

The long-term effects of obstetrical anal sphincter injury on pelvic floor function

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*“There are in fact two things, science and opinion;
the former begets knowledge, the latter ignorance.”*

- Hippocrates

To all the mothers, and the mothers to-be.

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Abstract

Background: As women live longer, the long-term effects of childbirth may negatively affect their quality of life and professional careers.

Aim: This thesis investigates the long-term effects of vacuum extraction (VE) and one and two obstetrical anal sphincter injuries (OASIs). Temporal trends of OASI incidence were compared in four countries with comparable national medical birth registers and healthcare systems.

Material and methods: The study cohorts consisted of women with one (Papers I and III) or two deliveries (Paper II and IV). National birth registers were used, and in Papers I, II, IV birth register data were linked to information from a questionnaire survey on current pelvic floor disorders (PFDs).

Results:

Paper I: OASI occurred three times more often during VE than spontaneous vaginal delivery (SVD). One OASI doubled the long-term prevalence of faecal incontinence (FI), irrespective of SVD or VE. The prevalence of other PFDs was similar after SVD and VE but lower after an acute caesarean section.

Paper II: The risk for a repeat OASI almost tripled after an OASI. The long-term prevalence of all components of FI doubled and tripled after one and two OASIs. Severe FI increased 3- and 5-fold.

Paper III: In 2004-2016, the incidence of OASI in primipara varied widely over time and between countries despite similar socio-economic conditions. Canada reported the highest and Austria the lowest rate of OASI. Only Norway reported a consistent and significant decrease in OASI incidence, which more than halved during the study period.

Paper IV: There was a significant trend of more frequent leakage, more severe grades of incontinence parameters, and an increasing impact of anal incontinence after one and two OASIs. The first and the second OASIs showed an equal cumulative effect on multiple self-reported outcome measures.

Conclusion: OASI was a potent risk factor for the prevalence, severity, and impact of long-term FI. Instrumental delivery was the leading risk factor for OASI. Perineal protection, when systematically and persistently applied, may lower the rate of OASI.

Keywords: Anal incontinence, caesarean section, faecal incontinence, pelvic organ prolapse, severity, urinary incontinence, vaginal delivery.

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SAMMANFATTNING PÅ SVENSKA

Bakgrund

Med en åldrande befolkning är bäckenbottendysfunktion bland kvinnor ett problem som kommer att kräva betydande och ökande hälsovårdande insatser. Bäckenbottendysfunktion är ett samlingsbegrepp för ett antal tillstånd, exempelvis urininkontinens, avföringsinkontinens och genitalt framfall, och utgör ett stort, globalt, hälsoproblem för kvinnor i alla åldrar men särskilt bland äldre. Den viktigaste riskfaktorn för framtida bäckenbottenbesvär är att ha genomgått en vaginal förlossning. Denna avhandling undersöker effekterna på bäckenbottenfunktionen 20 år efter förlossning med sugklocka och en eller två förlossningsorsakade skador på anus ringmuskel, en så kallad sfinkterskada. Förekomsten av sfinkterskada undersöks även i fyra länder med jämförbara nationella födelseregister av hög kvalitet samt statsfinansierade sjukvårdssystem.

Kvinnor lever idag ett yrkesaktivt liv under en lång period efter barnafödandet och många drabbade kvinnor riskerar därför allvarliga störningar socialt och i sin yrkesutövning. Mellan 10-20% av alla kvinnor i västvärlden genomgår en rekonstruktiv bäckenbottenoperation före 80 års ålder och efterfrågan ökar fortsatt. Etiologin till sena störningar i bäckenbottenfunktionen är multifaktoriell. Åldrande, genetiska faktorer, paritet, utdraget förlossningsförlopp, instrumentell förlossning, och förlossningsbristningar anses ha betydelse. Den främsta riskfaktorn för avföringsinkontinens bland kvinnor är en sfinkterskada.

