the puborectalis, and the pubococcygeus muscle (also termed puboviceralis) [1;20;21]. These subdivisions of the LA muscle have different origins and insertions and different muscle fibre directions [1;21]. The iliococcygus muscle forms the posterior part of the LA muscle (Figure 1), arising bilaterally from the arcus tendineus levator ani, spans the gap from one pelvic sidewall to the other, and is melded together and inserted into the iliococcygeal raphe [1;21]. The anterior and medial part of the LA muscle (Figure 1) , the pubococcygeus and the puborectalis muscle form a "U-shaped muscle sling", that originates from the right and left side of the pubic bone and loop back behind the rectum where they merge [1;21]. This sling borders an opening in the pelvic floor, the levator hiatus, allowing the passage of the urethra, rectum, and the vagina

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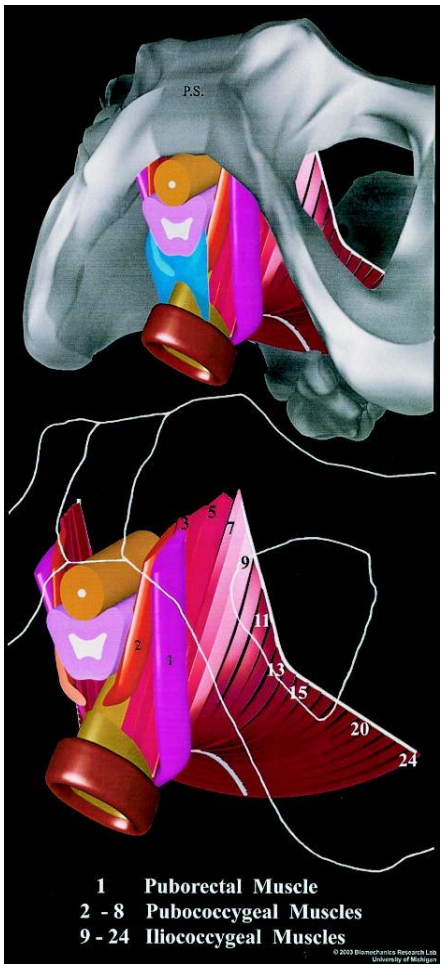

Figure 1. *(Top) Inferior three-quarter view, seen from the left, of the pelvic floor structures appearing behind the ischiopubic rami (gray). That portion of the perineal membrane (blue) connecting the most medial portion (2) of the pubococcygeus muscles to the perineal body is shown. The lateral portions of the perineal membrane have been removed. (Bottom) The pelvic bones (outlined from image above in white) and perineal body have been removed to show a close-up of the arrangement of the iliococcygeal, pubococcygeal, puborectalis muscles, as well as the urethra (umber), vagina (pink), and rectum (brown). Individual muscle bands are identified by a number inscribed near their origin on the arcus tendineus (white). The anal sphincters were segmented directly from in vivo magnetic resonance images, but neither the coapting effect of the venous plexus nor its covering anoderm are shown. © 2003 Biomechanics Research Lab, University of Michigan, Ann Arbor.*

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Pelvic floor muscle function

The 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 [1;17;22]. The function of this supportive system is illustrated by the "boat in dry dock theory" by Norton [22], 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 [23;24]. 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 [16;17].

The normal baseline activity of the PFM keeps the pelvic openings closed and keeps the pelvic floor elevated in a cranial direction [1;25]. 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 [1;16;17;26]. 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 [27].

Together with the urethral sphincter muscles, the PFM play an important role for maintaining urinary continence [1;16;17;26]. The mechanical supportive potential of the PFM is demonstrated by Miller et al [28]. By perineal ultrasound assessment, they found that a voluntary contraction of the PFM prior to and during a cough (a manoeuvre called the "Knack") resulted in a significant reduced displacement of the bladder neck [28]. Use of the "Knack" manoeuvre has also shown to significantly reduce urine loss among women with SUI [29;30].

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, faecal incontinence and pelvic organ prolapse) probably have a complex list of risk factors [7;31;32]. Factors that may lead to the development of pelvic floor impairment and dysfunction in women can according to Bump & Norton [7] be classified into the following four categories:

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

DeLancey et al [32] integrate factors affecting pelvic floor dysfunction into a "Integrated Lifespan Model" (Figure 2), 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. 3). Deterioration occurring with advancing age. Throughout the lifespan a decline of the functional reserve of the pelvic floors 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 [7;32].

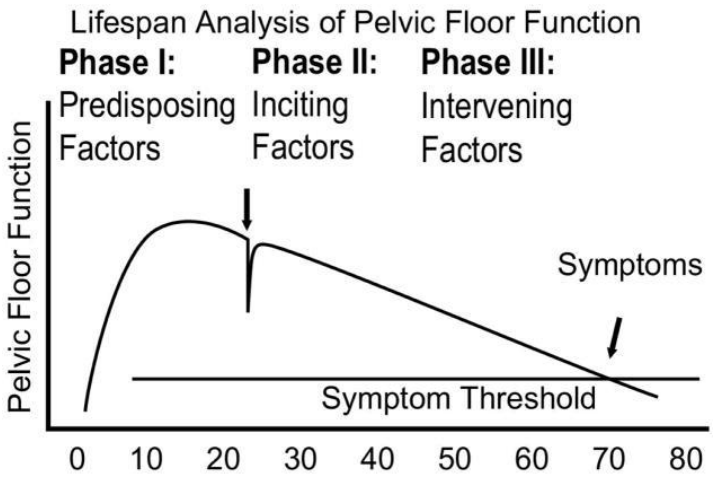

Figure 2. *Integrated lifespan analysis of pelvic floor function*
This graphical display of the abstract concept of pelvic floor function tracks the functional reserve throughout different phases of a woman's lifespan. Initially, pelvic floor structure growth in late teens leads to a fully developed pelvic floor. Vaginal birth affects pelvic floor function. Finally, age-related deterioration occurs until a symptom threshold is reached where the functional reserve present earlier in life is lost. (© DeLancey 2007).

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Urinary incontinence

Definition and classification

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: [33]

- **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."

Prevalence

A systematic literature review by Hunskaar et al [34] 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% [34]. 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 [35-37]. According to an updated review on UI prevalence by Milsom et al [6], do recent epidemiological studies report estimates on UI prevalence that places within the prevalence range reported by Hunskaar et al. [34]. 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 [6;34].

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 [6;34]. Higher period prevalence has been reported in parous than in nullipaous women [38-42]. The largest prospective population based study included in the review by Milsom et al [6] is the study published by Wesnes et al [42]. 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.

Prevalence after childbirth

The estimation of postpartum UI is, according to Milsom et al [6], challenged by study heterogeneity (study design and method, definition of UI, and sample studied.). In their review they therefore chose to summarise 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% to 30%. According to Milsom [6], 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 [43].

Etiology and pathophysiology of UI

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 [44]. Connective tissue, peripheral nerves and muscular structures are already during pregnancy subjected to hormonal, anatomical and morphological changes. During vaginal delivery, the above mentioned 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 [44]. The picture of possible causative factors and the pathophysiology of UI is 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 [44].

Pregnancy

Prospective observational studies have shown increased prevalence of UI from the first trimester to the second, and further into the third trimester [41;45;46]. 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 [41]. In an observational study by Hvidman et al [41], 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 [42] and after childbirth [47-49], which could be caused by increased intra-abdominal pressure and increased bladder pressure [50].

The PFM is considered to play a significant role in the continence control system [1;16;17;26], 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 [51-57], while some studies did not find such difference [58;59]. Two of the above-mentioned studies were on pregnant women [51;55]. In addition to significantly higher PFM strength, Mørkved et al [55] 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 [49;60-62].

Childbirth

Parity seems to be an increased risk factor for UI [31;39;46;63-65]. In a cross-sectional study of 27 900 women, Rørtveit et al [64] report a relative risk (RR) of UI of 2.2 (95% CI: 1.8 to 2.6) for primiparus women and 3.3 (95% CI: 2.4 to 4.4) for grand multiparous women. Altman et al [62] 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 [49] 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 [66] 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.

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 [17]. Peschers et al [67] investigated change in bladder neck mobility, during the Valsalva manoeuvre, 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 [68] and Dietz et al [69].

Meyer et al [70] found significantly higher bladder mobility, during the Valsalva manoeuvre, 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 & Freeman [71] followed nulliparous pregnant women with no pre-existing 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 remodelling [71;72].

A study on bladder neck mobility and tissue stiffness was performed by Howard et al [73]. Results from their study showed that primiparous women with SUI displayed similar bladder neck mobility during a cough and during a Valsalva manoeuvre ($p=0.49$), 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 ($p=0.001$).

Neural denervation

Neuromuscular impairment is associated with the development of incontinence. Smith et al [74] 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 [75-78]. In a biomechanical study by Lien et al [79], lengthening of pudendal nerve branches were 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 [77;80;81]. Such neural impairment may alter the muscle

morphology. In a study by Gilpin et al [82], biopsy samples from women with SUI showed a significant higher number of muscle fibres with pathological damage when compared to biopsy samples from continent women.

Weakening of the pelvic floor muscles

Vaginal delivery is considered as a main risk factor for weakening of the PFM [75;77;83-88]. Due to the extensive stretching of muscle fibres 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. However, there seems to be a paucity of prospective studies presenting clinical data on these PFM variables. A PubMed search prior to project start (January 2010), revealed three studies [51;68;89] investigating change in PFM strength from pregnancy to shortly after childbirth in relation to mode of delivery. Results from these three studies showed a significant reduction in PFM strength after vaginal delivery, but no significant decline after caesarean section.

Levator ani muscle defects

Vaginal delivery may stretch and load beyond the physiological properties of the PFM, which may lead to muscle fibre tearing and reduced contractile force. The bio-mechanical study by Lien et al [90] showed that muscle fibres 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 (Figure 3). Their findings showing a pronounced stretch and deformation of the medial part of the LA muscle is confirmed by Hoyte et al [91] and Parente et al [92].

During recent years, technical advancement within magnetic resonance and ultrasound imaging has enabled diagnosis of defects of the LA muscle [93]. 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 [83;86;93]. Imaging studies have shown that major LA muscle defects among primiparous women delivering vaginally could appear in 20-36% of the women [83;94]. The use of forceps [84] and length of the second stage [84;95] are associated with major LA muscle defects, whereas the importance of fetal head circumference and high fetal birth weight seems to be less clear [94-96].

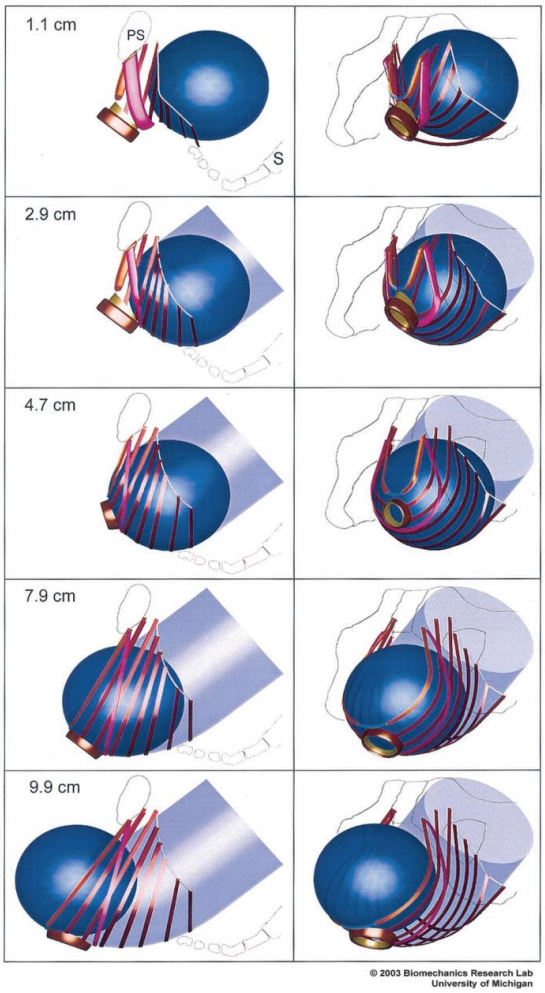

Figure 3. *Simulated effect of fetal head descent on the levator ani muscles in the second stage of labor. At top left, a left lateral view shows the fetal head (blue) located posteriorly and inferiorly to the pubic symphysis (PS) in front of the sacrum (S). The sequence of five images at left show the fetal head as it descends 1.1, 2.9, 4.7, 7.9, and 9.9 cm below the ischial spines as the head passes along the curve of Carus (indicated by the transparent, light blue, curved tube). The sequence of five images at right are front-left, three-quarter views corresponding to those shown at left. © 2003 Biomechanics Research Lab, University of Michigan, Ann Arbor.*

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Decreased strength is one of the most common symptoms following muscle tears within sport injuries [97]. Hence, decreased PFM strength in women with major LA muscle defects is expected, but has been sparsely investigated. A PubMed search prior to project start revealed two observational studies [98;99] in which PFM strength in women with and without LA muscle defects was assessed. Results from both studies showed significantly reduced PFM strength in women with LA muscle defects when compared to women without such defects. PFM strength was assessed by dynamometer in the study by DeLancey et al [98], and by digital palpation in the study by Dietz & Shek [99]. The mean age of women included in these two studies was ≥ 55 years and the parity range was from 0 to 12.

Major LA muscle defects have shown a marked effect on hiatal dimensions [100] and pelvic organ support [101] 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 [93;98;102;103], while the link between LA muscle defects and UI is debated. Two studies [83;94] 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 [102;104;105].

Muscle injury regeneration

The healing process of skeletal muscles

The healing process of a torn muscle has three phases: 1) the destruction phase, 2) the repair phase, and 3) the remodelling phase [97;106-108]. In the destruction phase, the rupture is followed by necrosis and formation of a hematoma. In the repair phase a phagocytosis of necrotised 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 revascularisation of the injured area initiated. During the remodelling phase, a further maturation of the regenerated myofibrils is implemented together with remodelling of the scar tissue, followed by recovery to functional capacity [97;106-108].

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 [109]. Early mobilisation is standard treatment after muscle injury within sports medicine, and training is believed to be important in speeding up tissue healing (repair and remodelling). This approach is supported by experimental studies showing that early mobilisation after a muscle injury may facilitates the following: More rapid capillary ingrowths, improved parallel orientation of the regenerating myofibrils, and improved tensile properties [97;106;108;110].

Pelvic floor muscle training

Several hypotheses have suggested that a trained PFM might reduce the risk of UI during pregnancy and after childbirth [111]. For example, a trained PFM may counteract the hormonally mediated increased laxity of the pelvic floor and the increased intra-abdominal pressure during pregnancy. A trained PFM may encompass a greater functional reserve so that childbirth does not cause the sufficient loss of muscle function to develop urinary leakage. Further, a trained PFM may recover better after childbirth as the appropriate neuromuscular motor patterns have already been learned [111].

PFM training (PFMT) and the importance of PFMT in restoring function after childbirth, was introduced as early as 1948 by Kegel [112]. In an uncontrolled clinical trial from 1952, he reported that 84% of his patients with UI were cured after performing PFMT [23]. According to Bø [113], there are two main rationales for why PFMT works:

- Women learn how to consciously pre-contract the PFM before and during situations causing increased abdominal pressure (e.g. coughing).
- Increased PFM strength and enhance hypertrophy takes place, building up long-lasting muscle volume to provide structural support.

Pre-contraction

During situations with increased abdominal pressure the supportive action of the PFM is believed to be important [1;16;17;26]. Miller et al [29] found that older women with SUI could acquire the skill of a well-timed PFM contraction just ahead of and during a cough ("The Knack"), and by this manoeuvre significantly reduce leakage. The positive effect of the Knack manoeuvre in reducing leakage during coughing, has later been confirmed both among nonpregnant and pregnant women [30]. The rationale to acquire such a skill is to prevent the urethra and bladder base from descending during increased abdominal pressure, and thereby prevent leakage. An actual stabilisation of the bladder neck by performing pre-contraction just ahead of and during a cough has subsequently been shown in observational studies, using perineal ultrasound, both among both nulliparous continent women [28;114] and older incontinent parous women [28].

Strength training

PFMT is designed to improve the conscious awareness of a correct PFM contraction and increase PFM strength, and thereby provide increased urethral compression and prevent urethral descent [113;115]. The PFM, as other skeletal muscles, respond to strength training by improved neuromuscular function, increased cross-sectional area, increased number of activated motor neurons, increased frequency of excitation, and improved muscle "tone", [113;115;116]. Specificity and overload are two fundamental principles that carefully must be addressed for effective strength training [115;117].

To improve a specific skill, that specific skill must be performed. To become a good skier you need to ski. To effectively improve PFM strength, specific PFM contraction performed in a correct manner needs to be carried out [115]. This compromises an inward lift and squeeze around the urethra, vagina and rectum [23;118;119]. Avoiding co-contraction of other muscles should be emphasised, as this may mask the actual strength of the PFM contraction being performed [115]. The principle of specificity also draws attention to the fact that a correct PFM contraction may be difficult to perform for some women. Studies on women with UI have actually shown that > 30% were unable to perform a correct PFM contraction [23;120-122], even after a brief verbal instruction on how to contract. Assessment of the ability to contract the PFM can easily be performed by visual observation and vaginal palpation [123;124]. Proper assessment, instruction and teaching on how to contract correctly, is considered as crucial in order to gain benefit from PFMT [125].

To achieve increased cross-sectional area and increased contractile force, the muscles need to be exposed to an overload that is larger than the common load encountered during everyday life [117]. Overload in PFMT can be achieved by performing close to maximal contractions, lengthening the holding periods for each contraction, increasing number of repetitions and number of sets completed, and reducing the rest intervals [115]. Strength training recommendations for skeletal muscles are 8-12 maximal contractions, 3-4 series, 3-4 times per week [13;116]. It takes time to achieve increased PFM strength, endurance and muscle volume [115], and The American College of Sports recommends the exercise duration period to be at least 15-20 weeks [13]. Strength training with contractions close to maximum and short rest intervals between the contractions usually also increase local muscle endurance as the muscle then is exposed to fatigue [27;126].

Effect of pelvic floor muscle training - evidence to date

To date there is Level I evidence of significant effect of supervised PFMT in the treatment of UI in the general female adult population [127;128], and Grade A recommendation for offering supervised PFMT as the first-line treatment for female stress, urge, or mixed UI [129;130].

During pregnancy

In the Cochrane review by Hay-Smith et al [111], the meta analysis of randomized controlled trials (RCTs) on primary prevention showed that PFMT during pregnancy (antenatal PFMT) reduced the likelihood of UI in late pregnancy by about 56% when compared to standard care or no treatment (RR=0.44, 95% CI: 0.30 to 0.65). Postpartum results (3-6 months) showed a reduced likelihood of UI by about 30% in favour of antenatal PFMT (RR=0.71, 95% CI: 0.52 to 0.97). Antenatal PFMT trials, including women with and without UI (mixed prevention and treatment trials), reduced the likelihood of UI by around 12% in late pregnancy (RR=0.88, 95% CI: 0.81 to 0.96). The National Institute for Health and Clinical Excellence (NICE) [129] and the 4th International Consultation on Incontinence [130] recommend that pregnant women having their first child should be offered supervised PFMT (Grade A recommendation).

After delivery (postpartum)

Trials on PFMT as treatment: The summarised effect of postpartum PFMT in treatment of UI presented in the Cochrane review by Hay-Smith et al [111] is based on three RCTs [131-133]. The pooled effect shows a significant risk reduction of UI in favour of PFMT; RR=0.79, 95% CI: 0.70 to 0.90. According to current guidelines and recommendations [129;130], should PFMT be offered as first line treatment to women with persistent UI symptoms after delivery (Grade A recommendation). The three primary studies in the above-mentioned Cochrane review [131-133] are presented in Table 1. The methodological quality of each study was assessed by using the PEDro Scale with a score range from 1-10 [134]. The given sum score to the three studies [131-133], ranged from 4-7, and their scoring on each PEDro Scale item is presented in Table 3.

The training participants in the study by Wilson & Herbison [131] and Glazener et al [132] were given instructions to perform 80-100 PFM contractions daily, while control participants received standard care. Dumoulin et al [133], compared weekly supervised PFMT (25 min) plus daily home training versus relaxation massage of the back and extremities during 8 consecutive weeks (Table 1). The study by Dumoulin et al [133] had no drop outs, while the other two studies had a considerable drop-out rate and did not meet the criteria of adequate follow up ≥ 85% (Table 3).

Trials on PFMT as prevention and treatment (mixed prevention and treatment trials):
The effect of postpartum PFMT in prevention and treatment of UI has, to date, been investigated in four RCTs [135-138] and in one matched controlled trial [139]. The results are conflicting. Two studies [137;139] showed a significant effect on reduced UI in favour of the PFMT group, while three studies [135;136;138] report no significant effect. Only RCTs are included in the Cochrane review [111]. The pooled risk ratio for UI 3-6 months postpartum, based on three RCTs [135;137;138], showed no effect of PFMT when compared to standard care (RR=0.97, 95% CI: 0.85 to 1.09). They report a similar pooled risk ratio for UI 6-12 months postpartum (RR=0.94, 95% CI: 0.75 to 1.16), which was based on two RCTs [136;137]. The four RCTs and the one matched controlled trial are all displayed in Table 2. Their method score (Table 3) ranged from 4-7 on the PEDro Scale [134]. The five studies presented in Table 2 include both primiparous and multiparous women with vaginal delivery, but was restricted to instrumental assisted vaginal delivery in two studies [137;138]. An additional inclusion criterion in the study by Chiarelli & Cockburn [137] was giving birth to a baby weighing 4000 g or more. The intervention started shortly after delivery (during hospital stay) in three studies [135;137;138], and 8 weeks after delivery in the other two studies [136;139]. The number of women included, content and dosage of the PFMT intervention, PFMT adherence, and drop-out rate varied greatly (Table 2). Mørkved and Bø [139] reported by far the highest risk reduction in favour of supervised PFMT, used a training protocol based on strength recommendations, and had 100% adherence in the PFMT group. Findings from their study showed a 50% reduced likelihood of UI in the training group four months after delivery (RR 0.50, 95% CI: 0.28 to 0.89).

For studies including both women with and without UI, the evidence is less clear. The lack of effect in studies included in the Cochrane review may be due to low intensity of the PFMT intervention and a low adherence [135;138].

Table 1. Studies evaluating effect of pelvic floor muscle training after delivery on any UI prevalence; studies including women with UI (treatment trials).

Study	Design	Subjects	Interventions	Outcomes at post-intervention Drop out Adherence	Quality of method PEDro Scale
Wilson & Herbison [131]	RCT (N=230) • Intervention (n=113) • Control (n=117) New Zealand	Primi- and multipara women With UI at inclusion With any delivery mode Included 3 months after delivery 190 / 230 (83%) with vaginal delivery at entry	Intervention: Received instruction by physiotherapist on 4 occasions (3, 4, 6 and 9 months after delivery). Vaginal perineometer used to teach awareness of PFM contractility. Basic programme of PFM exercises was 80-100 contractions daily. Basic cone programme was keeping cones of increasing weight for 15 min twice daily. Intervention participants were subdivided into: - PFM exercises - Cone training - PFM exercises + cone training Control: Usual postnatal care; PFM exercises taught by physiotherapist were offered during hospitalisation from 2nd day after delivery.	Self-reported UI 12 months after delivery • Intervention PFM exercises only: 9 / 19 (47.4%) • Control: 69 / 91 (75.8%) RR: 0.62 (0.38 to 1.02), p=0.059 Drop out: Intervention: n=59 (20 from PFM exercises, 22 from cone training, 17 from combination); Control n=26 89% in intervention and 65% in control reported to have performed PFMT last month 48% in intervention and 9% in control reported to have performed PFMT daily	Score: 4/10
Glazener et al [132]	RCT (N=747) • Intervention (n=371) • Control (n=376) New Zealand UK	Primi- and multipara women With UI at inclusion With any delivery mode Included 3 months after delivery	Intervention: Home visit by nurse; instructions about pelvic floor anatomy, PFM contraction regimens, and preparation exercises to identify the PFM were given. Participants were asked to follow a programme aiming at 80-100 contractions daily Control: Peripartum preparation, which sometimes included PFM exercises. Could seek medical advice	Self-reported UI 12 months after delivery • Intervention: 167 / 279 (59.9%) • Control: 169 / 245 (69.0%) RR: 0.87 (0.76 to 0.99), p=0.029 Drop out: Intervention n=92, Control n=131; 79% in intervention and 48% in control reported to have performed PFMT last month	Score: 7/10
Dumoulin et al [133]	RCT (N=64) • Intervention group I (n=21) • Intervention group II (n=23) • Control (n=20) Canada	Primi- and multipara women With SUI at inclusion Included 3 months after delivery	Intervention group I: Weekly sessions of supervised pelvic floor rehabilitation by trained physiotherapist over 8 consecutive weeks (15 min electrical stimulation, 25 min with PFMT) + home training 5 days / week. Intervention group II: Same as intervention group I + deep abdominal muscle training Control: 8 weekly session of relaxation massage for the back and extremities	Positive pad test (> 2 gram) 1 week post-intervention • Intervention group I: 6 / 20 (30.0%) • Intervention group II: 6 / 23 (26.1%) • Control: 19 / 19 (100.0%) RR Group I vs control: 0.32 (0.17 to 0.60), p=0.001 RR Group II vs control: 0.28 (0.14 to 0.54), p<0.001 Drop out: none from either group Adherence not reported	Score: 6/10

RCT=randomised controlled trial; PFM= pelvic floor muscle; PFMT=pelvic floor muscle training.
RR=relative risk are calculated for each study using the Mantel-Haenzel risk analysis, point estimates given with 95% confidence interval.
PEDro Scale score (ranging from 1-10) for rating methodological quality [134]. Items and scoring presented in Table 3.

Table 2. Studies evaluating effect of pelvic floor muscle training after delivery on UI prevalence, including women with and without UI (prevention & treatment trials).

Study	Design	Subjects	Interventions	Outcomes at post-intervention Drop out Adherence	Quality of method PEDro Scale
Sleep & Grant [135]	RCT (N=1800) • Intervention (n=900) • Control (n=900) UK	Primi- and multipara women With vaginal delivery With and without UI at inclusion Included within 24 hours after giving birth	Intervention: As controls + daily individually instruction /exercise from midwife at postnatal ward prior to discharge. Asked to repeat PFM exercises as often as they could and incorporate them in daily household tasks. Received telephone reminders. Training diary over the 4 weeks of intervention Control: Usual postnatal care; ordinary booklet from postnatal ward + physiotherapist offering instruction and PFMT during hospital stay prior to discharge (usually within 48 hours)	Self-reported UI 3 months after delivery • Intervention: 180 / 816 (22.1%) • Control: 175 / 793 (22.1%) RR: 1.00 (0.83 to 1.20), p=0.996 Drop out: Intervention n=84; Control n=107 58% in intervention group and 42 % in control reported to perform their PFMT 3 months after delivery	Score: 4/10
Mørkved & Bø [139]	Prospective matched controlled trial (N=198) • Intervention (n=99) • Control (n=99) Matched on age, parity, delivery mode Norway	Primi- and multipara women With vaginal delivery With and without UI at inclusion Included 8 weeks after delivery	Intervention: As controls + supervised PFMT class once a week led by physiotherapist + daily home training twice a day (sets of 8-12 maximal PFM contractions held for 6-8 sec). Training diary during the 8 weeks intervention Control: Usual postnatal care including costmary leaflet prior to discharge from postnatal ward. In addition to usual care they received instruction in how to contract the PFM correctly and ability to contract was assessed. Not discouraged from performing PFMT	UI 4 months after delivery 1) Structured interview • Intervention: 14 / 99 (14.1%) • Control: 28 / 99 (28.3%) RR: 0.50 (0.28 to 0.89), p=0.019 2) Positive pad test (> 2 gram) • Intervention: 3 / 99 (3.0%) • Control: 13 / 99 (13.1%) RR: 0.23 (0.07 to 0.78), p=0.019 Drop out: None from either group 100% adherence in PFMT; Controls: 65%, PFMT ≥ 3 times / week	Score: 4/10
Meyer et al [136]	Controlled trial with alternate allocation (N=107) • Intervention (n=51) • Control (n=56) Switzerland	Nulliparous women delivering vaginally With vaginal delivery With and without SUI at inclusion Included 9 weeks after delivery	Intervention: Pelvic floor education 12 sessions over 6 weeks including PFMT followed by biofeedback and electro-stimulation Control: No pelvic floor education during the intervention period (2-10 months after delivery)	Self-reported SUI 10 months after delivery • Intervention 6 / 51 (11.8%) • Control 8 / 56 (14.3%) RR: 0.82 (0.31 to 2.21), p=0.700 Drop out: None from either group Adherence not reported	Score: 4/10

Table 2. cont.

Chiarelli & Cockburn [137]	RCT (N=720) • Intervention (n=370) • Control (n=350) Australia	Primi- and multipara women With vaginal instrumental delivery With baby ≥ 4000 gram With and without UI at inclusion Included at hospital ward after giving birth	Intervention: "Promoting urinary incontinence" using the "Health believe model" and adherence strategies. Participants were seen by physiotherapist once during hospital stay and again 8 weeks after delivery. Intervention had both discussions and active intervention including individual tailored PFMT programme, "the knack", pelvic floor assessment, ongoing referral if necessary. Control: Usual care; ordinary booklet from hospital (no visit from a physiotherapist). Women were not restricted from training	Self-reported UI 3 months after delivery • Intervention 108 / 348 (31.0%) • Control: 125 / 328 (38.4%) Mean diff UI 7.4% (95% CI: 0.22% to 14.6%; p=0,004) OR: 0,65 (0,46 – 0,91; p=0,01)* RR: 0.81 (0.66 to 1.00), p=0.053 Drop out: Intervention n=22; Control n=22; PFMT reported 3 times or more / week: Intervention: 83.9%; Control: 57.6%	Score: 7/10
Ewings et al [138]	Nested RCT (N=234) • Intervention (n=117) • Control (n=117) UK	Primi- and multipara women With vaginal instrumental delivery With risk of UI With and without UI at inclusion Included from postnatal wards	Intervention: One to one instruction on PFMT at hospital, and invited to attend a PFM exercise group on two subsequent occasions 2 months and 4 months after delivery Control: Usual care; ordinary booklet from hospital + verbal promotion of PFMT	Self-reported UI 6 months after delivery • Intervention 54 / 90 (60.0%) • Control: 47 / 100 (47.0%) RR: 1.28 (0.98 to 1.67), p=0.074 Drop out: Intervention n=27; Control n=17 Intervention adherence: 20% attended 1st occasion of exercise 5% attended 2nd occasion of exercise	Score 6/10

*RCT=randomised controlled trial; PFM= pelvic floor muscle; PFMT=pelvic floor muscle training; *OR=odds ratio as reported in published article.*
RR=relative risk are calculated for each study using the Mantel-Haenzel risk analysis, point estimates given with 95% confidence interval.
PEDro Scale score (ranging from 1-10) for rating methodological quality [134]. Items and scoring presented in Table 3.

Table 3. Methodological quality score (PEDro Scale) of controlled trials evaluating effect of postpartum pelvic floor muscle training.

Study	Eligibility criteria specified	Random allocation	Concealed allocation	Groups similar at baseline	Blinding of subjects	Blinding of therapists	Outcome assessor blinded	Adequate follow up (≥85% of participants)	Intention to treat analysis	Between-group analyses	Point measures & variability measures	Total score
Wilson & Herbison [131]	+	+	?	+	÷	÷	?	÷	?	+	+	4/10
Glazener et al [132]	+	+	+	+	÷	÷	+	÷	+	+	+	7/10
Dumoulin et al [133]	+	+	?	+	÷	÷	+	+	÷	+	+	6/10
Sleep & Grant [135]	+	+	?	?	÷	÷	?	+	+	+	+	4/10
Mørkved & Bø [139]	+	÷	÷	+	÷	÷	÷	+	÷	+	+	4/10
Meyer et al [136]	+	?	?	+	÷	÷	?	+	÷	+	+	4/10
Chiarelli & Cockburn [137]	+	+	+	?	÷	÷	+	+	+	+	+	7/10
Ewings et al [138]	+	+	+	+	÷	÷	÷	÷	+	+	+	6/10

PEDro Scale score (ranging from 1-10) [134] for rating methodological quality presented with items and score. Each satisfied item (except the eligibility criteria) contributes 1 point to the total score (range 1-10) . Plus (+) given if criteria was satisfied; minus (÷) if criteria was not satisfied; question mark (?) if not clear whether criteria was satisfied.
Three studies included women with UI (treatment trials) [131-133] . Five trials included women with and without UI [135-139].

Basis for the aims of this dissertation

UI is highly prevalent in the female population [1;6] and one of the most common conditions seen in clinical gynaecological practice [7]. UI may significantly impact the physical, psychological and social well-being of those women affected [8;9]. Vaginal delivery is found to be independently associated with a significant long-term increase in prevalence of UI, regardless of maternal age and number of deliveries [49;62].

Evidence based recommendations (Grade A) propose that women having their first child should be offered supervised PFMT during pregnancy [129;130]. However there is scant knowledge to which degree pregnant women actually are practicing PFMT. There is further paucity on data regarding pregnant women's ability to contract their PFM correctly and sparse clinical data on their vaginal resting pressure, PFM strength and endurance. To our knowledge, only few studies [51;55] have investigated whether pregnant continent women have stronger PFM than incontinent counterparts.

Vaginal delivery is considered to be the main risk factor for weakening of the PFM [75;77;83-88]. However, only three studies [51;68;89] had, to our knowledge, clinically assessed change in PFM strength prospectively from pregnancy to postpartum in relation to mode of delivery prior to the start of this project. Further, UI in pregnant women before and after delivery has been associated with reduced PFM strength, but has been sparsely investigated.

Imaging studies have shown that major LA muscle defects among primiparous women delivering vaginally could appear within the range of 20-36% [83;94]. Impaired PFM function would be likely after such direct trauma to the LA muscle. However, clinical data are sparse on vaginal resting pressure, PFM strength and PFM endurance in women with and without major LA muscle defects. To our knowledge, had only two previous studies investigated PFM strength in women with and without LA muscle defects [98;99].

Current recommendation (Grade A) state that PFMT should be first line treatment for pregnant women and postpartum women with UI [129;130]. However, the evidence base for effect of postpartum PFMT in a "population-based approach" [111] including both women with and without UI is limited. Prior to project start, only four randomized controlled trials [135-138] and one matched controlled trial [139] had investigated the effect of postpartum PFMT, including both women with and without UI. The effect is not clear, and it has been suggested that mixed trials on prevention and treatment might be effective when the intervention is intensive enough [111]. The success of PFMT on UI in women with major defect of the LA muscle is still unknown.

Aims of the dissertation

The overall aim of this dissertation was to study PFM function in nulliparous pregnant women during pregnancy and after childbirth and to evaluate the effect of postpartum PFMT on UI in primiparous women with and without major defects of the LA muscle.

The PFM function variables assessed and studied were: Ability to contract, vaginal resting pressure, PFM strength and PFM endurance.

The specific aims were:

1. To investigate nulliparous pregnant women's knowledge about and practising of PFMT, assess their ability to perform a correct PFM contraction and their PFM function. Further, to compare vaginal resting pressure, PFM strength and PFM endurance in continent women versus women with UI (**Paper I**).

2. To study the impact of childbirth and mode of delivery on PFM function in terms of ability to contract, vaginal resting pressure, and PFM strength and PFM endurance by assessing change from mid-pregnancy to six weeks postpartum. Further, to investigate changes in vaginal resting pressure, PFM strength and PFM endurance from mid-pregnancy to six weeks postpartum in women with and without urinary incontinence (**Paper II**).

3. To assess whether women with major defects of the LA muscle after vaginal delivery are able to contract the PFM correctly. Further, to investigate vaginal resting pressure, PFM strength and PFM endurance six weeks after vaginal delivery in primiparous women with and without major defects of the LA muscle (**Paper III**).

4. To evaluate whether postpartum PFMT decreased the prevalence of UI (any frequency) in primiparous women with and without UI at the time of inclusion (mixed population), and further to perform stratified analyses on women with and without major LA muscle defects (**Paper IV**).

Materials and methods

Study design and sampling

The papers were based on a prospective cohort study **(Paper I-II)** and a RCT **(Paper III-IV)** performed at Akershus University Hospital in collaboration with the Norwegian School of Sport Sciences. The cohort study on 300 nulliparous pregnant women ran from mid–pregnancy to 12 months after delivery and included five assessment points: two during pregnancy and three after delivery (Figure 4). In the period January 2010 until April 2011 all nulliparous pregnant women scheduled for delivery at the hospital were invited to participate in our cohort study. They were invited via a letter sent out together with the written invitation for the regular ultrasound examination at gestational week 18. The RCT evaluating the effect of postpartum PFMT on UI was running from six weeks after delivery (baseline) to six months after delivery (post-intervention), with a follow up at 12 months after delivery (Figure 4). One hundred and seventy-five women were included six weeks after vaginal delivery, 139 women were recruited from the cohort study and 39 women were recruited from the maternity ward at Akershus University Hospital, or from community primary health care clinics within the geographical area of Akershus University Hospital (Figure 4).

The four papers (**I-IV**) had the following study designs and samples:

I. A cross-sectional study of 300 nulliparous pregnant women at mid-pregnancy (gestational week 18-22).

II. A prospective observational study of 277 nulliparous pregnant women followed from mid-pregnancy to six weeks after delivery (then as primiparous women).

III. A cross-sectional study of 175 primiparous women included in a randomised controlled trial (six weeks after vaginal delivery).

IV. A two-armed assessor blinded RCT including primiparous women six weeks after vaginal delivery delivery. The participants (n=175) were stratified on major LA muscle defects, verified by transperinal ultrasound.

