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# THE PELVIC FLOOR DURING PREGNANCY AND AFTER CHILDBIRTH, AND THE EFFECT OF PELVIC FLOOR MUSCLE TRAINING ON URINARY INCONTINENCE - A LITERATURE REVIEW

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**ABSTRACT**

Pregnancy and especially vaginal childbirth are risk factors for pelvic floor dysfunctions such as urinary incontinence (UI). The aim of this literature review was to give an overview of how the pelvic floor may be affected by pregnancy and childbirth, and further state the current evidence on pelvic floor muscle training (PFMT) on UI. 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. Trained pelvic floor muscles (PFM) may counteract the hormonally mediated increased laxity of the pelvic floor and the increased intra-abdominal pressure during pregnancy. Further, 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. Additionally, a trained PFM may recover better after childbirth as the appropriate neuromuscular motor patterns have already been learned. Evidence based guidelines recommend that pregnant women having their first child should be offered supervised PFMT, and likewise for women with persistent UI symptoms after delivery (Grade A recommendations). Conclusion: Several observational studies have demonstrated significantly higher PFM strength in continent women than in women having UI, and further that vaginal delivery weakens the PFM. Current evidence based guidelines state that PFMT can prevent and treat UI, and recommend strength training of the PFM during pregnancy and postpartum. .

## INTRODUCTION

Pregnancy and childbirth are associated with happiness and a positive life change for most women. However, pregnancy and childbirth can also be considered as risk periods for development of discomfort and morbidity especially related to the pelvic floor (1). Advancements in obstetric care during the 20$^{th}$ 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 (2).

Urinary incontinence (UI), faecal incontinence and pelvic organ prolapse are highly prevalent in the female population (1, 3) and are among the most common conditions seen in clinical gynaecological practice (4). These conditions may significantly impact the physical, psychological and social well-being of those women affected (5, 6). UI is shown to be a barrier for physical activity and exercise, (7-9), and may inhibit women from lifelong participation in regular physical activity (8, 9), which is important for maintaining health and fitness (10). A population based study conducted in Portland, Oregon, showed an 11% lifetime risk of undergoing surgery for UI or pelvic organ prolapse (11). The aim of this literature review was to give an overview of how the pelvic floor may be affected by pregnancy and childbirth, and further state the current evidence on pelvic floor muscle training (PFMT) on UI.

## METHODS

This literature review is narrative and do not have the methodological approach used when conducting systematic reviews (e.g. systematic searches, criteria for inclusion, and criteria for evaluating methodological quality).

We searched the MEDLINE/PubMed to capture relevant studies within the different topics covered in this overview. In addition we screened the reference lists of relevant studies and book chapters. To state the current evidence on PFMT on UI we searched the Cochrane Library for retrieving relevant Cochrane reviews and we searched the Cochrane Central Register of Controlled Trials for retrieving new trials not yet included in the most updated Cochrane review.

## THE PELVIC FLOOR

The urethra, bladder, vagina, uterus and bowel situated within the pelvis, are given structural support by pelvic floor structures arranged into a superficial and a deep layer of muscles and connective tissue (ligaments and fascia) (12). In addition to pelvic organ support, the pelvic floor maintains continence, permit urination, defecation, intercourse and vaginal birth (12).

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 (12). 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 (12-14).

The pelvic floor muscles (PFM) interact with the supportive ligaments and fasciae in order to maintain support of the pelvic organs, and protect the pelvic floor connective tissue from excessive loads (1, 14, 15). The function of this supportive system is illustrated by the "boat in dry dock theory" (15), where the PFM act as water in the dock floating the boat (pelvic organs) unloading the mooring (ligaments and fasciae) and holds 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 (16, 17). 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 (13, 14).

The normal baseline activity of the PFM keeps the pelvic openings closed and keeps the pelvic floor elevated in a cranial direction (1, 18). 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 this increased abdominal pressure by giving increased structural support and compression of the urethra (1, 13, 14, 19). 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 (20). This mechanical supportive potential of the PFM is demonstrated by Miller *et al* (21). 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 (21). Use of the "Knack" manoeuvre has also shown to significantly reduce urine loss among women with SUI (22, 23).