Metod

I Delarbete I identifierade Socialstyrelsen 9423 kvinnor ur det Svenska Medicinska Födelseregistret (MFR) som endast fött ett barn 1985-1988, vaginalt eller via kejsarsnitt. Kvinnorna tillsändes ett frågeformulär om bäckenbottensymptom 2008, vilket 5199 besvarade. Delarbete II och IV bygger på en undersökning av 11000 ur MFR slumpvis valda kvinnor som fött två barn vaginalt 1992-1998, samt samtliga kvinnor (469 st) som fått sfinkterskada vid båda sina förlossningar 1987-2000. Våren 2015 inbjöds kvinnorna till en enkätundersökning angående nuvarande bäckenbottenbesvär via Statistiska Centralbyrån. Av kvinnorna valde 7441 att delta, fördelat på kvinnor utan sfinkterskada (n=6760), sfinkterskada vid en av förlossningarna (n=357) och vid båda förlossningarna (n=324). I Delarbete I, II och IV kopplades MFR-uppgifter om graviditeter och förlossningar till respektive enkätsvar. Delarbete III bygger på registerdata från de Kanadensiska, Norska, Svenska och Österrikiska födelseregistren. Studien undersökte vaginala förstföderskor, med eller utan sugklocke- eller tångförlossning, med eller utan tidigare kejsarsnitt, gravida med bara ett foster, förlösta på sjukhus 2004-2016 i vecka 37+0 och med information om sfinkterskada (ja/nej). Totalt inkluderades 1 933 930 kvinnor, 923 357 från Kanada, 249 430 från Norge, 502 315 från Sverige och 258 828 från Österrike.

Resultat

Delarbete I: Förekomsten av sfinkterskada var nästan tre gånger så hög efter en sugklockeförlossning, jämfört med en vaginal förlossning utan

sugklocka. Efter en sfinkterskada rapporterade var tredje kvinna att hon hade avföringsläckage. Det var ingen skillnad i förekomsten av bäckenbottendysfunktion efter en förlossning med sugklocka jämfört med en spontan vaginal förlossning, men de som var förlösta med ett akut kejsarsnitt hade dock betydligt lägre förekomst av bäckenbottendysfunktion.

Delarbete II: Risken för att få sfinkterskada i en andra förlossning var nästan tre gånger så hög om man hade haft sfinkterskada vid första. Tjugo år efter den första förlossningen hade 12% av kvinnorna utan sfinkterskada någon gång avföringsläckage, 24% av dem med en och 36% av dem med sfinkterskada vid båda förlossningarna. Dessutom ökade risken för allvarligt avföringsläckage (besvärande läckage flera gånger i månaden eller mer) tre gånger efter en sfinkterskada och fem gånger efter två sfinkterskador, jämfört med kvinnor utan sfinkterskada. Mer än 80% av alla med sfinkterskada läckte gas eller avföring vid 60 års ålder.

Delarbete III: Förekomsten av sfinkterskada varierade märkbart över tid och mellan de fyra undersökta länderna. Generellt rapporterade Kanada den högsta förekomsten och Österrike den lägsta, men förekomsten ökade i båda länderna under den undersökta perioden. Från och med 2004 var det bara Norge som rapporterade en signifikant och fortsatt minskning i förekomsten av sfinkterskador, vilken till 2015 hade mer än halverats.

Delarbete IV: Två decennier efter två förlossningar med en eller två sfinkterskador hade kvinnorna en högre frekvens av läckagetillfällen, ett allvarligare läckage samt större subjektiv påverkan på sin livsföring på

grund av gas- och avföringsläckage, jämfört med kvinnor utan sfinkterskada. Den första och andra sfinkterskadan bidrog på ett kumulativt sätt till ökad svårighetsgrad och ökad subjektiv påverkan av gas- och avföringsläckage. Det fanns ingen skillnad i förekomst av annan bäckenbottendysfunktion mellan kvinnor utan sfinkterskada och kvinnor med en eller med två sfinkterskador.