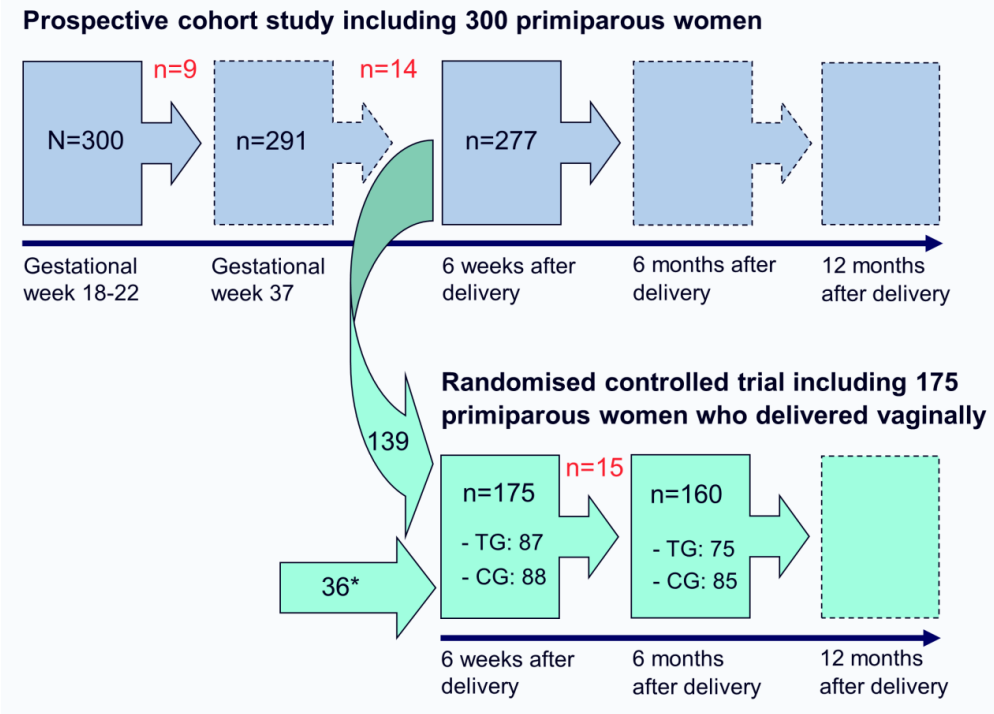

Figure 4. *Outline of the prospective cohort study (blue boxes) and the randomised controlled trial (green boxes) providing data for Paper I-IV. Study design, number of participants, and timing (points in time) of each paper were as follows:*

Paper I: Cross-sectional study, n=300, gestational week 18-22 (blue box with solid line).

Paper II: Prospective observational study, n=277, gestational week 18-22 and six weeks after delivery (blue boxes with solid lines). Gestational week 37 (blue box with broken line) was not included as manometer measurements of PFM function were not performed at this time.

Paper III: Cross-sectional study, n=175, six weeks after delivery (green box with solid line).

Paper IV: Randomised controlled trial, n=175, six weeks after delivery which is baseline, and six months after delivery which is post-intervention, (green boxes with solid lines). The 12 months follow up assessment (green box with broken line) is not included in this dissertation.

TG = Training group; CG = Control group; Red numbers = lost to follow-up.
**Participants recruited from the hospital maternity ward or from community primary health care clinics after giving birth.*

Inclusion criteria - clinical visits

Inclusion criteria

In **Paper I-II** the inclusion criteria were nulliparous pregnant women who were able to speak and understand any Scandinavian language. Exclusion criteria were: Multiple pregnancy, prior abortion /still birth after gestational week 16. In order to attend the study visit at six weeks after delivery in the prospective cohort study (**Paper II**), the women were to have given birth after gestational week 32. Women who experienced stillbirth were excluded.

In **Paper III-IV** we included primiparous women with and without UI who delivered vaginally after more than 32 weeks of gestation, and who were able to speak and understand any Scandinavian language. Women who had multiple pregnancy, or prior abortion / still birth after gestational week 16 were excluded (as in Paper I-II). An additional exclusion criterion for **Paper III-IV** was perineal tearing graded 3b, 3c or 4 during delivery. The rationale for this latter exclusion criterion was that women experiencing these severe perineal tears, including a substantial part of the anal sphincter, are routinely referred to a physiotherapist for PFMT. Ethically, these women could therefore not be randomised to the control group of the RCT.

Clinical visits

Timing of each clinical visit relative to the term of birth is presented in Figure 4. The timing of the three first visits was chosen on the basis of convenience for the participating women, as it could be combined with their routine pregnancy appointment and their routine postpartum appointment.

Sample size

The inclusion of 300 nulliparous pregnant women in the cohort study (Figure 4) was based on power calculations for detecting changes of hiatal dimensions. No power calculation was performed with regard to the expected difference in PFM function (vaginal resting pressure, PFM strength and PFM endurance) in women with and without UI, or for the expected change in PFM function from pregnancy to after childbirth in relation to mode of delivery (**Paper I-II**).

Paper III is a cross-sectional study of the 175 primiparous women included in the RCT (Figure 4). No power calculation was performed for the comparison of PFM function in women with and without major LA muscle defects. However, a substantial reduction in strength is one of the common symptoms following major muscle tears within sports injuries [97]. We therefore expected

that the number of cases with major LA muscle defects planned for in the RCT would provide a sufficient sample.

The power calculation for the randomised controlled trial (**Paper IV**) was based on a previous study performed within a similar setting [139], showing a 67% prevalence reduction of UI in the PFMT group compared to a 34% reduction in the control group, comprising 99 persons in each group. Assuming a similar difference among comparison groups with two-sided significance of < 0.05 and a power of 0.90, a total of 62 women would be required (31 in each group). As we planned for an additional stratified analysis among women with and without major LA muscle defects, and the fact that the effect of PFMT in women with such defects was unknown, the statistical advice was to aim for 80 women with- and 80 women without major LA muscle defects.

Data collection and measurement data

Demographics and other data obtained from electronic questionnaires

The participants received electronic questionnaires in conjunction with all clinical visits (Figure 4). Demographical data such as age, civil status and educational level were collected at their first visit. At their first visit they were asked retrospectively about pre-pregnancy weight, pre-pregnancy smoking, and pre-pregnancy UI. Prospectively we collected data about smoking, general physical activity, PFMT and UI from their first visit and onwards (Figure 4). Participants who were included six weeks after delivery were also asked retrospectively regarding status of the above items at mid-pregnancy. The project coordinator ensured that questionnaires were sent out at the right time and also that participants answered them. It was emphasized that the clinical visits at all five points in time (Figure 4) were done within a low variation of time.

Weight was measured at all visits for assessment of BMI (kg/m^2).

The above data items or a selection of them were used in descriptive statistics for **Paper I-IV**. Pre-pregnancy BMI was also used as co-variable in the regression analysis set up in **Paper II** and in **Paper III**.

Obstetric data

Data on delivery mode and other obstetric data were collected from the hospital's electronic birth records. Delivery mode was classified as: normal vaginal delivery, instrumental vaginal delivery (vacuum or forceps) and caesarean section (elective or emergency). Epidural analgesia was coded

"yes" or "no", with "yes" as continuous infusion with the possibility of top-ups. Duration of second stage of delivery was defined as the time-interval between full cervical dilatation and delivery of the child.

Delivery mode was the exposure (independent variable) in **Paper II**. Further, when investigating the role of obstetric variables on the change in PFM function from mid-pregnancy to six weeks after delivery, normal vaginal delivery was used as the reference delivery mode both for caesarean section and for instrumental assisted vaginal delivery, whereas length of total second stage > 60 min, the use of epidural, fetal birth weight, fetal head circumference and pre-pregnancy BMI were used as covariates in the regression model.

In **Paper III**, when investigating PFM function six weeks after delivery in women with and without major defects of the LA muscle, we controlled our findings for possible covariates by setting up a regression model with the following covariates: Instrumental assisted vaginal delivery, total second stage > 60 min, fetal birth weight, and pre-pregnancy BMI.

The selection of possible covariates was based on their correlation with the dependent variables, previous literature, and clinical judgement.

Ability to contract the pelvic floor muscles

At the first clinical visit, a physiotherapist gave all participants an individual teaching session in pelvic floor anatomy. They further received thorough instruction, feedback and practice in how to perform a correct PFM contraction. A PFM contraction without any movement of the pelvis or visible contraction of the glutei-, hip- or abdominal muscles was emphasised [118;119]. A correct contraction was defined as inward movement and squeeze around the urethra, vagina, and rectum [23;118;119], and was assessed by observation and palpation. Ability to contract the PFM was assessed by two trained physiotherapists. The clinical examinations were performed with the participant in a standardised supine crook lying position. Assessments were performed at mid-pregnancy and at all assessment points after delivery, but not at gestational week 37 (Figure 4). Descriptive statistics on women's ability to contract are included in all papers.

PFM function (vaginal resting pressure, PFM strength and PFM endurance)

Vaginal resting pressure, PFM strength and PFM endurance were measured by using an air filled vaginal balloon catheter (balloon size 6.7 x 1.7 cm) connected to a high precision pressure transducer (Camtech AS, Sandvika, Norway). At atmospheric pressure the vaginal balloon was set

to 0 cmH_2O for each subject before it was placed into the vagina. The middle of the balloon was positioned 3.5 cm inside the introitus [140]. Vaginal resting pressure was measured with the balloon positioned in the vagina without any voluntary PFM activity. PFM strength was measured as the difference between vaginal resting pressure and the squeeze pressure obtained at maximal voluntary contraction (Figure 5), and was reported as the mean of three maximal voluntary contractions. The method has been found to be reliable and valid if used with simultaneous observation of inward movement of the perineum/catheter during the contraction [118;119]. PFM endurance was defined as a sustained maximal contraction, and was quantified during the first 10 seconds as the area below the measurement curve (integral calculation) [141]. To minimise biases, the assessors (two physiotherapists) were trained ahead of the study and a rigorous protocol in standards of procedures was maintained. We aimed for high inter-rater agreement. Inter-observer values between the two investigators were calculated, and an intra-class correlation coefficient > 0.9 with no systematic differences between assessors was reached on eight independent datasets, both for vaginal resting pressure, PFM strength and PFM endurance. Assessors were blinded for current continence status (**Paper I-IV**) and for obstetric data (**Paper II, III, IV**).

Vaginal resting pressure, PFM strength and PFM endurance were primary outcome variables (dependent variables) when comparing women with and without UI in **Paper I and II**, when investigating the impact of mode of delivery **(Paper II),** and when comparing women with and without major LA muscle defects in **Paper III**. However, in **Paper IV** these PFM variables were so-called intervening causal variables ("mediators") acting on the cause-effect pathway between the intervention and outcome [142].

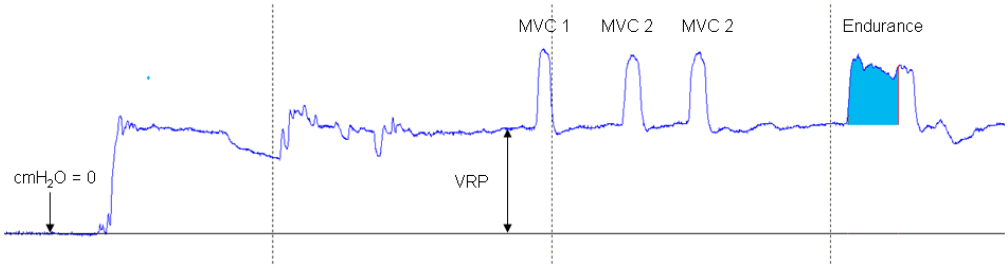

Figure 5. *Manometer measurements: VRP; PFM strength, measured as the mean of three maximal voluntary contractions (MVCs 1–3); and PFM endurance, measured as one sustained maximal contraction quantified during 10 seconds (integral calculation). From BJOG 2013;120(11):1423-29; Hilde G et al.; DOI: 10.1111/1471-0528.12321.*

Urinary incontinence (UI)

The International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form (ICIQ UI SF), applicable both for clinical practice and research developed by Avery et al [143] was used in this project. The ICIQ UI SF items encompass frequency of UI, the amount UI, its impact on quality of life (bother scale from 1-10), and the type of UI. The questionnaire is validated [143] and the Norwegian version of ICIQ-UI SF (Appendix 4) has undergone testing for linguistic validation and was found to be adequate for use [144;145]. Descriptive statistics on UI (frequency, amount, bother, and type) were performed in **Paper I**. Any leakage of UI (any frequency) versus no leakage of urine, obtained from the question "How often do you leak urine", was used when classifying the women as continent or incontinent. This dichotomization of UI was used to establish comparison groups in **Paper I and II**. Further, dichotomisation of UI (prevalence of UI at any frequency) was used as the dependent variable in **Paper IV** when evaluating the effect of postpartum PFMT.

The power calculation for the RCT (**Paper IV)** was based on such dichotomization of UI. Further, dichotomization of UI (prevalence of UI at any frequency) is primary outcome in Cochrane reviews on PFMT when pooling outcome data on UI.

The ICIQ UI SF was included in the electronic questionnaire sent out in conjunction with the clinical visits.

Pad test

A secondary outcome on UI in **Paper IV** was assessed by a pad test described and used by Mørkved and Bø [139]. After voiding, the women drank one litre of water. Thirty minutes later they wore a pre-weighted pad and performed a stress test as follows:

- Jumping up and down with maximal intensity for 30 seconds.
- Jumping with the legs in alternate abduction and adduction (Jumping Jacks) with maximal intensity for another 30 seconds.
- Coughing as hard as possible three times.

As in the study by Mørkved and Bø [139], a positive pad-test was set to a cut-off of 2 gram of leakage.

Assessment of major LA muscle defects

Tomographic imaging using three- and four-dimensional (3D/4D) transperineal ultrasound was used for diagnosing major defects of the LA muscle. This imaging technique made it possible to stratify for such defects in the RCT evaluating the effect of postpartum PFMT (**Paper IV**), and to compare PFM function in women with and without major LA muscle defects six weeks after delivery (**Paper III**). Two trained gynaecologists performed the transperineal ultrasound assessment by using the GE Kretz Voulson E8 (GE Healthcare AS, Oslo, Norway) with a 4-8 MHz curved array 3D/4D ultrasound transducer (RAB4-81/obstetric). The 3D/4D ultrasound volumes were acquired with the women in the same testing position as for the manometer measurements. Participants were asked to perform three attempts of maximal PFM contraction, and all three contractions were recorded. At the very end of the clinical visit, the acquired volume showing the best contraction was used for LA muscle defect assessment. This was defined as the volume with the largest reduction of the anterior-posterior diameter of the levator hiatus during maximal contraction. Identification of major LA muscle defects was then assessed by using tomographic imaging of the axial plane. The plane of minimal hiatal dimensions of the levator hiatus, defined as the plane with the shortest anterior-posterior diameter from the posterior-inferior margin of the symphysis to the rectal sling in the midsagittal plane, was used as the reference plane. Tomographic slices were obtained at 2.5 mm slice intervals from 5 mm caudally to 12.5 mm cranially to this reference plane producing eight slices [146;147]. Major defect of the LA muscle was diagnosed when an abnormal insertion of the muscle toward the pubic bone were present in all three central slices (Figure 6) as suggested by Dietz et al [146;147]; at the plane of minimal dimension and 2.5 mm and 5.0 mm cranially to it. Slices were scored as positive or negative for major LA muscle defects by direct visualization of the muscle attachment. In doubtful cases, measurement of the levator-urethral gap was used, with measurements > 2.5 cm regarded as abnormal [147]. Both investigators were trained gynaecologists with experience in 3D/4D transperineal ultrasound. Stær-Jensen et al [148] found good to very good intra- and inter-rater reliability for detecting major LA muscle defects shortly after childbirth in primiparous women when using the tomographic imaging method described above.

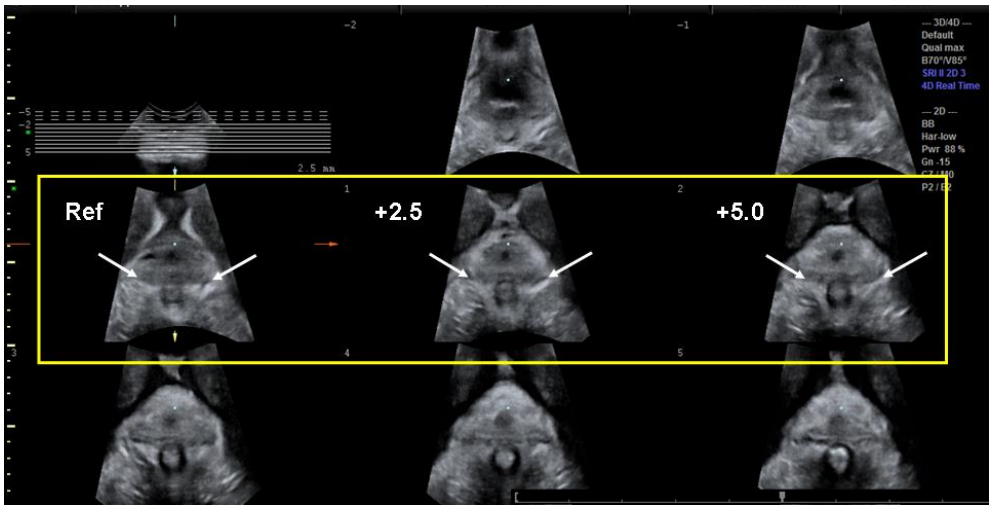

Figure 6. *Major bilateral defect of the levator ani (LA) muscle 6 weeks after delivery. Tomographic ultrasound in the axial plane of the levator hiatus, obtained with a 2.5-mm slice interval, from 5 mm caudally to 12.5 mm cranially. Major LA defect visualised as abnormal insertion (arrows) present in all three central slices (slices shown within yellow border). From BJOG 2013;120(11); Hilde G et al 2013; DOI: 10.1111/1471-0528.12321.*

Randomisation procedure and blinding of assessors

After the assessment of LA muscle defects being present or not, the participants were randomised into two groups in blocks of ten (**Paper IV**). The randomisation sequence was computer generated and thereafter concealed by opaque sealed envelopes. Allocation of participants to PFMT or control was administered outside the clinical room by the project coordinator (midwife). This procedure made blinding of outcome assessors (physiotherapists and gynaecologists) possible, and they were kept blinded for group allocation throughout the whole study.

Intervention

After randomisation, the training group received an exercise intervention for a period of 16 weeks (**Paper IV**). The exercise intervention protocol is described in detail by Bø et al 1990 [149;150] and by Mørkved and Bø [139]. The training group participants attended a supervised exercise class led by an experienced physical therapist once a week. PFMT was performed in different positions (lying, standing, kneeling and sitting) with legs abducted (Figure 7). Sets of 8-12, close to maximum PFM contractions aiming at a holding time of 6-8 seconds was performed, with an

additional 3-4 fast contractions added on top of the holding period [139;149;150]. Between the sets of PFM contraction, body awareness, breathing, relaxation, and strength exercises for the abdominal, back, arm and thigh muscles were performed to music. Additionally, the training group was prescribed to perform daily PFMT at home (three sets of 8-12 repetitions; close to maximum contractions). The training protocol is based on strength training recommendations [13;116]. An additional booklet and a DVD (www.corewellness.no) on PFMT were given to the exercise group for home training. Training adherence at home was recorded in a training diary as in Mørkved & Bø [139], whereas the physical therapist recorded group session adherence. Training participants were continuously motivated by the physical therapist to keep up their adherence to training classes and home training, and high performance during training was strongly emphasised. Beyond the customary leaflet (received from the postnatal ward) and the thorough initial instruction on how to contract correctly, the control group participants received no further intervention. They were not discouraged from doing PFMT on their own.

Figure 7. *Pelvic floor muscle training was performed in different positions with legs apart. With permission from Vitacon (norsk.vitacon.com)*

Statistics

Demographic and other descriptive variables were presented as means with standard deviations (SD) or as frequencies with percentages in all papers (**Paper I-IV**). For all four papers the level of statistical significance was set to < 0.05. Independent samples t-test and chi-square test were used to evaluate demographic differences between comparison groups (**Paper I-IV**).

Paper I: Independent samples t-test was used to analyse differences between continent women and incontinent women in PFM function (vaginal resting pressure, PFM strength and PFM endurance).

Paper II: Paired sample t-test was used to investigate change in PFM function (vaginal resting pressure, PFM strength and PFM endurance) from mid-pregnancy to six weeks after delivery within each delivery mode group (caesarean section, normal vaginal delivery, and instrumental assisted vaginal delivery). Differences between delivery modes in PFM function were analysed by One-way between groups analysis of variance. Standard multiple linear regression analysis was used to investigate the role of demographic and obstetric variables on the observed change of PFM measurements. One-way between groups analysis of variance was also used when analysing PFM function in women with and without UI.

Paper III: Independent samples t-test was used to analyse differences between women with major LA defects and women without major LA defects in PFM function (vaginal resting pressure, PFM strength and PFM endurance). Standard multiple and standard simple linear regression analysis was applied to control findings in PFM measurements for possible covariates.

Paper IV: Mantel-Haenszel relative risk analysis was used to evaluate between-group differences on prevalence of self-reported UI (any frequency) and prevalence of a positive pad test.

When analysing between-group differences on continuous data of urinary leakage obtained from women with a pad test > 2 g, the Mann-Whitney U test was used as these data were not normally distributed.

Paired sample t-test (within-group) and independent samples t-test (between-groups) on the PFM function variables (vaginal resting pressure, PFM strength; PFM endurance) were performed in order to investigate within-group change and between-groups differences in these intervening causal variables ("mediators") [142].

Intention to treat was the principal analysis. Missing values for continuous data were imputed by using the baseline value plus added change observed in the corresponding control group. For categorical data (self-reported UI) the approach of "last observation carried forward" was used. The overall analysis included the total study sample. In addition, stratified analyses for the stratum of women with major LA muscle defects and the stratum of women without such defects were performed. A "per protocol analysis" was also carried out, in which drop-outs, training participants with an exercise adherence ≤ 80%, and participants with a new pregnancy at the clinical visit six months after delivery were excluded.

Data in all four papers were analysed using SPSS software version 15 (SPSS Inc, Chicago, IL). Review Manager 5.1 was used for the Mantel-Haenszel relative risk analysis in **Paper IV**.

Ethics

- The cohort study in Figure 4, providing data for **Paper I-II** was approved by the Regional Committees for Medical Research Ethics (REK South East 2009/170) (Appendix 1) and the Norwegian Social Science Data Services (2799026) (Appendix 1), and registered at ClinicalTrials.gov (NCT01045135).
- The RCT in Figure 4, providing data for **Paper III-IV** was approved by the Regional Committees for Medical Research Ethics (REK South East 2009/289a) (Appendix 2) and the Norwegian Social Science Data Services (2799004) (Appendix 2), and registered at ClinicalTrials.gov (NCT01069484).
- All participants gave written informed consent before entering the above mentioned studies (Appendix 3).
- The ethical standards of WMA Declaration of Helsinki - Ethical Principles for Medical Research Involving Human Subjects [151] were followed.

Main results

Paper I

The cross-sectional study at mid-pregnancy (gestational week 18-22) involved 300 nulliparous pregnant women with a mean age of 28.7 years (SD=3.9). Of the 300 women, 88.7% had heard of PFMT at mid-pregnancy, 35% of them performed PFMT ≥ once a week, and 15% ≥ three times per week. The most frequently reported sources of information were leaflets/magazines/newspapers, followed by midwife/nurse, friend, physiotherapist, fitness class, physician, DVDs, and finally antenatal class. After thorough instruction, the assessment of ability to contract the PFM showed that 12 of the 300 women (4.0%) were unable to contract the PFM correctly, of whom ten were straining. Thirty-five percent (104 of 300) reported UI at gestational week 18–22; 27.0% reported to leak once a week or less, 4.3% two to three times per week, 1.3% once a day, and 2.0% several times per day. For the women reporting UI at any frequency (n=104), mean score on the ICIQ-UI-SF bother scale (ranging from 0-10) was 1.2 (SD1.6), with 43 of the 104 incontinent women scoring zero. PFMT once a week or more was reported by 48% of the 104 women reporting UI, and by 28% of the 196 continent women. Corresponding figures for regular PFMT three times or more per week were 21% and 12% respectively. Women continent for urine had significantly higher PFM strength and PFM endurance when compared with women having UI, with mean differences of 6.6 cmH_2O (95% CI: 2.3 to 10.8, p=0.003), and 41.5 cmH_2Osec (95% CI: 9.8 to 73.1, p=0.010), respectively. No between-group difference was found for vaginal resting pressure (2.3 cmH_2O, 95% CI: 0.0 to 4.6, p=0.054).

Paper II

The prospective study following nulliparous pregnant women from mid-pregnancy to six weeks after delivery counted 277 women with a mean age of 28.7 years (SD=4.3). Mean gestational week at the first study visit was 21 (SD 1.4), ranging from gestational week 17-25. At the study visit after delivery, the mean postpartum week was 6.2 (SD 1.0), ranging from 3-11 weeks postpartum. Eleven of 277 women (3.9%) did not contract the PFM correctly at mid-pregnancy. The corresponding number six weeks after delivery was 12 of 277 (4.3%). Five women who were able to contract their PFM correctly at mid-pregnancy had lost the ability to contract after delivery, whereas four women unable to contract correctly at mid-pregnancy had learned to contract correctly at the postpartum visit.

Of the 277 women, 193 (69.7%) had a normal vaginal delivery, 45 (16.2%) had an instrumental assisted vaginal delivery (41 with vacuum and 4 with forceps), and 39 (14.1%) delivered by caesarean section (29 emergency, 10 elective). The women who had elective caesarean section were excluded from further analysis of PFM function. Within the group of women with emergency caesarean section, the only parameter that changed significantly from mid-pregnancy to six weeks after delivery was vaginal resting pressure, which was reduced by 10% ($p=0.003$). Within the group of women with normal vaginal delivery, the vaginal resting pressure was reduced by 29%, PFM strength by 54% and endurance by 53% from mid-pregnancy to six weeks after delivery ($p<0.001$ for all measures). Within the group of women with instrumental vaginal delivery, the vaginal resting pressure was reduced by 30%, PFM strength by 66%; and endurance by 65% ($p<0.001$ for all measures).

The One-way between group analysis of variance, showed that women who delivered vaginally had a significantly larger reduction of vaginal resting pressure, PFM strength and PFM endurance from mid-pregnancy to six weeks after delivery than women who delivered by caesarean section. Comparison between women with caesarean section and women with normal vaginal delivery showed a mean difference in change from mid-pregnancy to six weeks after delivery of -7.9 cmH_2O in vaginal resting pressure (95% CI: -11.7 to -4.1, $p<0.001$), -14.5 cmH_2O in PFM strength (CI: -20.7 to -8.2, $p<0.001$), and -100.5 cmH_2Osec in PFM endurance (95% CI: -151.8 to -49.2, $p<0.001$). Comparison between women with caesarean section and women with instrumental vaginal delivery showed a mean difference in change of -9.4 cm H_2O in vaginal resting pressure (95% CI: -13.9 to -4.8, $p<0.001$), -18.5 cmH_2O in PFM strength (95% CI: -26.0 to -11.0, $p<0.001$), and -128.7 cmH_2Osec in PFM endurance (95%CI: -190.0 to -67.4, $p<0.001$). Results showed no significant differences when comparing the group of women with normal vaginal delivery versus the group of women with instrumental assisted vaginal delivery. The multiple linear regression analysis investigating the role of age, pre-pregnancy BMI, length of second stage > 60 min, the use of epidural, fetal birth weight, and head circumference, showed that delivery mode was the most important factor for changes in PFM variables.

Four different groups with UI were compared when investigating change in PFM function in women with and without UI: Women who were continent at both mid-pregnancy and 6 weeks after delivery (n=122) vs. women with de novo UI six weeks after delivery (n=48) vs. women with UI at mid-pregnancy but continent six weeks after delivery (n=36) vs. women who had UI at both points in time. Between group comparisons (One-way between group analysis of variance) showed no significant differences in change from mid-pregnancy to six weeks after

delivery (all comparisons), neither for vaginal resting pressure (p>0.05), PFM strength (p>0.05), nor PFM endurance (p>0.05). Results showed that women continent for urine both at mid-pregnancy and six weeks after delivery had significantly higher PFM strength and better endurance than counterparts with UI at both points in time: When comparing these two groups, the mean difference in PFM strength was 9.4 cmH_2O (p=0.006) at mid-pregnancy, and 6.8 cmH_2O at six weeks postpartum (p=0.006). For PFM endurance the mean difference was 64.9 cmH_2Osec (p=0.013) at mid-pregnancy, and 48.1 cmH_2Osec (p=0.010) six weeks after delivery.

Paper III

The cross-sectional study six weeks after delivery (mean 6.1 weeks; SD 0.9 weeks; range 4-9 weeks) had a study sample of 175 primiparous women who had delivered vaginally; 139 women were recruited from the ongoing cohort study, and 39 were recruited after childbirth (Figure 4). Their mean age was 29.8 years. Eighty percent of the women (140 of 175) had a normal vaginal delivery, and 20% (35 of 175) had an instrumental vaginal delivery (33 with vacuum and 2 with forceps). Our study sample had 55 women diagnosed with major LA muscle defects, and 120 women with no major defects.

After thorough instruction, feedback, and practise on how to contract correctly, seven of the 175 women (4%) were not able to contract the PFM correctly, four of them had major LA muscle defects. Women with major LA muscle defects (n=55) had 47% lower PFM strength and 47% lower PFM endurance compared to women without major LA muscle defects (n=120). When comparing women without versus women with major LA muscle defects the mean difference for strength was 7.5 cmH_2O (95% CI: 5.1 to 9.9, P < 0.001), and for PFM endurance 51.2 cmH_2Osec (95% CI: 32.8 to 69.6, P < 0.001). No between group difference was found for vaginal resting pressure (P < 0.670). In the multiple linear regression analysis, adjustments were made for instrumental vaginal delivery, total second stage > 60 min, infant birth weight and pre-pregnancy body mass index (BMI). Results showed that adjusted and crude unstandardized regression coefficients for major LA muscle defects were similar, which support the robustness of the estimates for mean differences in PFM measures presented above.

Paper IV

The participants included in the RCT (n=175) were the same as in Paper III (Figure 4). The number of participants and the number of women with major defects in each arm (PFMT group and control group) is shown in Figure 8. The level of high education (college/university) was significantly higher in the control group (81.7%) than in the PFMT group (73.6%); p=0.01. No significant between-group differences were found for other demographics (age, BMI, civil status), level of regular general physical activity, or regular PFMT (p>0.05).

Seven of the 175 women (4%) were not able to contract the PFM correctly at baseline; four of them were allocated to the training arm (three having major LA muscle defects), and three to the control arm (one having major LA muscle defects). At the post-intervention test six months after delivery 15 women (8.6%) were lost to follow up; 12 (13.8%) from the PFMT group, and three (3.4%) from the control group (Figure 8).

Home training diaries and the exercise class attendance records of the training group participants completing the trial, showed that 96% (72 of 75) reached an adherence level of 80%, both for class sessions and for daily home training. Training adherence in the control group was not registered through training diaries. However, when asked retrospectively through the post-test questionnaire about a weekly average of PFMT during the intervention period, 16.5% of the control participants reported PFMT three times or more per week.

The percentage of women with UI (any frequency) at baseline was 39.1% in the training group and 50.0% in the control group; the between-group difference was not statistically significant. At the post-intervention test (six months after delivery), the UI prevalence was 34.5% in the training group and 38.6% in the control group; the between-group difference was not significant (RR 0.89, 95% CI: 0.60-1.32, p=0.57). Similar non-significant between-group differences were found in the subgroup analyses for the major LA muscle defect stratum (RR 0.89, 95% CI: 0.51-1.56, p=0.68) and for the no major defect stratum (RR 0.90, 95% CI: 0.53-1.52, p=0.70). Pad test results showed no significant between-group differences (p>0.05). The "per protocol analysis" did not alter these results. A total of 12 women developed UI during the study period (self-reported UI); seven from the training group (one with and six without major LA muscle defects), and five from the control group (three with and two without major LA muscle defects).

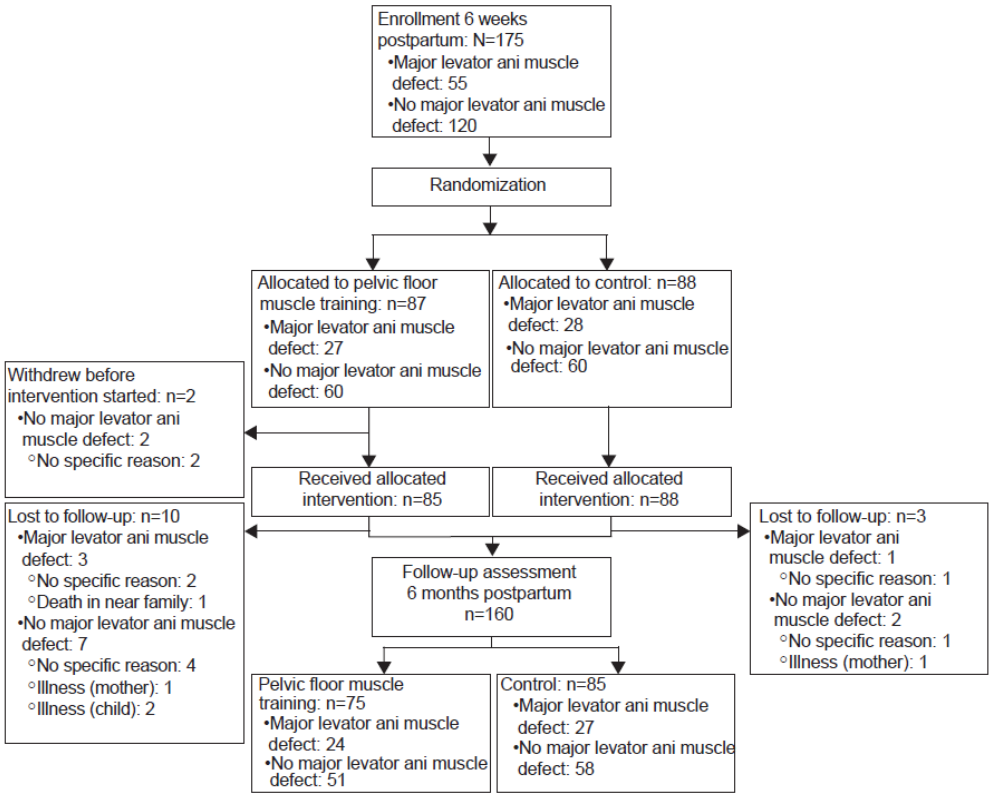

Figure 8. *Flowchart of participants trough each stage of the randomised trial. From Obstet Gynecol 2013;122:1231–8; Hilde G et al; DOI: 10.1097/AOG.0000000000000012*

Discussion

Summary of main findings

The vast majority of the 300 pregnant women included in the cohort study knew about PFMT at mid-pregnancy. Overall, one third of the pregnant women performed PFMT once a week or more. Among women reporting UI (n=104), 48% performed PFMT once a week or more. Continent pregnant women at mid-pregnancy had significantly higher PFM strength and endurance when compared to pregnant women with UI at mid-pregnancy. For women who underwent caesarean section, no changes in PFM strength and endurance from mid-pregnancy to six weeks after delivery were found, but a significant and pronounced reduction in all PFM measurements was found for women who had delivered vaginally. Women who were continent at both mid-pregnancy and six weeks after delivery had significantly higher PFM strength and endurance than their counterparts being incontinent at both points in time. Cross-sectional data six weeks after delivery, showed that women with major LA muscle defects (n=55) had pronounced lower PFM strength and endurance than women without major defects (n=120), but no difference in vaginal resting pressure was found. In the RCT, the results showed no significant effect of postpartum PFMT on UI prevalence (any frequency) six months after vaginal delivery. Stratified analysis of women with and without major LA muscle defects showed similar non-significant results.

Methodological considerations

Study design

The first three papers in this dissertation were observational studies, two of them had a cross-sectional design (**Paper I and III**), and one had a prospective cohort design (**Paper II**). **Paper IV** was an experimental study with a randomised controlled design.

Paper I was descriptive in nature. We wanted to investigate whether nulliparous pregnant women knew about PFMT, what their sources for knowledge about PFMT were, and whether they performed PFMT. Further we assessed their ability to contract, their PFM function (vaginal resting pressure, PFM strength and PFM endurance), and finally compared PFM function in women with and without UI. A cross-sectional design is well suited for such purposes. However, it is important to note that a cross-sectional study design such as in **Paper I** provides a

"snapshot" at one point in time, which means that the temporal relationship between PFM strength and UI is unclear, and provides limited evidence of causation [152;153].

In **Paper II** we prospectively followed nulliparous pregnant women from mid-pregnancy to six weeks after delivery, and investigated the impact of delivery mode on vaginal resting pressure, pelvic floor muscle strength and PFM endurance. However, each of these variables may be influenced by other variables as well [154]. A standard multiple linear regression analysis was therefore applied in order to investigate the role of demographic and obstetric variables on the change of PFM, this analysis adds strength to the study. Vaginal delivery turned out to have the most significant impact on PFM function, but PFM strength and endurance was also affected by a second stage lasting more than 60 minutes.

In the cross-sectional study six weeks after delivery (**Paper III**), comparing women with and without major LA muscle defects, a standard multiple regression analysis was used to control our findings for possible covariates. The similarities found between adjusted and crude unstandardized regression coefficients support the robustness of our reported estimates for mean differences on vaginal resting pressure, PFM strength and PFM endurance. The cross-sectional design may present a limitation as women with and without major LA may have differed with respect to PFM function already before childbirth. This is elaborated when discussing results from Paper III.