## 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 (4, 24, 25). Factors that may lead to the development of pelvic floor impairment and dysfunction in women can, according to Bump & Norton, be classified into the following four categories (4):

**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* (25) integrate factors affecting pelvic floor dysfunction into a "Integrated Lifespan Model" (Figure 1), 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 (4, 25).

## URINARY INCONTINENCE

*"Urinary incontinence (symptom)"* has been defined as "Complaint of involuntary loss of urine" (26). The subtypes, stress-, urgency- and mixed urinary incontinence have the following definitions (26):

"*Stress (urinary) incontinence*: Complaint of involuntary leakage on effort or physical exertion (e.g., sporting activities), or on sneezing or coughing."
*"Urgency (urinary) incontinence*: Complaint of involuntary loss of urine associated with urgency."
*Mixed (urinary) incontinence*: Complaint of involuntary loss of urine associated with urgency and also with effort or physical or on sneezing or coughing."

**Prevalence**

A systematic literature review by Hunskaar *et al* (27) 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% (27). 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 (28-30). According to an updated review on UI prevalence by Milsom *et al* (3), do recent epidemiological studies report estimates on UI prevalence that places within the prevalence range reported by Hunskaar *et al*. (27). The most common type of UI reported by young and middle-aged women is stress urinary incontinence (SUI), while older women are more likely to report mixed urinary Incontinence (MUI) and urgency urinary incontinence (UUI) (3, 27).

**Prevalence during pregnancy**

Studies on 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 (27). Higher period prevalence has been reported in parous than in nulliparous women (31-35). The prospective population based study (n=43279) by Wesnes *et al* (35), which was part of the Norwegian Mother and Child Cohort Study reported the following results: The 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* (36), 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. 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 *et al* (36), 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 (37). In a recent systematic review (38), in which 33 population based studies were included, the mean prevalence of UI at any frequency was estimated to 31% (95% CI 30-33%) during the three first months postpartum after vaginal delivery. This estimate showed small changes during the first postpartum year.

## ETIOLOGY AND PATHOPHYSIOLOGY OF URINARY INCONTINENCE

UI also occurs in nulliparous women. However, pregnancy and childbirth are considered main etiological factors for the development of UI (39). 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 (39). 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 (39).

### Pregnancy

Prospective observational studies have shown increased prevalence of UI from the first trimester to the second, and further into the third trimester (34, 40, 41). 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 (34). In the observational study by Hvidman *et al* (34), 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 (35) and after childbirth (42-44), which could be caused by increased intra-abdominal pressure and increased bladder pressure (45).

The PFM is considered to play a significant role in the continence control system (1, 13, 14, 19), 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 (46-53), while some studies did not find such difference (54, 55). Three of the above-mentioned studies were on pregnant women (46, 50, 53). In addition to significantly higher PFM strength, Mørkved *et al* (50) 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 (44, 56-58).

**Childbirth**

Parity seems to be an increased risk factor for UI (24, 32, 41, 59-61). In a cross-sectional study of 27 900 women, Rørtveit *et al* (60) reported a relative risk (RR) of UI on 2.2 (95% CI: 1.8 to 2.6) for primiparus women and 3.3 (95% CI: 2.4 to 4.4) for women with parity of four or more. Altman *et al* (58) included 304 primiparous women and followed them 10 years prospectively. They found vaginal delivery to be independently associated with a significant long-term increase of SUI and UUI, regardless of maternal age and number of deliveries. This is supported by Viktrup *et al* (44) 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* (62) the prevalence of postpartum SUI after caesarean section was compared with vaginal delivery. Based on data from six cross-sectional studies in this review by Press *et al*, caesarean section reduced the risk of postpartum SUI from 16% to 10% (odds ratio (OR) 0.56; 95% CI: 0.45-0.68) while data from the 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 (14). Peschers *et al* (63) 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* (64) and Dietz *et al* (65).