Konklusion

Att ha haft sfinkterskada var associerat med en mycket hög risk för avföringsläckage två decennier efter förlossningen. Förekomsten, svårighetsgraden samt den subjektiva påverkan av gas- och avföringsläckage ökade kumulativt efter en och två sfinkterskador. Förekomsten av sfinkterskada var tre gånger så hög efter en förlossning med sugklocka, jämfört med en förlossning utan sugklocka, och även efter en första förlossning med sfinkterskada. Sfinkterskadan verkar till viss del vara undvikbar eftersom förekomsten varierade stort mellan undersökta länder. En nationell handlingsplan för återkommande och strukturerad utbildning och träning av alla personalkategorier verkar vara avgörande för att sänka förekomsten av sfinkterskador. Att undvika sfinkterskada skulle sannolikt signifikant minska förekomsten, svårighetsgraden och den subjektiva påverkan av gas- och avföringsläckage, samt senarelägga dess debut.

LIST OF PAPERS

This thesis is based on the following papers, which will be referred to in the text by their roman numerals.

- I. Long-term effects of vacuum extraction on pelvic floor function: a cohort study in primipara. Nilsson I, Åkervall S, Milsom I, Gyhagen M. *Int Urogynecol J* 2016;27:1051-6.
- II. Symptoms of fecal incontinence two decades after no, one, or two obstetrical anal sphincter injuries. Nilsson IEK, Åkervall S, Molin M, Milsom I, Gyhagen M. *Am J Obstet Gynecol* 2021;224(3):276.e1-276.e23.
- III. Temporal trends in obstetric anal sphincter injury from the first vaginal delivery in Austria, Canada, Norway, and Sweden. Gyhagen M, Ellström Engh M, Husslein H, Koelbl H, Nilsson IEK, Schulz J, Wagg A, Milsom I. *Acta Obstet Gynecol Scand* 2021;100(11):1969-1976.
- IV. Severity and impact of accidental bowel leakage two decades after no, one, or two sphincter injuries. Nilsson IEK, Åkervall S, Molin M, Milsom I, Gyhagen M. *Am J Obstet Gynecol* 2023; doi: <https://doi.org/10.1016/j.ajog.2022.11.1312>.
Online ahead of print.

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ABBREVIATIONS

ABL	Accidental bowel leakage
ACS	Acute caesarean section
AI	Anal incontinence
aOR	Adjusted odds ratio
BMI	Body mass index
CI	Confidence interval
CS	Caesarean section
EAS	Externa anal sphincter
ECS	Elective caesarean delivery
IAS	Internal anal sphincter
ICD	International classification of disease
ICS	International continence society
ICI	International consultation on incontinence
IGI	Isolated gas incontinence
IUGA	International urogynecological association
FI	Faecal incontinence
LAM	Levator ani muscle
LUTS	Lower urinary tract symptoms
MBR	Swedish medical birth register
MRI	Magnetic resonance imaging
MUI	Mixed urinary incontinence

OASI	Obstetrical anal sphincter injury
OAB	Overactive bladder
OR	Odds ratio
RCT	Randomised controlled trial
RR	Risk ratio
SPAR	The Swedish state personal address register
PFD	Pelvic floor disorders
POP	Pelvic organ prolapse
SAS	Statistical analysis system
sPOP	Symptomatic pelvic organ prolapse
SUI	Stress urinary incontinence
SVD	Spontaneous vaginal delivery
SWEPOP	The Swedish pregnancy, obesity, and pelvic floor surveys
TPR	Total population register
UI	Urinary incontinence
UUI	Urge urinary incontinence
VBAC	Vaginal birth after caesarean section
VD	Vaginal delivery
VE	Vacuum extraction

1 INTRODUCTION

From a global perspective, life expectancy is increasing, and more women will live active lives and continue to work long after their childbearing years. As women live longer, more of them will experience the long-term effects of childbirth, negatively affecting their quality of life and professional careers.