Participants in in the RCT (**Paper IV**) were randomly allocated into two groups. The randomisation sequence was computer generated and concealed (opaque envelopes). A random allocation of participants to treatment or control intervention ensures that allocation happens by chance alone. This type of allocation is considered vital for the internal validity of clinical trials, as this procedure protects against selection bias and increases the likelihood of an equal distribution of confounding factors in the groups being compared [155]. The randomised controlled design is therefore considered to provide the highest level of evidence when assessing the effect of an intervention [156]. The use of sealed opaque envelopes is an additional important procedure as this protects against manipulation of the randomisation sequence [157]. In a systematic review by Kunz et al [158] it was shown that non-randomised trials and randomised trials with inadequate allocation concealment tend, on average, to result in larger estimates of effect when compared to randomised trials with proper allocation concealment. We had an imbalance between comparison groups on reported UI at baseline with a higher UI prevalence in the control group than in the PFMT group, this may represent a limitation. However, this difference was not statistically

significant. Blinding of the outcome assessors for group allocation adds an important strength to our study, as such blinding protects against detection bias [155;157]. Blinding or masking the participants and therapists, making them unaware of the treatment assignments, would protect against performance bias [155;157], but is of course not possible in exercise trials. High drop-out rate also represents a threat to the internal validity in clinical trials [157;159]. In our trial, the drop-out rate was less than 15%, which is acceptable [134;159]. However, the drop-out in our trial might not be random, as 12 women dropped out from the training group, but only three from the control group. Intention to treat analysis, with imputation of lost outcome data, was the principal analysis. This adds strength to the study by keeping the groups similar apart from random variation [160].

Assessment methods

Choice of assessment tools to be used in research should be judged on their responsiveness, reliability and validity [161].

Urinary incontinence (UI)

Assessment of UI was used as descriptive statistics in **Paper I**, to establish comparison groups of "no UI" versus "UI of any frequency" in **Paper I-II**, and as outcome (UI prevalence of any frequency) in **Paper IV**.

ICIQ UI SF is the recommended instrument for self-report of UI [162], and is used in **Paper I-II, IV**. The ICIQ UI SF is considered to be a brief and robust questionnaire to assess the symptoms and impact of UI, and has shown good construct validity, acceptable convergent validity, as well as good reliability in terms of stability and internal consistency [143]. It has further been shown that this questionnaire can be easily completed with very low levels of missing data.

A pad-test was used as a secondary outcome for UI in the RCT (**Paper IV**). The applied pad-test did not have a standardised bladder volume, this represents a limitation because of variation in initial urine load [163]. The randomised design and blinding of outcome assessors, do however, protect against systematic bias for this secondary outcome of UI.

Pelvic floor muscle function

The PFM function variables studied in in this dissertation were vaginal resting pressure, PFM strength and PFM endurance. These variables were in all four papers assessed by a squeeze pressure device, a vaginal balloon catheter, connected to a pressure transducer with high precision (Camtech AS, Sandvika, Norway). The method has been found to be reliable and valid if used with simultaneous observation of inward movement of the perineum/catheter during the contraction [118;119]. To minimise biases, the two assessors were trained ahead of the study and a rigorous protocol in standards of procedures was kept. The protocol involved cautious teaching of the participants in not using the glutei-, hip- or abdominal muscles when voluntary contracting their PFM, as this could alter the manometer assessments due to increased abdominal pressure [119;164]. Further, we used a standardized test position and the participants were given standardized instructions ahead of and during assessment of PFM strength and PFM endurance [124].

Measurements of squeeze pressure (manometer) is a common method, both in clinical practice and research, to measure PFM strength and endurance [124]. Dynamometers is an alternative assessment method, and may have an advantage over pressure transducers as they measure force directly [165], however they are not commercially available.

Involuntary PFM contraction during an increase of abdominal pressure (automatic function), e.g. during a cough, is an important aspect of PFM function to be assessed, but is not included in this dissertation. In our cohort study and our RCT, we performed transperinal ultrasound assessment of automatic PFM function during "huff" manoeuvre (fast maximal expiration) [166] at all clinical visits. However, these ultrasound data have yet to be analysed and published.

Levator ani muscle defect

The use of 3D/4D transperineal ultrasound imaging has recently become more widely available. This imaging method is regarded as an important research tool for assessment of morphology and functions of the PFM, and can be performed with minimal discomfort to the patient and at little cost [167]. This imaging technique has proven to correlate well with MRI findings both when assessing hiatal dimensions of the LA muscle [168;169] and when diagnosing major LA muscle defects [170]. Tomographic imaging using transperineal 3D/4D ultrasound has shown high-retest agreement when assessing major defects of the LA muscle [93;102]. Further, a recent reliability study by Stær-Jensen et al [148], performed within this project, showed high intra- and inter-rater reliability for this imaging method also when diagnosing major LA muscle defects shortly after childbirth.

However, discriminating between major LA defects and haematoma might in some cases be challenging shortly after childbirth [171], this represents a limitation as it may cause false positives.

Blinding of assessors

All assessors (two physiotherapists, two gynaecologists) were blinded to the participants' continence status (reported via the electronic questionnaire) and obstetric data, this blinding adds strength to all the papers.

Variation in time around clinical visits

In general we achieved a low variation of time around each clinical visit which represents an added strength for our studies. The actual timing of the clinical visits during pregnancy and after childbirth is of relevance, because of the expected altered pelvic floor function during pregnancy, and the expected gradual natural remission after delivery [44].

Selection of subjects

In the period January 2010 until April 2011 all nulliparous pregnant women scheduled for delivery at Akershus University Hospital were invited to participate in our study. The study sample in the cohort study providing data for **Paper I-II** and in the study sample in the RCT providing data for **Paper III-IV** were comparable to the total population of nulliparous pregnant women scheduled for delivery at Akershus University Hospital (n=2 621) with respect to age and civil status. However, the study participants had a higher level of education, which limits generalization of our results. Seventy-five % of the women included in **Paper I-II** had a college or university education versus 51% in the total population scheduled for delivery at our hospital, and the corresponding percentages for **Paper III-IV** were 82% versus 51%. Furthermore, the inclusion criterion regarding language does represent a selection of participants and limits the generalisation of our results. Only women who were able to speak and understand Scandinavian languages were included. It is estimated that 1/6 of the 2 621 nulliparous pregnant women scheduled for delivery at Akershus University Hospital during the inclusion period were not eligible due to the language criterion.

Knowledge and practicing of pelvic floor muscle training (Paper I)

The vast majority in this study knew about PFMT at mid-pregnancy. Of the included nulliparous pregnant women 105 of 300 (35%) reported to perform PFMT once a week or more. This figure is within the range found in other cross-sectional studies, where reported proportions of PFMT during pregnancy (once a week or more) range from 16-58% [172-176]. Of the pregnant women reporting UI, 48% performed PFMT once a week or more, and 21% performed PFMT three times per week or more. These low numbers of reported PFMT may reflect the low bother score reported (ICIQ-UI-SF) and that women seem to tolerate some UI as part of being pregnant. Further, most women may not know that antenatal UI could increase the risk of UI postpartum.

Relatively few of the pregnant women reported health personnel as being their source of information on PFMT. This may reflect the fact that supervised PFMT is not a significant part of regular antenatal care service [177]. It may further reflect that pelvic floor risks and efficacy of PFMT is less counselled than factors such as weight gain, high blood pressure, preterm labour, etc. [178]. Results from RCTs summarised in the Cochrane review by Hay-Smith et al [111] and later updated by Boyle et al [179] have provided Level I evidence for the recommendation of antenatal PFMT [129;130]. According to the recommendation given by the 4th International Consultation on Incontinence of 2009 [130], women having their first child should be offered supervised PFMT and intensive antepartum PFMT to prevent postpartum UI (Grade A recommendation). This recommendation has been retained in the 5th International Consultation on Incontinence of 2013 [180]. However, it takes time to translate research findings into daily practice, and research on guideline dissemination and implementing strategies has shown that changing practice is not an easy task [181;182]. Some health care providers may also be hesitant about implementing antenatal PFMT as they believe PFMT may make the PFM too strong and less elastic, resulting in a prolonged second stage of labour [183]. However, this myth has been contradicted by three RCTs [184-186] and a large cohort study [175] showing that antenatal PFMT neither prolonged the second stage nor obstructed labour. In addition to the above evidence, prospective cohort data from our project published by Bø et al [187] showed that strong PFM were not disadvantageous for vaginal delivery.

Not knowing how to perform PFM contractions may be a barrier for performing PFMT [172]. Examination by palpation showed that 4% of the women (12 out of 300) were unable to contract their PFM correctly even after thorough instruction and practice including vaginal palpation and feedback. Our low number of incorrect PFM contraction is in line with Mørkved et al [55], but in contrast to studies reporting ratios ≥ 30% [23;120-122]. One explanation for the low number in our

study and in the study by Mørkved et al [55] may be the inclusion of nulliparous pregnant women only, whereas studies reporting high ratios of incorrect contractions included parous women with pelvic floor complaints. Parous women are likely to have an increased risk of pelvic floor trauma which may affect their ability to contract the PFM correctly. Another explanation is that studies differ with regard to the degree of teaching before the final assessment of the ability to contract. In our study and in the study by Mørkved et al [55] the participants were given several attempts and guidance before the registration of ability to contract. After giving a brief verbal instruction, Bump et al [122] found that 12 out of 47 women (25%) were straining instead of contracting correctly. In our study, 10 out of 300 women were straining after thorough instruction and clinical guidance. Such findings support the view that clinical assessment of the ability to contract is important before starting a PFMT programme [125;172;177].

Our findings showed low numbers of women performing regular PFMT during pregnancy which is in accordance with previous studies. This indicates that current evidence-based recommendations (Grade A) on supervised PFMT during pregnancy need to be better implemented in antenatal health care. Additionally, general fitness classes during pregnancy should include PFMT on a regular basis.

Childbirth and pelvic floor muscle function (Paper II and III)

Muscle fibres might be stretched up to three times their resting length as the fetal head is crowning [90], and nerves innervating the LA muscle might exhibit a strain of 35% [79]. Further, major LA muscle defects among primiparous women delivering vaginally have been shown to appear within 13-36% of the women [83;94;188]. It is therefore not surprising that measurements on vaginal resting pressure, PFM strength and PFM endurance six weeks postpartum were significantly influenced by mode of delivery (**Paper II**), and further that differences were seen when comparing women with and without major LA muscle defects (**Paper III**). However, before initiation and start of the current study project (January 2010), few studies had presented clinical data on vaginal resting pressure, PFM strength and PFM endurance in relation to delivery mode and major LA muscle defects.

Mode of delivery (Paper II)

Until the start of this project, a PubMed search revealed three previous studies assessing change in PFM strength prospectively from pregnancy to postpartum [51;68;89]. During the project period three additional prospective studies have to our knowledge been published [189-191]. Except for the

study by Caroci et al [190] and Meyer et al [68] counting 226 and 149 nulliparous pregnant women respectively, the sample sizes of the above-cited studies were small, ranging from 20 to 75 participants. When assessing PFM function, the above-cited studies used either digital palpation [51;89;189;190], manometer [68;89;190;191] or electromyography [189]. Five of these studies report significantly decreased PFM strength after vaginal delivery in primiparous women [51;68;89;189;191], whereas Caroci et al [190] report no such strength reduction. None of the above-mentioned studies found any significant decline in PFM strength after caesarean section. Botelho et al [189] actually reported increased PFM contractility when comparing antenatal values (3rd trimester) versus values 45 days after caesarean section (p=0.003).

To our knowledge the study presented in **Paper II**, is one of the largest studies (n=277) presenting prospective data on vaginal resting pressure, PFM strength and endurance from pregnancy to postpartum on women delivering their first child. However, low number of emergency caesarean section (n=29) and instrumental vaginal deliveries (n=45) may represent a limitation. This is also a challenge for comparable studies, with caesarean group sizes ranging from 5 to 37 women [51;68;89;189-191]. Another limitation for our study could be that the women were examined in mid-pregnancy, but not again closer to delivery. A possible contribution of changes late in pregnancy can therefore not be taken into account. However, we found no changes in PFM strength and endurance within the emergency CS group, indicating that the impact of late pregnancy events on outcome measures seems unlikely. A lack of a priori power calculation is a weakness and might weaken this statement as the CS group is small.

In **Paper II** we chose to discuss and compare our findings with the four studies using manometer measurements (vaginal squeeze pressure) when assessing PFM function [68;89;190;191]. Our results showed that PFM strength was not significantly changed in the group of women who delivered by caesarean section, but significantly and pronounced in the group of women who delivered vaginally. Our findings are in contrast to results reported by Caroci et al [190] for women delivering vaginally, but they are in line with results reported by Peschers et al [89], Meyer et al [68] and Sigurdardottir et al [191]. Although these three latter studies and our study all showed significant and marked decrease in PFM strength for primiparous women delivering vaginally, the reported percentual decrease in strength differs. One explanation could be the use of different squeeze pressure apparatus making direct comparison difficult [124;192]. A possible variation in study samples with respecet to demographics and and obstetric variables could also explain some of the variation. Difference in PFM measurements from pregnancy to after childbirth can be influenced by a "testing effect" obtained from first- to second assessment. To minimize this effect, we

emphasized a thorough teaching on how to contract correctly before the final PFM assessment at the first clinical visit. We do not know how this was handled in the above mentioned studies. Finally, the difference in timing for clinical assessment during pregnancy (i.e. mid-pregnancy or late pregnancy) as well as length of recovery after childbirth, before the second assessment, may influence PFM change measurements. This assumption is supported by Peschers et al [89] as they also measured PFM strength shortly after delivery (2-8 days) and found a significant improvement from this point in time to 6-10 weeks postpartum.

To which extent the PFM recover in the first postpartum year is of great interest, as well as whether high quality postpartum PFMT would add more than natural recovery alone. Elenskaia et al [193] included 403 pregnant women (182 nulliparous and 221 multiparous), followed them from the second trimester to one year postpartum, and assessed PFM function by using manometer. They found that PFM strength recovered completely at one year postpartum in both primiparous and multiparous women irrespective of delivery mode. Additional studies are needed to see whether their findings can be confirmed or not.

Levator ani muscle defects (Paper III)

This cross-sectional study six weeks after delivery provides data at one point in time, which may represent a limitation. One may question whether primiparous women with and without major LA muscle defects, after their first vaginal delivery, also differed in vaginal resting pressure, PFM strength and PFM endurance before delivery. As 139 of the included women in Paper III were recruited from the cohort study that started at mid-pregnancy, we had the opportunity to look into antenatal PFM measurements in 139 cases (44 with and 95 without major LA defects at six weeks postpartum). An independent samples t-test performed on these 139 women showed no significant differences in antenatal PFM measurements when comparing women who were later diagnosed either with or without major LA defects: neither for vaginal resting pressure ($p=0.745$), PFM strength ($p=0.836$) nor PFM endurance ($p=0.399$).

A PubMed search before starting this project revealed two previous studies assessing PFM strength in women with and without major LA muscle defects [98;99]. Two recent studies have been published during our project period [194;195]. Our results, showing significant reduction in PFM strength in women with major LA muscle defect when compared to women without major defect, are in line with all four studies mentioned above [98;99;194;195]. Dynamometer was used for assessment of PFM strength in two of these four studies [98;195], digital palpation in one study [99], and transperineal ultrasound in one study [194]. Dietz et al [196] further found that women with major

LA muscle defects reported perceiving weakness of the PFM. As in our study, Brincat et al [195] included primiparous women only. Brincat et al [195] also found a difference between groups for vaginal resting pressure (p=0.03), which is in contrast to our findings. A direct comparison between our study and the study by Brincat et al [195] is however difficult, as the time span for remission differs. We assessed the PFM function six weeks after delivery, whereas Brincat et al [195] performed the assessments 9-12 months after delivery. Haematoma early after delivery in combination with major LA defect might explain why we in contrast to Brincat et al [195] found no group differences for vaginal resting pressure. A direct comparison may further be limited by the use of different methods when assessing PFM strength.

Training is believed to be important in speeding up tissue healing [97;108], and early active rehabilitation is standard treatment after muscle injury within sports medicine. Most women with major LA defects in the present study were able to contract the PFM. This also indicates a potential capacity for non-injured muscle fibres to compensate for loss in muscle strength even early after delivery. The success of PFMT for women with major muscle defects in the pelvic floor, was to our knowledge not known when publishing **Paper III**, but was planned for in our project protocol and investigated in our RCT. The results of PFMT for women with major LA muscle defects with UI as the primary outcome are presented in **Paper IV**.

Pelvic floor muscle function and UI (Paper I and II)

The cross-sectional study at mid- pregnancy (**Paper I**) showed that nulliparous pregnant women continent for urine had significantly higher PFM strength and endurance than those who reported urine leakage. Our results are in line with cross-sectional findings reported by two previous studies on nulliparous pregnant women [51;55]. Sampselle [51] included 20 nulliparous pregnant women at gestational week 32-36, followed them to six weeks after delivery, and assessed PFM strength by digital palpation. In the cross-sectional study by Mørkved et al [55], 103 nulliparous pregnant women at gestational week 20 were included and their PFM strength was assessed by using manometer.

Other cross-sectional studies on different study samples than ours [53;54;56-59] and one case-control study [52] have shown divergent results on urinary continence status and PFM strength. Direct comparison of study results is limited due to sample heterogeneity e.g. parity, definition and assessment of UI, and method used when assessing PFM strength.

In **Paper II** we only found a significant difference in PFM strength and endurance when comparing those who were continent for urine at both points in time (mid-pregnancy and six weeks after delivery) versus those who were incontinent at both points in time. Our results are in line with Sampselle [51]. Findings indicate that PFM strength and endurance are of importance for staying continent during pregnancy and childbirth, and that there might be some carryover effect of antenatal PFM strength with regards to continence status [51]. Our comparison groups in **Paper II** were similar with respect to age, pre-pregnancy BMI, and regular antenatal PFMT. However, the picture of possible risk factors in the development or maintenance of UI is complex [7;31;32]. Other confounders or covariates not accounted for might represent a limitation and interpretation of results must be done with caution.

Effect of postpartum pelvic floor muscle training on UI (paper IV)

As stated in the Cochrane review by Hay-Smith [111] and also in the updated version by Boyle et al from 2012 [179], it is possible that postpartum PFMT in a mixed population of parous women with and without UI might be effective when the intervention is intensive enough. One strength of the present RCT (**Paper IV**), assessing the effect of postpartum PFMT, is the use of a training protocol based on strength training recommendations [13;116;197], and skilled physical therapists supervising the group training sessions. This PFMT protocol has proved to be successful in previous studies; both in treatment trials of UI [149;150], in prevention trials of UI [198;199], and in a mixed trial on prevention and treatment of UI [139]. To our knowledge, this is the first study performing stratified analysis of the effect of postpartum PFMT on UI prevalence in women with and without major LA muscle defects. The statistical advice was to aim for 80 women in the stratum with such defects, but we managed to include only 55, this may represent a limitation for the subgroup analyses. In general our effect estimates have wide confidence intervals, due to the rather optimistic effect size planned for. However, as the between-group differences were minimal or non-existent, a type 2 error is unlikely. Limitation with regards to generalizability is discussed above (under general methodological considerations), however, an additional limitation for generalizability of our overall analyses in this RCT (n=175) may be that the present study had more women with major LA muscle defects when compared to the general primiparous population at Akershus University Hospital.

Our findings, showing no significant effect of postpartum PFMT on prevention and treatment of UI, are in line with three previous RCTs [135;136;138], but in contrast to the RCT by Chiarelli and Cockburn [137] and the matched controlled study by Mørkved and Bø [139].

Chiarelli & Cockburn [137] evaluated efficacy of adherence strategies and the "Health belief model", and found a significant effect in favour of an intervention containing two educational practice sessions led by a physical therapist and a booklet to promote PFMT. They included 820 women, and in contrast to our study their participants were primiparous and multiparous women, and they included only women with vacuum-assisted delivery whose babies had a birth weight above 4000g.

The pelvic floor intervention in the present study was the same as the intervention applied in the study by Mørkved and Bø [139], but the findings are surprisingly different. Findings from their study give a relative risk (RR) on UI of 0.50 in favour of the PFMT group (95% CI: 0.28, 0.89), which is a statistically significant and considerably strong effect, but the confidence limits are wide. Control groups in both the present study and the study by Mørkved and Bø [139] reported to perform PFMT during the intervention period. Despite this, Mørkved and Bø [139] found significant effect between groups, while we did not. A direct comparison of results is limited by differences in study design. Our study has a randomised and assessor-blinded design, included only primiparous women, and assessed UI by the ICIQ UI SF. Mørkved and Bø [139] had a matched controlled design, their study was not assessor-blinded, they included a mix of primiparous and multiparous women, and assessed UI through a structured interview. Additional differences were number of drop-outs: In our study 12 women dropped out from the training group and three from the control group, whereas Mørkved and Bø [139] had no drop-outs. Further, our study most likely has more women with major LA muscle defects due to the inclusion of two strata (55 with major defects and 120 without).

No differences in PFM vaginal resting pressure, PFM strength or PFM endurance were found between comparison groups, either at baseline or at post-intervention. No interventional effect on PMF strength and endurance was surprising, as the intervention was based on strength training recommendations and a high adherence in the training group was achieved. It is important to bear in mind that the controls in this study had the same thorough instructions in how to contract the PFM correctly as the training participants, which may have served as a strong incentive to perform PFMT among the controls. In addition, most people are disappointed when they are motivated to participate in a training study, but are randomised to a control group. Controls in our RCT may therefore have exercised more than they would have done following usual care, and they might represent women who are more eager to train than eligible women who said no to participate in our RCT on postpartum PFMT.

An UI prevalence of 34.5% in the PFMT group and 38.6% in the control group must be considered as high six months after delivery. One possible explanation might be an increased general physical activity level from six weeks after delivery to six months after delivery. We expected that supervised PFMT would counteract this, but were not able to show this in the present study. Another explanation for the high UI prevalence may be impairment of the urethral sphincter system. According to DeLancey [200] more research is needed to better understand and evaluate the relative importance of the urethral supportive system (PFM interacting with the supportive ligaments and fasciae) and the sphincter system (external and internal sphincter muscles and vascular elements within the submucosa) in relation to urinary incontinence.

Our results on UI prevalence blend in with the results from previous RCTs on postpartum PFMT including women with and without UI (mixed prevention and treatment trials). According to Clarke et al [201], clinical trials should begin and end with systematic reviews of the relevant evidence available. The review by Hay-Smith et al [111] was the basis and incentive for executing the current RCT. During the project period, the review by Hay-Smith et al [111] was updated by Boyle et al [179], but with the same mixed prevention and treatment trials as earlier (no new studies were published). In the forest plot below (Figure 9), our trial results on UI are pooled with the results from the three previous RCTs reporting UI in the mid-postnatal period as outcome [135;137;138]. The pooled effect when our study results are included gives a RR of 0.98 (95% CI: 0.81 to 1.19), which is literally the same relative risk as the one reported by Boyle et al (RR 1.00 (95% CI: 0.79 to 1.26) [179].

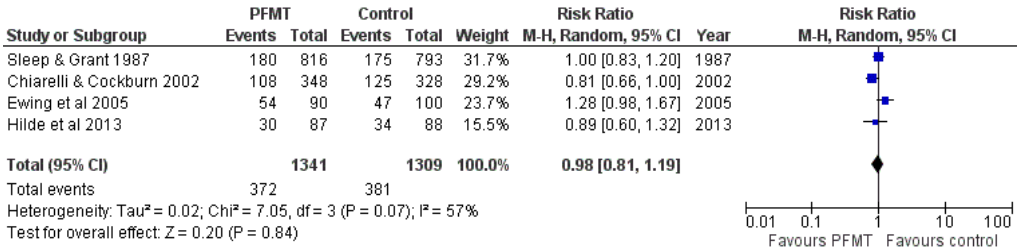

Figure 9. *Pooled effect of postpartum PFMT for prevention and treatment of incontinence, based on randomised controlled trials reporting urinary incontinence in the mid-postnal period as outcome*

Trials on postpartum PFMT including women with and without UI (prevention and treatment trials) seem to be less successful than trials aiming either at prevention or treatment. Future trials should therefore probably be more targeted towards certain groups of women. An individually supervised exercise intervention might be more successful than a class-based intervention when targeting, for instance, women with major muscle defect, poor pelvic floor muscle function, or more severe UI.

Conclusions

Based on the results presented in **Paper I-IV** the following conclusions can be drawn:

1. Most nulliparous pregnant women knew about PFMT, but only 35% performed PFMT once a week or more. Incontinent nulliparous pregnant women had weaker PFM than their continent counterparts. More emphasis on information of PFM function and PFMT is warranted during pregnancy.

2. Pronounced reductions in vaginal resting pressure, PFM strength, and PFM endurance were found from mid-pregnancy to six weeks after delivery for women who delivered vaginally. Women continent for urine both at mid-pregnancy and six weeks after delivery had stronger and more endurant PFM than their counterparts being incontinent at both points in time.

3. Primiparous women with major LA defects after vaginal delivery had pronounced lower PFM strength and endurance than women without major defects. However, most women with major LA defects were able to contract the PFM. This indicates a potential capacity by non-injured muscle fibres to compensate for loss in muscle strength even at an early stage after delivery.

4. Postpartum PFMT did not decrease UI prevalence six months after delivery in primiparous women. Stratified analysis on women with and without major LA muscle defects showed similar non-significant results.

Further research

- There is a need for further research to examine clinical guideline implementation on pelvic floor exercises and the adherence of recommendations. There is a need for studies exploring possible barriers for guideline implementation both during pregnancy and after childbirth. Such barriers may give valuable knowledge in the development of more effective implementation strategies. Such strategies should also address coaches and fitness instructors offering fitness classes during pregnancy and after childbirth.

- To which extent the PFM recover in the first postpartum year is of great interest, and further whether postpartum PFMT of high quality would add more than natural recovery alone. Clinical data presenting data on the recovery of vaginal resting pressure, PFM strength and PFM endurance is still limited. The study by Elenskaia et al (2011) showed that PFM strength recovered completely at one year postpartum in both primiparous and multiparous women irrespective of delivery mode. Additional studies are needed to see whether their findings can be confirmed or not.

- Involuntary PFM contraction during an increase of abdominal pressure (automatic function), e.g. during a cough, is an important aspect of PFM function. There is a need for studies assessing automatic function and its change during pregnancy, change from pregnancy to after childbirth, and change throughout the first year postpartum. Future RCT on PFMT should also incorporate assessment of automatic function when evaluating the effect of PFMT.

- Mixed prevention and treatment trials on postpartum PFMT seem to be less successful than trials aiming either at prevention or treatment. Future trials should therefore probably be more targeted towards certain groups of women. An individual supervised exercise intervention might be more successful than a class-based intervention when targeting for instance women with major muscle defect, poor pelvic floor muscle function, or more severe UI.

References

(1) Ashton-Miller JA, Delancey JO. Functional anatomy of the female pelvic floor. Ann N Y Acad Sci 2007; 1101:266-296.

(2) Index Mundi. World Birth rate. http://www.indexmundi.com/world/birth_rate.htm Online source [accessed Oct 7th 2013].

(3) Materna Medical Inc. Clinical Need. http://www.maternamedical.com/clinical-need.html Online source [accessed Oct 7th 2013] . 2011.

(4) Norwegian Institute of Public Health. Medical Birth Registry. http://mfr-nesstar.uib.no/mfr/ Online source [accessed Oct 7th 2013] . 2013.

(5) Handa VL, Harris TA, Ostergard DR. Protecting the pelvic floor: obstetric management to prevent incontinence and pelvic organ prolapse. Obstet Gynecol 1996; 88(3):470-478.

(6) 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. Paris: Health Publication Ltd, 2009: 35-111.

(7) Bump RC, Norton PA. Epidemiology and natural history of pelvic floor dysfunction. Obstet Gynecol Clin North Am 1998; 25(4):723-746.

(8) Norton PA, MacDonald LD, Sedgwick PM, Stanton SL. Distress and delay associated with urinary incontinence, frequency, and urgency in women. BMJ 1988; 297(6657):1187-1189.

(9) Hunskaar S, Vinsnes A. The quality of life in women with urinary incontinence as measured by the sickness impact profile. J Am Geriatr Soc 1991; 39(4):378-382.

(10) Bø K, Hagen R, Kvarstein B, Larsen S. Female stress urinary incontinence and participation in different sport and social activities. Scandinavian J Sports Sci 1989;(11):117-121.

(11) Brown WJ, Miller YD. Too wet to exercise? Leaking urine as a barrier to physical activity in women. J Sci Med Sport 2001; 4(4):373-378.

(12) Nygaard I, Girts T, Fultz NH, Kinchen K, Pohl G, Sternfeld B. Is urinary incontinence a barrier to exercise in women? Obstet Gynecol 2005; 106(2):307-314.

(13) Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA et al. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Med Sci Sports Exerc 2007; 39(8):1423-1434.

(14) Olsen AL, Smith VJ, Bergstrom JO, Colling JC, Clark AL. Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol 1997; 89(4):501-506.

(15) 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 Churchill livingstone, 2005: 1357-1371.

(16) DeLancey JO. Structural aspects of the extrinsic continence mechanism. Obstet Gynecol 1988; 72(3 Pt 1):296-301.

(17) DeLancey JO. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol 1994; 170(6):1713-1720.

(18) Shafik A, Doss S. Surgical anatomy of the somatic terminal innervation to the anal and urethral sphincters: role in anal and urethral surgery. J Urol 1999; 161(1):85-89.

(19) Grigorescu BA, Lazarou G, Olson TR, Downie SA, Powers K, Greston WM et al. Innervation of the levator ani muscles: description of the nerve branches to the pubococcygeus, iliococcygeus, and puborectalis muscles. Int Urogynecol J Pelvic Floor Dysfunct 2008; 19(1):107-116.

(20) DeLancey JO. The anatomy of the pelvic floor. Curr Opin Obstet Gynecol 1994; 6(4):313-316.

(21) Kearney R, Sawhney R, Delancey JO. Levator ani muscle anatomy evaluated by origin-insertion pairs. Obstet Gynecol 2004; 104(1):168-173.

(22) Norton PA. Pelvic floor disorders: the role of fascia and ligaments. Clin Obstet Gynecol 1993; 36(4):926-938.

(23) 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.

(24) 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):167-174.

(25) 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):153-160.

(26) 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.

(27) 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: Churchill Livingstone Elsevier, 2007: 119-132.

(28) Miller JM, Perucchini D, Carchidi LT, DeLancey JO, Ashton-Miller J. Pelvic floor muscle contraction during a cough and decreased vesical neck mobility. Obstet Gynecol 2001; 97(2):255-260.

(29) 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):870-874.

(30) 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):773-782.

(31) 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):1460-1470.

(32) 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):610-615.

(33) 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.

(34) 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 June 26-29,2004. Plymouth, UK: Health Publication Ltd, 2005: 255-312.

(35) 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):473-480.

(36) 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):230-234.

(37) Minassian VA, Stewart WF, Wood GC. Urinary incontinence in women: variation in prevalence estimates and risk factors. Obstet Gynecol 2008; 111(2 Pt 1):324-331.

(38) 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.

(39) 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):400-402.

(40) Mørkved S, Bo K. Prevalence of urinary incontinence during pregnancy and postpartum. Int Urogynecol J Pelvic Floor Dysfunct 1999; 10(6):394-398.

(41) 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):278-283.

(42) Wesnes SL, Rortveit G, Bo K, Hunskaar S. Urinary incontinence during pregnancy. Obstet Gynecol 2007; 109(4):922-928.

(43) 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):863-872.

(44) 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. Paris: Helarh Publicationa Ltd, 2009: 255-330.

(45) 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):266-273.

(46) 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.

(47) 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):154-161.

(48) 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):359-362.

(49) Viktrup L, Rortveit G, Lose G. Risk of stress urinary incontinence twelve years after the first pregnancy and delivery. Obstet Gynecol 2006; 108(2):248-254.

(50) Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. J Intern Med 1997; 241(1):71-79.

(51) Sampselle CM. Changes in pelvic muscle strength and stress urinary incontinence associated with childbirth. J Obstet Gynecol Neonatal Nurs 1990; 19(5):371-377.

(52) 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 Invest 1996; 41(4):269-274.

(53) 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):208-215.

(54) 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):668-674.

(55) 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):384-389.

(56) 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):624-630.

(57) 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):1917-1922.

(58) 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):1028-1032.

(59) 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):171-174.

(60) Hvidman L, Foldspang A, Mommsen S, Nielsen JB. Postpartum urinary incontinence. Acta Obstet Gynecol Scand 2003; 82(6):556-563.

(61) 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):923-927.

(62) 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):873-878.

(63) 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):419-425.

(64) 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):1004-1010.

(65) Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. Obstet Gynecol 2006; 107(6):1253-1260.

(66) Press JZ, Klein MC, Kaczorowski J, Liston RM, von DP. Does cesarean section reduce postpartum urinary incontinence? A systematic review. Birth 2007; 34(3):228-237.

(67) Peschers U, Schaer G, Anthuber C, Delancey JO, Schuessler B. Changes in vesical neck mobility following vaginal delivery. Obstet Gynecol 1996; 88(6):1001-1006.

(68) 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):613-618.

(69) Dietz HP, Clarke B, Vancaillie TG. Vaginal childbirth and bladder neck mobility. Aust N Z J Obstet Gynaecol 2002; 42(5):522-525.

(70) 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):138-146.

(71) King JK, Freeman RM. Is antenatal bladder neck mobility a risk factor for postpartum stress incontinence? Br J Obstet Gynaecol 1998; 105(12):1300-1307.

(72) 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; 16:381-382.

(73) 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):535-540.

(74) 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.

(75) Snooks SJ, Setchell M, Swash M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet 1984; 2(8402):546-550.

(76) 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):24-28.

(77) Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol 1990; 97(9):770-779.

(78) 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):1358-1360.

(79) 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):1669-1676.

(80) Snooks SJ, Swash M, Henry MM, Setchell M. Risk factors in childbirth causing damage to the pelvic floor innervation. Int J Colorectal Dis 1986; 1(1):20-24.

(81) Sultan AH, Kamm MA, Hudson CN. Pudendal nerve damage during labour: prospective study before and after childbirth. Br J Obstet Gynaecol 1994; 101(1):22-28.

(82) 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.

(83) 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.

(84) 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):144-149.

(85) Dietz HP. Pelvic floor trauma following vaginal delivery. Curr Opin Obstet Gynecol 2006; 18(5):528-537.

(86) 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: 93-105.

(87) Ashton-Miller JA, Delancey JO. On the biomechanics of vaginal birth and common sequelae. Annu Rev Biomed Eng 2009; 11:163-176.

(88) 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):1186-1195.

(89) Peschers UM, Schaer GN, Delancey JO, Schuessler B. Levator ani function before and after childbirth. Br J Obstet Gynaecol 1997; 104(9):1004-1008.

(90) 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.

(91) 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.

(92) 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.

(93) Dietz HP. Quantification of major morphological abnormalities of the levator ani. Ultrasound Obstet Gynecol 2007; 29(3):329-334.

(94) Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. Obstet Gynecol 2005; 106(4):707-712.

(95) 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):91-97.

(96) 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):1175-1181.

(97) 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):317-331.

(98) 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.

(99) Dietz HP, Shek C. Levator avulsion and grading of pelvic floor muscle strength. Int Urogynecol J Pelvic Floor Dysfunct 2008; 19(5):633-636.

(100) Shek KL, Dietz HP. The effect of childbirth on hiatal dimensions. Obstet Gynecol 2009; 113(6):1272-1278.

(101) 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):141-144.

(102) Dietz HP, Steensma AB. The prevalence of major abnormalities of the levator ani in urogynaecological patients. BJOG 2006; 113(2):225-230.

(103) Dietz HP, Simpson JM. Levator trauma is associated with pelvic organ prolapse. BJOG 2008; 115(8):979-984.

(104) 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):239-245.

(105) 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):967-972.

(106) Bodine-Fowler S. Skeletal muscle regeneration after injury: an overview. J Voice 1994; 8(1):53-62.

(107) Lieber RL. Skeletal muscle response to injury. In: Lieber RL, editor. Skeletal muscle structure, function, and plasticity. Lippincott Williams & Wilkins, 2002: 287-341.

(108) Jarvinen TA, Jarvinen TL, Kaariainen M, Kalimo H, Jarvinen M. Muscle injuries: biology and treatment. Am J Sports Med 2005; 33(5):745-764.

(109) 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):158-159.

(110) Lovering R. Physical therapy and related interventions. In: Tiidus PM, editor. Skeletal muscle damage and repair. Champaign, IL: Human Kinetics, 2007: 219-230.

(111) Hay-Smith J, Morkved S, Fairbrother KA, Herbison GP. Pelvic floor muscle training for prevention and treatment of urinary and faecal incontinence in antenatal and postnatal women. Cochrane Database Syst Rev 2008;(4):CD007471.

(112) Kegel AH. Progressive resistance exercise in the functional restoration of the perineal muscles. Am J Obstet Gynecol 1948; 56(2):238-248.

(113) Bø K. Pelvic floor muscle training is effective in treatment of female stress urinary incontinence, but how does it work? Int Urogynecol J Pelvic Floor Dysfunct 2004; 15(2):76-84.

(114) Peschers UM, Vodusek DB, Fanger G, Schaer GN, DeLancey JO, Schuessler B. Pelvic muscle activity in nulliparous volunteers. Neurourol Urodyn 2001; 20(3):269-275.

(115) Bø K. Pelvic floor muscle exercise for the treatment of stress urinary incontinence: An exercise physiology perspective. Int Urogynecol J 1995; 6:282-291.

(116) DiNubile NA. Strength training. Clin Sports Med 1991; 10(1):33-62.

(117) Åstrand PO, Rodahl K, Dahl AA, Strømme SB. Physical training. Textbook of work physiology: Physiological bases of exercise. Windsor: Human Kinetics, 2003: 313-368.

(118) Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. I. Reliability of vaginal pressure measurements of pelvic muscle strength. Neurourol Urodyn 1990; 9:471-477.

(119) Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. II. Validity of vaginal pressure measurements of pelvic muscle strength- The necessity of supplementary methods for control of correct contraction. Neurourol Urodyn 1990; 9:479-487.