Meyer *et al* (66) 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 (67) 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 (67, 68).

A study on bladder neck mobility and tissue stiffness was performed by Howard *et al* (69). 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 ($p=0.002$). 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* (70) 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 (71-74). In a biomechanical study by Lien *et al* (75), 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 (73, 76, 77). Such neural impairment may alter the muscle morphology. In a study by Gilpin *et al* (78),

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 a main risk factor for weakening of the PFM (71, 73, 79-84). Due to the extensive stretching of muscle fibres and the likelihood of muscle denervation it is not surprising that vaginal delivery may lead to impaired PFM strength and endurance, and that caesarean section may protect the PFM. A PubMed search gave seven studies (46, 64, 85-89) investigating change in PFM strength from pregnancy to shortly after childbirth in relation to mode of delivery. Except from one study (87), the other six studies showed a significant reduction in PFM strength after vaginal delivery in primiparous women, 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. 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 (Figure 2) either unilaterally or bilaterally (79, 82, 93). Imaging studies have shown that major LA muscle defects among primiparous women delivering vaginally could appear in 13-36% of the women (79, 94, 95). The use of forceps (80) and length of the second stage (80, 96) 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, 97).

Decreased strength is one of the most common symptoms following muscle tears within sport injuries (98). Hence, decreased PFM strength in women with major LA muscle defects is expected, but has been sparsely investigated. A PubMed search revealed five observational

studies (99-103) in which PFM strength in women with and without LA muscle defects was assessed. Results from all five studies showed significantly reduced PFM strength in women with LA muscle defects when compared to women without such defects. Dynamometer was used for assessment of PFM strength in two of these five studies (99, 102), digital palpation in one study (100), transperineal ultrasound in one (101), and manometer in one (103). These studies did also differ in age and parity of the women included.

Major LA muscle defects have shown a marked effect on hiatal dimensions (104-106) and pelvic organ support (106) 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, 99, 107, 108), while the link between LA muscle defects and UI is debated. Two studies (79, 94) report a significant association between LA muscle defects and SUI in the postpartum period, whereas contradictory findings are reported for this link in studies on women with mixed parity and mean age > 50 years (107, 109, 110).

## MUSCLE INJURY REGENERATION

The healing process of a torn muscle has three phases: 1) the destruction phase, 2) the repair phase, and 3) the remodelling phase (98, 111, 112). 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 (98, 111, 112).

### 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 (113). 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 (98, 111, 112, 114).

## PELVIC FLOOR MUSCLE TRAINING

Several hypotheses have suggested that a trained PFM might reduce the risk of UI during pregnancy and after childbirth (115). 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 (115).

The importance of PFMT in restoring function after childbirth, was introduced as early as 1948 by Kegel (116). In an uncontrolled clinical trial from 1952, he reported that 84% of his patients with UI were cured after performing PFMT (16). According to Bø (117), 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 enhanced 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, 13, 14, 19). Miller *et al* (22) 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 (23). 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 by using perineal ultrasound in observational studies, both among nulliparous continent women (21, 118) and older incontinent parous women (21).

### 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 (117, 119). 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", (117, 119, 120). Specificity and overload are two fundamental principles that carefully must be addressed for effective strength training (119, 121).

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 (119). This compromises an inward lift and squeeze around the urethra, vagina and rectum (16, 17). Avoiding co-contraction of other muscles should be emphasised, as this may mask the actual strength of the PFM contraction being performed (119). 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 (16, 122-124), 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 (125, 126). Proper assessment, instruction and teaching on how to contract correctly, is considered as crucial in order to gain benefit from PFMT (127).

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 (121). 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 (119).

Strength training recommendations for skeletal muscles are 8-12 maximal contractions, 3-4 series, 3-4 times per week (10, 120, 128). It takes time to achieve increased PFM strength, endurance and muscle volume (119), and The American College of Sports Medicine recommends the exercise duration period to be at least 15-20 weeks (10). 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 (20, 129).