Pelvic floor disorder is an umbrella term for several conditions, the most common being urinary incontinence, pelvic organ prolapse, and faecal incontinence. Urinary incontinence in women has been reported to occur in 25-45% and symptomatic pelvic organ prolapse and faecal incontinence in 5-10% (1-3). These conditions are common and disabling; treatment options are still suboptimal (4).

With an ageing female population, pelvic floor disorders will pose an even greater challenge and increase demands on healthcare systems (5). Currently, one in five women in welfare states, at 80 years of age, will have undergone surgery for urinary incontinence and symptomatic pelvic organ prolapse (6,7). Given the scale and consequences of these problems, it is unsatisfactory that there is scarce information and conflicting results on the long-term prevalence, severity and impact of childbirth and serious obstetric events on pelvic floor function (8-12).

The fear of sustaining a permanent injury to the pelvic floor at birth increases the demand for healthcare professionals to be well-informed

about potential sequelae, not only in the short-term but also in the long-term (13). Vaginal delivery may constitute severe trauma to the pelvic floor, and an obstetrical anal sphincter injury (OASI) is the leading risk factor for faecal incontinence in parous women (3,14). In Sweden, the incidence rate of OASI was 7% in the first, 2% in the second, and 1% in the third delivery from 1999 to 2011, according to the Medical Birth Register (15).

Anal incontinence is common during the first months after vaginal delivery (16-18). However, many women with early problems will recover within the first year (19-22), only to subsequently experience reoccurrence several years after delivery. The prevalence of faecal incontinence in women without OASI has been reported to be 5% to 12% (23,24), and 14% to 29% (23,25) after one OASI. The prevalence of faecal incontinence after two OASIs was 24% in one register-based study from Denmark (26).

Qualitative studies have demonstrated multiple serious consequences of faecal incontinence, such as limitations to daily life activities, social debilitation, and isolation (27,28). In two large national studies, faecal incontinence severely impacted the quality of life in 23–39% of afflicted women (29,30). Surgical treatment with secondary sphincter repair has shown disappointing long-term results following a short-time improvement in incontinence (31).

Common risk factors for OASI are nulliparity, instrumental delivery (vacuum and forceps assisted delivery), high infant birth weight, the first vaginal delivery after caesarean section, and midline episiotomy (32-40). A recent meta-analysis showed that vacuum-assisted delivery increased the risk ratio for OASI by 2.6 and forceps delivery by 3.2 compared to spontaneous vaginal delivery (41). In 2020, 13% of all nulliparous women in Sweden were delivered by vacuum extraction, whilst forceps delivery was used nationwide in only ~50 deliveries per year (42).

There are still significant gaps in knowledge about the association between OASI and faecal incontinence later in life. A review of 16 studies showed that seven reports did not find an association between OASI and bowel incontinence (43). Since there is no generally accepted prediction model for clinical use to determine the individual risk for OASI, it is still often excused as an inevitable event, impossible to foresee (44,45).

With this background, the studies of this thesis aimed to investigate the long-term effects of one and two obstetrical anal sphincter injuries and vacuum extraction on pelvic floor function, using national register-based data and self-reported information from large questionnaire surveys in women with one or two vaginal deliveries. In addition, temporal trends of the incidence of OASI were compared in countries with comparable high-quality national medical birth registers and state-funded healthcare systems in women at their first vaginal delivery.

1.1 THE ANATOMY OF THE PELVIC FLOOR IN WOMEN

The pelvic floor consists of striated muscles, connective tissue, and ligaments attached to the bones of the pelvis. It forms a dome that supports the pelvic organs, their outlets, and the lower intra-abdominal viscera (Figures 1 and 2), and aids urinary and faecal continence function (46-48). As the pelvic floor relaxes, it allows for less resistance in the respiratory system, and together with the abdominal and gluteal muscles, it supports the human body's upright position (49,50). In addition, the pelvic floor is activated during sexual arousal and orgasm (47,51).