(120) Benvenuti F, Caputo GM, Bandinelli S, Mayer F, Biagini C, Sommavilla A. Reeducative treatment of female genuine stress incontinence. Am J Phys Med 1987; 66(4):155-168.

(121) Bø K, Larsen S, Oseid S, Kvarstein B, Hagen R, Jørgensen J. Knowledge about and ability to correct pelvic floor muscle exercises in women with urinary stress incontinence. Neurourol Urodyn 1988; 7:261-262.

(122) Bump RC, Hurt WG, Fantl JA, Wyman JF. Assessment of Kegel pelvic muscle exercise performance after brief verbal instruction. Am J Obstet Gynecol 1991; 165(2):322-327.

(123) Messelink B, Benson T, Berghmans B, Bo K, Corcos J, Fowler C et al. Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence Society. Neurourol Urodyn 2005; 24(4):374-380.

(124) Bø K, Sherburn M. Measurement of pelvic floor muscle function and strength and pelvic organ prolapse: Vaginal squeeze pressure measurement. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-Based Physical Therapy for the Pelvic Floor. Edinburgh: Churchill Livingstone Elsevier, 2007: 63-68.

(125) Bø K, Mørkved S. Pelvic floor and exercise science: Motor learning. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-Based Physical Therapy for the Pelvic Floor. Edinburgh: Churchill Livingstone Elsevier, 2007: 113-119.

(126) Kraemer WJ, Ratamess NA. Fundamentals of resistance training: progression and exercise prescription. Med Sci Sports Exerc 2004; 36(4):674-688.

(127) Hay-Smith EJ, Bo Berghmans LC, Hendriks HJ, de Bie RA, van Waalwijk van Doorn ES. Pelvic floor muscle training for urinary incontinence in women. Cochrane Database Syst Rev 2001;(1):CD001407.

(128) Hay-Smith EJ, Dumoulin C. Pelvic floor muscle training versus no treatment, or inactive control treatments, for urinary incontinence in women. Cochrane Database Syst Rev 2006;(1):CD005654.

(129) National Institute for Health and Clinical Exellence. Urinary incontinence: the management of urinary incontinence in women, Guideline 40. London, UK: National Institute for Health and Clinical Excellence, 2006.

(130) Hay-Smith J, Berghmans BBK, Dumoulin C, Hagen S, Moore K, N'Dow J et al. Adult Conservative Management. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence: 4th International Consultation on Incontinence. Paris: Health Publication Ltd, 2009: 1025-1120.

(131) Wilson PD, Herbison GP. A randomized controlled trial of pelvic floor muscle exercises to treat postnatal urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct 1998; 9(5):257-264.

(132) Glazener CM, Herbison GP, Wilson PD, MacArthur C, Lang GD, Gee H et al. Conservative management of persistent postnatal urinary and faecal incontinence: randomised controlled trial. BMJ 2001; 323(7313):593-596.

(133) Dumoulin C, Lemieux MC, Bourbonnais D, Gravel D, Bravo G, Morin M. Physiotherapy for persistent postnatal stress urinary incontinence: a randomized controlled trial. Obstet Gynecol 2004; 104(3):504-510.

(134) Maher CG, Sherrington C, Herbert RD, Moseley AM, Elkins M. Reliability of the PEDro scale for rating quality of randomized controlled trials. Phys Ther 2003; 83(8):713-721.

(135) Sleep J, Grant A. Pelvic floor exercises in postnatal care. Midwifery 1987; 3(4):158-164.

(136) Meyer S, Hohlfeld P, Achtari C, De GP. Pelvic floor education after vaginal delivery. Obstet Gynecol 2001; 97(5 Pt 1):673-677.

(137) Chiarelli P, Cockburn J. Promoting urinary continence in women after delivery: randomised controlled trial. BMJ 2002; 324(7348):1241.

(138) Ewings P, Spencer S, Marsh H, O'Sullivan M. Obstetric risk factors for urinary incontinence and preventative pelvic floor exercises: cohort study and nested randomized controlled trial. J Obstet Gynaecol 2005; 25(6):558-564.

(139) Mørkved S, Bo K. The effect of postpartum pelvic floor muscle exercise in the prevention and treatment of urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct 1997; 8(4):217-222.

(140) Bø K. Pressure measurements during pelvic floor muscle contractions: the effect of different positions of the vaginal measuring device. Neurourol Urodyn 1992; 11:107-113.

(141) Brækken IH, Majida M, Ellstrom EM, Holme IM, Bo K. Pelvic floor function is independently associated with pelvic organ prolapse. BJOG 2009; 116(13):1706-1714.

(142) Bauman AE, Sallis JF, Dzewaltowski DA, Owen N. Toward a better understanding of the influences on physical activity: the role of determinants, correlates, causal variables, mediators, moderators, and confounders. Am J Prev Med 2002; 23(2 Suppl):5-14.

(143) Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P. ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn 2004; 23(4):322-330.

(144) International Consultation on Incontinence Modular Questionnaire (ICIQ). ICIQ-UI Short Form. http://www.iciq.net/ICIQ-UIshortform.html Online source [accessed Aug 5th 2009].

(145) Abrams P, Avery K, Gardener N, Donovan J. The International Consultation on Incontinence Modular Questionnaire: www.iciq.net. J Urol 2006; 175(3 Pt 1):1063-1066.

(146) Dietz HP, Shek KL. Tomographic ultrasound imaging of the pelvic floor: which levels matter most? Ultrasound Obstet Gynecol 2009; 33(6):698-703.

(147) Dietz HP, Bernardo MJ, Kirby A, Shek KL. Minimal criteria for the diagnosis of avulsion of the puborectalis muscle by tomographic ultrasound. Int Urogynecol J 2011; 22(6):699-704.

(148) Staer-Jensen J, Siafarikas F, Hilde G, Braekken IH, Bo K, Engh ME. Pelvic floor muscle injuries 6 weeks post partum-an intra- and inter-rater study. Neurourol Urodyn 2013; 32(7):993-997.

(149) Bø K, Hagen RH, Kvarstein B, Jørgensen J, Larsen S. Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. III. Effects of two different degrees of pelvic floor muscleexercises. Neurourol Urodyn 1990; 9:489-502.

(150) Bø K, Talseth T, Holme I. Single blind, randomised controlled trial of pelvic floor exercises, electrical stimulation, vaginal cones, and no treatment in management of genuine stress incontinence in women. BMJ 1999; 318(7182):487-493.

(151) World Medical Association. WMA Declaration of Helsinki - Ethical Principles for Medical Research Involving Human Subjects. 2008 http://www.wma.net/en/30publications/10policies/b3/ Online source [accessed Oct 7th 2013].

(152) Grimes DA, Schulz KF. An overview of clinical research: the lay of the land. Lancet 2002; 359(9300):57-61.

(153) Mann C. Observational research methods. Research design II: cohort, cross sectional, and case-control studies. Emerg Med J 2003; 20:54-60.

(154) Altman DG. Relation between several variables. Practical statistics for medical research. London: Chapmann & Hall, 1992: 325-361.

(155) Altman DG. Clinical trials. Practical statistics for medical research. London: Chapman & Hall, 1992: 440-474.

(156) Howick J, Chalmers I, Glasziou P, Greenhalgh T, Heneghan C, Liberati A et al. OCEBM Levels of Evidence Working Group. "The Oxford 2011 Levels of Evidence". Oxford Centre for Evidence-Based Medicine, 2011. http://www.cebm.net/index.aspx?o=5653. Online Source [accessed Oct 10th 2013].

(157) Higgins JPT, Altman DG, Sterne JAC. Assessing risk of bias in included studies: Introduction to sources of bias in clinical trials. In: Higgins JPT, Green S, editors. Cochrane Handbokk for Systematic Reviews of Interventions. The Cochrane Collaboration, www.cochrane-handbook.org, 2011.

(158) Kunz R, Vist G, Oxman AD. Randomisation to protect against selection bias in healthcare trials. Cochrane Database Syst Rev 2007;(2):MR000012.

(159) Herbert RD. Chritical appraisal of randomized trials and systematic reviews of the effects of physical therapy interventions for the pelvic floor. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-Based Physical Therapy for the Pelvic Floor. Edinburgh: Churchill Livingstone Elsevier, 2007: 9-19.

(160) Hollis S, Campbell F. What is meant by intention to treat analysis? Survey of published randomised controlled trials. BMJ 1999; 319(7211):670-674.

(161) Bø K, Sherburn M. Measurement of pelvic floor muscle function and strength and pelvic organ prolapse: Introduction. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-based Physical Therapy for the Pelvic Floor. Edinburgh: Churchill Livingstone Elsevier, 2007: 47-49.

(162) Kelleher C, Staskin D, Cherian P, Cotterill N, Coyne K, Kopp Z et al. Patient-Reported Outcome Assessment. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence: 5th International Consultation on Incontinence. Paris: ICUD-EAU, 2013: 389-427.

(163) Lose G, Rosenkilde P, Gammelgaard J, Schroeder T. Pad-weighing test performed with standardized bladder volume. Urology 1988; 32(1):78-80.

(164) Peschers UM, Gingelmaier A, Jundt K, Leib B, Dimpfl T. Evaluation of pelvic floor muscle strength using four different techniques. Int Urogynecol J Pelvic Floor Dysfunct 2001; 12(1):27-30.

(165) Dumoulin C, Gravel D, Bourbonnais D, Lemieux MC, Morin M. Reliability of dynamometric measurements of the pelvic floor musculature. Neurourol Urodyn 2004; 23(2):134-142.

(166) Brækken IH, Majida M, Ellstrom-Engh M, Dietz HP, Umek W, Bo K. Test-retest and intra-observer repeatability of two-, three- and four-dimensional perineal ultrasound of pelvic floor muscle anatomy and function. Int Urogynecol J Pelvic Floor Dysfunct 2008; 19(2):227-235.

(167) Dietz HP. Measurement of pelvic floor muscle function and strength and pelvic organ prolapse: Ultrasound in the assessment of pelvic floor muscle and pelvic organ descent. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, editors. Evidence-Based Physical Therapy for the Pelvic Floor. Edinburgh: Churchill Livingstone Elsevier, 2007: 81-92.

(168) Majida M, Braekken IH, Bo K, Benth JS, Engh ME. Validation of three-dimensional perineal ultrasound and magnetic resonance imaging measurements of the pubovisceral muscle at rest. Ultrasound Obstet Gynecol 2010; 35(6):715-722.

(169) Kruger JA, Heap SW, Murphy BA, Dietz HP. Pelvic floor function in nulliparous women using three-dimensional ultrasound and magnetic resonance imaging. Obstet Gynecol 2008; 111(3):631-638.

(170) Zhuang RR, Song YF, Chen ZQ, Ma M, Huang HJ, Chen JH et al. Levator avulsion using a tomographic ultrasound and magnetic resonance-based model. Am J Obstet Gynecol 2011; 205(3):232-238.

(171) van Delft K, Shobeiri A, Sultan AH, Thakar R. Haematomas may masquerade as levator ani muscle defects. 37th Annual Meeting IUGA, Brisbane; The International Urogynecological Association, 2012. Abstract.

(172) Chiarelli P, Murphy B, Cockburn J. Women's knowledge, practises, and intentions regarding correct pelvic floor exercises. Neurourol Urodyn 2003; 22(3):246-249.

(173) Whitford HM, Alder B, Jones M. A cross-sectional study of knowledge and practice of pelvic floor exercises during pregnancy and associated symptoms of stress urinary incontinence in North-East Scotland. Midwifery 2007; 23(2):204-217.

(174) Bø K, Haakstad AH, Voldner N. Do pregnant women exercise their pelvic floor muscles? Int Urogynecol J Pelvic Floor Dysfunct 2007; 18(7):733-736.

(175) Bø K, Fleten C, Nystad W. Effect of antenatal pelvic floor muscle training on labor and birth. Obstet Gynecol 2009; 113(6):1279-1284.

(176) Mason L, Glenn S, Walton I. The prevalence of stress incontinence during pregnancy and following delivery. Midwifery 1999; 15(2):120-128.

(177) Mason L, Glenn S, Walton I, Hughes C. The instruction in pelvic floor exercises provided to women during pregnancy or following delivery. Midwifery 2001; 17(1):55-64.

(178) McLennan MT, Melick CF, Alten B, Young J, Hoehn MR. Patients' knowledge of potential pelvic floor changes associated with pregnancy and delivery. Int Urogynecol J Pelvic Floor Dysfunct 2006; 17(1):22-26.

(179) Boyle R, Hay-Smith EJ, Cody JD, Morkved S. Pelvic floor muscle training for prevention and treatment of urinary and faecal incontinence in antenatal and postnatal women. Cochrane Database Syst Rev 2012; 10:CD007471.

(180) Moore K, Dumoulin C, Bradley C, Burgio K, Chambers T, Hagen S et al. Adult Conservative Management. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence: 5th International Consultation on Incontinence. Paris: ICUD-EAU, 2013: 1101-1305.

(181) Grimshaw JM, Thomas RE, MacLennan G, Fraser C, Ramsay CR, Vale L et al. Effectiveness and efficiency of guideline dissemination and implementation strategies. Health Technol Assess 2004; 8(6):iii-72.

(182) Grimshaw J, Eccles M, Tetroe J. Implementing clinical guidelines: current evidence and future implications. J Contin Educ Health Prof 2004; 24 Suppl 1:S31-S37.

(183) Horsley K. Fitness in the child-bearing years. In: Sapsford R, Bullock-Saxton J, Markwell S, editors. Women's health. A textbook for physiotherapists. New York (NY): Thieme, 2011: 168-191.

(184) Salvesen KA, Morkved S. Randomised controlled trial of pelvic floor muscle training during pregnancy. BMJ 2004; 329(7462):378-380.

(185) Agur W, Steggles P, Waterfield M, Freeman R. Does antenatal pelvic floor muscle training affect the outcome of labour? A randomised controlled trial. Int Urogynecol J Pelvic Floor Dysfunct 2008; 19(1):85-88.

(186) Dias LA, Driusso P, Aita DL, Quintana SM, Bo K, Ferreira CH. Effect of pelvic floor muscle training on labour and newborn outcomes: a randomized controlled trial. Rev Bras Fisioter 2011; 15(6):487-493.

(187) Bø K, Hilde G, Jensen JS, Siafarikas F, Engh ME. Too tight to give birth? Assessment of pelvic floor muscle function in 277 nulliparous pregnant women. Int Urogynecol J 2013; 24(12):2065-2070.

(188) Shek KL, Dietz HP. Intrapartum risk factors for levator trauma. BJOG 2010; 117(12):1485-1492.

(189) 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):1258-1261.

(190) 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):2424-2433.

(191) Sigurdardottir T, Steingrimsdottir T, Arnason A, Bo K. Pelvic floor muscle function before and after first childbirth. Int Urogynecol J 2011.

(192) Bø K, Sherburn M. Evaluation of female pelvic-floor muscle function and strength. Phys Ther 2005; 85(3):269-282.

(193) Elenskaia K, Thakar R, Sultan AH, Scheer I, Beggs A. The effect of pregnancy and childbirth on pelvic floor muscle function. Int Urogynecol J 2011; 22(11):1421-1427.

(194) 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):861-867.

(195) 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):1491-1495.

(196) Dietz HP, Shek KL, Chantarasorn V, Langer SE. Do women notice the effect of childbirth-related pelvic floor trauma? Aust N Z J Obstet Gynaecol 2012; 52(3):277-281.

(197) Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee IM et al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc 2011; 43(7):1334-1359.

(198) Mørkved S, Bo K, Schei B, Salvesen KA. Pelvic floor muscle training during pregnancy to prevent urinary incontinence: a single-blind randomized controlled trial. Obstet Gynecol 2003; 101(2):313-319.

(199) Stafne S, Salvesen K, Romundstad P, Torjusen I, Morkved S. Does regular exercise including pelvic floor muscle training prevent urinary and anal incontinence during pregnancy? A randomised controlled trial. BJOG 2012; 119(10):1270-1280.

(200) DeLancey JO, Miller JM, Kearney R, Howard D, Reddy P, Umek W et al. Vaginal birth and de novo stress incontinence: relative contributions of urethral dysfunction and mobility. Obstet Gynecol 2007; 110(2 Pt 1):354-362.

(201) Clarke M, Hopewell S, Chalmers I. Clinical trials should begin and end with systematic reviews of relevant evidence: 12 years and waiting. Lancet 2010; 376(9734):20-21.

Paper I

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ORIGINAL ARTICLE

Continence and pelvic floor status in nulliparous women at midterm pregnancy

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Abstract
Introduction and hypothesis A Cochrane review recommends antenatal pelvic floor muscle training (PFMT) in urinary incontinence (UI) prevention. The aim of the study was to investigate nulliparous pregnant women's knowledge about and practising of PFMT, their pelvic floor muscle (PFM) function, and ability to contract correctly. It was hypothesized that continent women had higher PFM strength and endurance than women with UI.
Methods Three hundred nulliparous women at gestational week 18–22 were included in a cross-sectional study. Vaginal resting pressure, maximum voluntary contraction, and PFM endurance were measured by manometer. UI was assessed by International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form (ICIQ-UI-SF). Comparisons of PFM function in continent women and women with UI were analyzed using independent-samples *t* test. Mean differences with 95 % confidence interval (CI) are presented.
Results Of 300 women, 89 % had heard of PFMT at mid pregnancy, and 35 % performed PFMT once or more a week. After thorough instruction 4 % were unable to contract correctly. Thirty-five percent reported UI, of whom 48 % performed PFMT once or more a week. Continent women had significantly higher PFM strength and endurance when compared with women having UI, with mean differences of 6.6 cmH_2O (CI 2.3–10.8, $p=0.003$), and 41.5 cmH_2Osec (CI 9.8–73.1, $p=0.010$), respectively. No difference was found for vaginal resting pressure ($p=0.054$).
Conclusions Most nulliparous pregnant women knew about PFMT. Thirty-five percent performed PFMT once or more a week. Incontinent nulliparous pregnant women had weaker PFM than their continent counterparts. More emphasis on information regarding PFM function and PFMT is warranted during pregnancy.

Keywords Exercise · Urinary incontinence / prevention and control · Pelvic floor · Pregnancy · Prenatal care · Strength training

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Introduction

A Cochrane review concluded that pregnant women without prior urinary incontinence (UI) who exercise the pelvic floor muscles (PFM) are 56 % less likely to report UI in late pregnancy and 30 % less likely to report UI by 6 months postpartum and recommends antenatal PFM training (PFMT) for preventing UI [1]. However, to date, there is scant knowledge about to which degree nulliparous pregnant women are practicing PFMT, their ability to perform a correct contraction, and further PFM function in relation to UI. Cross-sectional studies from different countries show that proportions of pregnant women performing PFMT once

a week or more vary from 16–58 % [2–6]. None of these studies assessed the ability to perform a correct PFM contraction. Being able to contract correctly, defined as a squeeze around the pelvic openings and an inward lift [7], is necessary to achieve effective PFMT. Studies including women with pelvic floor dysfunction show that≥30 % are unable to contract the PFM correctly at their first consultation [7–9]. Some observational studies show significantly higher PFM strength in continent women compared with women having UI [10–16], whereas other studies reported nonsignificant differences for this comparison [17, 18]. These studies differed both in sample and method used for assessing PFM strength. Only two studies [10, 11] were performed on pregnant women. Sampselle [11] studied 20 nulliparous women at gestational week 32–36 and assessed PFM strength by digital palpation. Mørkved et al. [10] assessed 103 nulliparous women at gestational week 20 and PFM strength using manometry. The aims of the study reported here were to: (1) investigate nulliparous pregnant women's knowledge about and practising of PFMT, (2) assess their PFM function and ability to perform a correct PFM contraction, and (3) compare vaginal resting pressure, PFM strength, and endurance in continent women versus women with UI.

Methods

This was a cross-sectional study of 300 nulliparous women at gestational week 18–22 participating in an ongoing prospective cohort at Akershus University Hospital, Norway. In the cohort study, all nulliparous women scheduled for delivery at this hospital from January 2010 until April 2011 were invited to participate. The study was approved by the Regional Medical Ethics Committee (2009/170), Norwegian Social Science Data Services (2799026), and registered at ClinicalTrials.gov (NCT01045135). All participants gave written informed consent before entering the study.

Inclusion

Inclusion criteria were nulliparous women giving birth at Akershus University Hospital, being able to speak and understand Scandinavian languages. Exclusion criteria were multiple pregnancy and prior delivery (abortion) after gestational week 16. Background data, sources of information regarding PFMT, frequency of PFMT at the point of midpregnancy, practicing of precontraction of the PFM before coughing/sneezing, and continence status were collected through an electronic questionnaire in conjunction with participants' first clinical visit at gestational week 18–22.

UI

International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form (ICIQ-UI-SF) was included in the electronic questionnaire and used to assess prevalence and frequency of urinary incontinence and its impact on quality of life [19]. ICIQ-UI-SF has been shown to have good construct validity, acceptable convergent validity, and good reliability [19]. Women were assessed as continent if they answered "never" to the question: "How often do you leak urine"? Type of UI was assessed by answers given for: "When does urine leak"?

PFMT

Women were asked in the questionnaire whether they performed PFMT, defined as training the muscles surrounding the urethra, vagina, and rectum. They were also asked about frequency of training.

PFM function

At the first visit, participants were taught how to perform a correct PFM contraction defined as inward movement and squeeze around the pelvic openings [7, 20]. PFM contraction without any movement of the pelvis or visible contraction of the glutei, hip, or abdominal muscles was emphasized [20]. The instructions continued during observation and vaginal palpation with the participant in a supine crook-lying position. The ability to perform a correct PFM contraction after thorough instruction, feedback, and practice was assessed on the basis of palpation and observation [21]. Measurements of vaginal resting pressure, PFM strength, and endurance were undertaken by a vaginal balloon connected to a high-precision pressure transducer (Camtech AS, Sandvika, Norway) [20]. The device was positioned with the middle of the balloon 3.5 cm inside the introitus [20, 21]. PFM strength was measured as the difference between vaginal resting pressure and pressure obtained at maximal voluntary contraction (MVC) and reported as the mean of three contractions. The method has been found to be reliable and valid if used with simultaneous observation of inward movement of the perineum/catheter during the contraction [20, 21]. Vaginal resting pressure was measured as the difference between atmospheric pressure and vaginal pressure at rest, without any voluntary PFM activity. PFM endurance was defined as a sustained maximal contraction and was quantified during the first 10 sec as the area below the measurement curve (integral calculation) [22]. Endurance was measured after one attempt. The atmospheric pressure toward the balloon was set to 0 cmH_2O for each participant before it was placed into the vagina. The physiotherapist assessing PFM function (GH) was blinded to participants' continence status.

Statistical analysis

Statistical analysis was performed using SPSS version 15. Background and descriptive variables are presented as frequencies with percentages or means with standard deviations (SD). Data show normal distribution for vaginal resting pressure, PFM strength, and endurance, and independent-samples *t* test was used to analyze differences between continent women and women reporting UI. Independent-samples *t* test and chi-square test were used to evaluate differences in demographic characteristics between comparison groups. *P* values <0.05 were considered significant.

Results

Characteristics of the study group are given in Table 1. Of the included women, 88.7 % had heard of of PFM training. Leaflet/magazine/newspaper were the most frequently reported source of information, followed by midwife/nurse, friend, physiotherapist, fitness class, physician, DVDs, and antenatal class (Table 2). Nineteen percent of the women reported "other" sources for PFMT knowledge. The most frequent other source was the Internet (7 %), followed by information obtained during own education (4.7 %) and from own mother (2.3 %).

Table 3 shows frequency of PFMT among women studied. When asked whether they performed a precontraction of the PFM before coughing and sneezing, 34 % reported they did, 21.3 % reported that they did not, and 44.7 % did not know. Ninety-four percent reported to be able to contract the PFM, whereas 6 % were unsure. Ninety-two percent reported to be able to voluntarily stop the urine stream when voiding. After thorough instruction, feedback, and practice on how to contract correctly, clinical examination by observation and palpation showed that 12 of 300 women (4 %) did not contract correctly, of whom ten were straining.

Table 1 Demographic characteristics of nulliparous women and differences between comparison groups: no urinary incontinence (No UI), urinary incontinence of all types (UI), and stress urinary incontinence (SUI). Numbers with percentages (%), mean with standard deviation (SD)

	Total sample (*n*=300)	No UI (*n*=196)	UI (*n*=104)	SUI (*n*=97)	No UI vs UI (*P* value)	No UI vs SUI (*P* value)
Age (years)	28.7 (4.3)	28.5 (4.3)	29.0 (4.3)	28.9 (4.6)	0.362	0.466
Prepregnancy BMI (kg/m^2)	23.9 (3.9)	23.8 (3.9)	24.3 (3.9)	24.5 (4.0)	0.282	0.077
Education level						
College/university	226 (75.3%)	146 (74.5%)	80 (76.9%)	71 (73.2%)	0.745	0.871
Primary/high school/other	74 (24.7%)	50 (25.5%)	24 (23.1%)	26 (26.8%)		
Marital status						
Married/cohabitant	287 (95.7%)	190 (96.9%)	97 (93.3%)	91 (93.8%)	0.235	1.000
Single	13 (4.3%)	6 (3.1%)	7 (6.7%)	6 (6.2%)		
Smoking prepregnancy						
No	223 (74.3%)	149 (76.0%)	74 (71.2%)	69 (71.1%)	0.436	0.526
Sometimes/daily	77 (25.7%)	47 (24.0%)	30 (28.8%)	28 (28.9%)		
Smoking during present pregnancy						
No	284 (94.7%)	189 (96.4%)	95 (91.3%)	88 (90.7%)	0.111	0.100
Sometimes/daily	16 (5.3%)	7 (3.6%)	9 (8.7%)	9 (9.3%)		
UI prepregnancy						
No	254 (84.7%)	194 (99.0%)	60 (57.7%)	62 (63.9%)	<0.001	<0.001
Yes	46 (15.3%)	2 (1.0%)	44 (42.3%)	35 (36.1%)		
General exercise, gestational week 18–22						
< 1 time per week	125 (41.7%)	77 (39.3%)	48 (46.2%)	46 (47.4%)	0.305	0.291
1 time per week	38 (12.7%)	32 (16.3%)	6 (5.8%)	9 (9.3%)	0.015	0.244
2 times per week	44 (14.7%)	23 (11.7%)	21 (20.2%)	19 (19.6%)	0.072	0.109
≥ 3 times per week	93 (30.9%)	64 (32.7%)	29 (27.9%)	23 (23.7%)	0.472	0.131

BMI body mass index

No UI: answered "never" to the question "How often do you leak urine"? (Question 3 in ICIQ-UI-SF). Comparison No UI versus UI is based on this question

SUI: answered "leaks when you cough or sneeze" OR "leaks when you are physically active/exercising" to the question "When does urine leak"? (Question 6 in ICIQ-UI-SF). Comparison No UI versus SUI is based on this question

Table 2 Sources of information about pelvic floor muscle training at gestational week 18–22 among nulliparous women. Multiple answers were possible

Sources	No. (%)
Leaflet/magazine/newspaper	120 (40%)
Midwife/nurse	83 (27.7%)
Friend	81 (27.0%)
Physiotherapist	44 (14.7%)
Fitness class (fitness instructor)	37 (12.3%)
Physician	31 (10.3%)
DVD	12 (4.0%)
Antenatal class	2 (0.7%)
Other	57 (19.0%)

Thirty-five percent reported UI at gestational week 18–22, whereas 27.0 % reported to leak once a week or less, 4.3 % two to three times per week, 1.3 % once a day, and 2.0 % several times per day. When leaking urine, one third of the women reported to leak a small amount and 1 % a moderate to large amount. Women having UI scored 1.2 (SD 1.6) on the bother scale (ICIQ-UI-SF), with 43 of 104 scoring zero. The ICIQ-UI-SF sum score for women leaking urine was 4.6 (SD 2.3, range 1–14). Among those reporting UI, stress urinary incontinence (SUI) was most prevalent (85.8 %), followed by urgency urinary incontinence (UUI) (29.2 %), mixed urine incontinence (MUI) (18.6 %), postmicturition dribble (8.8 %), no obvious reason (5.3 %), and leakage during sleep (1.8 %). As participants could check all subtypes that applied, the cumulative percentage is >100. Forty-eight percent of women having UI at midpregnancy (n=104) reported to perform PFMT once a week or more compared with 28 % of the continent women (n=196). Corresponding figures for PFMT three times per week or more were 21 % and 12 %, respectively. Mean vaginal resting pressure, MVC, and endurance are described in Table 4. Continent women (no UI) were significantly stronger and had higher PFM endurance when compared with women having UI (regardless of type), but no significant differences were found for vaginal resting pressure (Table 4). When comparing continent women versus those having SUI, significant differences in favor of the continent group were found for all PFM measures (Table 4). The above comparison groups showed no significant difference for age, prepregnancy BMI, education level, marital status, smoking before or smoking during pregnancy or general physical exercise at the frequencies two times or more per week, but a significantly greater proportion of those having UI at gestational week 18–22 also had UI before the current pregnancy (Table 1).

Table 3 Frequency of pelvic floor muscle training (PFMT) among nulliparous women at gestational week 18–22

PFMT	Frequency no. (%)
Never	150 (50%)
When needed	45 (15.0%)
Once per week	29 (9.7%)
1–2 times per week	30 (10.0%)
3 times per week	29 (9.7%)
Every day	17 (5.7%)

Discussion

The vast majority of the pregnant women in this study knew about PFMT at gestational week 18–22, posting leaflet/magazine/newspaper as the most dominant source of information. However, only one third of the women performed PFMT once a week or more. After thorough instruction including vaginal palpation, only 12 of 300 were unable to perform a correct PFM contraction. One third of the women reported UI at midpregnancy. Continent women had significantly higher PFM strength and endurance when compared with women having UI. Only half of the women reporting UI were doing PFMT once a week or more.

Strengths of this study were the number of participants both attending the clinical examination and answering the questionnaire; use of a responsive, reliable, and valid method to assess vaginal resting pressure, PFM strength, endurance [20, 21], and UI [19]; and further blinding of the assessor to continence status. To our knowledge, this study has the largest sample on nulliparous women assessing PFM function. However, one study, including both nulliparous and multiparous women had a larger study sample, with 487 women [12]. Our study has a population-based approach, as all nulliparous women scheduled for delivery at Akershus University Hospital were invited to participate. Our study sample was comparable with the total population of nulliparous women scheduled for delivery at this hospital during the inclusion period (n=2,621) with respect to age (mean 28.7 and 28.4 years, respectively) and for being married/cohabitant (95.7 % and 92.7 %, respectively). They differed in educational status, as 75.3 % of our study sample had higher education (college/university), compared with 50.8 % in the total population. A limitation of the study was the criterion of being able to speak and understand Scandinavian languages, making generalization to other language groups difficult. It is estimated that 1/6 of the 2,621 nulliparous women were not eligible due to their language. We have no other data regarding reason for declining to participate in the study.

A relatively small proportion (one third) of all included women reported to perform PFMT once a week or more. This is a figure within the range found in earlier cross-sectional

Table 4 Vaginal resting pressure, pelvic floor muscle (PFM) strength, and endurance in nulliparous women at gestational week 18−22 [mean with standard deviation (SD)]. Difference between women reporting no urinary incontinence (No UI), urinary incontinence (UI) and stress urinary incontinence (SUI) [mean difference with 95% confidence interval (CI)]

	Total (n=300)	No UI* (n=196)	UI (n=104)	SUI** (n=97)	No UI versus UI		No UI versus SUI	
					Mean diff (95% CI)	P value	Mean diff (95% CI)	P value
Vaginal resting pressure (cmH_2O)	43.0 (9.8)	43.8 (10.1)	41.5 (9.2)	41.1 (8.7)	2.3 (0.0–4.6)	0.054	2.8 (0.4–5.1)	0.024
PFM strength (cmH_2O)	35.5 (18.0)	37.7 (18.0)	31.2 (17.4)	30.3 (17.2)	6.6 (2.3–10.8)	0.003	7.7 (3.3–12.1)	0.001
PFM endurance ($cmH_2Osec.$)	245 (133.8)	259.6 (132.0)	218.1 (133.6)	212.2 (132.5)	41.5 (9.8–73.1)	0.010	50.6 (18.0–83.3)	0.002

PFM strength is reported as the mean of three maximal voluntary contractions. PFM endurance is reported after one attempt of sustained maximal contraction quantified during 10 sec

No UI: answered "never" to the question "How often do you leak urine"? (Question 3 in ICIQ–UI-SF)

SUI: answered "leaks when you cough or sneeze" OR "leaks when you are physically active/exercising" to the question "When does urine leak"? (Question 6 in ICIQ-UI-SF)

studies during pregnancy [2–6]. The variation in numbers of women performing regular PFMT might reflect that teaching PFMT during pregnancy is not yet a significant part of regular antenatal care service [23]. According to McLennan et al. [24], general pregnancy topics such as weight gain, high blood pressure, preterm labor, etc. seems to be counselled more frequently than pelvic floor risks and efficacy of PFMT. In this study, relatively few women reported health personnel as a source of PFMT. Although results from single randomized controlled trials with positive results of PFMT have been available for some years, the Cochrane review recommending antenatal PFMT [1] has only been available for a few years. It takes time to implement research into clinical practice, and this may be one explanation of why so few women were doing regular PFMT, even though they reported UI. However, the implementation of antenatal PFMT might also be restrained by health care providers claiming that PFMT may make the PFM too strong and less elastic, resulting in a prolonged second stage of labor [25]. However, this myth has recently been contradicted by two randomized controlled trials and a large cohort study showing that antenatal PFMT neither prolonged the second stage nor obstructed labor [5, 26, 27].

One barrier to PFMT might be lack of knowledge about how to perform PFM contractions [2]. Examination by palpation showed that 4 % were not able to contract their PFM correctly, even after thorough instruction, feedback, and practice. Four percent might be considered low when compared with studies reporting ratios≥30 % [7–9]. An explanation for low numbers of incorrect PFM contractions in our study and in the study by Mørkved et al. [10] might be the inclusion of nulliparous women only, whereas studies reporting high ratios of incorrect contractions included parous women with pelvic floor complaints. Parous women who have delivered vaginally have an increased risk of pelvic floor trauma, e.g. PFM tears, disruption of the pelvic fascial supports, and pudendal nerve injury [28], which may result in reduced ability to contract the PFM correctly. An additional factor that makes direct comparison from study to study difficult is that studies differ in the degree of teaching on how to contract the PFM correctly before the final assessment. In this study and the study from Mørkved et al. [10], the registration of ability to contract was done after several attempts and guidance during vaginal palpation. Clinical assessment of incorrect PFM contraction corresponded well with the women's own evaluation in our study, as 6 % percent reported they were unsure whether they contracted correctly and 8 % that they were unable to voluntary stop the urine stream when voiding. However, the study confirms that some women are straining instead of contracting correctly, and support that clinical assessment of ability to contract is important before starting a PFMT program [2, 23].

Continence status seemed to influence motivation for PFMT to some extent, as 48 % of women having UI reported performing regular PFMT, compared with 28 % of the asymptomatic women. However, the number of incontinent women performing PFMT is low. One explanation might be the low bother score (ICIQ-UI-SF) among women who reported UI, whereas 43 of 104 women reported that UI did not at all interfere with their daily life. This might reflect that pregnant women may tolerate some UI as a part of being pregnant. However, most pregnant women might not know that having UI during pregnancy increases the risk of UI postpartum [1, 24]. Such information, followed by evidence-based recommendation for antenatal PFMT [1], needs to be implemented in clinical practice and provided to pregnant women on a regular basis.

UI at gestational week 18–22 was prevalent in one third of the women in our study. Further, our findings support the findings from Mørkved et al. [10] and Sampselle [11] showing that continent nulliparous pregnant women have significantly stronger PFM and higher endurance when compared with women having UI. A direct comparison with other

observational studies reporting somewhat divergent results regarding PFM strength and continence status [12–18] is difficult because of variation in study sample due to parity and status regarding pelvic floor dysfunction. Furthermore, methods for assessing PFM function differ from study to study. Two previously published studies [11, 12] used digital palpation as the only measurement method when assessing PFM strength. This method has been questioned regarding responsiveness, reliability, and validity [29, 30].

In the context of low adherence to antenatal PFMT in this study, an interesting finding was that a higher proportion of incontinent women (48 %) performed PFMT once a week compared with continent women (28 %), but they had weaker PFM. Corresponding figures for PFMT three times per week or more were 21 % and 12 %, respectively. Although participants reported to have trained the PFM, they may not have performed supervised training or learned how to do a correct contraction or received feedback of their contraction. Further, we do not know the intensity of their contractions, and most importantly, we do not know their baseline values for PFM strength and endurance. It is important to note that reporting to exercise is not the same as the effectiveness of training, as it is possible to exercise regularly but still have little effect of training. Hence, effectiveness of a training program must be based on randomized controlled trials with actual measurement of improvement in the targeted training variable, e.g., PFM strength.

Our results support reports that incontinent women have weaker PFM and that relatively few of them exercise the PFM. This indicates that clinical assessment of PFM function, assessment of ability to contract, and PFMT needs to be implemented in antenatal health care and that health professionals regularly need to be provided with new scientific knowledge regarding the PFM and PFMT. Additionally, general fitness classes during pregnancy should include PFMT on a regular basis. To better understand the natural course of PFM function and the influence of pregnancy and childbirth, there is also a need for cohort studies running from early pregnancy into the postpartum period.

Conclusion

Most of the 300 nulliparous pregnant women in this study knew about PFMT, but only 35 % exercised once a week or more at midpregnancy. After thorough instruction, including vaginal palpation, 96 % were able to perform a correct contraction. At gestational week 18–22, 35 % of the women had UI. Women with UI had weaker PFM than their continent counterparts. More emphasis on PFM function and the advantage of PFMT is warranted during pregnancy.

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Conflicts of interest None.