Specific recommendations for exercise during pregnancy and after childbirth have been given by several guidelines (130, 131). Strength training for large muscle groups is recommended, but the prescription of frequency, intensity and duration of strength training is vague. According to the review of guidelines performed by Evenson *et al* (130, 131), only few of them (132-135) recommend PFMT, and the recommendation is only posted after childbirth.

## EVIDENCE FOR EFFECT OF PELVIC FLOOR MUSCLE TRAINING

Based on current evidence presented in a recent Cochrane review by Dumoulin (136), PFMT is better than no treatment, placebo drug, or inactive control treatments for women with UI (any type). The current evidence support the widespread recommendation (Grade A) for offering supervised PFMT as the first-line treatment for female stress, urgency, or mixed UI (136, 137).

### During pregnancy

In the Cochrane review by Boyle *et al* (138) 22 randomised controlled trials (RCTs) involving 8485 women (4231 PFMT, 4254 controls) were included in the analysis. Results showed that pregnant women with no prior UI (prevention) who were randomised to intensive antenatal PFMT were about 30% less likely to report UI up to six months postpartum than women randomised to usual antenatal care or no PFMT (RR 0.71, 95% CI 0.54 to 0.95, combined result of 5 RCTs). The results of seven trials showed a statistically significant reduced risk of UI in late pregnancy favouring PFMT in a mixed population (women with and without incontinence symptoms at inclusion) (RR 0.74, 95% CI 0.58 to 0.94, combined result of 7 RCTs).). The findings of Boyle et al (138) on PFMT during pregnancy are supported by three recent RCTs (139-141). Two of them stated a preventive effect (140, 141) and one trial (139) stated effect on a mixed population (prevention and treatment approach). The 5$^{th}$ International Consultation on Incontinence (137) recommend that pregnant women having their first child should be offered supervised PFMT (Grade A recommendation). To date there is not enough evidence to conclude on the long term effect.

### Postpartum

The Cochrane review by Boyle (138) concluded that postnatal women with persistent UI (treatment) three months postpartum and who were randomised to PFMT were about 40% less likely to report UI 12 months postpartum than women randomised to usual antenatal care or no PFMT (RR 0.60, 95% CI 0.35 to 1.03, combined result of 3 RCTs). More intensive

programs with more supervision showed greater effect. Two recent RCTs on PFMT for treatment of UI after childbirth (142, 143) support the findings reported by Boyle et al (138).

Based on the conducted RCTs to date, the extent to which mixed prevention and treatment approaches on PFMT in the postnatal period is effective is less clear (that is, offering advice on PFMT to all pregnant or postpartum women whether they have incontinence symptoms or not. It is possible that mixed prevention and treatment approaches might be effective when the intervention is intensive enough (138). A recent RCT on postpartum PFMT including women with and without UI (144), in which the applied training protocol was based on current strength training recommendations, found no effect. The pooled effect on UI based on data from this recent trial and the former mixed prevention and treatment trials 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) (138).

**DISCUSSION**

Due to the extensive stretching of muscle fibres and the likelihood of muscle denervation it is not surprising that vaginal delivery may lead to impaired PFM strength, which is confirmed by six of seven observational studies (46, 64, 85-89) assessing change in pelvic floor muscle strength from pregnancy to after childbirth. Although the six studies (46, 64, 85, 86, 88, 89) showed a significant and marked decrease in PFM strength in primiparous women delivering vaginally, a direct comparison is difficult due to different assessment methods used, which was digital palpation (46, 85-87), manometer (64, 85, 87-89), and electromyography (86). Another difficulty for a direct comparison is the timing of assessment during pregnancy (e.g: mid pregnancy or late pregnancy) and length of recovery after delivery before the second assessment. The influence of length of recovery is shown by Peschers *et al* (85), as they found a significant improvement of PFM strength within the timespan shortly after childbirth (2-8 days) to 6-8 weeks after delivery.