The female pelvis consists of the following:

- I. The ligaments and outlets (urethra, vagina, and anus) of the pelvic organs (Figure 1 and Figure 2) and the endopelvic fascia (a fibrous connective tissue) which attaches the organs to the pelvic walls (Figure 3).
- II. The pelvic diaphragm (the levator ani and coccygeus muscles) (Figures 1 and 2).
- III. The perineal membrane (a one-layer muscle and connective tissue complex) (not in figure).
- IV. The superficial layer of supporting muscles (the external anal sphincter, the superficial transverse perineal muscle, the bulbospongiosus, and the ischiocavernosus muscles) (Figure 4).

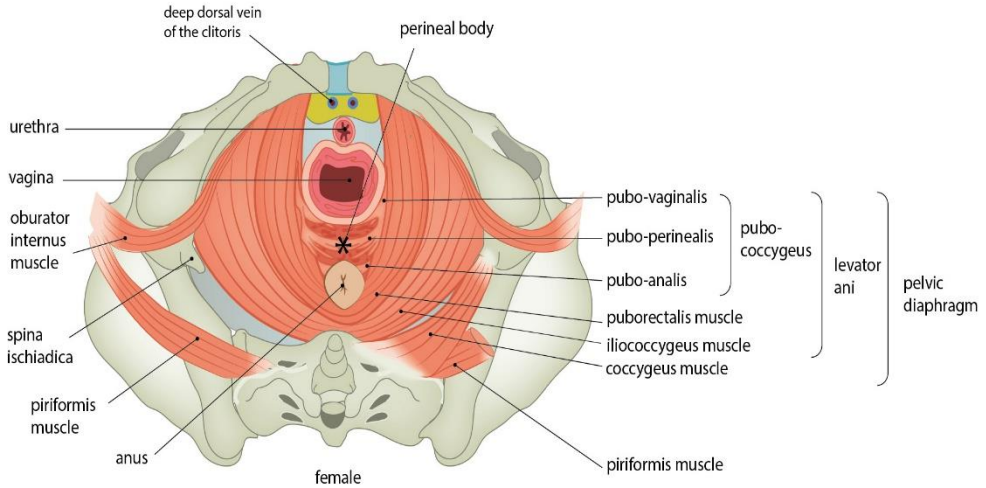


Figure 1. "Slagter - Drawing Inferior view of the female pelvic diaphragm 2 - English labels" at AnatomyTOOL.org by Ron Slagter, LUMC, Marco DeRuiter, LUMC and O.P. Gobée, LUMC, license: CC BY-NC-SA 4.0.

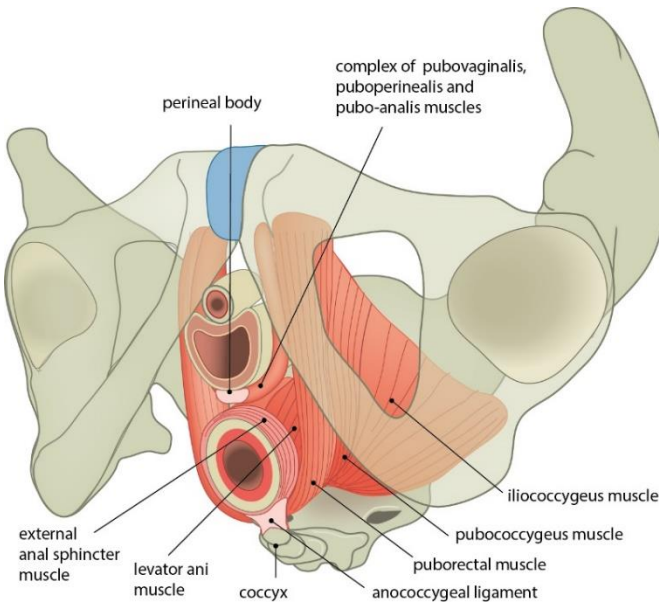


Figure 2. "Left inferior view of levator ani and external anal sphincter muscles - English labels" at AnatomyTOOL.org by Ron Slagter, LUMC and Marco DeRuiter, LUMC, license: CC BY-NC-SA 4.0