References

1. Hay-Smith J, Mørkved S, Fairbrother KA, Herbison GP (2008) Pelvic floor muscle training for prevention and treatment of urinary and faecal incontinence in antenatal and postnatal women. Cochrane Database Syst RevCD007471
2. Chiarelli P, Murphy B, Cockburn J (2003) Women's knowledge, practises, and intentions regarding correct pelvic floor exercises. Neurourol Urodyn 22:246–249
3. Whitford HM, Alder B, Jones M (2007) A cross-sectional study of knowledge and practice of pelvic floor exercises during pregnancy and associated symptoms of stress urinary incontinence in North-East Scotland. Midwifery 23:204–217
4. Bø K, Haakstad AH, Voldner N (2007) Do pregnant women exercise their pelvic floor muscles? Int Urogynecol J Pelvic Floor Dysfunct 18:733–736
5. Bø K, Fleten C, Nystad W (2009) Effect of antenatal pelvic floor muscle training on labor and birth. Obstet Gynecol 113:1279–1284
6. Mason L, Glenn S, Walton I (1999) The prevalence of stress incontinence during pregnancy and following delivery. Midwifery 15:120–128
7. Kegel AH (1952) Stress incontinence and genital relaxation; a nonsurgical method of increasing the tone of sphincters and their supporting structures. Ciba Clin Symp 4:35–51
8. Benvenuti F, Caputo GM, Bandinelli S, Mayer F, Biagini C, Sommavilla A (1987) Reeducative treatment of female genuine stress incontinence. Am J Phys Med 66:155–168
9. Bump RC, Hurt WG, Fantl JA, Wyman JF (1991) Assessment of Kegel pelvic muscle exercise performance after brief verbal instruction. Am J Obstet Gynecol 165:322–327
10. Mørkved S, Salvesen KA, Bø K, Eik-Nes S (2004) Pelvic floor muscle strength and thickness in continent and incontinent nulliparous pregnant women. Int Urogynecol J Pelvic Floor Dysfunct 15:384–389
11. Sampselle CM (1990) Changes in pelvic muscle strength and stress urinary incontinence associated with childbirth. J Obstet Gynecol Neonatal Nurs 19:371–377
12. Samuelsson E, Victor A, Svardsudd K (2000) Determinants of urinary incontinence in a population of young and middle-aged women. Acta Obstet Gynecol Scand 79:208–215
13. Morin M, Bourbonnais D, Gravel D, Dumoulin C, Lemieux MC (2004) Pelvic floor muscle function in continent and stress urinary incontinent women using dynamometric measurements. Neurourol Urodyn 23:668–674
14. Thompson JA, O'Sullivan PB, Briffa NK, Neumann P (2006) 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 17:624–630
15. Shishido K, Peng Q, Jones R, Omata S, Constantinou CE (2008) Influence of pelvic floor muscle contraction on the profile of vaginal closure pressure in continent and stress urinary incontinent women. J Urol 179:1917–1922
16. Hahn I, Milsom I, Ohlsson BL, Ekelund P, Uhlemann C, Fall M (1996) Comparative assessment of pelvic floor function using vaginal cones, vaginal digital palpation and vaginal pressure measurements. Gynecol Obstet Invest 41:269–274

17. Bø K, Stien R, Kulseng-Hanssen S, Kristofferson M (1994) Clinical and urodynamic assessment of nulliparous young women with and without stress incontinence symptoms: a case-control study. Obstet Gynecol 84:1028–1032
18. Sartore A, Pregazzi R, Bortoli P, Grimaldi E, Ricci G, Guaschino S (2003) Assessment of pelvic floor muscle function after vaginal delivery. Clinical value of different tests J Reprod Med 48:171–174
19. Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P (2004) ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn 23:322–330
20. Bø K, Hagen RH, Kvarstein B, Larsen S (1990) Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. I. Reliability of vaginal pressure measurements of pelvic muscle strength. Neurourol Urodyn 9:471–477
21. Bø K, Hagen RH, Kvarstein B, Larsen S (1990) Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. II. Validity of vaginal pressure measurements of pelvic muscle strength- The necessity of supplementary methods for control of correct contraction. Neurourol Urodyn 9:479–487
22. Braekken IH, Majida M, Ellstrom EM, Holme IM, Bø K (2009) Pelvic floor function is independently associated with pelvic organ prolapse. BJOG 116:1706–1714
23. Mason L, Glenn S, Walton I, Hughes C (2001) The instruction in pelvic floor exercises provided to women during pregnancy or following delivery. Midwifery 17:55–64
24. McLennan MT, Melick CF, Alten B, Young J, Hoehn MR (2006) Patients' knowledge of potential pelvic floor changes associated with pregnancy and delivery. Int Urogynecol J Pelvic Floor Dysfunct 17:22–26
25. Horsley K (2011) Fitness in the child-bearing years. In: Sapsford R, Bullock-Saxton J, Markwell S (eds) Women's health. A textbook for physiotherapists. Thieme, New York, pp 168–191
26. Salvesen KA, Mørkved S (2004) Randomised controlled trial of pelvic floor muscle training during pregnancy. BMJ 329:378–380
27. Agur W, Steggles P, Waterfield M, Freeman R (2008) Does antenatal pelvic floor muscle training affect the outcome of labour? a randomised controlled trial. Int Urogynecol J Pelvic Floor Dysfunct 19:85–88
28. Dietz HP (2006) Pelvic floor trauma following vaginal delivery. Curr Opin Obstet Gynecol 18:528–537
29. Bø K, Sherburn M (2005) Evaluation of female pelvic-floor muscle function and strength. Phys Ther 85:269–282
30. Bø K, Finckenhagen HB (2001) Vaginal palpation of pelvic floor muscle strength: inter-test reproducibility and comparison between palpation and vaginal squeeze pressure. Acta Obstet Gynecol Scand 80:883–887

Paper II

GENERAL GYNECOLOGY

Impact of childbirth and mode of delivery on vaginal resting pressure and on pelvic floor muscle strength and endurance

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OBJECTIVE: We sought to study impact of delivery mode on vaginal resting pressure (VRP) and on pelvic floor muscle (PFM) strength and endurance, and whether these measurements differed in women with and without urinary incontinence.

STUDY DESIGN: We conducted a cohort study following 277 nulliparous women from midpregnancy to 6 weeks postpartum. Manometer was used for PFM measurements; differences were analyzed by *t* test (within groups) and analysis of variance (between groups).

RESULTS: Only VRP changed significantly (10% reduction, $P = .001$) after emergency cesarean section. After normal and instrumental vaginal delivery, VRP was reduced by 29% and 30%; PFM strength by 54% and 66%; and endurance by 53% and 65%, respectively. Significant differences for all PFM measures ($P < .001$) were found when comparing cesarean vs normal and instrumental vaginal delivery, respectively. Urinary continent women at both time points had significantly higher PFM strength and endurance than incontinent counterparts ($P < .05$).

CONCLUSION: Pronounced reductions in VRP and in PFM strength and endurance were found after vaginal delivery. Continent women were stronger than incontinent counterparts.

Key words: mode of delivery, pelvic floor muscle strength and endurance, pregnancy and childbirth, urinary incontinence, vaginal resting pressure

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The pelvic floor muscles (PFM) play a significant role in the continence control system and pelvic organ support.[1] The most established risk factor for pelvic floor dysfunction and weakening of the PFM is vaginal delivery.[2-9] During vaginal childbirth, PFM, nerves, and connective tissue are forcibly stretched, compressed, and bruised. Neurophysiologic studies have shown that vaginal deliveries cause partial denervation of the pelvic floor striated muscles in most women,[8-10] whereas imaging studies have shown major defect of the most medial part of the PFM, the pubococcygeus muscle, within the range 13–36% among primiparous women delivering vaginally.[2,11,12] Hence, it is likely that the impact of vaginal childbirth may lead to reduced vaginal resting pressure (VRP) and reduced PFM strength and endurance, and that cesarean section (CS) may protect the PFM.

To date there is a paucity of knowledge regarding the change in VRP and in PFM strength and endurance from pregnancy to postpartum. Studies assessing change in PFM strength from pregnancy to shortly after childbirth (3 days to 12 weeks postpartum) in relation to mode of delivery have used either digital palpation,[13-15] manometry,[14,16-18] or electromyography.[13] Except for the studies of Caroci et al[14] and Meyer et al[18] counting 226 and 149 nulliparous women, respectively, the sample sizes of the above-cited studies were small, ranging from 20–75 participants. This leaves very few women in each group of delivery mode. Results from above-cited studies are conflicting and none of the published studies addressed change in VRP when comparing modes of delivery. The cohort study by Elenskaia et al,[19] including 182 nulliparous women during the second trimester, was not included as a comparable study as they discriminate between delivery modes only at their last study visit, taking place 12 months postpartum.

Pregnancy and childbirth are considered main etiological factors in the development of urinary incontinence (UI).[20] UI in nulliparous women before and after delivery has been associated with reduced PFM strength[15,18,21] and endurance.[15,21] However, only 2 of these studies[15,18] had a prospective design, following up 20 and 149 nulliparous women from pregnancy to after childbirth, respectively.

The aims of the present study were to: (1) study impact of childbirth and mode of delivery on PFM function in terms of ability to contract, VRP, and PFM strength and endurance from midpregnancy to 6 weeks postpartum; and (2) investigate changes in VRP and in PFM strength and endurance from midpreg-

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nancy to 6 weeks postpartum in women with and without UI.

Materials and Methods

This is a prospective cohort study following 300 nulliparous pregnant women from midpregnancy to 6 weeks postpartum. All nulliparous women scheduled for delivery at Akershus University Hospital, Norway from January 2010 through April 2011 were invited to participate, and they were recruited in connection with the routine ultrasound examination in gestational week 18–22 (midpregnancy).

Nulliparous women with a singleton pregnancy who could speak and understand Scandinavian languages were included. Women with a prior abortion after gestational week 16 were excluded. To attend the study visit at 6 weeks postpartum, the women had to give birth after gestational week 32. Women with stillbirth were excluded. The time point 6 weeks postpartum was chosen on the basis of convenience for the participating women, as it could be combined with their routine postpartum appointment.

Demographic data were collected through an electronic questionnaire in conjunction with participants' first clinical visit, which was taking place shortly after routine ultrasound examination at gestational week 18–22. Data on delivery mode and other obstetric variables were collected from the hospital's electronic medical records.

PFM measurements

During the first visit, participants were taught how to perform a correct PFM contraction. PFM contraction without any movement of the pelvis or visible contraction of the gluteal, hip, or abdominal muscles was emphasized as described in Bø et al.[22] All examinations were performed with the participant in a standardized supine crook lying position. Correct contraction was assessed on the basis of palpation and observation and defined as inward movement and squeeze around the pelvic floor openings.[22,23] VRP and strength and endurance of the PFM were measured by using an air-filled vaginal balloon connected to a high-precision pressure transducer (Camtech AS, Sandvika, Norway). The middle of the balloon was positioned 3.5 cm inside the introitus.[24] PFM strength was calculated as the mean of 3 maximal voluntary contractions. The method has been found to be reliable and valid if used with simultaneous observation of inward movement of the perineum/catheter during the contraction.[22,25] VRP was measured with the balloon positioned in the vagina without any voluntary PFM activity. PFM endurance was defined as a sustained maximal contraction, and was quantified during the first 10 seconds as the area below the measurement curve (integral calculation).[26] The balloon was set to 0 cm H_2O for each subject before it was placed into the vagina. Changes (Δ) in VRP and in PFM strength and endurance from midpregnancy (visit 1) to 6 weeks postpartum (visit 2) were recorded as ΔVRP, ΔPMF strength, and ΔPFM endurance. The 2 assessors were blinded for delivery data at the second visit. To minimize biases in assessment and manometer measurement, the assessors (both physiotherapists) were trained ahead of the study and a rigorous protocol in standards of procedures was kept.

UI

International Consultation on Incontinence Questionnaire (ICIQ) UI Short Form (SF) was included in the electronic questionnaire. ICIQ UI SF has been shown to have good construct validity, acceptable convergent validity, and good reliability.[27] Women were defined as continent when answering "never" to the question: "How often do you leak urine?" (ICIQ UI SF).

Statistical analysis

Statistical analysis was performed using software (version 15; SPSS, Inc, Chicago, IL). Background and descriptive variables are presented as frequencies with percentages or means with SD. Changes from midpregnancy to 6 weeks postpartum within group regarding VRP and PFM strength and endurance were analyzed using Paired-sample *t* test for normally distributed data and Wilcoxon signed rank test for nonnormally distributed data. Differences between delivery modes and differences between incontinent and continent women were analyzed by using 1-way between-groups analysis of variance if data qualified for a normal distribution. If not Kruskal-Wallis test was used. Standard multiple linear regression was used to analyze the role of demographic and obstetric variables on the change of PFM measurements. *P* values $< .05$ were considered significant.

This study is part of a prospective cohort. The sample size of 300 was a result of power calculation on change in hiatal dimensions of the levator ani muscle from pregnancy to postpartum (using 3-/4-dimensional ultrasound), and not VRP and PFM strength and endurance.

Institutional review board

The study was approved by the Regional Medical Ethics Committee (2009/170) and Norwegian Social Science Data Services (2799026), and registered at ClinicalTrials.gov (NCT01045135). All subjects gave written informed consent before entering the study.

Results

Three hundred nulliparous pregnant women were included at midpregnancy and 277 were seen again as primiparous at 6 weeks postpartum. Of the 23 (7.7%) women not attending the clinical examination postpartum, 10 delivered at another hospital, 9 did not want to continue, 3 had a stillbirth, and 1 was excluded due to delivery <32 weeks of gestation. Characteristics of the study sample attending both clinical visits (n = 277) are shown in Table 1. Mean gestational week at the first study visit was 21 (SD 1.4), ranging from gestational week 17–25. After delivery, the mean postpartum week was 6.2 (SD 1.0), ranging from 3–11 weeks postpartum.

Eleven (3.9%) of 277 women did not contract the PFM correctly at midpregnancy. At the visit 6 weeks postpartum 4 of those 11 had learned to contract correctly; 3 had a normal vaginal delivery (NVD) and 1 had CS. Seven of those 11 were still unable to perform a correct contraction; 6 had NVD and 1 had CS. Further, 5 women contracting the PFM correctly at midpregnancy had lost the

TABLE 1
Characteristics of nulliparous women included at gestational week 21 (n = 277)

Characteristic	Value
Age, y	28.7 (4.3)
Prepregnancy BMI, kg/m^2	23.8 (3.9)
Education level	
College or university	209 (75.5%)
Primary school, high school, or other	68 (24.5%)
Marital status	
Married or cohabitant	265 (95.7%)
Single	12 (4.3%)
Smoking prepregnancy	
No	207 (74.7%)
Yes	70 (25.3%)

Continuous variables given as mean with SD. Categorical variables given as numbers with percentages.
BMI, body mass index.

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ability to contract after delivery; 2 had a NVD and 3 had an instrumental vaginal delivery (IVD). This leaves 12 of 277 women (4.3%) not contracting correctly 6 weeks postpartum.

Of the 277 women 69.7% had NVD, 16.2% had IVD (41 with vacuum, 4 with forceps), and 14.1% delivered by CS (29 emergency, 10 elective). The women having elective CS were excluded from further analysis (Tables 2-5). Five of the 29 women having emergency CS were in second stage of labor before CS was performed. Indication was fetal distress in 1 case and protracted total second stage in the remaining 4 cases.[28]

Measurements on VRP and PFM strength and endurance within delivery mode groups are presented in Table 2 and between delivery modes in Table 3. Within the CS group a 10% reduction in VRP, 12% reduction in PFM strength, and 11% reduction in endurance was found, however change was statistically significant for resting pressure only (Table 2). Within-groups changes for both NVD and IVD were highly significant for all PFM measures ($P < .001$) (Table 2).

Mean differences in change from midpregnancy to 6 weeks postpartum were significant for all PFM measurements when comparing CS vs NVD and CS vs IVD (Table 3). VRP was reduced by 29% in the NVD group and by 30% in the IVD group: respectively 2.8 and 3.2 times more than the reduction found in the CS group. For the same comparison PFM strength was reduced by 54% (NVD) and 66% (IVD): respectively 4.3 and 5.2 times more than the CS group. Finally, PFM endurance was reduced by 53% (NVD) and 65% (IVD): respectively 4.5 and 5.5 times more than the CS group. When comparing women with NVD and IVD, there were no significant differences in changes from midpregnancy to 6 weeks postpartum in VRP or in PFM strength or endurance (Table 3).

Table 4 shows that prepregnancy body mass index influenced change in resting pressure from midpregnancy to 6 weeks postpartum, whereas PFM strength and endurance were influenced by a total second stage lasting >60 minutes.

Thirty-six percent (94 of 264) reported UI at midpregnancy, and 40% (106 of 264) at 6 weeks postpartum. Of the 94 women having UI at midpregnancy, 58 still had UI 6 weeks postpartum, whereas 36 became continent (Table 5). Of the 170 women having no UI at midpregnancy, 122 were still continent at 6 weeks postpartum, whereas 48 reported de novo UI. Women with no UI at both time points had significantly higher PFM strength and endurance than their incontinent counterparts (Table 5). No significant differences were found when comparing women with de novo UI postpartum and women with persistent UI postpartum (UI at both time points) and continent women (no UI both time points), respectively (Table 5). The 4 comparison groups did not differ in age, prepregnancy body mass index (1-way analysis of variance), or antenatal PFM training (PFMT) ≥3 times per week (χ^2 test for independence).

COMMENT

Analyzing the data for different delivery modes, we found no changes in PFM strength or endurance for the CS group, but a significant and pronounced reduction in all PFM measurements for vagi-

TABLE 2
Delivery modes and pelvic floor muscle function; within-group differences (n = 267)

	CS (n = 29)				NVD (n = 193)				IVD (n = 45)			
Variable PFM measures	Gestational wk 21, mean (SD)	6 wk postpartum, mean (SD)	Mean difference (95% CI)	*P* value	Gestational wk 21, mean (SD)	6 wk postpartum, mean (SD)	Mean difference (95% CI)	*P* value	Gestational wk 21, mean (SD)	6 wk postpartum, mean (SD)	Mean difference (95% CI)	*P* value
VRP, cmH$_2$0	43.8 (12.6)	39.5 (12.1)	4.3 (1.6–7.0)	.003	42.2 (9.2)	30.1 (6.8)	12.2 (11.0–13.3)	< .001	44.8 (8.7)	31.1 (9.6)	13.6 (11.4–15.9)	< .001
PFM strength, cmH$_2$0	35.3 (18.3)	30.9 (16.0)	4.4 (−0.5 to 9.4)	.077	35.3 (18.7)	16.4 (12.3)	18.9 (17.0–20.8)	< .001	34.8 (16.8)	11.8 (8.3)	22.9 (18.9–27.0)	< .001
PFM endurance, cmH$_2$0sec	250.9 (134.2)	222.3 (140.0)	28.6 (−19.7 to 76.9)	.236	243.8 (137.5)	114.7 (85.7)	129.1 (113.7–144.4)	< .001	243.3 (126.1)	86.0 (68.4)	157.3 (126.9–187.7)	< .001

Paired-sample *t* test. Women with elective CS (n = 10) not included.
CI, confidence interval; *CS*, cesarean section (emergency only); *IVD*, instrumental vaginal delivery (vacuum and forceps); *NVD*, normal vaginal delivery; *PFM*, pelvic floor muscle; *VRP*, vaginal resting pressure.
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TABLE 3
Delivery modes and pelvic floor muscle function; between-group differences (n = 267)

Variable PFM measurements	CS (n = 29) vs NVD (n = 193)		CS (n = 29) vs IVD (n = 45)		NVD (n = 193) vs IVD (n = 45)	
	Mean difference (95% CI)	*P* value	Mean difference (95% CI)	*P* value	Mean difference (95% CI)	*P* value
VRP gestational wk 21, cmH_2O	1.6 (−2.9 to 6.0)	.689	−1.0 (−6.3 to 4.4)	.906	−2.5 (−6.2 to 1.2)	.247
VRP 6 wk postpartum, cmH_2O	9.4 (5.7–13.2)	< .001	8.4 (3.9–12.9)	< .001	−1.0 (−4.2 to 2.1)	.718
ΔVRP, cmH_2O	−7.9 (−11.7 to −4.1)	< .001	−9.4 (−13.9 to −4.8)	< .001	−1.5 (−4.6 to 1.7)	.505
PFM strength gestational wk 21, cmH_2O	0.0 (−8.6 to 8.6)	1.000	0.6 (−9.7 to 10.9)	.991	0.5 (−6.6 to 7.7)	.982
PFM strength 6 wk postpartum, cmH_2O	14.5 (8.7–20.2)	< .001	19.1 (12.2–25.9)	< .001	4.6 (−0.2 to 9.4)	.061
ΔPFM strength, cmH_2O	−14.5 (−20.7 to −8.2)	< .001	−18.5 (−26.0 to −11.0)	< .001	−4.0 (−9.2 to 1.2)	.160
PFM endurance gestational wk 21, cmH_2Osec	7.2 (−56.3 to 70.7)	.962	7.6 (−68.4 to 83.6)	.970	0.4 (−52.4 to 53.2)	1.000
PFM endurance 6 wk postpartum, cmH_2Osec	107.7 (65.2–150.2)	< .001	136.3 (85.5–187.2)	< .001	28.6 (−6.7 to 64.0)	.138
ΔPFM endurance, cmH_2Osec	−100.5 (−151.8 to −49.2)	< .001	−128.7 (−190.0 to −67.4)	< .001	−28.2 (−70.8 to 14.4)	.265

One-way between-groups analysis of variance. Women with elective CS (n = 10) not included. PFM strength is reported as the mean of 3 maximal voluntary contractions. PFM endurance is reported after 1 attempt of sustained maximal contraction quantified during 10 seconds.
CI, confidence interval; *CS*, cesarean section (emergency only); *IVD*, instrumental vaginal delivery (vacuum and forceps); *NVD*, normal vaginal delivery; *PFM*, pelvic floor muscle; *VRP*, vaginal resting pressure; Δ, change between gestational week 21 and 6 weeks postpartum.
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nal delivery modes (NVD and IVD). Women who were continent both at midpregnancy and 6 weeks postpartum had significantly higher PFM strength and endurance than their incontinent counterparts. Multivariate analysis showed that delivery mode was the most important factor for change in PFM variables.

Strengths of the present study were the number of participants followed up from pregnancy to after childbirth, few losses to follow-up (7.7%), and that the participants had their clinical examination within a low variation of time, both at midpregnancy and 6 weeks postpartum. To our knowledge this is one of the largest cohort studies presenting clinical data on VRP and on PFM strength and endurance in women delivering their first child. An added strength is the use of a high-precision manometer to evaluate change in PFM function, a method found to be responsive, reliable, and valid.[22,25] Further, the assessors were blinded for delivery mode. A limitation is the low number of women with CS (n = 29) and instrumental assisted deliveries (n = 45). This is also a challenge for comparable studies, having a cesarean group sizes ranging from 5–37 women.[13-18] Another limitation could be that the women were examined in midpregnancy but not again closer to delivery. A possible contribution of changes late in pregnancy can therefore not be taken into account. However, we found no changes in PFM strength and endurance within the CS group, indicating that the impact of late pregnancy events on outcome measures seems unlikely. A lack of priori power calculation is a weakness and might weaken this statement as the CS group is small. However, according to the study by Sigurdardottir et al,[17] a group size of 8 women was needed to detect changes in PFM strength associated with childbirth.

Our study sample was comparable to the total population of nulliparous women (n = 2621) scheduled for delivery at Akershus University Hospital during the inclusion period with respect to age (mean of 28.7 and 28.4 years, respectively) and being married/cohabitant (95.7% and 92.7%, respectively). Further, the CS rate was similar: 14.1% in our study sample and 16.9% in the total nulliparous population at our hospital. However, our study sample differed in educational status as 75.5% of our participants had higher education (college/university), compared to 50.8% in the total population of nulliparous women. The latter may be linked to the inclusion criterion of being able to speak and understand Scandinavian languages.

At both clinical visits, examination by palpation and observation showed that 4% were unable to contract their PFM correctly, which is in line with the study by Mørkved et al,[21] but might be considered low when compared to studies reporting ≥30%.[23,29-31] However, direct comparison from study to study is difficult due to heterogeneity in study samples and the degree of instruction on how to contract the PFM correctly before the final assessment. In this study and the study from Mørkved et al,[21] the registration of ability to contract was done after

TABLE 4
Role of demographic and obstetric variables on change in pelvic floor muscle function (n = 267)

	Factor					
	ΔVRP, cmH_2O		ΔPFM strength, cmH_2O		ΔPFM endurance, cmH_2Osec	
Variable	B coefficient (95% CI)	*P* value	B coefficient (95% CI)	*P* value	B coefficient (95% CI)	*P* value
CS[a]	−8.0 (−11.3 to −4.6)	< .001	−16.1 (−21.6 to −10.6)	< .001	−111.5 (−156.6 to −66.5)	< .001
IVD[a]	1.2 (−1.6 to 4.1)	.403	1.4 (−3.3 to 6.1)	.546	9.6 (−29.1 to 48.2)	.626
Age	0.1 (−0.1 to 0.4)	.270	0.229 (−0.2 to 0.6)	.251	1.5 (−1.7 to 4.7)	.366
Prepregnancy BMI	−0.5 (−0.7 to −0.2)	.001	−0.4 (−0.8 to 0.1)	.101	−3.6 (−7.2 to −0.2)	.049
Length of total second stage >60 min	1.7 (−0.4 to 3.9)	.117	6.9 (3.3–10.5)	< .001	52.5 (23.0–82.0)	.001
Epidural	−0.2 (−2.3 to 2.00)	.886	−0.8 (−4.3 to 2.8)	.668	−12.0 (−41.3 to 17.3)	.422
Fetal birthweight	0.0 (0.0–0.0)	.546	0.0 (0.0–0.0)	.694	0.0 (−0.1 to 0.0)	.478
Head circumference	−0.8 (−1.6 to 0.0)	.062	0.6 (−0.8 to 2.0)	.399	6.7 (−4.6 to 18.1)	.244

Standard multiple linear regression. Women with elective CS (n = 10) not included.
BMI, body mass index; *CI*, confidence interval; *CS*, cesarean section (emergency only); *IVD*, instrumental vaginal delivery (vacuum and forceps); *PFM*, pelvic floor muscle; *VRP*, vaginal resting pressure; Δ, change.

[a] Normal vaginal delivery as reference.

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several attempts and guidance by the physiotherapist during vaginal palpation.

PFM and innervating nerves are forcibly stretched during vaginal deliveries. Muscle fibers might be stretched up to 3 times their resting length as the fetal head is crowning,[32] and nerves innervating the levator ani might exhibit a strain of 35%.[33] It is therefore not surprising that measurements on VRP and on PFM strength and endurance 6 weeks postpartum were significantly influenced by mode of delivery, with vaginal delivery as the strongest predictor. Our results showing that PFM strength was not influenced by CS, but significantly influenced and pronounced by vaginal delivery, are in contrast to the findings by

TABLE 5
Urinary continence and pelvic floor muscle function; between-group differences (n = 264)

PFM measure	1. No UI[a] at any time point (n = 122)	2. No UI[a] gestational wk 21 but UI 6 wk postpartum (n = 48)	3. UI gestational wk 21 but no UI[a] 6 wk postpartum (n = 36)	4. UI at both time points (n = 58)	*P* value
VRP gestational wk 21, cmH_2O	44.0 (10.3)	42.4 (7.9)	42.0 (8.2)	41.2 (10.1)	> .05 (all comparisons)
VRP 6 wk postpartum, cmH_2O	32.6 (8.7)	29.9 (6.4)	31.1 (8.3)	29.8 (9.4)	> .05 (all comparisons)
ΔVRP, cmH_2O	11.4 (9.0)	12.6 (7.2)	10.9 (8.1)	11.4 (8.7)	> .05 (all comparisons)
PFM strength gestational wk 21, cmH_2O	38.2 (17.0)	36.4 (21.0)	34.6 (19.4)	28.8 (16.2)	.006 (1 vs 4) > .05 (all other comparisons)
PFM strength 6 wk postpartum, cmH_2O	19.3 (14.8)	15.8 (11.5)	20.0 (13.0)	12.5 (9.4)	.006 (1 vs 4) .032 (3 vs 4) > .05 (all other comparisons)
ΔPFM strength, cmH_2O	18.9 (14.8)	20.6 (14.4)	14.6 (13.7)	16.3 (12.8)	> .055 (all comparisons)
PFM endurance gestational wk 21, cmH_2Osec	264.5 (121.9)	251.9 (158.6)	245.9 (147.2)	199.6 (123.3)	.013 (1 vs 4) > .05 (all other comparisons)
PFM endurance 6 wk postpartum, cmH_2Osec	135.0 (105.7)	117.0 (96.3)	138.5 (93.7)	86.9 (73.6)	.010 (1 vs 4) > .05 (all other comparisons)
ΔPFM endurance, cmH_2Osec	129.5 (120.5)	134.9 (113.4)	107.4 (122.4)	112.7 (96.8)	> .05 (all comparisons)

One-way between-groups analysis of variance: mean with SD, statistical difference between groups given as *P* value. Women with elective cesarean section (n = 10) not included; additional 3 participants had missing data on no UI/UI, total n = 264. PFM strength is reported as the mean of 3 maximal voluntary contractions. PFM endurance is reported after 1 attempt of sustained maximal contraction quantified during 10 seconds.
PFM, pelvic floor muscle; *UI*, urinary incontinence; *VRP*, vaginal resting pressure; Δ, change between gestational week 21 and 6 weeks postpartum.

[a] Answered "never" to question "How often do you leak urine?" (question 3; International Consultation on Incontinence Questionnaire UI Short Form).

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Caroci at al,[14] but in line with findings in other comparable studies using manometry.[16-18] Sigurdardottir et al,[17] following up 36 nulliparous women from midpregnancy to 6-12 weeks postpartum, found a reduction in PFM strength by 49% in the NVD group and 64% in the IVD group. Peschers et al[16] found a 35% decline in PFM strength from late pregnancy to 6-10 weeks postpartum in primiparous women delivering vaginally (n = 25), but for multiparous women delivering vaginally (n = 20) they found that PFM strength at 6-10 weeks postpartum returned to strength measurements from late pregnancy. Finally, Meyer et al[18] following up 149 nulliparous women from pregnancy to 9 weeks postpartum found a decline of 14% in the NVD group (n = 91) and 35% in the IVD group (n = 25, all forceps deliveries).

Difference in VRP and in PFM strength and endurance from pregnancy to after childbirth can be influenced by a testing effect obtained from first to second assessment. To minimize this effect we emphasized thorough instruction on how to contract correctly before the final assessment at the first clinical visit. How this was handled in the above-mentioned studies[16-18] we do not know, but testing effect might be a factor explaining variance of change in PFM measurements seen in the different studies. Additionally, variance in time point of the first assessment (ie, midpregnancy or late pregnancy) and further length of recovery after childbirth before the second assessment may influence change measurements. This assumption is supported by Peschers et al[16] as they also measured PFM strength shortly after delivery (2-8 days) and found a significant improvement from this time point to 6-10 weeks postpartum.

In contrast to our results, Sigurdardottir et al[17] reported no significant difference in VRP when comparing vaginal deliveries with CS. A possible explanation for divergent findings might be a lack of statistical power in the study by Sigurdardottir et al,[17] as their study sample only counted 5 women with CS.

No significant differences in change from midpregnancy to 6 weeks postpartum were found when comparing NVD vs IVD. This was unexpected since IVD and the use of forceps have been shown to be associated with levator trauma.[12,34] A plausible explanation for this nonsignificant difference might be that only 4 of the 45 instrumental deliveries in our study were forceps assisted. Interestingly, Shek and Dietz[35] found that vacuum-assisted delivery had less impact on the PFM when compared to NVD.

How well VRP and PFM strength and endurance recover in the postpartum year is of great interest, and further, whether PFMT of high quality would add more than natural recovery alone. To our knowledge, Elenskaia et al[19] conducted to date the only study with long-term follow-up. They found that PFM strength recovered completely at 1 year postpartum in both primiparous and multiparous women irrespective of delivery mode. Additional studies are needed to see whether their findings can be confirmed or not.

Change in VRP and in PFM strength and endurance from midpregnancy to 6 weeks postpartum showed no significant differences for any of the PFM measurements when comparing women with and without UI. However, our results showed that women who were continent both at midpregnancy and 6 weeks postpartum had significantly higher PFM strength and endurance than their incontinent counterparts. Our findings are supported by Mørkved et al[21] and Sampselle.[15] Our results indicate that PFM strength and endurance are of importance for staying continent during pregnancy and after childbirth. Even though PFM strength and endurance are reduced just as much for continent women as for women with UI, continent women at midpregnancy seem to be better off at 6 weeks because they have a better starting point regarding PFM strength and endurance.

The evidence on effect of postpartum PFMT in prevention and treatment of UI is conflicting.[36] Changes in PFM measures in our study do not explain de novo UI, which might be explained by factors not explored in this study such as overall position of the pelvic floor, levator hiatus area, and general descent of the pelvic floor during increase in intraabdominal pressure. However, our results link PFM strength and endurance with UI both at midpregnancy and 6 weeks postpartum, and it seems like women with no UI and higher PFM strength and endurance at midpregnancy can cope better with the pronounced decline in strength and endurance after vaginal delivery. This does address the need of a proper clinical examination of PFM function and pelvic floor dysfunction both during pregnancy and after childbirth.

In addition to a general weakness of the PFM, partly attributable to excessive stretching during childbirth, muscle, peripheral nerve, and connective tissue injuries may play an important role in reduction of PFM function.[7] So far, there is scant knowledge about the association between diagnosed PFM injuries and VRP, PFM strength, and PFM endurance. However, a cross-sectional study[37] on women with pelvic organ prolapse found larger hiatal dimensions both at rest and at maximal contraction among women diagnosed with major PFM defects when compared to women without such muscle defect.

To which degree injured PFM respond to training is still not known, and needs to be tested in a high-quality randomized controlled trial. ■

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REFERENCES

1. 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.

2. 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;101:46-53.

3. Kearney R, Miller JM, Ashton-Miller JA, Delancey JO. Obstetric factors associated with levator ani muscle injury after vaginal birth. Obstet Gynecol 2006;107:144-9.

4. Dietz HP. Pelvic floor trauma following vaginal delivery. Curr Opin Obstet Gynecol 2006; 18:528-37.

5. DeLancey JO, Ashton-Miller JA. MRI of intact and injured female pelvic floor muscles. In: Bø

K, Berghmans B, Mørkved S, Van Kampen M, eds. Evidence-based physical therapy for the pelvic floor. Edinburgh: Churchill Livingstone Elsevier; 2007:93-105.
6. 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;52:1186-95.
7. Ashton-Miller JA, Delancey JO. On the biomechanics of vaginal birth and common sequelae. Annu Rev Biomed Eng 2009;11:163-76.
8. Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol 1990;97:770-9.
9. Snooks SJ, Setchell M, Swash M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet 1984;2:546-50.
10. 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;77:1358-60.
11. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. Obstet Gynecol 2005;106:707-12.
12. Shek KL, Dietz HP. Intrapartum risk factors for levator trauma. BJOG 2010;117:1485-92.
13. 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;29:1258-61.
14. Caroci AS, Riesco ML, Sousa WS, 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 cesarean childbirth). J Clin Nurs 2010;19:2424-33.
15. Sampselle CM. Changes in pelvic muscle strength and stress urinary incontinence associated with childbirth. J Obstet Gynecol Neonatal Nurs 1990;19:371-7.
16. Peschers UM, Schaer GN, Delancey JO, Schuessler B. Levator ani function before and after childbirth. Br J Obstet Gynaecol 1997;104:1004-8.
17. Sigurdardottir T, Steingrimsdottir T, Arnason A, Bo K. Pelvic floor muscle function before and after first childbirth. Int Urogynecol J 2011;22:1497-503.
18. Meyer S, Schreyer A, De GP, Hohlfeld P. The effects of birth on urinary continence mechanisms and other pelvic-floor characteristics. Obstet Gynecol 1998;92:613-8.
19. Elenskaia K, Thakar R, Sultan AH, Scheer I, Beggs A. The effect of pregnancy and childbirth on pelvic floor muscle function. Int Urogynecol J 2011;22:1421-7.
20. Mørkved S. Evidence for pelvic floor physical therapy for urinary incontinence during pregnancy and after childbirth. In: Bø K, Berghmans B, Mørkved S, Van Kampen M, eds. Evidence-based physical therapy for the pelvic floor. Edinburgh: Churchill Livingstone Elsevier; 2007:317-36.
21. 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;15:384-9.
22. Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of female stress urinary incontinence, I: reliability of vaginal pressure measurements of pelvic muscle strength. Neurourol Urodyn 1990;9:471-7.
23. Kegel AH. Stress incontinence and genital relaxation; a nonsurgical method of increasing the tone of sphincters and their supporting structures. Ciba Clin Symp 1952;4:35-51.
24. Bø K. Pressure measurements during pelvic floor muscle contractions: the effect of different positions of the vaginal measuring device. Neurourol Urodyn 1992;11:107-13.
25. Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of female stress urinary incontinence, II: validity of vaginal pressure measurements of pelvic muscle strength–the necessity of supplementary methods for control of correct contraction. Neurourol Urodyn 1990;9:479-87.
26. Brækken IH, Majida M, Ellstrom EM, Holme IM, Bo K. Pelvic floor function is independently associated with pelvic organ prolapse. BJOG 2009;116:1706-14.
27. Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P. ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn 2004;23:322-30.
28. American College of Obstetrics and Gynecology Committee on Practice Bulletins–Obstetrics. ACOG practice bulletin no. 49, December 2003: dystocia and augmentation of labor. Obstet Gynecol 2003;102:1445-54.
29. Benvenuti F, Caputo GM, Bandinelli S, Mayer F, Biagini C, Sommavilla A. Reeducative treatment of female genuine stress incontinence. Am J Phys Med 1987;66:155-68.
30. Bø K, Larsen S, Oseid S, Hagen R, Jørgensen J. Knowledge about and ability to correct pelvic floor muscle exercises in women with urinary stress incontinence. Neurourol Urodyn 1988;7:261-2.
31. Bump RC, Hurt WG, Fantl JA, Wyman JF. Assessment of Kegel pelvic muscle exercise performance after brief verbal instruction. Am J Obstet Gynecol 1991;165:322-7.
32. Lien KC, Mooney B, Delancey JO, Ashton-Miller JA. Levator ani muscle stretch induced by simulated vaginal birth. Obstet Gynecol 2004;103:31-40.
33. Lien KC, Morgan DM, Delancey JO, Ashton-Miller JA. Pudendal nerve stretch during vaginal birth: a 3D computer simulation. Am J Obstet Gynecol 2005;192:1669-76.
34. Kearney R, Fitzpatrick M, Brennan S, et al. Levator ani injury in primiparous women with forceps delivery for fetal distress, forceps for second stage arrest, and spontaneous delivery. Int J Gynaecol Obstet 2010;111:19-22.
35. Shek KL, Dietz HP. The effect of childbirth on hiatal dimensions. Obstet Gynecol 2009;113:1272-8.
36. Hay-Smith J, Morkved S, Fairbrother KA, Herbison GP. Pelvic floor muscle training for prevention and treatment of urinary and fecal incontinence in antenatal and postnatal women. Cochrane Database Syst Rev 2008;4:CD007471.
37. 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;23:707-14.