Studies providing clinical data on PFM strength after vaginal delivery, have shown an aggravated decline in muscle strength when the LA muscle has been subjected to major muscle defects (99-103). Again is a direct comparison between studies difficult due to different assessment methods and different timing of the postpartum clinical assessment.

The above mentioned clinical data showing decreased PFM strength after vaginal delivery and after major LA muscle defects, the eight studies (46-53) versus two studies (54, 55) showing that continent women have stronger PFM than incontinent counterparts, and further a prevalence of UI within the range 32-64% during pregnancy (27) and 31% during the first year postpartum (38) makes a good rationale for strengthening the PFM both during pregnancy and after childbirth. Several RCTs have already addressed the effect of PFMT during pregnancy and after childbirth and their findings are summarised in the Cochrane review by Boyle *et al* (138). Boyle *at al* conclude that PFMT during pregnancy is efficient both for treatment of UI (treatment trials) and when aiming both for prevention and treatment (mixed prevention and treatment approach), whereas PFMT postpartum seems effective for treatment of UI (treatment trials) but not clear when aiming both for prevention and treatment (mixed prevention and treatment approach). Their conclusions are supported by a recent systematic review performed by Mørkved and Bø (145), and by six recent published RCTs (139-144). Two of these recent RCTs (139, 143) were included in the review by Mørkved and Bø (145).

Trials on postpartum PFMT including women with and without UI (mixed prevention and treatment approach) 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 (e.g. women with bladder neck hypermobility, a large baby, or forceps assisted delivery) (138). Further, 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 (144).

Due to a strong dose-response relationship in exercise training, the effect size from training is dependent on type of exercise, dosage of training and adherence to the exercise protocol (146). Mørkved and Bø (145) conclude that the most optimal dosage for PFMT is not known, but state that a PFMT protocol following general strength training recommendations, emphasising close to maximum contractions over a period of at least eight weeks can be recommended. On the other hand are current guidelines for physical activity vague on their recommendation regarding dosage on strength training during pregnancy, and according to Evenson *et al* (130) is more work needed to clarify the safest prescription. However, it is important to note that these guidelines are geared towards general strength straining involving large muscle groups. Exercise guidelines during pregnancy warn against activities in the

supine position due to chance of decreased cardiac output, orthostatic hypotension and increased pressure towards vena cava (130) However, PFMT can easily be performed in other positions e.g. sitting, kneeling or standing.

There has been some concern that a tight and strong pelvic floor might obstruct labor and result in instrumental delivery, perineal trauma and / or injury of peripheral nerves, connective tissue and muscles (147) Three RCTs reported that PFMT did not negatively affect ability to deliver vaginally (148-150). In addition, one large cohort study did not find any negative effect of reported PFMT during pregnancy on childbirth (151). In a smaller cohort study of 300 nulliparous women it was found that a higher vaginal resting pressure at mid-pregnancy was associated with prolonged second stage of labour, whereas PFM strength and endurance had no influence on any birth outcomes (152).

To which extent the PFM recover in the first postpartum year is of great interest. Elenskaia et al (153) 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. A recent longitudinal study by Stær-Jensen et al (154), following 300 primiparous women from mid-pregnancy to one year postpartum, used transperineal ultrasound when assessing recovery of the LA muscle in terms of levator hiatus area. Results showed that the LA muscle was able to recover from the effect of pregnancy and childbirth, although not all women recovered to their pregnancy level. The results from this longitudinal study also showed that most of the recovery occurred during the first six months postpartum.

**CONCLUSION**

Several observational studies have demonstrated significantly higher PFM strength in continent women than in women having UI, and further that vaginal delivery weakens the PFM. Current evidence based guidelines state that PFMT can prevent and treat UI, and recommend strength training of the PFM during pregnancy and postpartum.

## LIST OF ABBREVATIONS

UI: Urinary incontinence

LA: levator ani

PFM: Pelvic floor muscles

SUI: stress urinary incontinence

MUI: mixed urinary incontinence

UUI: urgency urinary incontinence

RR: relative risk

OD: odds ratio

PFMT: pelvic floor muscle training

## CONFLICT OF INTEREST.

Declared none.