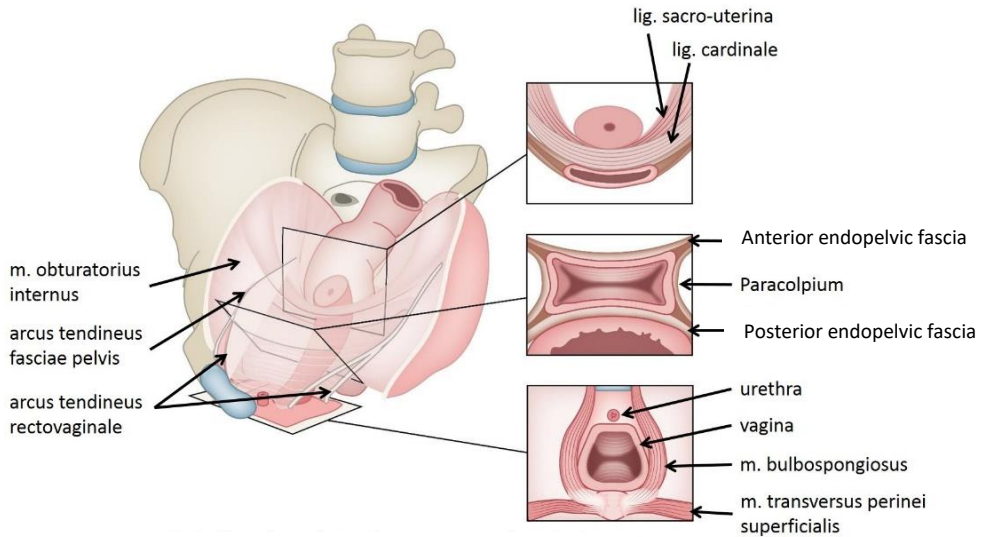


Figure 3. "Delancey's three levels of pelvic support – Dutch labels" at AnatomyTOOL.org by Ron Slagter, LUMC and Marco DeRuiter, LUMC, license: CC BY-NC-SA 4.0. Simplified by IEK Nilsson 2022.

The main muscle of the pelvic floor, the levator ani (levator ani muscle, LAM), consists of three muscles, the pubococcygeus, the puborectalis, and the iliococcygeus muscle (Figures 1 and 2) (48,52). The puborectalis muscles encircle the levator hiatus, the opening in the pelvic diaphragm, where the urethra, vagina, and anus pass. Muscle fibres from the pubococcygeus and the puborectalis are directly interconnected to the vagina, the perineal body, the anal canal, and anal sphincters (46-48). The LAM and the coccygeus muscle predominantly consist of type 1 striated muscle fibres (aerobic); in the pelvic diaphragm, they are constantly activated, decreasing the tension on the pelvic floor ligaments, and preventing them from being overstretched. The constant tonus of the pelvic diaphragm, together with the endopelvic fascia, gives the pelvic floor its dome shape. The tonus also

closes the levator hiatus, aiding continence and the stability of the pelvic organs (Figures 1 and 2). During anesthetization, or in cadavers, the dome shape is lost, and the pelvic floor has a more basin-like shape (46,48).