Paper III

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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

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Objective To investigate ability to contract, vaginal resting pressure (VRP), pelvic floor muscle (PFM) strength and PFM endurance 6 weeks after vaginal delivery in primiparous women, with and without major defects of the levator ani (LA) muscle.

Design Cross-sectional comparative study.

Setting Akershus University Hospital, Norway.

Sample A cohort of 175 singleton primiparous women delivering vaginally after more than 32 weeks of gestation.

Methods Major LA defects were assessed by 3D/4D transperineal ultrasound at maximal PFM contraction, using tomographic imaging. VRP, PFM strength and PFM endurance were measured vaginally by manometer. Data were analysed by independent-samples Student's *t* test, chi-square test, and standard multiple and simple linear regression.

Main outcome measures VRP, PFM strength and PFM endurance.

Results Of the women included in the study, 4% were not able to contract their PFM 6 weeks after delivery. Women with major LA defects (n = 55) had 47% lower PFM strength and 47% lower endurance when compared with women without major LA defects (n = 120). Mean differences were 7.5 cmH_2O (95% CI 5.1–9.9, $P < 0.001$) and 51.2 cmH_2O seconds (95% CI 32.8–69.6, $P < 0.001$), respectively. These estimates were unchanged by adjustment in multivariable linear regression for potentially confounding demographic and obstetric factors. No difference was found regarding VRP (P = 0.670).

Conclusions Women with major LA defects after vaginal delivery had pronounced lower PFM strength and endurance than women without such defects; however, most women with major LA defects were able to contract the PFM. This indicates a potential capacity by non-injured muscle fibres to compensate for loss in muscle strength, even at an early stage after delivery.

Keywords Levator ani defects, muscle endurance, muscle strength, pelvic floor muscles, vaginal delivery, vaginal resting pressure.

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Introduction

Pelvic floor trauma in conjunction with vaginal delivery can involve injuries of the perineum, vagina, anal sphincter, pelvic floor muscles and innervating nerves.[1] The levator ani (LA) muscle plays a significant role for pelvic organ support and for staying continent.[2] A biomechanical study by Lien and colleagues[3] showed that muscle fibres of the pubovisceral muscle (PVM), the most medial component of the LA muscle, might be stretched up to three times their resting length as the fetal head is crowning.

During recent years technical advancement within magnetic resonance and ultrasound imaging has enabled diagnosis of major defects of the LA muscle. Major defects of the LA muscle are often defined as an abnormal insertion of this muscle towards the pubic bone, visually seen as a complete loss of visible muscle attachment at this specific site, either unilaterally or bilaterally.[4–7] Major LA defects

among primiparous women delivering vaginally, have shown to appear within the range of 13–36%.[4,8,9]

Major LA defects have shown a marked effect on hiatal dimensions and pelvic organ support,[10–12] which in turn might be explanatory factors for pelvic floor dysfunction, especially pelvic organ prolapse. To date there is scant knowledge about the influence of major LA defects on vaginal resting pressure (VRP), pelvic floor muscle (PFM) strength and PFM endurance. A search on PubMed revealed only one such previous study with a clinical assessment of PFM strength in primiparous women.[13] Hence, the purpose of the present study was to assess whether women with major defects of the LA muscle after vaginal delivery were able to contract the PFM correctly. Furthermore, we investigated VRP, PFM strength and PFM endurance 6 weeks after vaginal delivery in primiparous women, with and without major defects of the LA muscle.

Methods

This is a cross-sectional study of baseline data from a randomised controlled trial (RCT) including first-time mothers 6 weeks after vaginal delivery, in which they were stratified according to whether a major defect of the LA muscle was present or not. The study took place at Akershus University Hospital, Norway. Participants were recruited from a continuing cohort study at the hospital,[14] or in conjunction with the routine medical visit at 6 weeks postpartum. Inclusion criteria were singleton primiparous women delivering vaginally after more than 32 weeks of gestation, and who could speak and understand Scandinavian languages. Women who had a prior abortion or stillbirth after gestational week 16, or who had a perineal tear graded 3b, 3c, or 4 during delivery, were excluded from the study. The reasoning for this latter exclusion criterion was that women having these severe perineal tears, including a substantial part of the anal sphincter, are routinely referred to a physiotherapist for PFM training. Ethically, these women could therefore not be randomised to the control group of the above mentioned RCT.

The study was approved by the Regional Medical Ethics Committee (REK South East 2009/289a), Norwegian Social Science Data Services (2799004), and was registered at ClinicalTrials.gov (NCT01069484). All subjects gave written informed consent before entering the study.

Ability to contract, VRP, PFM strength and PFM endurance

Ability to contract, VRP, PFM strength and PFM endurance were assessed with the participant in a supine crook lying position, after voiding. Contraction without any movement of the pelvis or visible contraction of the glutei, hip or abdominal muscles was emphasised.[15] The ability to perform a correct PFM contraction after thorough instruction, feedback and practise was assessed on the basis of palpation and observation.[16] A correct PFM contraction was defined as an inward movement and squeeze around the urethra, vagina and rectum.[15,17] Measurements of VRP, PFM strength and PFM endurance were undertaken by an air-filled vaginal balloon connected to a high-precision pressure transducer (Camtech AS, Sandvika, Norway).[15] At atmospheric pressure the balloon was set to 0 cmH_2O for each subject before it was placed into the vagina. The device was positioned with the middle of the balloon 3.5 cm inside the introitus.[18] VRP was measured with the balloon positioned in the vagina without any voluntary PFM activity. PFM strength was measured as the difference between VRP and pressure obtained at maximal voluntary contraction (MVC), and was reported as the mean of three contractions (Figure 1). The method has been found to be reliable and valid if used with simultaneous observation of inward movement of the perineum/catheter during the contraction.[15,16] PFM endurance was defined as a sustained maximal contraction, and was quantified during the first 10 seconds as the area below the measurement curve (integral calculation).[19] Endurance was measured and assessed after one attempt (Figure 1). All PFM measurements took place before assessing whether the woman had major defects of the LA muscle or not.

Major defects of the LA muscle

With the participant in the same position as for the manometer measurements, transperineal ultrasound was performed using the GE Kretz Voulson E8 (GE Healthcare AS, Oslo, Norway) with a 4–8 MHz curved array 3D/4D ultrasound transducer (RAB4-81/obstetric). Major defects

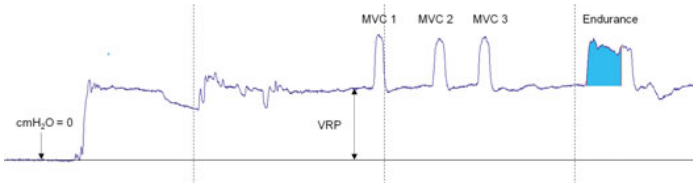

Figure 1. Manometer measurements: VRP; PFM strength, measured as the mean of three maximal voluntary contractions (MVCs 1–3); and PFM endurance, measured as one sustained maximal contraction quantified during 10 seconds (integral calculation).

of the LA muscle were assessed using tomographic imaging of the axial plane at maximal PFM contraction. The plane of minimal hiatal dimensions of the levator hiatus, defined as the plane with the shortest anterior–posterior diameter from the posterior–inferior margin of the symphysis to the rectal sling in the midsagittal plane, was used as the reference plane. Tomographic slices were obtained at 2.5-mm slice intervals, from 5 mm caudally to 12.5 mm cranially to this reference plane, producing eight slices.[7,20] A major defect of the LA muscle was diagnosed when an abnormal insertion of the muscle towards the pubic bone was present in all three central slices (Figure 2), as suggested by Dietz and colleagues[7,20]: at the plane of minimal dimension, and 2.5 and 5.0 mm cranially to it. Slices were scored as positive or negative for major LA defects by direct visualisation of the muscle attachment. In doubtful cases measurements of the levator–urethral gap was used, with measurements >2.5 cm regarded as abnormal.[7]

Two physiotherapists (G.H. and K.G.) assessed the ability to contract the PFM and performed manometer measurements, whereas two gynaecologists (J.S.J. and F.S.) performed transperineal ultrasound and assessed whether major defects of the LA muscle were apparent or not. To minimise biases, all assessors were trained ahead of the study, and a rigorous protocol for the standard of procedures was maintained.

Background data were collected through an electronic questionnaire. Delivery data were collected from the women's electronic medical birth records. All assessors were blinded to data collected from these sources.

Statistical analysis

Statistical analysis was performed using SPSS 15 (SPSS Inc, Chicago, IL, USA). Demographic characteristics and delivery data are presented as frequencies, with percentages or means with standard deviations (SD). To analyse differences between women with and without major LA muscle defects, the independent-samples Student's t test was used for continuous data, and the chi-square test was used for categorical data. Standard multiple and simple linear regression was used to control findings in PFM measurements for possible covariates. $P < 0.05$ was considered to be significant. The selection of possible covariates was based on existing literature, clinical reasoning, and between-group differences on delivery data with $P < 0.20$. If covariates intercorrelated with a Pearson correlation >0.6, the covariate selected for the model was based on correlation with dependent variables and clinical judgement. If covariates did not meet the assumption of linearity, they were to be dichotomised with the following cut-off: body mass index (BMI), 25 kg/m^2; infant birth weight, 4000 g; second stage, 60 minutes.

Results

One hundred and seventy-five primiparous women delivering vaginally were included in the study 6 weeks after delivery (mean, 6.1 weeks; SD 0.9 weeks; range, 4–9 weeks). The characteristics of the study sample are presented in Table 1. Eighty percent of the women (140 of 175) had a normal vaginal delivery, and 20% (35 of 175) had an instrumental vaginal delivery (33 with vacuum and two with forceps). Our sample had 55 women in the group diagnosed with major defects of the LA muscle, and 120 women in the group with no major defects (Table 2).

After thorough instruction, feedback, and practise on how to contract correctly, seven of the 175 women (4%) were not able to contract the PFM correctly. Four of these women had major LA muscle defects.

Women with major LA muscle defects had 47% lower PFM strength and 47% lower endurance, when compared

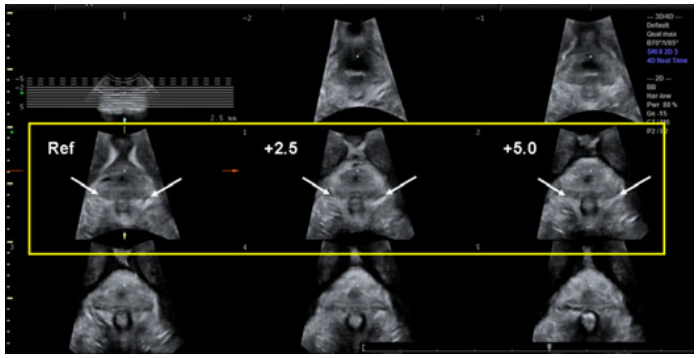

Figure 2. Major bilateral defect of the levator ani (LA) muscle 6 weeks after delivery. Tomographic ultrasound in the axial plane of the levator hiatus, obtained with a 2.5-mm slice interval, from 5 mm caudally to 12.5 mm cranially. Major LA defect visualised as abnormal insertion (arrows) present in all three central slices (slices shown within yellow border).

Table 1. Demographic characteristics and delivery data of the primiparous women participating (n = 175)

	Total sample (n = 175)	1, no major LA defects (n = 120)	2, major LA defects (n = 55)	P 1 vs 2
Demographic variables				
Age	29.8 (SD 4.1)	29.7 (4.1)	29.9 (4.0)	0.857
Prepregnancy BMI (kg/m^2)	23.8 (SD 4.0)	24.2 (4.0)	23.1 (3.8)	0.104
Education level				
College or university	143 (81.7%)	100 (83.3%)	43 (78.2%)	0.543
Primary school, high school, other	32 (18.3%)	20 (16.7%)	12 (21.8%)	
Marital status				
Married or cohabitant	166 (94.9%)	114 (95.0%)	52 (94.5%)	1.000
Single	9 (5.1%)	6 (5.0%)	3 (5.5%)	
Smoking pre-pregnancy				
No	137 (78.3%)	96 (80%)	41 (74.5%)	0.539
Yes	38 (21.7%)	24 (20%)	14 (25.5%)	
Delivery variables				
Induction				
No	152 (86.9%)	105 (87.5%)	47 (85.5%)	0.896
Yes	23 (13.1%)	15 (12.5%)	8 (14.5%)	
Episiotomy				
No	127 (72.6%)	90 (75.0%)	37 (67.3%)	0.378
Yes	48 (27.4%)	30 (25.0%)	18 (32.7%)	
Perineal tear <3b				
No	95 (54.3%)	63 (52.5%)	32 (58.2%)	0.591
Yes	80 (45.7%)	57 (47.5%)	23 (41.8%)	
IVD (forceps or vacuum)				
No	140 (80.0%)	101 (84.2%)	39 (70.9%)	0.067
Yes	35 (20.0%)	19 (15.8%)	16 (29.1%)	
Length of second stage (min)*	68.8 (46.3)	64.0 (45.6)	79.0 (46.5)	0.048
Infant birthweight (g)	3462.5 (454.2)	3413.4 (437.8)	3569.7 (474.6)	0.034
Infant head circumference (cm)**	34.8 (1.6)	34.7 (1.5)	35.1 (1.6)	0.067

Independent-sample Student's t test and chi-square test: continuous variables given as means with standard deviations (SDs); categorical variables given as numbers with percentages (%). All P-values are two-sided. LA, levator ani; BMI, body mass index; IVD, instrumental vaginal delivery.
*Missing data on three women (from group with major LA defects).
**Missing data on one infant (from group with major LA defects).

with women without major defects 6 weeks after delivery. However, there was no significant difference in VRP in women with and without major defects (Table 2). In the multiple linear regression analysis, adjustments were made for instrumental vaginal delivery, total second stage >60 minutes, infant birthweight, and pre-pregnancy BMI. Our results showed that the adjusted and crude unstandardised regression coefficients for major LA defects were similar (Table 3).

Discussion

Main findings

Most women with major defects of the LA muscle after vaginal delivery were able to contract the PFM. Women with major defects had pronounced lower PFM strength and endurance than women without major defects. No difference in VRP was found between women with or without major LA muscle defects. Similarities between adjusted and crude unstandardised regression coefficients in the linear regression analysis support the robustness of the estimates for mean differences presented in Table 2.

Strengths and weaknesses

The strengths of the present study were the inclusion of a homogeneous group regarding parity and the short time span after delivery (range, 4–9 weeks). A mix of primi- and multiparous women would have introduced the aspect that some women might have had the major defect of the LA muscle for a longer period of time. Blinding the assessors for delivery data also strengthens the study. An additional strength is the use of a high-precision tool to evaluate VRP, PFM strength and PFM endurance: a method found to be responsive, reliable, and valid.[15,16] Three-dimensional transperineal ultrasound imaging of the LA muscle has been shown to correlate well with magnetic resonance

Table 2. Vaginal resting pressure (VRP), pelvic floor muscle (PFM) strength and PFM endurance, 6 weeks after vaginal delivery in primiparous women, with and without major defects of the levator ani (LA) muscle (n = 175)

	No major LA defects (n = 120)	Major LA defects (n = 55)	Mean difference	P
VRP (cmH_2O)	29.2 (6.4)	28.7 (9.0)	0.5 (−1.8, 2.9)	0.670
PFM strength (cmH_2O)	15.9 (11.0)	8.4 (5.2)	7.5 (5.1, 9.9)	<0.001
PFM endurance (cmH_2O seconds)	109.3 (80.7)	58.1 (42.4)	51.2 (32.8, 69.6)	<0.001

PFM strength is reported as the mean of three maximal voluntary contractions. PFM endurance is reported after one attempt of sustained maximal contraction, quantified during 10 seconds. Independent-samples Student's t test: data expressed as means with standard deviation (SD), mean difference with 95% confidence interval, and corresponding P-value. All P-values are two-sided.

imaging (MRI) findings.[21] The method used for detecting major LA muscle defects, tomographic imaging, did show good intra- and inter-rater reliability shortly after childbirth in a reliability study performed on women participating in the present study.[22] However, discriminating between major LA defects and haematoma might be challenging in some cases.[23] The number of women included was based on the power calculation for the RCT in particular, and not on the present cross-sectional study. This may represent a weakness of the study. However, a substantial decrease in strength is one of the common symptoms following major muscle tears within sports injuries.[24] We therefore expected that 55 cases of major muscle defects would ensure enough statistical power to detect whether a true difference in VRP, PFM strength and PFM endurance, in women with and without major defects of the LA muscle were present.

A cross-sectional study provides data on one time point. This may represent a weakness, as one may question whether women with and without major defects of the LA after their first vaginal delivery also differed in VRP, PFM strength and PFM endurance, ahead of the delivery. In this study 139 women were recruited from a cohort study that started mid-pregnancy (mean, 21 weeks of gestation; range, 18–25 weeks of gestation). This gave us the opportunity to retrospectively look at antenatal PFM measurements performed on these 139 women (44 with and 95 without major defects at 6 weeks postpartum). An independent-

Table 3. The role of delivery variables and pre-pregnancy body mass index (BMI) on vaginal resting pressure (VRP), pelvic floor muscle (PFM) strength and PFM endurance (n = 175)

Independent variables		Dependent variables		
		VRP (cmH_2O)	PFM strength (cmH_2O)	PFM endurance (cmH_2O seconds)
	a c	Adjusted β coefficient Crude β coefficient	Adjusted β coefficient Crude β coefficient	Adjusted β coefficient Crude β coefficient
Major LA defect	a	−0.366 (−2.772 to 2.040)	−7.078 (−10.201 to −3.955)**	−50.381 (−73.314 to −27.447)**
	c	−0.509 (−2.863 to 1.845)	−7.520 (−10.609 to −4.432)**	−51.214 (−74.020 to −28.409)**
IVD	a	0.766 (−2.064 to 3.595)	−5.591 (−9.264 to −1.918)*	−42.781 (−69.752 to −15.810)*
	c	1.057 (−1.672 to 3.786)	−5.679 (−9.399 to −1.959)*	−40.200 (−67.471 to −12.929)*
Total second stage >60 min***	a	0.673 (−1.611 to 2.957)	3.563 (0.598 to 6.527)*	33.233 (11.466 to 55.000)*
	c	0.772 (−1.447 to 2.991)	1.436 (−1.645 to 4.526)	18.559 (−3.906 to 41.023)
Infant birth weight (g)	a	0.001 (−0.002 to 0.003)	<0.000 (−0.003 to 0.004)	0.006 (−0.018 to 0.030)
	c	0.001 (−0.001 to 0.004)	−0.001 (−0.004 to 0.003)	<0.000 (−0.025 to 0.025)
Pre-pregnancy BMI (kg/m^2)	a	0.434 (0.160 to 0.709)*	0.327 (−0.029 to 0.683)	1.527 (−1.089 to 4.143)
	c	0.441 (0.174 to 0.707)*	0.412 (0.034 to 0.791)*	2.116 (−0.673 to 4.905)

Standard multiple and simple linear regression: Data expressed as adjusted (a) and crude (c) unstandardised regression coefficients (β) with 95% confidence interval (CIs).
LA, levator ani; IVD, instrumental vaginal delivery using vacuum or forceps.
*P < 0.05.
**P < 0.001 (P values are two-sided).
***Missing data on three women (from group with major LA muscle defects).

samples Student's t test performed on these 139 women showed no significant differences in antenatal PFM measurements when comparing women who later were diagnosed either with or without major LA defects: not for VRP ($P = 0.745$), PFM strength ($P = 0.836$), or PFM endurance ($P = 0.399$). This cross-sectional study provides baseline data of an RCT containing two strata: women with and without major defects of the LA. Importantly, the number of women with major defects of the LA must therefore not be read as prevalence data.

Major defects of the LA muscle have been linked to pelvic organ prolapse in particular, for stress urinary incontinence the evidence is contradictory, and for fecal incontinence the evidence is limited.[25] Our results showing significant reduction in PFM strength in women with major LA muscle defects when compared with women without major defects are in line with former studies.[13,26–28] Two of these studies used dynamometers,[13,26] one used digital palpation,[27] and one used 3D/4D transperineal ultrasound.[28] Dietz and colleagues[29] found that women with major defects of the LA reported perceiving weakness of the PFM. Of the above-mentioned studies measuring PFM strength, Brincat and colleagues[13] was the one study including primiparous women only. In line with our findings they found that women with major defects of the LA had significantly lower PFM strength than women without major defects ($P = 0.050$). In contrast to our study, Brincat and colleagues[13] also found a difference between groups at rest ($P = 0.03$). A direct comparison is, however, difficult, as the time spans for remission do differ. We assessed PFM function at 6 weeks postpartum, whereas Brinkat and colleagues[13] performed the assessments at 9–12 months postpartum. Haematoma early after delivery, in combination with major LA defects, might explain why we found no group differences for VRP, in contrast to Brincat and colleagues[13]

Interpretation

Early active rehabilitation is standard treatment after musculoskeletal injury within sports medicine, and training is believed to be important in speeding up tissue healing.[24] Experimental studies have found that early mobilisation after muscle injury facilitates more rapid capillary ingrowths, improves parallel orientation of the regenerating myofibrils, improves tensile properties, and stimulates the change of satellite cells to the formation of muscle cells.[24,30,31]

Most women with major LA defects in the present study were able to contract the PFM. This indicates a potential capacity by non-injured muscle fibres to compensate for a loss in muscle strength even at this early stage after delivery. The success of PFM training in women with major muscle defects in the pelvic floor is still not known, and must be addressed in future RCTs of high interventional and methodological quality.

Conclusion

Shortly after vaginal delivery, women with major LA defects had pronounced lower PFM strength and endurance than women without such defects. However, most women with major LA defects were able to contract the PFM. This indicates a potential capacity by non-injured muscle fibres to compensate for loss in muscle strength, even at this early stage after delivery. The effect of early active rehabilitation needs to be addressed in future clinical trials.

Disclosure of interests

The authors have no conflicts of interest to declare.

Contribution to authorship

All of the authors were involved in project development and design of the study, acquisition of data and interpretation of results, and drafting the article and revising it critically. All authors approved the final version of the submitted article. KB has been the main supervisor of the study.

Details of ethics approval

Regional Committees for Medical Research Ethics, South-Eastern Norway (REK South East): ref. no. 2009/289a; 2 December 2012.

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References

1 Ashton-Miller JA, DeLancey JO. On the biomechanics of vaginal birth and common sequelae. *Annu Rev Biomed Eng* 2009;11:163–76.
2 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.
3 Lien KC, Mooney B, DeLancey JO, Ashton-Miller JA. Levator ani muscle stretch induced by simulated vaginal birth. *Obstet Gynecol* 2004;103:31–40.
4 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;101:46–53.

5 DeLancey JO, Ashton-Miller JA. 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*. London, UK: Churchill Livingstone Elsevier; 2007. pp. 93–105.

6 Dietz HP. Quantification of major morphological abnormalities of the levator ani. *Ultrasound Obstet Gynecol* 2007;29:329–34.

7 Dietz HP, Bernardo MJ, Kirby A, Shek KL. Minimal criteria for the diagnosis of avulsion of the puborectalis muscle by tomographic ultrasound. *Int Urogynecol J* 2011;22:699–704.

8 Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. *Obstet Gynecol* 2005;106:707–12.

9 Shek KL, Dietz HP. Intrapartum risk factors for levator trauma. *BJOG* 2010;117:1485–92.

10 Shek KL, Dietz HP. The effect of childbirth on hiatal dimensions. *Obstet Gynecol* 2009;113:1272–8.

11 Majida M, Brækken IH, Bø 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;23:707–14.

12 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;114:141–4.

13 Brincat CA, DeLancey JO, Miller JM. Urethral closure pressures among primiparous women with and without levator ani muscle defects. *Int Urogynecol J* 2011;22:1491–5.

14 Hilde G, Stær-Jensen J, Ellström Engh M, Brækken IH, Bø K. Continence and pelvic floor status in nulliparous women at midterm pregnancy. *Int Urogynecol J* 2012;23:1257–63.

15 Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. I. Reliability of vaginal pressure measurements of pelvic muscle strength. *Neurourol Urodyn* 1990;9:471–7.

16 Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of of female stress urinary incontinence. II. Validity of vaginal pressure measurements of pelvic muscle strength- The necessity of supplementary methods for control of correct contraction. *Neurourol Urodyn* 1990;9:479–87.

17 Kegel AH. Stress incontinence and genital relaxation; a nonsurgical method of increasing the tone of sphincters and their supporting structures. *Ciba Clin Symp* 1952;4:35–51.

18 Bø K. Pressure measurements during pelvic floor muscle contractions: the effect of different positions of the vaginal measuring device. *Neurourol Urodyn* 1992;11:107–13.

19 Braekken IH, Majida M, Ellström Engh M, Holme IM, Bø K. Pelvic floor function is independently associated with pelvic organ prolapse. *BJOG* 2009;116:1706–14.

20 Dietz HP, Shek KL. Tomographic ultrasound imaging of the pelvic floor: which levels matter most? *Ultrasound Obstet Gynecol* 2009;33:698–703.

21 Majida M, Braekken IH, Bø K, Benth JS, Engh ME. Validation of three-dimensional perineal ultrasound and magnetic resonance imaging measurements of the pubovisceral muscle at rest. *Ultrasound Obstet Gynecol* 2010;35:715–22.

22 Staer-Jensen J, Siafarikas F, Hilde G, Braekken IH, Bø K, Engh ME. Pelvic floor muscle injuries 6 weeks post partum-an intra- and inter-rater study. *Neurourol Urodyn* 2012. doi:10.1002/nau.22337. [Epub ahead of print].

23 Van Delft K, Shobeiri A, Sultan AH, Thakar R. Haematomas may masquerade as levator ani muscle defects. 37th Annual Meeting of the International Urogynecological Association, Brisbane 2012 2012.

24 Jarvinen TA, Jarvinen TL, Kaariainen M, Aarimaa V, Vaittinen S, Kalimo H, et al. Muscle injuries: optimising recovery. *Best Pract Res Clin Rheumatol* 2007;21:317–31.

25 Schwertner-Tiepelmann N, Thakar R, Sultan AH, Tunn R. Obstetric levator ani muscle injuries: current status. *Ultrasound Obstet Gynecol* 2012;39:372–83.

26 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;109:295–302.

27 Dietz HP, Shek C. Levator avulsion and grading of pelvic floor muscle strength. *Int Urogynecol J Pelvic Floor Dysfunct* 2008;19:633–6.

28 Steensma AB, Konstantinovic ML, Burger CW, de Ridder D, Timmerman D, Deprest J. Prevalence of major levator abnormalities in symptomatic patients with an underactive pelvic floor contraction. *Int Urogynecol J* 2010;21:861–7.

29 Dietz HP, Shek KL, Chantarasorn V, Langer SE. Do women notice the effect of childbirth-related pelvic floor trauma? *Aust N Z J Obstet Gynaecol* 2012;52:277–81.

30 Bodine-Fowler S. Skeletal muscle regeneration after injury: an overview. *J Voice* 1994;8:53–62.

31 Lovering R. Physical therapy and related interventions. In: Tiidus PM, editor. *Skeletal Muscle Damage and Repair*. Champaign, IL: Human Kinetics; 2007. pp. 219–30.

Paper IV

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Original Research

Postpartum Pelvic Floor Muscle Training and Urinary Incontinence

A Randomized Controlled Trial

Gunvor Hilde, PT, Jette Stær-Jensen, MD, Franziska Siafarikas, MD, Marie Ellström Engh, PhD, MD, and Kari Bø, PhD, PT

OBJECTIVE: To evaluate whether postpartum pelvic floor muscle training decrease prevalence of any urinary incontinence (UI) in primiparous women with and without UI at inclusion (mixed population) and further to perform stratified analyses on women with and without major levator ani muscle defects.

METHODS: A two-armed assessor-blinded randomized controlled trial including primiparous women 6 weeks after vaginal delivery was conducted. Participants were stratified on major levator ani muscle defects, verified by transperineal ultrasonography, and thereafter randomly allocated to training or control. All participants were taught to contract the pelvic floor muscles. The control participants received no further intervention, whereas training participants attended a weekly supervised pelvic floor muscle training class and performed daily home exercise for 16 weeks. Primary outcome was self-reported UI analyzed by relative risk.

RESULTS: We included 175 women, 55 with major levator ani muscle defects and 120 without. Prevalence of UI at baseline was 39.1% in the training group (n=87) and 50% among those in the control group (n=88). Fifteen women (8.6%) were lost to follow-up. At 6 months after delivery (postintervention), 34.5% and 38.6% reported UI in the training and control groups, respectively. Relative risk analysis of UI gave a nonsignificant effect size of 0.89 (95% confidence interval [CI] 0.60–1.32). Results were similar for the stratum with and without major levator ani muscle defects, 0.89 (95% CI 0.51–1.56) and 0.90 (95% CI 0.53–1.52), respectively.

CONCLUSIONS: Postpartum pelvic floor training did not decrease UI prevalence 6 months after delivery in primiparous women. Stratified analysis on women with and without major levator ani muscle defects showed similar nonsignificant results.

CLINICAL TRIAL REGISTRATION: ClinicalTrials.gov, www.clinicaltrials.gov, NCT01069484.

(Obstet Gynecol 2013;122:1231–8)
DOI: 10.1097/AOG.0000000000000012

LEVEL OF EVIDENCE: I

To date there is Level I evidence, Grade A recommendation that pelvic floor muscle training is effective in treatment of female stress, urgency, or mixed urinary incontinence (UI).[1] Pregnancy and especially vaginal births are established risk factors for development of UI, and stretch and tears of peripheral nerves, connective tissue, and pelvic floor muscles may contribute to weakness of the pelvic floor.[2] Mean prevalence of UI at any frequency is estimated to be 31% (95% confidence interval [CI] 30–33%) during the 3 first months postpartum after vaginal delivery.[3] This estimate showed small changes during the first year postpartum.[3]

In a recent Cochrane review it was estimated that women with UI postpartum receiving pelvic floor muscle training were 40% less likely to report UI 12 months after delivery than women receiving no treatment or usual care.[4] However, to date only four

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randomized controlled trials[5–8] and one matched controlled trial[9] have investigated the effect of postpartum pelvic floor muscle training in trials including women with and without UI, so-called mixed trials,[4] and the effect is ambiguous and unclear.[4]

Recent research using ultrasonography and magnetic resonance imaging have demonstrated that 13–36% of primiparous women may present with major defects of the levator ani muscle after vaginal delivery.[10–12] Early active rehabilitation is standard treatment after muscle injury within sports medicine, and training is believed to be important in speeding up tissue healing.[13] However, the success of pelvic floor muscle training in women with major muscle defects in the pelvic floor is still unknown.

The main aim of the present study was to evaluate whether postpartum pelvic floor muscle training decreased the prevalence of UI (any frequency) in primiparous women with and without UI at the time of inclusion (mixed population) and further to perform stratified analyses on women with and without major levator ani muscle defects.

MATERIALS AND METHODS

This assessor-blinded two-armed randomized controlled trial (pelvic floor muscle training compared with control) stratified on major levator ani muscle defects was conducted at Akershus University Hospital, Norway, from February 2010 to May 2012. Participants were recruited from a cohort study at the hospital or in conjunction with the routine medical visit 6 weeks after delivery. Inclusion criteria were singleton primiparous women who delivered vaginally after more than 32 weeks of gestation and able to speak and understand Scandinavian languages. Women having a prior abortion or stillbirth after gestational week 16, serious illness to the mother or neonate, or perineal tearing graded as 3b, 3c, or 4 were excluded. The rationale for this latter criterion was that women having these severe perineal tears are routinely referred to a physical therapist for pelvic floor muscle training. Ethically, these women could not be allocated to the control group.

The study was approved by the Regional Medical Ethics Committee (REK South East 2009/289a), Norwegian Social Science Data Services (2799004), and registered at ClinicalTrials.gov (NCT01069484). All participants gave written informed consent before entering the study.

Power calculation was based on a former study performed within a similar setting[9] showing a 67% prevalence reduction of UI in the pelvic floor muscle training group compared with a 34% reduction in the control group with 99 persons in each group. Assuming a similar difference among comparison groups with two-sided significance of $<.05$ and a power of 0.9, a total of 62 women would be needed (31 in each group). Because we planned for an additional stratified analysis among women with and without major levator ani muscle defects, respectively, and the fact that the effect of pelvic floor muscle training in women with such defects is unknown, the statistical advice was to aim for 80 women in each stratum.

The participants were evaluated by questionnaire, ultrasonography, and manometer 6 weeks after delivery (baseline) and 6 months after delivery (postintervention). Before inclusion, all participants had received a customary written leaflet from the postnatal ward before discharge containing information about and encouragement to perform regular postpartum pelvic floor muscle training. When included 6 weeks after delivery, all women received thorough individual instructions in how to perform a correct pelvic floor muscle contraction (including vaginal palpation and feedback). A correct contraction was defined as inward movement and squeeze around the pelvic floor openings[14,15] and assessed by observation and palpation.[16] All clinical examinations were performed with the participants in a standardised crook lying position.

Two gynecologists performed transperineal ultrasonography using the GE Kretz Voulson E8 system with a 4- to 8-MHz curved array three-dimensional and four-dimensional ultrasound transducer (RAB4-81/obstetric). Major defects of the levator ani muscle were diagnosed using tomographic ultrasound imaging of the axial plane at maximal pelvic floor muscle contraction. A major defect of the medial anterior part of the levator ani muscle was diagnosed when an abnormal insertion of the muscle toward the pubic bone was present at the plane of minimal dimension and 2.5 mm and 5.0 mm cranially to it, as suggested by Dietz et al.[17,18] In a reliability study performed shortly after childbirth, this diagnostic method showed good to excellent intrarater and interrater reliability.[19]

The participants were stratified on major levator ani muscle defects being present or not at the very end of the baseline assessment and thereafter randomized into two groups (training or control) in blocks of 10. The randomization sequence was computer-generated and concealed. Allocation of participants was administered outside the clinical room by a project midwife keeping the outcome assessors blinded for group allocation. After randomization, the training group

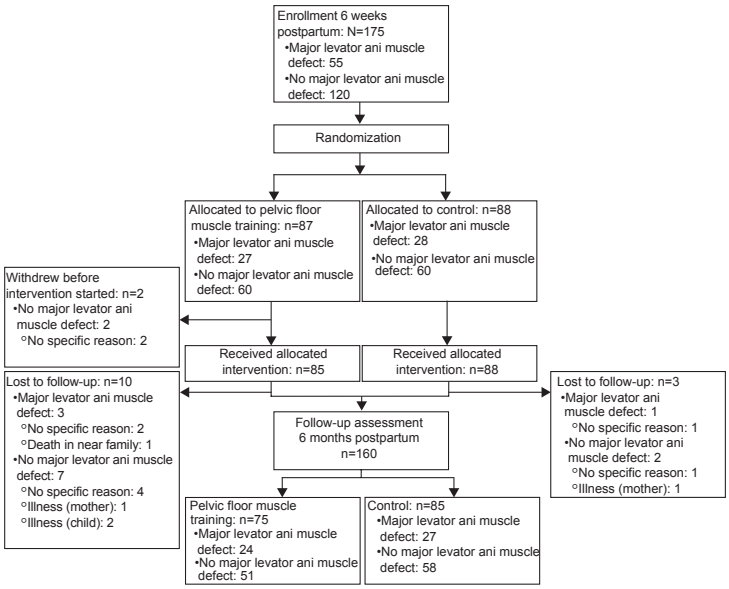

Fig. 1. Flowchart of participants through each stage of the randomized trial.

Hilde. Postpartum Pelvic Floor Muscle Training. Obstet Gynecol 2013.

attended an exercise intervention for a period of 16 weeks (starting 6-8 weeks after delivery). Once a week the training participants attended a supervised exercise class led by an experienced physical therapist. The exercise class protocol is described in detail by Bø et al[20] and Mørkved and Bø[9] Additionally, the

Table 1. Characteristics of Included Primiparous Woman at Baseline (6 Weeks After Delivery)

Characteristic	Total Sample (n=175)	Training Group (n=87)	Control Group (n=88)	One vs Two *P* Value
Age (y)	29.8±4.1	29.5±4.3	30.1±4.0	.38
BMI (kg/m^2)	25.7±4.0	26.0±4.1	25.3±3.9	.26
Education				
College or university	143 (81.7)	64 (73.6)	79 (89.8)	.01
Primary school, high school, other	32 (18.3)	23 (26.4)	9 (10.2)	
Civil status				
Married or cohabitant	166 (94.9)	80 (92.0)	86 (97.7)	.10
Single	9 (5.1)	7 (8.0)	2 (2.3)	
Major defect of the levator ani muscle	55 (31.4)	27 (31.0)	28 (31.8)	1.00
Physical activity of at least 30 min 3 times or more/wk*	49 (28.3)	20 (23.5)	29 (33.0)	.23
Pelvic floor muscle training 3 times or more/wk*	63 (36.4)	26 (30.6)	37 (42.0)	.16
UI†	78 (44.6)	34 (39.1)	44 (50.0)	.15
Once/wk or less	52 (29.7)	27 (31.0)	25 (28.4)	
2–3 times/wk	13 (7.4)	5 (5.7)	8 (9.1)	
Once/d	7 (4.0)	1 (1.1)	6 (6.8)	
Several times/d	6 (3.4)	1 (1.1)	5 (5.7)	

Data are mean±standard deviation or n (%)unless otherwise specified.
BMI, body mass index; UI, urinary incontinence.
* Total n=173; missing data on two women, both from training group (valid percent-reported).
† International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form.