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**FIGURE LIST**

**Figure 1: 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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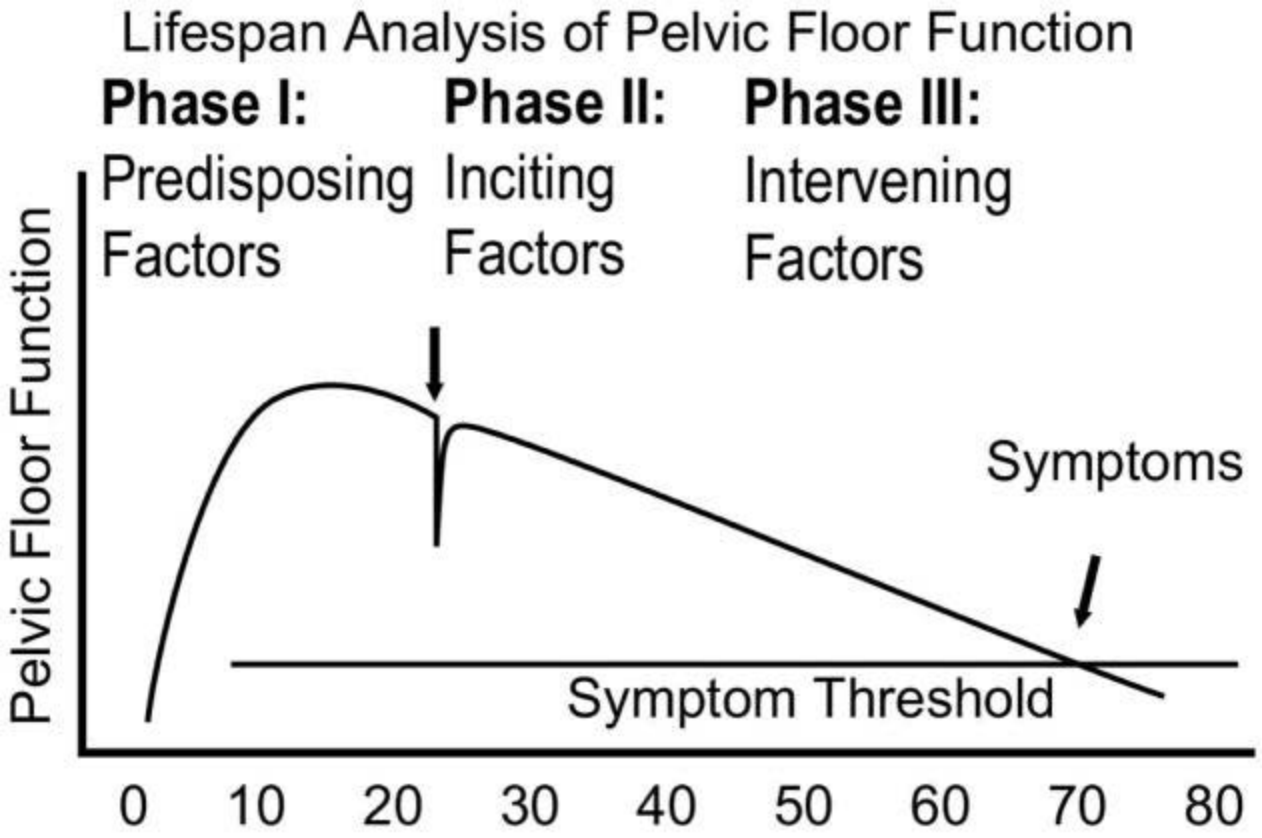
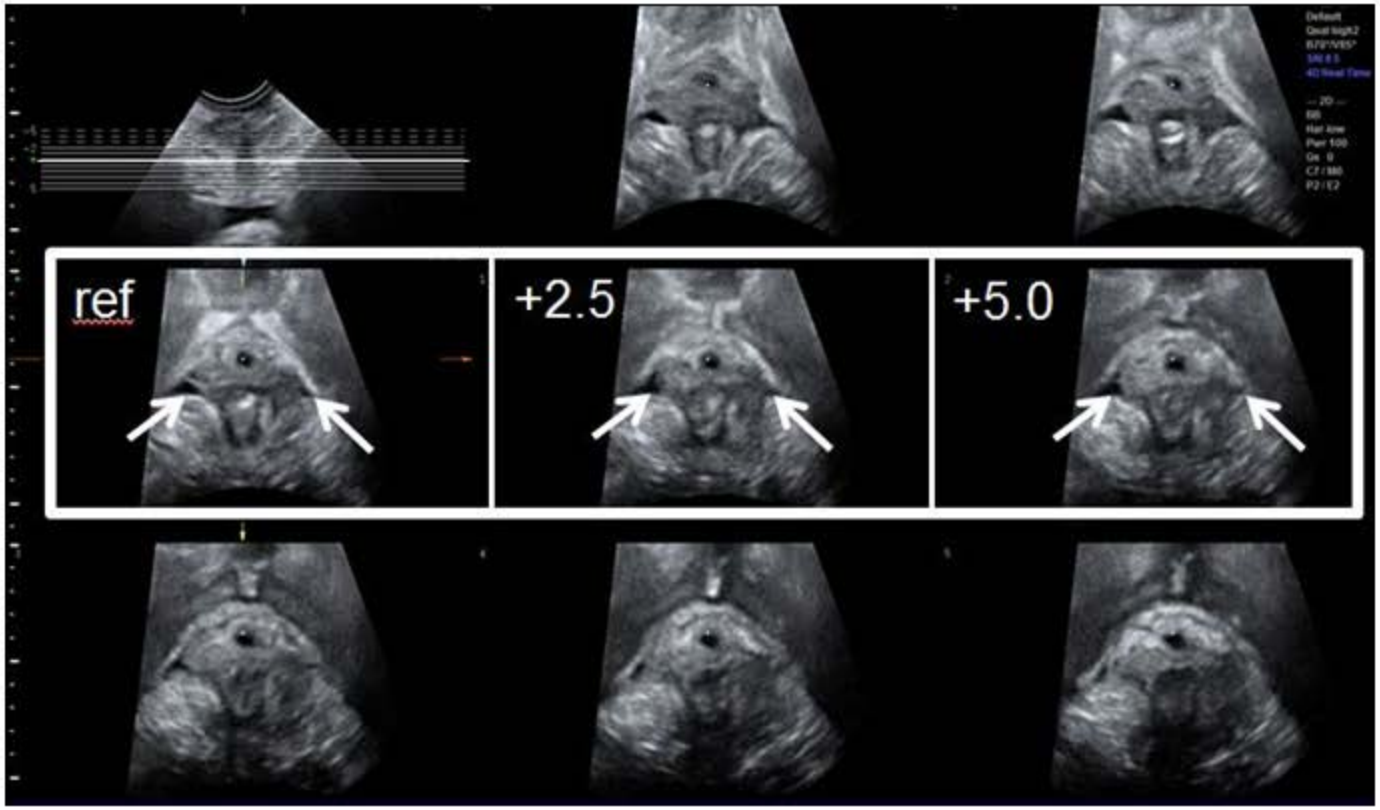
**Figure 2.** Tomographic ultrasound in the axial plane of the levator hiatus, obtained with a 2.5-mm slice interval, from 5 mm caudally to 12.5 mm cranially. Major LA defect visualized as abnormal insertion (arrows) present in all three central slices (slices shown within white rectangle).


Lifespan Analysis of Pelvic Floor Function
Phase I:
Predisposing Factors
Phase II:
Inciting Factors
Phase III:
Intervening Factors
Pelvic Floor Function
Symptoms
Symptom Threshold
0
10
20
30
40
50
60
70
80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