Table 2. Effect of Postpartum Pelvic Floor Muscle Training on Urinary Incontinence in Primiparous Women With or Without Major Levator Ani Muscle Defects

	Total Study Sample (n=175)				Major Levator Ani Muscle Defects (n=55)			
	Training (n=87)	Control (n=88)	Between-Group Comparisons	*P*	Training (n=27)	Control (n=28)	Between-Group Comparisons	*P*
UI 6 wk after delivery	34/87 (39.1)	44/88 (50.0)	RR: 0.78 (0.56–1.09)	.15	13/27 (48.1)	14/28 (50.0)	RR: 0.96 (0.56–1.65)	.89
UI 6 mo after delivery	30/87 (34.5)	34/88 (38.6)	RR: 0.89 (0.60–1.32)	.57	12/27 (44.4)	14/28 (50.0)	RR: 0.89 (0.51–1.56)	.68
Positive pad test 6 wk after delivery	27/87 (31.0)	34/88 (38.6)	RR: 0.80 (0.53–1.21)	.29	10/27 (37.0)	12/28 (42.9)	RR: 0.86 (0.45–1.66)	.66
Positive pad test 6 wk after delivery	19/87 (21.8)	23/88 (26.1)	RR: 0.84 (0.49–1.42)	.51	11/27 (40.7)	12/28 (42.9)	RR: 0.95 (0.51–1.77)	.87
Pad test (g)* 6 wk after delivery	8.0 (2.0–46.0)	10.0 (2.0–76.0)	U: 432.00, Z: −0.40	.69	7.0 (2.0–34.0)	10.0 (2.0–38.0)	U: 55.50, Z: −0.30	.77
Pad test (g)* 6 mo after delivery	4.0 (2.0–80.0)	6.0 (2.0–114.0)	U: 213.50, Z: −0.13	.90	3.6 (2.0–80.0)	6.0 (2.0–114.0)	U: 59.50, Z: −0.41	.69

UI, urinary incontinence; RR, relative risk; U, Mann-Whitney U statistic; Z, normalized statistics of U.
Data are n/N (%), median (range) [minimum–maximum] or relative risk (95% confidence interval) unless otherwise specified.
Six weeks after delivery is baseline, and 6 months after delivery is postintervention. The principle of intention-to-treat with imputation of lost outcome data was applied when analyzing the data. Categorical data analyzed by Mantel-Haenszel risk analysis.
Positive pad test is a weighted pad above 2g
* Continuous data (only data for women with a pad test greater than 2 g) were not normally distributed and therefore analyzed by Mann-Whitney *U* test. All *P* values are two-sided.

training group was prescribed to perform daily pelvic floor muscle training at home (three sets of 8 to 12 contractions close to maximum). Training adherence at home was recorded in a training diary, whereas the physical therapist recorded group session adherence. Beyond the customary leaflet and the thorough initial instruction on how to contract correctly, the control group received no further intervention.

The primary outcome was UI assessed by The International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form. Women were assessed as incontinent if they reported to leak urine (any frequency). The International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form has been shown to have good construct validity, acceptable convergent validity, and good reliability.[21] A secondary outcome on UI was assessed by a pad test described by Mørkved and Bø[9]; the cutoff value for a positive test was 2 g.

Vaginal resting pressure, pelvic floor muscle strength, and pelvic floor muscle endurance were assessed by two physical therapists using a vaginal balloon connected to a high precision pressure transducer.[14] Vaginal resting pressure was measured without any voluntary pelvic floor muscle activity. Pelvic floor muscle strength was measured as the difference between vaginal resting pressure and pressure obtained at maximal voluntary contraction. The method has been found to be reliable and valid.[14,16] Pelvic floor muscle endurance was defined as a sustained maximal contraction quantified during the first 10 seconds.[22]

Background data were collected through electronic questionnaires, and delivery data were collected from the women's electronic medical birth records. Assessors were blinded from these data.

Statistical analysis was performed using SPSS 15/Review Manager 5.1. Within- and between-group comparisons on continuous data were analyzed by Student's *t* test if data qualified for a normal distribution. If not, Wilcoxon signed rank test or Mann-Whitney *U* test was used. χ^2 test and Mantel-Haenszel (relative risk ratio) were used to evaluate between-group differences on categorical data. *P* values $<.05$ were considered significant.

Intention to treat was the principal analysis. Missing values for continuous data were imputed by using the baseline value plus added change observed in the corresponding control group. For categorical data (self-reported UI), the approach of "last observation carried forward" was used. In addition to the overall analysis including the total study sample, stratified analyses for those with and without major levator ani muscle defects, respectively, were performed. A "per protocol analysis" was also carried out, in which training participants with an exercise adherence of less than 80%, drop outs, and participants with a new pregnancy at the clinical visit 6 months after delivery were excluded.

RESULTS

A total of 175 singleton primiparous women who delivered vaginally were included in the study 6 weeks after delivery (mean 6.1 week, standard deviation

No Major Levator Ani Muscle Defects (n=120)			
Training (n=60)	Control (n=60)	Between-Group Comparisons	*P*
21/60 (35.0)	30/60 (50.0)	RR: 0.70 (0.46–1.07)	.10
18/60 (30.0)	20/60 (33.3)	RR: 0.90 (0.53–1.52)	.70
17/60 (28.3)	22/60 (36.7)	RR: 0.77 (0.46–1.30)	.33
8/60 (13.3)	11/60 (18.3)	RR: 0.73 (0.31–1.68)	.46
8.0 (2.0–46.0)	7.0 (2.0–76.0)	U: 175.50, Z: −0.33	.74
8.0 (2.0–46.0)	6.0 (2.0–68.0)	U: 42.00, Z: −0.17	.87

0.9, range 3.9–8.7 weeks), 55 in the stratum with major levator ani muscle defects and 120 in the stratum with no such defects. Numbers of participants randomized to pelvic floor muscle training and control and the flow throughout the trial is shown in Figure 1. Characteristics of the study sample at baseline 6 weeks after delivery, before intervention started, are given in Table 1. For generalizability, the total population of primiparous women (n=2,621) scheduled for delivery at Akershus University Hospital during the inclusion period had a mean age of 28.4 years, 92.7% were married or cohabitant, and 50.8% had higher education (college or university).

Seven of the 175 women (4%) were not able to contract the pelvic floor muscles correctly at baseline. Four of them were allocated to the training arm (three having major levator ani muscle defects) and three to the control arm (one having major levator ani muscle defects). At baseline, before the intervention started, the percentage of women reporting to perform pelvic floor muscle training three times or more per week was higher in the control group than in the training group (Table 1).

At the postintervention test 6 months after delivery (mean 6.1 months, standard deviation 0.8, range 4.9–8.3 months), 15 (8.6%) women were lost to follow-up, 12 (13.8%) from the pelvic floor muscle training group and three (3.4%) from the control group (Fig. 1). No adverse effects were reported from the pelvic floor muscle training participants. Among the 15 women lost to follow-up, there was a higher percentage (46.7%) with lower education than among the 160 women completing the trial (15.6%; *P*=.01). For all other demographic variables listed in Table 1, the difference between those lost to follow-up and those completing the trial were not significant (*P*>.05).

Home training diaries and the exercise class attendance records showed that 96% of the training group participants completing the trial (72/75) reached an adherence level of 80%, both for class sessions and for daily home training. Training adherence in the control group was not registered through training diaries. However, when asked retrospectively about a weekly average of pelvic floor muscle training during the intervention period through the posttest questionnaire, 16.5% of the control participants reported to have trained three times or more per week.

Total study sample prevalence of UI at any frequency was 44.6% at baseline; 29.7% reported to leak urine once a week or less often, and 14.8% reported to leak two to three times per week or more often. The corresponding prevalence numbers postintervention were 36.6% for UI at any frequency, 26.3% for once a week or less often, and 10.3% for two to three times per week or more often. The percentages of women with UI (any frequency) in the training group and the control group at baseline and postintervention are shown in Table 1. At postintervention, the overall analysis (training compared with control, n=175) of any self-reported UI gave a nonsignificant relative risk of 0.89 (95% CI 0.60–1.32, *P*=.569). Similar figures were found in the subgroup analyses of the major levator ani muscle defect stratum (n=55) and the no major defect stratum (n=120) (Table 2). Pad test results showed no significant difference between comparison groups either (Table 2). The "per protocol analysis" did not alter the results. A total of 12 women developed UI during the study period (self-reported UI), seven from the training group (one with and six without major levator ani muscle defect) and five from the control group (three with and two without major levator ani muscle defect).

The manometer measurements showed no significant differences between comparison groups (training compared with control) at baseline or at postintervention. Mean differences at the postintervention test (overall analyses, n=175) were 1.3 cm H_2O for vaginal resting pressure (95% CI −1.0 to 3.6; *P*=.257), 3.3 cm H_2O for floor muscle strength (95% CI −1.4 to 8.0; *P*=.172), and 29.8 cm H_2O sec for endurance (95% CI −10.6.0 to 70.2, *P*=.148). Within-group analyses showed a significant increase in pelvic floor muscle strength and endurance from baseline to postintervention (*P*<.001). Strength increased by 15.7 cm H_2O within the training group and by 12.1 cm H_2O within the control group, whereas endurance increased by 145.6 cm H_2O sec and 111.7 cm H_2O sec, respectively. Similar figures were found for the strata with and without major levator ani muscle defects (Fig. 2).

DISCUSSION

In this study no significant effect of postpartum pelvic floor muscle training on UI in primiparous women was found 6 months after delivery. Stratified analysis on women with and without major levator ani muscle defects showed similar nonsignificant results.

Strengths of the present study were stratification on major levator ani muscle defects, blinding of outcome assessors, use of a validated and reliable questionnaire to assess self-reported UI,[21] and a high precision tool to evaluate vaginal resting pressure, pelvic floor muscle strength, and pelvic floor muscle endurance.[14,16] Further strengths are the use of intention-to-treat analysis, skilled physical therapists supervising the group training sessions, and the use of a training protocol based on strength training recommendations[23] shown to be successful in former studies, in treatment of UI,[20,24] in prevention of UI,[25,26] and in a mixed trial evaluating treatment and prevention of UI.[9] A limitation is that the dropout is probably not random, because 12 women dropped out from the training group but only three from the control group. An imbalance between comparison groups on reported UI at baseline may also present a limitation, but the difference was not statistically significant. The statistical advice was to aim for 80 women with major levator ani defects, but we managed to include only 55, which may present a limitation for the subgroup analyses. In general our effect estimates have wide CIs as a result of the rather optimistic effect size planned for. However, the between-group differences were minimal or nonexistent, and a type 2 error is therefore unlikely. A limitation for generalizability of our overall analyses (n=175) may be that the present study had more women major levator ani defects and higher education when compared with the general primiparous population.

Our findings, showing no significant effect of postpartum pelvic floor muscle training on prevention and treatment of UI, are in line with three former randomized controlled trials,[5,6,8] but in contrast to the randomized trial by Chiarelli and Cockburn[7] and the matched controlled study by Mørkved and Bø.[9]

Chiarelli and Cockburn[7] evaluated efficacy of adherence strategies and the "health belief model," and found a significant effect in favor of an intervention containing two educational practice sessions led by a physical therapist and a booklet to promote postpartum pelvic floor muscle training. They included 820 women, and, in contrast to our study, their participants were primiparous and multiparous women with vacuum-assisted delivery whose neonates had birth weights above 4,000 g.

The pelvic floor intervention in the present study was the same as applied in the study by Mørkved and Bø,[9] but the findings are surprisingly different. Findings from their study give a relative risk on UI of 0.50 in favor of the pelvic floor muscle training group (95% CI 0.28–0.89). This is a statistically significant and considerably strong effect, but the confidence limits are wide. Both the present study and the study by Mørkved and Bø[9] had control groups reporting pelvic floor muscle training during the intervention period. Despite that Mørkved and Bø[9] found a significant effect both within and between groups, we did not. A direct comparison of results is limited by differences in study design. The present study has a randomized and assessor-blinded design, included only primiparous women, and assessed UI by the International Consultation on Incontinence Questionnaire Urinary Incontinence Short Form. Mørkved and Bø[9] had a matched controlled design, the study was not assessor-blinded, they included a mix of primiparous and multiparous women, and assessed UI by a structured interview. The estimated effect size of an intervention might be influenced by the methodologic design applied. It has been shown that nonrandomized trials and randomized trials with inadequate allocation concealment on average tend to result in larger estimates of effect when compared with randomized trials with proper allocation concealment.[27] Further discrepancies were that 12 women dropped out from the training group in our study, whereas Mørkved and Bø[9] had no dropouts. Additionally, our study may have more women with major levator ani muscle defects as a result of the inclusion of two strata (55 with major defects and 120 without).

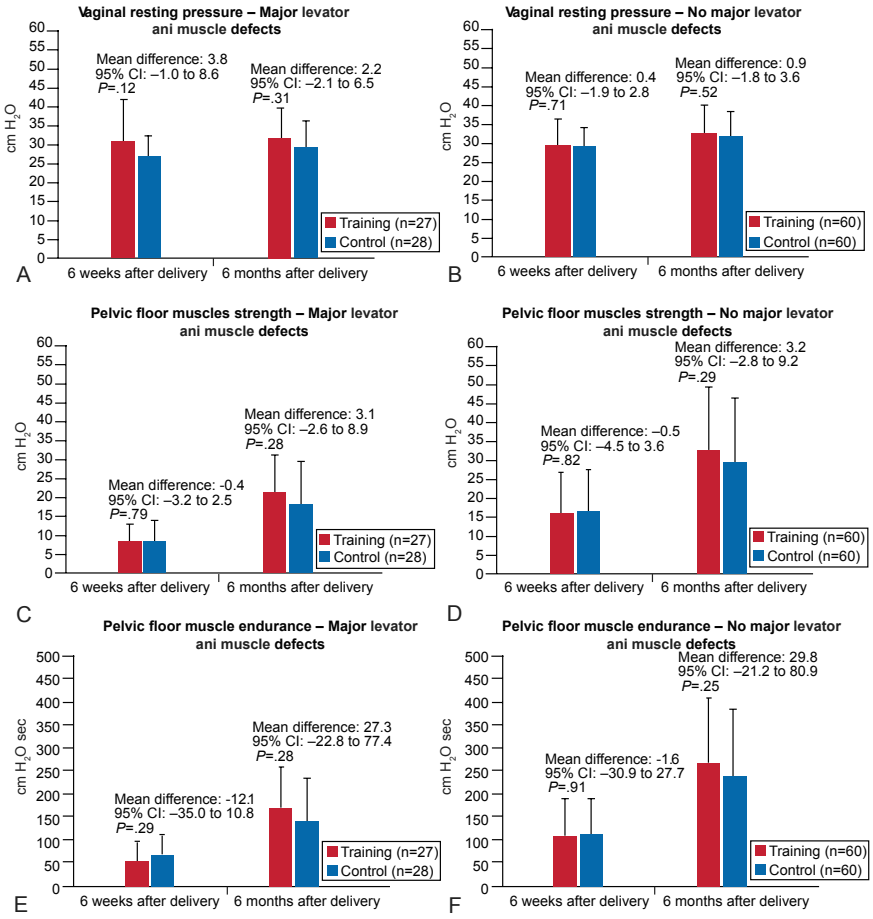

Fig. 2. Effect of postpartum pelvic floor muscle training on vaginal resting pressure (**A–B**), pelvic floor muscle strength (**C–D**), and pelvic floor muscle endurance (**E–F**). Stratified analysis on women with and without levator ani muscle defects. Six weeks after delivery is baseline and 6 months after delivery is postintervention. The principle of intention to treat with imputation of lost outcome data was applied when analyzing the data. Data are mean with standard deviation. Between-group differences analyzed by independent-samples *t* test; data expressed as mean difference with 95% confidence interval (CI) and corresponding *P* value. All *P* values are two-sided.

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A prevalence of 34.5% in the pelvic floor muscle training group and 38.6% in the control group must be considered high 6 months after delivery. Our results showing no effect of postpartum pelvic floor muscle training on UI in the early postpartum period have to be interpreted with caution because they seem contraintuitive, and the long-term effect of our intervention remains to be reported. However, our results blends in with the results from former randomized controlled trials on postpartum pelvic floor muscle training including women with and without UI (mixed trials). They seem to be less successful than trials aiming either at prevention or treatment. Future trials should therefore probably be more targeted toward certain groups of women.[1] An individual supervised exercise intervention might be more successful than a class-based intervention when targeting for instance women with major muscle defect, poor pelvic floor muscle function, or more severe UI.

REFERENCES

1. Abrams P, Cardozo L, Khoury S, Wein A, eds. Incontinence. 5th International Consultation on Incontinence, Paris; February 2012;1108–37.
2. Ashton-Miller JA, DeLancey JO. On the biomechanics of vaginal birth and common sequelae. Annu Rev Biomed Eng 2009; 11:163–76.
3. Thom DH, Rortveit G. Prevalence of postpartum urinary incontinence: a systematic review. Acta Obstet Gynecol Scand 2010;89:1511–22.
4. Boyle R, Hay-Smith EJ, Cody JD, Morkved S. Pelvic floor muscle training for prevention and treatment of urinary and faecal incontinence in antenatal and postnatal women. The Cochrane Database of Systematic Reviews 2012, Issue 10. Art. No.:CD007471. DOI: 10.1002/14651858.CD007471.pub2.
5. Sleep J, Grant A. Pelvic floor exercises in postnatal care. Midwifery 1987;3:158–64.
6. Meyer S, Hohlfeld P, Achtari C, De GP. Pelvic floor education after vaginal delivery. Obstet Gynecol 2001;97:673–7.
7. Chiarelli P, Cockburn J. Promoting urinary continence in women after delivery: randomised controlled trial. BMJ 2002; 324:1241.
8. Ewings P, Spencer S, Marsh H, O'Sullivan M. Obstetric risk factors for urinary incontinence and preventative pelvic floor exercises: cohort study and nested randomized controlled trial. J Obstet Gynaecol 2005;25:558–64.
9. Mørkved S, Bø K. The effect of postpartum pelvic floor muscle exercise in the prevention and treatment of urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct 1997;8:217–22.
10. 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;101:46–53.
11. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. Obstet Gynecol 2005;106:707–12.
12. Shek KL, Dietz HP. Intrapartum risk factors for levator trauma. BJOG 2010;117:1485–92.
13. Jarvinen TA, Jarvinen TL, Kaariainen M, Aarimaa V, Vaittinen S, Kalimo H, et al. Muscle injuries: optimising recovery. Best Pract Res Clin Rheumatol 2007;21:317–31.
14. Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of female stress urinary incontinence. I. Reliability of vaginal pressure measurements of pelvic muscle strength. Neurourol Urodyn 1990;9:471–7.
15. Kegel AH. Stress incontinence and genital relaxation; a nonsurgical method of increasing the tone of sphincters and their supporting structures. Ciba Clin Symp 1952;4:35–51.
16. Bø K, Hagen RH, Kvarstein B, Larsen S. Pelvic floor muscle exercise for the treatment of female stress urinary incontinence. II. Validity of vaginal pressure measurements of pelvic muscle strength–the necessity of supplementary methods for control of correct contraction. Neurourol Urodyn 1990;9:479–87.
17. Dietz HP, Bernardo MJ, Kirby A, Shek KL. Minimal criteria for the diagnosis of avulsion of the puborectalis muscle by tomographic ultrasound. Int Urogynecol J 2011;22:699–704.
18. Dietz HP, Shek KL. Tomographic ultrasound imaging of the pelvic floor: which levels matter most? Ultrasound Obstet Gynecol 2009;33:698–703.
19. Staer-Jensen J, Siafarikas F, Hilde G, Braekken IH, Bø K, Engh ME. Pelvic floor muscle injuries 6 weeks post partum–an intra- and inter-rater study. Neurourol Urodyn 2013;32: 993–7.
20. Bø K, Hagen RH, Kvarstein B, Jørgensen J, Larsen S. Pelvic floor muscle exercise for the treatment of female stress urinary incontinence. III. Effects of two different degrees of pelvic floor muscle exercises. Neurourol Urodyn 1990;9:489–502.
21. Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P. ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn 2004; 23:322–30.
22. Braekken IH, Majida M, Ellström Engh M, Holme IM, Bø K. Pelvic floor function is independently associated with pelvic organ prolapse. BJOG 2009;116:1706–14.
23. Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee IM, et al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc 2011;43: 1334–59.
24. Bø K, Talseth T, Holme I. Single blind, randomised controlled trial of pelvic floor exercises, electrical stimulation, vaginal cones, and no treatment in management of genuine stress incontinence in women. BMJ 1999;318:487–93.
25. Mørkved S, Bø K, Schei B, Salvesen KA. Pelvic floor muscle training during pregnancy to prevent urinary incontinence: a single-blind randomized controlled trial. Obstet Gynecol 2003;101:313–9.
26. Stafne S, Salvesen K, Romundstad P, Torjusen I, Mørkved S. Does regular exercise including pelvic floor muscle training prevent urinary and anal incontinence during pregnancy? A randomised controlled trial. BJOG 2012;119:1270–80.
27. Kunz R, Vist G, Oxman AD. Randomisation to protect against selection bias in healthcare trials. The Cochrane Database of Systematic Reviews 2007, Issue 2. Art. No.: MR000012. DOI: 10.1002/14651858.MR000012.pub2.

Appendix 1

Cohort study
Letters of approval

Regional Committees for Medical Research Ethics
Norwegian Social Science Data Services

UNIVERSITETET I OSLO

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Dato: 04.08.09
Deres ref.:
Vår ref.: 2009/170

Vedr. svar på merknader for studien "Bekkenbunn skader ved fødsel målet med tre og fire-dimensjonalt ultralyd."

Vi viser til svar på merknader av 13.07.09. med følgende vedlegg: Revidert informasjonsskriv

Komiteen behandlet svar på merknader 04.08.09. Prosjektet er vurdert etter gjeldende regelverk med tilhørende forskrifter.

Komiteen finner at våre merknader er tilfredsstillende besvart. Komiteen kan imidlertid ikke ta standpunkt til spørsmålet om fremlegging er nødvendig for å foreta en ekstra undersøkelse uten å motta supplerende opplysninger. I praksis kan dette sannsynligvis håndteres som en enkel endringssøknad.

Vedtak:
Prosjektet godkjennes som nå fremlagt. Eventuell endring av prosjektet imøtesees i form av en endringssøknad.

REK har gått over til elektronisk saksbehandling og fått ny saksportal: http://helseforskning.etikkom.no. Vi ber om at svar på merknader og henvendelser til REK sendes inn via denne portalen eller på epost: post@helseforskning.etikkom.no. Vennligst oppgi REKs saksnummer.

Med vennlig hilsen

Stein A. Evensen (sign.)
Professor dr.med.
leder

Ingrid Middelthon
komitésekretær

Brevet er godkjent elektronisk

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Dato: 13.10.2009
Deres ref.:
Vår ref.: 2009/170

Bekkenbunn skader ved fødsel målet med tre og fire-dimensjonalt ultralyd.

Vi viser til søknad av 25.09.2009 for det ovenfor nevnte forskningsprosjekt.

Prosjektleder er dr. med. Marie Ellstrøm Engh.

Forskningsansvarlig er Akershus Universitetssykehus.

Endringene innebærer:
Det søkes om å gjøre en ekstra undersøkelse av bekkenbunnen rundt 37. graviditetsuke hos de førstegangsfødende som allerede er rekruttert til studien.

Komiteen har vurdert endringssøknaden og godkjenner prosjektet slik det nå foreligger med hjemmel i helseforskningsloven § 11.

Tillatelsen er gitt under forutsetning av at prosjektet gjennomføres slik det er beskrevet i søknaden og oppdatert protokoll, og de bestemmelser som følger av helseforskningsloven med forskrifter.

Dersom det skal gjøres endringer i prosjektet i forhold til de opplysninger som er gitt i søknaden må prosjektleder sende endringsmelding til REK. Vi gjør oppmerksom på at hvis endringene er vesentlige må prosjektleder sende ny søknad, eller REK kan pålegge at dette gjøres.

Forskningsprosjektets data skal oppbevares forsvarlig, se personopplysningsforskriften kapittel 2, og Helsedirektoratets veileder for «Personvern og informasjonssikkerhet i forskningsprosjekter innenfor helse- og omsorgssektoren», http://www.norsk-helsenett.no/informasjonssikkerhet/bransjenormen/Personvern%20og%20informasjonssikkerhet%20i%20forskningsprosjekter%20v1.pdf

Vedtak:
Prosjektet godkjennes slik det nå foreligger.

REK har gått over til elektronisk saksbehandling og fått ny saksportal: http://helseforskning.etikkom.no. Vi ber om at svar på merknader og henvendelser til REK

sendes inn via denne portalen eller på epost: post@helseforskning.etikkom.no. Vennligst oppgi REKs saksnummer.

Med vennlig hilsen

Stein A. Evensen (sign.)
Professor dr.med.
leder

Ingrid Middelthon (sign.)
komitésekretær

Brevet er godkjent elektronisk

<MELDESKJEMA[1] for forskningsstudier, kvalitetssikring og annen aktivitet som medfører behandling av personopplysninger som er melde- eller konsesjonspliktig i henhold til helseregisterloven og personopplysningsloven med forskrifter. Dette gjelder også bruk av biologisk materiale i forskning.

Utfylt skjema lagres på disk og sendes til ellef.mork@ahus.no som vedlegg til e-post sammen med eventuelt informasjonsskriv. Biobank: Ta kontakt med biobankkoordinator Randi Otterstad (epost: Randi.Otterstad@ahus.no).

1 INFORMASJON OM PROSJEKTANSVARLIG OG PROSJEKTLEDER (SØKEREN)	
A. PROSJEKTANSVARLIG (div direktør/klinikksjef):	
Navn og stilling: Pål Wiik	Klinikk/avdeling: Kirurgisk Divisjon
Telefonnummer: 67969099	E-postadresse: Pal.Wiik@ahus.no
B. PROSJEKTLEDER[2]	
Navn og stilling: Marie Ellstrøm Engh, første amenuensis, overlege KK Ahus	Klinikk/avdeling hvor prosjektet gjennomføres: Kvinneklinikken
Telefonnummer: 67964541	E-postadresse: m.e.engh@medisin.uio.no
C. MULTISENTERSTUDIE	
Er prosjektet en multisenterstudie? ☐ Ja x Nei Dersom **ja**, angi øvrige virksomheter som deltar: Skal noen av disse også ha kopi av elektronisk database/informasjon som etableres i prosjektet? ☐ Ja ☐ Nei	
D. ANNEN DATABEHANDLINGSANSVARLIG ENN AKERSHUS UNIVERSITETSSYKEHUS HF[3]	
Er prosjektet organisert fra et legemiddelfirma eller annen ekstern virksomhet? ☐ Ja x Nei Dersom **ja**, angi virksomhetens navn (Kopi av konsesjonen/godkjenning skal sendes personvernombudet, og prosjektet skal meldes til personvernombudet som meldepliktig prosjekt, dvs skjemaet fylles ut med unntak av punkt 5.4): Skal den eksterne også ha kodelisten/navnelisten over deltakere? ☐ Ja ☐ Nei	
2 PROSJEKTETS NAVN/TITTEL	
Bekkenbunnsskader ved graviditet og fødsel undersøkt ved 3 og 4 dimensjonal ultralyd	
3 Finansiering av prosjektet	
☐ Nei x Ja Hvis ja – hvor (NFR, HSØ etc): HSØ og NFR Prosjektnr/kostnadsted: 2799026/ 90002	
4 BESKRIV FORMÅLET MED BEHANDLINGEN/PROSJEKTET[4]	
Formålet er å beskrive de endringene som skjer med bekkenbunnen hos gravide dels i siste halvdel av graviditeten men også etter fødselen. Disse endriger ønsker vi å sette op mot fødselsforløp og mulige tiltak under fødselen. Vi vil studere om de anatomiske forhold i bekkenbunnen kan være prediktive for fødselsforløpet og for mulige skader. Vi ønsker derved å opnå å bli bedre til å forebygge skader ved fødselen.	

5 AVKLARING FOR KONSESJON ELLER MELDING[5]

a) Kobling

x Ja, det benyttes kobling mot forskriftsregulerte registre, som for eksempel fødselsregister, kreftregister eller dødsårsaksregister, eller interne konsesjonsbelagte registre.
Hvis **ja**, beskriv hvilke registre: DIPS og PARTUS

b) Store datasett

Angi totalt antall inkluderte: 200

☐ Ja, studien inkluderer et stort omfang av personer og/eller data – dvs mer enn 5000 og/eller opplysninger av svært inngripende karakter.

c) Varighet

Angi antall år opplysningene vil bli lagret, inkludert oppbevaring for etterprøving[6]: 50

6 RETTSLIG GRUNNLAG FOR BEHANDLING AV PERSONOPPLYSNINGENE[7]

6.1 Samtykke

Skal det innhentes skriftlig samtykke fra den registrerte?	x Ja	☐ Nei
Skal det innhentes skriftlig samtykke fra andre enn den registrerte?	☐ Ja	x Nei
Skal det søkes Helsedirektoratet om unntak fra taushetsplikten?	☐ Ja	x Nei

ELLER

6.2 Intern kvalitetssikring av pasientbehandling.[8]

☐ Ja, prosjektet oppfyller helsepersonelloven § 26. Opplysningene må være slettet eller anonymisert før eventuell publisering av resultater. Må publiseres som kvalitetssikring, ikke som forskning. Det kreves ikke samtykke (ref. punkt 5.1). Personopplysningsloven § 33 4. ledd gir unntak for konsesjon, men krever melding. Det er ikke krav til samtykke, men pasienter som har reservert seg mot slik bruk av opplysningene skal respekteres.

ELLER

6.3 Annet som hjemler melding, angi årsak/hjemmel:

6.4 Andre tillatelser

X Fremleggingsplikt for De regionale komiteer for medisinsk forskningsetikk[9]

☐ Søknadsplikt til Statens legemiddelverk

☐ Bioteknologiloven kommer til anvendelse (det utføres genetiske undersøkelser hvor deltakeren gis tilbakemelding om resultatet)[10]

7 PROSJEKTPERIODE

Studiestart (dd.mm.åååå):	Studieslutt(dd.mm.åååå)[11]:	Sletting/anonymisering av data (dd.mm.åååå) 01092059
01092009	31122015	Beskriv hvordan data vil bli slettet/anonymisert: HØKH vil stå ansvarlig for sletting av Data. Projektansvarlig vil bli kontaktet før sletting av data.

8 HUMANT, BIOLOGISK MATERIALE

Medfører prosjektet bruk av humant, biologisk materiale som tas kun for denne studien eller fra en diagnostisk biobank? ☐ Ja x Nei

Dersom ja:

,

Opprettes forskningsbiobanken fra en eksisterende biobank? ☐ Ja ☐ Nei

Hvis ja, navn på biobank: Biobankregisternr.:

Opprettes forskningbiobanken som en ny biobank ? ☐ Ja ☐ Nei

Ansvarshavende person for biobanken (Biobankloven §7)[12]:

Forskningsbiobankens navn:

Forskningsbiobankens innhold (vev, blod og lignende):

Skal biologisk materiale til forskning utleveres fra Ahus til en ekstern biobankansvarlig institusjon

Dersom ja: Kontakt biobankkoordinator

Ved avsluttet prosjekt:

Hva skjer med biobankmaterialet?: ☐ Materialet destrueres ☐ Materialet føres tilbake til eksisterende biobank Annet:

Hva skjer med forskningsdata utledet av biobankmaterialet?:

9 DETALJER OM PROSJEKTETS INFORMASJONSBEHANDLING

9.1 Type personopplysninger behandlingen skal omfatte:

9.1.1 Ikke-sensitive personopplysninger

Identifikasjonsopplysninger
- X Navn, adresse, fødselsdato
- X Fødselsnummer (11 siffer)
- ☐ Fingeravtrykk, iris
- ☐ Annet:

Opplysninger om tredjepersoner
- ☐ Navn, adresse, fødselsdato
- ☐ Fødselsnummer (11 siffer)
- ☐ Annet:

9.1.2 Sensitive personopplysninger (jf. personopplysningsloven § 2 nr. 8)

Prosjektet omfatter opplysninger om
- x rasemessig eller etnisk bakgrunn, eller politisk, filosofisk eller religiøs oppfatning
- ☐ at en person har vært mistenkt, siktet, tiltalt eller dømt for en straffbar handling
- X helseforhold
- X seksuelle forhold

Presiser nærmere:

Rasemessig bakgrunn vil bli registrert, da dette kan ha betydning for elastisitet i bekkenleddene og bekkenbunnen og for organmobilitet i bekkenet.

Helseopplysninger vil omfatte: Sosial status, utdanning, inntekt, type yrke og grad av belastning i yrke, røykeadferd, fysisk aktivitetsnivå, BMI, gynekologiskt status inkludert gradering av prolaps (POPQ), ultralyd data av bekkenbunnsmorfologi, styrkemåling av bekkenbunnmuskultatur, subjektive symptomer fra ulike validerte spørreskjema på dysfunksjon i bekkebunnen. I tillegg vil relevante journaldata fra DIPS/PARTUS bli innhentet (f.eks. lengde på fødselsforløp, barnets hodeomkrets, eventuell instrumentell fødsel, skade i bekkenbunnen). Eksklusjonskriterier i studien er prematur fødsel (< 32 uker), alvorlig sykdom hos mor eller barn. Disse opplysningene innhentes også fra DIPS / PARTUS ved Ahus.

Seksuelle forhold: Dysfunksjon i bekkenbunnen (for eksempel inkoninens og prolaps) som følge av graviditet og fødsel kan få følger for seksualfunksjon. Standardiserte internasjonale spørreskjema vil bli benyttet for å innhente opplysninger om dette.

9.2 Utvalg

Behandlingen omfatter opplysninger om (beskriv også eventuell kontrollgruppe):

☐ Ansatte i egen virksomhet ☐ Elever/studenter/ barnehagebarn ☐ Pasienter ☐ Tilfeldig utvalgte

☐ Adgangskontrollerte ☐ Medlemmer ☐ Kunder/klienter/brukere X Seleksjonsutvalgte

☐ Friske frivillige Dersom det skal gis godtgjørelse, beskriv nærmere:

Inkluderer utvalget personer med begrenset samtykkekompetanse, eks mindreårige, demente eller annet? ☐ Ja x Nei

Dersom **ja**, forklar:

9.3 Innsamling av opplysningene

Hvordan samles personopplysningene inn?

X Manuelt x Elektronisk (bilde og tekst) ☐ Videoopptak ☐ Lydopptak ☐ Annet (beskriv hvordan):

Hvor innhentes personopplysningene fra? X Fra den registrerte selv x Annet (beskriv hvor fra): DIPS og PARTUS

Hvis uttrekk av forskningsdata, hvem er ansvarlig for uttrekk og anonymisering/avidentifisering av data:

X HØKH ☐ SEIM x Andre – oppgi hvem: Projektkoordinator, jordmor Tone Breines Simonsen

9.4 Utlevering av opplysningene

Blir personopplysningene gjort tilgjengelige/utlevert til andre? ☐ Ja x Nei

Dersom **ja**, oppgi mottakeres navn og adresse, samt hvilken rolle mottakeren har i prosjektet:

Dersom mottaker skal være databehandler må det inngås databehandleravtale.
Er det inngått slik avtale? Dersom **ja**, legg ved avtale. ☐ Ja ☐ Nei

Hva blir overført?

☐ Informasjon med navn, personnummer eller annet som entydig angir det enkelte individ

☐ Anonymisert informasjon

☐ Avidentifisert informasjon. Forklar i så fall hvordan kryssreferanseliste beskyttes dersom dette ikke er likt som i pkt 8.6:

Hvordan oversendes informasjonen?

☐ Personlig overlevering

☐ CD sendt med rekommandert post

☐ Registreres på sikret web-side hos mottaker

☐ Legges ut på sikret område for nedlasting av mottaker

☐ Annet. Nærmere beskrivelse:

9.5 Lagring og behandling av opplysninger

Hvordan lagres opplysningene?

X Elektronisk:

- X Egen forskningsserver ved Ahus
- X Lokal PC
- ☐ Annen virksomhet – oppgi hvem
- ☐ Forskningsserver ved UiO
- ☐ Annet[13]. Angi navn på server:

x På papir. Forklar hvordan dette sikres mot uvedkommende: I låst skab på Tone Breines Simonsen sit kontor

☐ På video, tape eller annet opptak. Beskriv hvordan dette er sikret og om personen kan identifiseres:

☐ Annet. Forklar:

9.6 Gjenfinning av opplysningene

Hvordan gjenfinnes opplysningene? (Bruk av direkte identifisering som personnummer og navn skal forsøkes unngått)

☐ Opplysningene lagres med navn, personnummer eller annet som entydig angir det enkelte individ

X Opplysningene lagres avidentifisert (ved bruk av krysslister, kodelister, løpenummer eller lignende)

Hvordan er krysslister/kodelister beskyttet/lagret? Forklar: Kun autorisert personell knyttet til prosjektet (Tone Breines Simonsen) har adgang til navneliste med kodenøkkel som gjør det mulig å finne tilbake til deltagere i prosjektet. Liste med navn, adresse, personnummer og mobilnummer,samt løpenummer i studien oppbevares elektronisk på HØKH sitt forskernett. Samtykke-erklæringene inkl. ark med personopplysninger oppbevares i låst skap på Tone sitt kontor

10 DATO FOR UTFYLLING

Meldeskjemaet er forelagt divisjonsdir/ klinikk-/ forskningsansvarlig x Ja

Sted og dato	**Utfylt av:**
Akershus Universitetets Sykehus, 24.09.09	Jette Stær-Jensen

11 BEHANDLING AV PERSONVERNOMBUD	
Skal det sendes søknad om konsesjon til Datatilsynet?	
Ja, det må sendes søknad om konsesjon til Datatilsynet. Jfr POL §33) ☐	
Nei, ikke nødvendig – oppgi begrunnelse: ☒ PROSJEKT GODKJENT AV REK.	
Sted og dato: AHUS 28.09.09	Navn personvernombud:

12 GODKJENNING FOR OPPRETTELSE AV REGISTER/PROSJEKT (fylles ut av direktør ved Forskningssenteret)	
Anmodning om opprettelse av forskningsregister er:	
Godkjent (skjema sendes personvernombud) ☒	
Avslått (skjema returneres avsender) ☐	
Sted og dato: Nordbyhagen 300909	Navn forskningsdirektør:

Aktuelle rutiner og retningslinjer for Forskning ved Ahus er tilgjengelig via EQS – Forskning og utvikling.

Merknader

[1] Tilpasset fra Datatilsynets meldeskjema

[2] Prosjektleder er ansvarlig for at studien formaliseres i henhold til gjeldende lovbestemmelser. Hvis prosjektleder ikke er ansatt ved Akershus universitetssykehus HF (Ahus) må det oppgis navn på den Ahus-ansatte som er ansvarlig for at studien formaliseres korrekt.

[3] For alle studier som startes i regi av Ahus og som bruker pasientdata som utgår fra Ahus vil normalt databehandlingsansvarlig være Forskingsdirektør ved Ahus. Hvis det foretas en utlevering av data til ekstern institusjon, skal navnet på denne virksomheten skrives her.

[4] Behovet for konsesjon/melding er knyttet opp til hvilket formål man har med behandlingen av personopplysningene. Pasientjournalsystemet er meldt i sin helhet, og har lovhjemlet formål. Når informasjon i journalsystemet skal benyttes til andre formål må konsesjon eller alternativt ny melding vurderes og man må angi formålet med den nye bruken/behandlingen av personopplysningene. Formulering av formålet er derfor viktig. Tilsvarende gjelder for annen innsamling og behandling av pasient-/personopplysninger. Formålet må samsvare med det som beskrives i samtykket fra hver enkelt person som deltar i studien.

[5] Ett av de tre hovedpunktene under må være oppfylt for at studien skal være meldepliktig, og unntatt fra konsesjon:

1. Prosjektet er omfattet av personopplysningsforskriften §7-27. (Punkt a må være oppfylt, samt enten b eller c)
   a) Prosjektet er tilrådd av personvernombud. For prosjekter med medisinsk eller helsefaglig forskning skal prosjektet i tillegg være godkjent av REK.
   b) Ikke stort omfang, men lang varighet og identifiserbart, eller
   c) store datasett og tilfredsstillende avidentifisert eller pseudonymisert.
2. Prosjektet/behandlingen har hjemmel i lov og utføres i regi av organ i stat eller kommune (eks. kvalitetssikring etter helsepersonellovens § 26) – se personopplysningsloven § 33, fjerde ledd.
3. Prosjektet er regulert i forskrift som spesielt angir at det er unntatt fra konsesjonsplikt eller underlag meldeplikt (f.eks. de sentrale helseregisterforskriftene)

Frafallsanalyser (analyser av fordelinger over utdanning, inntekt og ytelser m.m. blant fremmøtte og ikke-fremmøtte for å beregne betydningen av frafallet) er også unntatt dersom de er basert på samtykke.

[6] Data skal lagres i en viss tid etter at prosjektet er ferdigstilt (analyse er gjennomført) for mulig etterprøving. I forskningsstudier skal data lagres 5 år (Norsk Lægemiddelforening) etter publisering, og for klinisk utprøving skal data lagres i minst 15 år etter innsendt sluttrapport til SLV. Enkelte større tidsskrifter krever 10 års oppbevaring for etterprøving. Data kan ikke oppbevares etter prosjektslutt for kvalitetssikring. Dersom forskningsprosjektet er finansiert av Norges forskningsråd, skal sluttrapport og prosjektdata arkiveres på betryggende måte i minimum 10 år etter avslutning av prosjektet (se punkt 5.3 i Norges forskningsråds generelle kontraktsvilkår).

[7] Som hovedregel skal skriftlig informert samtykke innhentes.

[8] Kvalitetssikring er intern kvalitetskontroll av diagnostiske og behandlingsmessige metoder som har som formål å forbedre diagnostiseringen og behandlingen av pasientene ved sykehuset.

[9] Samtlige biomedisinske forskningsprosjekter hvor det inngår forsøk på mennesker, og som ikke er av en slik art at det regnes som en del av vanlig etablert behandlingsprosedyre. Det gjelder både terapeutisk og ikke-terapeutisk forskning på pasienter og friske forsøkspersoner. Det skal foreligge en hypotese og en protokoll.

[10] Når det skal gis tilbakemelding om genetiske resultater skal deltagerne informeres før, under og etter det utføres genetiske analyser. Det er ikke aktuelt å gi tilbakemelding til barn.

[11] Når prosjektet er ferdigstilt. Dette inkluderer innsamling, analyse/vurdering, artikkelskriving/konklusjon.
[12] Hver biobank skal ha en ansvarshavende person med medisinsk eller biologisk utdannelse av høyere grad. Dette vil normalt være klinikksjef eller prosjektleder .
[13] Krever gjennomføring og godkjenning av risikovurdering

Appendix 2

Randomised controlled trial
Letters of approval

Regional Committees for Medical Research Ethics
Norwegian Social Science Data Services

UNIVERSITETET I OSLO

DET MEDISINSKE FAKULTET

Professor dr.scient Kari Bø
Norges idrettshøgskole
Postboks 4014 Ullevål Stadion
0806 Oslo

Regional komité for medisinsk og helsefaglig forskningsetikk Sør-Øst A (REK Sør-Øst A)
Postboks 1130 Blindern
NO-0318 Oslo

Telefon: 22 84 46 66
Telefaks: 22 85 05 90
E-post: jorgen.hardang@medisin.uio.no
Nettadresse: http://helseforskning.etikkom.no

Dato: 29.09.09
Deres ref.:
Vår ref.: 2009/289a

2009/289a Effekt av bekkenbunnstrening etter fødsel

Vi viser til epost av 24.09.2009 vedlagt revidert informasjonsskriv samt epost av 28.09.2009 fra Gunnvor Hilde vedlagt svar på komiteens merknader.

Komiteen har ingen merknader til revidert informasjonsskriv.

Komiteen tar til orientering at vilkår for godkjenning er oppfylt

Med vennlig hilsen

Gunnar Nicolaysen (sign)
Professor
Leder

Jørgen Hardang
Komitésekretær

UNIVERSITETET I OSLO

DET MEDISINSKE FAKULTET

Professor dr.scient Kari Bø
Norges idrettshøgskole
Postboks 4014 Ullevål Stadion
0806 Oslo

Regional komité for medisinsk og helsefaglig forskningsetikk Sør-Øst A (REK Sør-Øst A)
Postboks 1130 Blindern
NO-0318 Oslo

Telefon: 22 84 46 66
Telefaks: 22 85 05 90
E-post: post@helseforskning.etikkom.no
Nettadresse: http://helseforskning.etikkom.no

Dato: 02.12.09
Deres ref.:
Vår ref.: 2009/289a

2009/289a Effekt av bekkenbunnstrening etter fødsel

Vi viser til viser til innsendte endringer per e-post for ovennevnte studie, mottatt 19.11.2009; samt e-post mottatt 26.11.2009. Vedlagt følger også en forespørsel om deltakelse i forskningsprosjektene.

Prosjektleder er Kari Bø.

Det forutsettes at forskningsansvarlig er Norges idrettshøgskole. Komiteen gjør oppmerksom på at forskningsansvarlig etter helseforskningsloven § 4 er institusjon eller annen juridisk eller fysisk person som har det overordnede ansvaret for forskningsprosjektet, og som har de nødvendige forutsetninger for å kunne oppfylle den forskningsansvarliges plikter etter denne loven.

Endringene innebærer følgende:
Endret intervensjonsvarighet fra 3 til 4 måneder, bruk av tradisjonell treningsdagbok i stedet for bruk av SMS trac, bruk av ICIQ spørreskjema i stedet for bruk av Mouritsen, i tillegg til innføringen av en PAD test.

Komiteen har vurdert endringssøknaden og har ingen forskningsetiske innvendinger mot endringen av prosjektet. Komiteen godkjenner prosjektet slik det nå foreligger med hjemmel i helseforskningsloven § 11.

Tillatelsen er gitt under forutsetning av at prosjektet gjennomføres slik det er beskrevet i søknad og protokollen, og de bestemmelser som følger av helseforskningsloven med forskrifter.

Forskningsprosjektets data skal oppbevares forsvarlig, se personopplysningsforskriftens kap. 2, og Helsedirektoratets veileder for "Personvern og informasjonssikkerhet i forskningsprosjekter innenfor helse- og omsorgssektoren" (http://www.helsedirektoratet.no/samspill/informasjonssikkerhet/norm_for_informasjonssikkerhet_i_helsesektoren_232354).

Tillatelsen gjelder til 01.05.2013. Prosjektet skal sende sluttmelding på eget skjema (se helseforskningsloven § 12) senest et halvt år etter prosjektslutt.

Med vennlig hilsen

Gunnar Nicolaysen (sign.)
Professor
Leder REK sør-øst A

Øyvind Grønnie Olsen
Jurist/rådgiver REK sør-øst
Fungerende komitésekretær

MELDESKJEMA[1] for forskningsstudier, kvalitetssikring og annen aktivitet som medfører behandling av personopplysninger som er melde- eller konsesjonspliktig i henhold til helseregisterloven og personopplysningsloven med forskrifter. Dette gjelder også bruk av biologisk materiale i forskning.

Utfylt skjema lagres på disk og sendes til ellef.mork@ahus.no som vedlegg til e-post sammen med eventuelt informasjonsskriv. Biobank: Ta kontakt med biobankkoordinator Randi Otterstad (epost: Randi.Otterstad@ahus.no).

1 INFORMASJON OM PROSJEKTANSVARLIG OG PROSJEKTLEDER (SØKEREN)

A. PROSJEKTANSVARLIG (div direktør/klinikksjef):

Navn og stilling:	Klinikk/avdeling:
Pål Wiik	Kirurgisk Divisjon
Telefonnummer:	E-postadresse:
67969099	pal.wiik@ahus.no

B. PROSJEKTLEDER[2]

Navn og stilling:	Klinikk/avdeling hvor prosjektet gjennomføres:
Kari Bø, professor, dr. scient, fysioterapeut	Kvinneklinikken
Telefonnummer:	E-postadresse:
23 26 20 09	kari.bo@nih.no

C. MULTISENTERSTUDIE

Er prosjektet en multisenterstudie? ☐ Ja xNei
Dersom **ja**, angi øvrige virksomheter som deltar:

Skal noen av disse også ha kopi av elektronisk database/informasjon som etableres i prosjektet? ☐ Ja ☐ Nei

D. ANNEN DATABEHANDLINGSANSVARLIG ENN AKERSHUS UNIVERSITETSSYKEHUS HF[3]

Er prosjektet organisert fra et legemiddelfirma eller annen ekstern virksomhet? ☐ Ja x Nei

Dersom **ja**, angi virksomhetens navn (Kopi av konsesjonen/godkjenning skal sendes personvernombudet, og prosjektet skal meldes til personvernombudet som meldepliktig prosjekt, dvs skjemaet fylles ut med unntak av punkt 5.4):

Skal den eksterne også ha kodelisten/navnelisten over deltakere? ☐ Ja ☐ Nei

2 PROSJEKTETS NAVN/TITTEL

Effekt av bekkenbunnstrening etter fødsel for kvinner med og uten skade i bekkenbunnsmuskulatur

3 Finansiering av prosjektet

☐ Nei x Ja Hvis ja – hvor (NFR, HSØ etc):

Prosjektnr/kostnadsted: 2799004 / 90005

4 BESKRIV FORMÅLET MED BEHANDLINGEN/PROSJEKTET[4]

Hensikten med denne randomiserte kontrollerte studien er å evaluere effekt av bekkenbunnstrening etter fødsel hos førstegangsfødende med og uten skade i bekkenbunn. Forekomst av inkontinens, underlivsprolaps, styrke og skadetilheling av bekkenbunnsmuskulatur er valgte effektmål.

5 AVKLARING FOR KONSESJON ELLER MELDING[5]

a) Kobling

x Ja, det benyttes kobling mot forskriftsregulerte registre, som for eksempel fødselsregister, kreftregister eller dødsårsaksregister, eller interne konsesjonsbelagte registre.
Hvis **ja**, beskriv hvilke registre: PARTUS, Dips

b) Store datasett

Angi totalt antall inkluderte: 200

☐ Ja, studien inkluderer et stort omfang av personer og/eller data – dvs mer enn 5000 og/eller opplysninger av svært inngripende karakter.

c) Varighet

Angi antall år opplysningene vil bli lagret, inkludert oppbevaring for etterprøving[6]: 50

6 RETTSLIG GRUNNLAG FOR BEHANDLING AV PERSONOPPLYSNINGENE[7]

6.1 Samtykke

Skal det innhentes skriftlig samtykke fra den registrerte?	x Ja	☐ Nei
Skal det innhentes skriftlig samtykke fra andre enn den registrerte?	☐ Ja	x Nei
Skal det søkes Helsedirektoratet om unntak fra taushetsplikten?	☐ Ja	x Nei

ELLER

6.2 Intern kvalitetssikring av pasientbehandling.[8]

☐ Ja, prosjektet oppfyller helsepersonelloven § 26. Opplysningene må være slettet eller anonymisert før eventuell publisering av resultater. Må publiseres som kvalitetssikring, ikke som forskning. Det kreves ikke samtykke (ref. punkt 5.1). Personopplysningsloven § 33 4. ledd gir unntak for konsesjon, men krever melding. Det er ikke krav til samtykke, men pasienter som har reservert seg mot slik bruk av opplysningene skal respekteres.

ELLER

6.3 Annet som hjemler melding, angi årsak/hjemmel:

6.4 Andre tillatelser

x Fremleggingsplikt for De regionale komiteer for medisinsk forskningsetikk[9]

☐ Søknadsplikt til Statens legemiddelverk

☐ Bioteknologiloven kommer til anvendelse (det utføres genetiske undersøkelser hvor deltakeren gis tilbakemelding om resultatet)[10]

7 PROSJEKTPERIODE

Studiestart (dd.mm.åååå):	Studieslutt(dd.mm.åååå)[11]:	Sletting/anonymisering av data (dd.mm.åååå): 01092059
01.09.2009	31.12.2015	Beskriv hvordan data vil bli slettet/anonymisert: HØKH vil stå ansvarlig for sletting av Data. Projektansvarlig vil bli kontaktet før sletting av data.

8 HUMANT, BIOLOGISK MATERIALE

Medfører prosjektet bruk av humant, biologisk materiale som tas kun for denne studien eller fra en diagnostisk biobank? ☐ Ja x Nei

Dersom ja:

,

Opprettes forskningsbiobanken fra en eksisterende biobank? ☐ Ja ☐ Nei

Hvis **ja**, navn på biobank: Biobankregisternr.:

Opprettes forskningbiobanken som en ny biobank ? ☐ Ja ☐ Nei

Ansvarshavende person for biobanken (Biobankloven §7)[12]:

Forskningsbiobankens navn:

Forskningsbiobankens innhold (vev, blod og lignende):

Skal biologisk materiale til forskning utleveres fra Ahus til en ekstern biobankansvarlig institusjon

Dersom ja: Kontakt biobankkoordinator

Ved avsluttet prosjekt:

Hva skjer med biobankmaterialet?: ☐ Materialet destrueres ☐ Materialet føres tilbake til eksisterende biobank Annet:

Hva skjer med forskningsdata utledet av biobankmaterialet?:

9 DETALJER OM PROSJEKTETS INFORMASJONSBEHANDLING

9.1 Type personopplysninger behandlingen skal omfatte:

9.1.1 Ikke-sensitive personopplysninger

Identifikasjonsopplysninger
- xNavn, adresse, fødselsdato
- x Fødselsnummer (11 siffer)
- ☐ Fingeravtrykk, iris
- ☐ Annet:

Opplysninger om tredjepersoner
- ☐ Navn, adresse, fødselsdato
- ☐ Fødselsnummer (11 siffer)
- ☐ Annet:

9.1.2 Sensitive personopplysninger (jf. personopplysningsloven § 2 nr. 8)

Prosjektet omfatter opplysninger om
- x rasemessig eller etnisk bakgrunn, eller politisk, filosofisk eller religiøs oppfatning
- ☐ at en person har vært mistenkt, siktet, tiltalt eller dømt for en straffbar handling
- x helseforhold
- x seksuelle forhold

Presiser nærmere:

Rasemessig bakgrunn vil bli registrert, da dette kan ha betydning for elastisitet i bekkenleddene og bekkenbunnen og for organmobilitet i bekkenet.

Helseopplysninger vil omfatte: Sosial status, utdanning, inntekt, type yrke og grad av belastning i yrke, røykeadferd, fysisk aktivitetsnivå, BMI, gynekologiskt status inkludert gradering av prolaps (POPQ), ultralyd data av bekkenbunnsmorfologi, styrkemåling av bekkenbunnmuskultatur, subjektive symptomer fra ulike validerte spørreskjema på dysfunksjon i bekkebunnen. I tillegg vil relevante journaldata fra DIPS/PARTUS bli innhentet (f.eks. lengde på fødselsforløp, barnets hodeomkrets, eventuell instrumentell fødsel, skade i bekkenbunnen). Eksklusjonskriterier i studien er prematur fødsel (< 32 uker), keisersnitt, alvorlig sykdom hos mor eller barn. Disse opplysningene innhentes også fra DIPS / PARTUS ved Ahus.

Seksuelle forhold: Dysfunksjon i bekkenbunnen (for eksempel inkoninens og prolaps) som følge av graviditet og fødsel kan få følger for seksualfunksjon. Standardiserte internasjonale spørreskjema vil bli benyttet for å innhente opplysninger om dette (ICIQ-Group: http://www.iciq.net/)

9.2 Utvalg

Behandlingen omfatter opplysninger om (beskriv også eventuell kontrollgruppe):

☐ Ansatte i egen virksomhet ☐ Elever/studenter/ barnehagebarn ☐ Pasienter ☐ Tilfeldig utvalgte

☐ Adgangskontrollerte ☐ Medlemmer ☐ Kunder/klienter/brukere x Seleksjonsutvalgte

☐ Friske frivillige Dersom det skal gis godtgjørelse, beskriv nærmere:

Inkluderer utvalget personer med begrenset samtykkekompetanse, eks mindreårige, demente eller annet? ☐ Ja x Nei

Dersom **ja**, forklar:

9.3 Innsamling av opplysningene

Hvordan samles personopplysningene inn?

x Manuelt x Elektronisk (bilde og tekst) ☐ Videoopptak ☐ Lydopptak ☐ Annet (beskriv hvordan):

Hvor innhentes personopplysningene fra? x Fra den registrerte selv x Annet (beskriv hvor fra): DIPS og PARTUS

Hvis uttrekk av forskningsdata, hvem er ansvarlig for uttrekk og anonymisering/avidentifisering av data:

xHØKH ☐ SEIM x Andre – oppgi hvem: Prosjektkoordinator Tone Breines Simonsen, jordmor, prosketkoordinator

9.4 Utlevering av opplysningene

Blir personopplysningene gjort tilgjengelige/utlevert til andre? ☐ Ja x Nei

Dersom **ja**, oppgi mottakeres navn og adresse, samt hvilken rolle mottakeren har i prosjektet:

Mottaker:

Dersom mottaker skal være databehandler må det inngås databehandleravtale.
Er det inngått slik avtale? Dersom **ja**, legg ved avtale. ☐ Ja ☐ Nei

Hva blir overført?

☐ Informasjon med navn, personnummer eller annet som entydig angir det enkelte individ

☐ Anonymisert informasjon

☐ Avidentifisert informasjon. Forklar i så fall hvordan kryssreferanseliste beskyttes dersom dette ikke er likt som i pkt 8.6:

Hvordan oversendes informasjonen? –

☐ Personlig overlevering

☐ CD sendt med rekommandert post

☐ Registreres på sikret web-side hos mottaker

☐ Legges ut på sikret område for nedlasting av mottaker

☐ Annet. Nærmere beskrivelse:

9.5 Lagring og behandling av opplysninger

Hvordan lagres opplysningene?

x Elektronisk:
- x Egen forskningsserver ved Ahus
- x Lokal PC
- ☐ Annen virksomhet – oppgi hvem
- ☐ Forskningsserver ved UiO
- ☐ Annet[13]. Angi navn på server:

x På papir. Forklar hvordan dette sikres mot uvedkommende: I låst skap (Tone Breines Simonsen sitt kontor?)

☐ På video, tape eller annet opptak. Beskriv hvordan dette er sikret og om personen kan identifiseres:

☐ Annet. Forklar:

9.6 Gjenfinning av opplysningene

Hvordan gjenfinnes opplysningene? (Bruk av direkte identifisering som personnummer og navn skal forsøkes unngått)

☐ Opplysningene lagres med navn, personnummer eller annet som entydig angir det enkelte individ

x Opplysningene lagres avidentifisert (ved bruk av krysslister, kodelister, løpenummer eller lignende)

Hvordan er krysslister/kodelister beskyttet/lagret? Kun autorisert personell knyttet til prosjektet (Tone Breines Simonsen) har adgang til navneliste med kodenøkkel som gjør det mulig å finne tilbake til deltagere i prosjektet. Liste med navn, adresse, personnummer og mobilnummer,samt løpenummer i studien oppbevares elektronisk på HØKH sitt forskernett. Samtykke-erklæringene inkl. ark med personopplysninger oppbevares i låst skap på Tone sitt kontor.

10 DATO FOR UTFYLLING

Meldeskjemaet er forelagt divisjonsdir/ klinikk-/ forskningsansvarlig x Ja

Sted og dato	**Utfylt av:**
Akershus Universitetssykehus, 24.09.09	Gunvor Hilde

11 BEHANDLING AV PERSONVERNOMBUD	
Skal det sendes søknad om konsesjon til Datatilsynet?	
Ja, det må sendes søknad om konsesjon til Datatilsynet. Jfr POL §33) ☐	
Nei, ikke nødvendig – oppgi begrunnelse: ☐ X **Med hjemmel i personopplysningsforskr § 7-12 og helseregisterloven § 36 har Datatilsynet ved oppnevning av personvernombud ved Ahus fritatt sykehuset for meldeplikten.**	
Sted og dato 20.11.2009	**Navn personvernombud:**

12 GODKJENNING FOR OPPRETTELSE AV REGISTER/PROSJEKT (fylles ut av direktør ved Forskningssenteret)	
Anmodning om opprettelse av forskningsregister er:	
Godkjent (skjema sendes personvernombud) ☒	
Avslått (skjema returneres avsender) ☐	
Sted og dato 23/11 09	**Navn forskningsdirektør:**

Aktuelle rutiner og retningslinjer for Forskning ved Ahus er tilgjengelig via EQS – Forskning og utvikling.

Merknader

[1] Tilpasset fra Datatilsynets meldeskjema

[2] Prosjektleder er ansvarlig for at studien formaliseres i henhold til gjeldende lovbestemmelser. Hvis prosjektleder ikke er ansatt ved Akershus universitetssykehus HF (Ahus) må det oppgis navn på den Ahus-ansatte som er ansvarlig for at studien formaliseres korrekt.

[3] For alle studier som startes i regi av Ahus og som bruker pasientdata som utgår fra Ahus vil normalt databehandlingsansvarlig være Forskingsdirektør ved Ahus. Hvis det foretas en utlevering av data til ekstern institusjon, skal navnet på denne virksomheten skrives her.

[4] Behovet for konsesjon/melding er knyttet opp til hvilket formål man har med behandlingen av personopplysningene. Pasientjournalsystemet er meldt i sin helhet, og har lovhjemlet formål. Når informasjon i journalsystemet skal benyttes til andre formål må konsesjon eller alternativt ny melding vurderes og man må angi formålet med den nye bruken/behandlingen av personopplysningene. Formulering av formålet er derfor viktig. Tilsvarende gjelder for annen innsamling og behandling av pasient-/personopplysninger. Formålet må samsvare med det som beskrives i samtykket fra hver enkelt person som deltar i studien.

[5] Ett av de tre hovedpunktene under må være oppfylt for at studien skal være meldepliktig, og unntatt fra konsesjon:

1. Prosjektet er omfattet av personopplysningsforskriften §7-27. (Punkt a må være oppfylt, samt enten b eller c)
   a) Prosjektet er tilrådd av personvernombud. For prosjekter med medisinsk eller helsefaglig forskning skal prosjektet i tillegg være godkjent av REK.
   b) Ikke stort omfang, men lang varighet og identifiserbart, eller
   c) store datasett og tilfredsstillende avidentifisert eller pseudonymisert.
2. Prosjektet/behandlingen har hjemmel i lov og utføres i regi av organ i stat eller kommune (eks. kvalitetssikring etter helsepersonellovens § 26) – se personopplysningsloven § 33, fjerde ledd.
3. Prosjektet er regulert i forskrift som spesielt angir at det er unntatt fra konsesjonsplikt eller underlag meldeplikt (f.eks. de sentrale helseregisterforskriftene)

Frafallsanalyser (analyser av fordelinger over utdanning, inntekt og ytelser m.m. blant fremmøtte og ikke-fremmøtte for å beregne betydningen av frafallet) er også unntatt dersom de er basert på samtykke.

[6] Data skal lagres i en viss tid etter at prosjektet er ferdigstilt (analyse er gjennomført) for mulig etterprøving. I forskningsstudier skal data lagres 5 år (Norsk Lægemiddelforening) etter publisering, og for klinisk utprøving skal data lagres i minst 15 år etter innsendt sluttrapport til SLV. Enkelte større tidsskrifter krever 10 års oppbevaring for etterprøving. Data kan ikke oppbevares etter prosjektslutt for kvalitetssikring. Dersom forskningsprosjektet er finansiert av Norges forskningsråd, skal sluttrapport og prosjektdata arkiveres på betryggende måte i minimum 10 år etter avslutning av prosjektet (se punkt 5.3 i Norges forskningsråds generelle kontraktsvilkår).

[7] Som hovedregel skal skriftlig informert samtykke innhentes.

[8] Kvalitetssikring er intern kvalitetskontroll av diagnostiske og behandlingsmessige metoder som har som formål å forbedre diagnostiseringen og behandlingen av pasientene ved sykehuset.

[9] Samtlige biomedisinske forskningsprosjekter hvor det inngår forsøk på mennesker, og som ikke er av en slik art at det regnes som en del av vanlig etablert behandlingsprosedyre. Det gjelder både terapeutisk og ikke-terapeutisk forskning på pasienter og friske forsøkspersoner. Det skal foreligge en hypotese og en protokoll.

Appendix 3

Study information and consent forms

Cohort study

Randomised controlled trial

FORESPØRSEL OM DELTAKELSE I FORSKNINGSPROSJEKTENE:

Kartlegging av hvordan bekkenbunnen påvirkes av graviditet og fødsel ved hjelp av ultralyd
&
Effekt av bekkebunnstrening etter fødsel

Bakgrunn og hensikt
Dette er et spørsmål om du som er førstegangsfødende vil delta i forskningsprosjekter for å kartlegge endringer i bekkenbunnen under graviditet, fødsel og barseltid, og videre undersøke effekt av bekkenbunnstrening etter fødsel.

Svangerskap og fødsel er først og fremst forbundet med positive forventninger og glede, men utgjør også risiko for skader i bekkenbunnen. Man vet at fødselen påvirker bekkenbunnen, men nyere undersøkelser kan tyde på at en del av forandringene også skjer i løpet av graviditeten. Ved bruk av ultralyd og måling av styrke kan man kartlegge endringer i bekkenbunnen som følge av mekanisk belasting og hormonell påvirkning. Skader i bekkenbunnen kan føre til urinlekkasje, avføringslekkasje, smerter og underlivsprolaps (fremfall/nedsynking av underlivet). Heldigvis går flesteparten av disse skadene over av seg selv, men det antas at bekkenbunnstrening kan forebygge og behandle dette ytterligere. Hensikten med studiene er:

Kartleggingsstudien: Kartlegge endringer i bekkenbunnen for å kunne forebygge og behandle skader i forbindelse med svangerskap og fødsel
Treningsstudien: Undersøke om bekkenbunnstrening kan gi bedre skadetilheling, forebygge og behandle urinlekkasje, avføringslekkasje og underlivsprolaps.

I forbindelse med rutinemessig ultralydundersøkelse i uke 18-20 vil vi spørre om du vil delta i disse studiene. Du kan velge å delta i en eller begge. Dersom du vil delta, blir du bedt om å fylle ut et spørreskjema. Dette tar ca 20 minutter.

Hva innebærer kartleggingsstudien: Vi ønsker å inkludere 200 førstegangsfødende kvinner. Første undersøkelse foregår i graviditetsuke 20-22 med oppfølgningsundersøkelser i graviditetsuke 37, 6 uker, 6 og 12 mnd etter fødsel. Undersøkelsene foregår ved gynekologisk poliklinikk på Ahus og tar ca 1 t. Du blir instruert i riktig bekkenbunnssammentrekning av fysioterapeut, som undersøker om du gjør dette riktig ved å kjenne i ytre del av skjeden. Muskelstyrke i bekkenbunnen måles ved vaginal trykkmåling.
Deretter vil gynekologen foreta en ultralydundersøkelse av bekkenbunnen der ultralydapparatet tas utvendig mot underlivet. I tillegg foretas en gynekologisk undersøkelse.

Hva innebærer treningsstudien: 200 førstegangsfødende som har født vaginalt med og uten skader i bekkenbunnen vil bli spurt om å delta. Du blir ved loddtrekning trukket ut til å være med i enten en treningsgruppe eller en kontrollgruppe. Studien innebærer de samme undersøkelsene som i kartleggingsstudien. I tillegg inngår en stresstest for urinlekkasje (PAD test). Blir du trukket ut til treningsgruppen vil du få veiledet trening i gruppe hos fysioterapeut 1 gang i uken og ellers gjennomføre daglig egentrening. Hjemmetreningen tar ca 15 min. Treningen starter 6-8 uker etter fødsel og varer i 4 måneder. Registrering av hjemmetreningen gjøres via treningsdagbok. De som kommer i kontrollgruppen vil få samme oppfølging som barselkvinner får i dag, dvs. skriftlig informasjon om bekkenbunnstrening. Gynekolog og fysioterapeut ved Ahus skal ikke vite om du er med i trenings- eller kontrollgruppen.

Mulige fordeler og ulemper: Som deltager i disse studiene vil du få en grundigere undersøkelse av bekkenbunnen og eventuelle skader enn det som er vanlig i dag. Hvis ønskelig kan du få utført rutinekontrollene i graviditetsuke 37 og 6 uker etter fødsel ved Ahus i stedet for hos jordmor / fastlege. Helsepersonell med spesialkompetanse innen kvinnehelse foretar undersøkelsene og svarer på eventuelle spørsmål. Alle vil bli instruert i riktig bekkenbunnsammentrekning. De undersøkelsene som inngår i studiene er ikke forbundet med risiko for skade eller bivirkninger hverken hos deg eller det ufødte barnet. Som deltager må du møte til flere undersøkelser enn det som er vanlig, og treningsgruppen må investere noe mer tid til trening.

Hva skjer med informasjonen om deg: Informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studiene. Behandlere og undersøkere har taushetsplikt. Alle opplysningene vil bli oppbevart avidentifisert og behandles uten navn, fødselsnummer eller andre direkte gjenkjennende opplysninger. Kun autorisert personell knyttet til prosjektet har adgang til navneliste med kodenøkkel som gjør det mulig å finne tilbake til deg. Det vil ikke være mulig å identifisere deg når studienes resultater publiseres.

I tillegg til data fra undersøkelsene hos gynekolog, fysioterapeut og spørreskjema vil vi innhente relevante journaldata (f.eks. lengde på fødselsforløp, eventuell instrumentell fødsel, barnets hodeomkrets).

Sier du ja til å delta i studiene, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har rett til å få korrigert eventuelle feil. Når studiene er avsluttet vil opplysningene bli oppbevart i en avidentifisert database i 50 år for videre oppfølgning dersom du gir tillatelse til dette. Opplysningene kan kun hentes ut hvis det blir aktuelt med en oppfølgningsstudie, i så fall vil du få en ny henvendelse om samtykke til dette.

Frivillig deltakelse: Dersom du ønsker å delta, undertegner du samtykkeerklæring (siste 2 sider). Du kan når som helst og uten å oppgi noen grunn trekke deg fra studiene. Dette vil ikke få konsekvenser for din videre behandling. Ønske du å trekke deg kontakter du Tone Breines Simonsen, jordmor ved Ahus og koordinator for studiene Kontor: 67 96 85 16, mobil: 900 68 626, e-post: tosi@ahus.no

Ansvar og finansiering. Studiene gjøres i samarbeid mellom Akershus Universitetssykehus og Norges idrettshøgskole. Studiene finansieres helt av uavhengige forskningsmidler fra Norges Forskningsråd og Helse Sør Øst. Studiene er godkjent av Regional komité for medisinsk forskningsetikk, og meldt til Personvernombudet ved Akershus Universitetssykehus.

Hovedansvarlig for kartleggingsstudien er Marie Ellström Engh, dr.med., førsteamanuensis, overlege ved Kvinneklinikken Ahus. For mer informasjon om denne studien kan du kontakte Jette Stær-Jensen, gynekolog, doktorgradsstudent ved Kvinneklinikken Ahus. Mobil 41 14 00 32, e-post: jett@ahus

Hovedansvarlig for treningsstudien er Kari Bø, dr.scient, fysioterapeut, professor ved Norges idrettshøgskole, seksjon for idrettsmedisin. For mer informasjon om denne studien kan du kontakte Gunvor Hilde, fysioterapeut, doktorgradsstudent ved Norges idrettshøgskole, seksjon for idrettsmedisinske fag. Mobil 41 36 60 45, e-post: gunvor.hilde@nih.no

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Doktorgradsstudent
gynekolog

Akershus universitetssykehus

NORGES IDRETTSHØGSKOLE

Samtykkeerklæring – Kartleggingsstudie

Jeg har mottatt skriftlig og muntlig informasjon og er villig til å delta i studien ” *Kartlegging av hvordan bekkenbunnen påvirkes av graviditet og fødsel ved hjelp av ultralyd”.*

Jeg er innforstått med at undersøkelsesdata oppbevares anonymisert i en database for fremtidig forskning

Navn…… Dato……..

Akershus universitetssykehus

NORGES IDRETTSHØGSKOLE

Samtykkeerklæring - Treningsstudie

Jeg har mottatt skriftlig og muntlig informasjon og er villig til å delta i studien ” *Effekt av bekkebunntrening etter fødsel”.*

Jeg er innforstått med at undersøkelsesdata oppbevares anonymisert i en database for fremtidig forskning

Navn…… Dato……..……

Appendix 4

Questionnaire (selected part only)

International Consultation on
Incontinence Questionnaire Urinary Incontinence Short Form (ICIQ UI SF)

Initial number

ICIQ-SF (Norwegian)

KONFIDENSIELT

DAG MÅNED ÅR

Today's date

Mange mennesker lekker urin av og til. Vi forsøker å finne ut hvor mange mennesker som lekker urin og hvor mye dette plager dem. Vi er takknemlige om du vil besvare følgende spørsmål. (Vi vil gjerne vite hvordan du har hatt det, gjennomsnittlig, de siste 4 ukene).

1 Vennligst skriv inn din fødselsdato: DAG MÅNED ÅR

2 Du er *(kryss av i korrekt firkant)***:** Kvinne ☐ Mann ☐

3 Hvor ofte lekker du urin? *(Kryss av i èn boks)*

aldri ☐	0
omtrent èn gang i uken eller sjeldnere ☐	1
2 – 3 ganger i uken ☐	2
ca. 1 gang per dag ☐	3
flere ganger per dag ☐	4
hele tiden ☐	5

4 Vi vil gjerne vite hvor mye urin du tror du lekker.
Hvor mye urin lekker du vanligvis (enten du bruker beskyttelse eller ikke)?
(Kryss av i en rute)

ikke noe ☐	0
en liten menge ☐	2
en moderat mengde ☐	4
en stor mengde ☐	6

5 Hvor mye påvirker urinlekkasje ditt hverdagsliv?
Vær vennlig, sett en ring rundt et tall mellom 0 (ikke i det hele tatt) og 10 (mye)

0 1 2 3 4 5 6 7 8 9 **10**
ikke i det hele tatt svært mye

ICI-Q score: sum scores 3+4+5 ☐☐

6 Når lekker du urin? *(Vennligst kryss av alt som passer for deg)*

aldri, jeg lekker ikke urin ☐
lekker før jeg når toalettet ☐
lekker når jeg hoster eller nyser ☐
lekker når jeg sover ☐
lekker når jeg er fysisk aktiv/trimmer ☐
lekker når jeg er ferdig med å late vannet og har tatt på meg klærne ☐
lekker uten noen opplagt grunn ☐
lekker hele tiden ☐

Mange takk for at du besvarte disse spørsmålene.