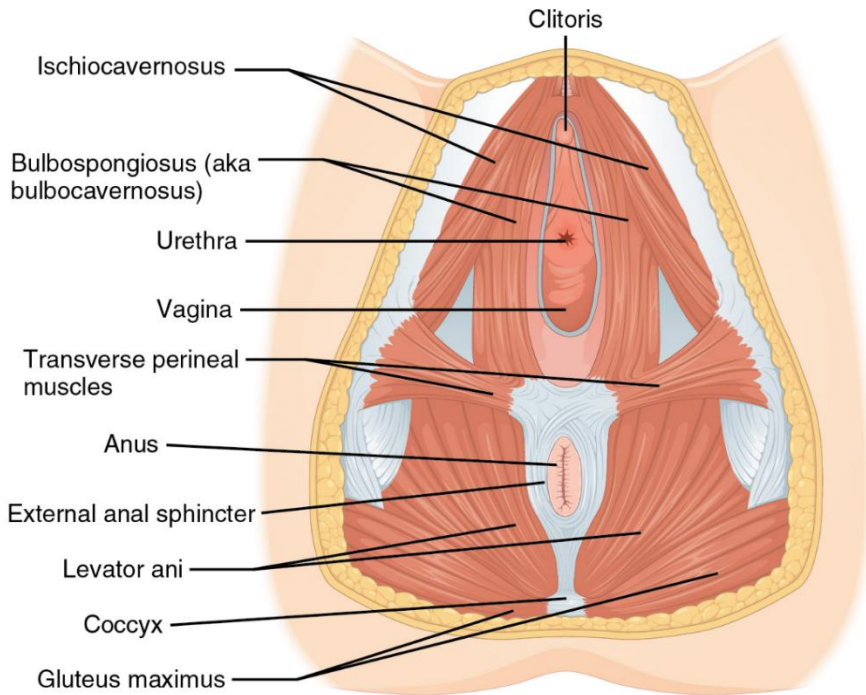


Figure 4. "OpenStax AnatPhys fig.11.20 - **Muscles of the Female Perineum - English labels**" at AnatomyTOOL.org by OpenStax, license: CC BY 4.0. Source: book 'Anatomy and Physiology', <https://openstax.org/details/books/anatomy-and-physiology>.

The most central muscles for the continence of faeces are the anal sphincter complex and the LAM. Its main components are the voluntarily controlled external anal sphincter (EAS) and the involuntarily autonomously controlled internal anal sphincter (IAS) (Figures 2 and 5).

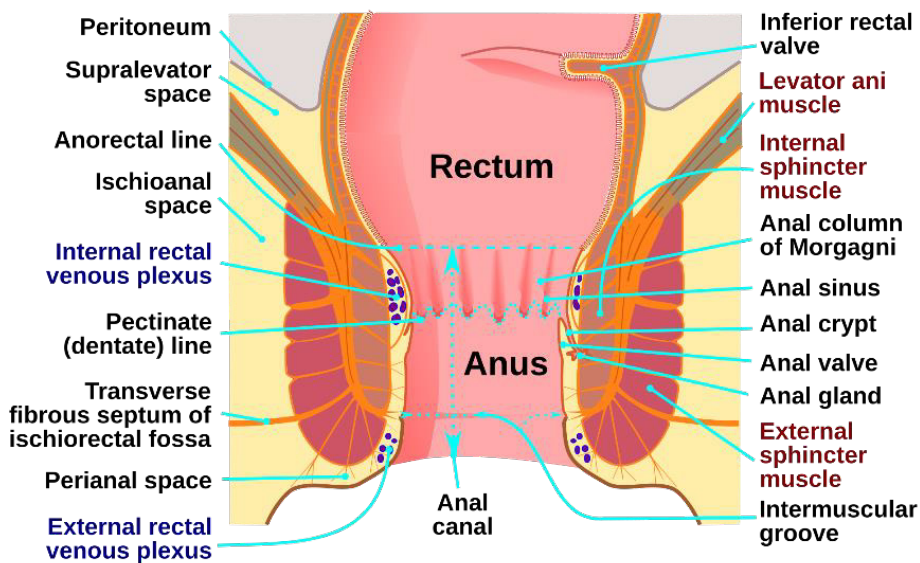


Figure 5. "Jmarchn - Drawing Human Anus - English labels" at AnatomyTOOL.org by Jordi Marchn, license: CC BY-SA 4.0

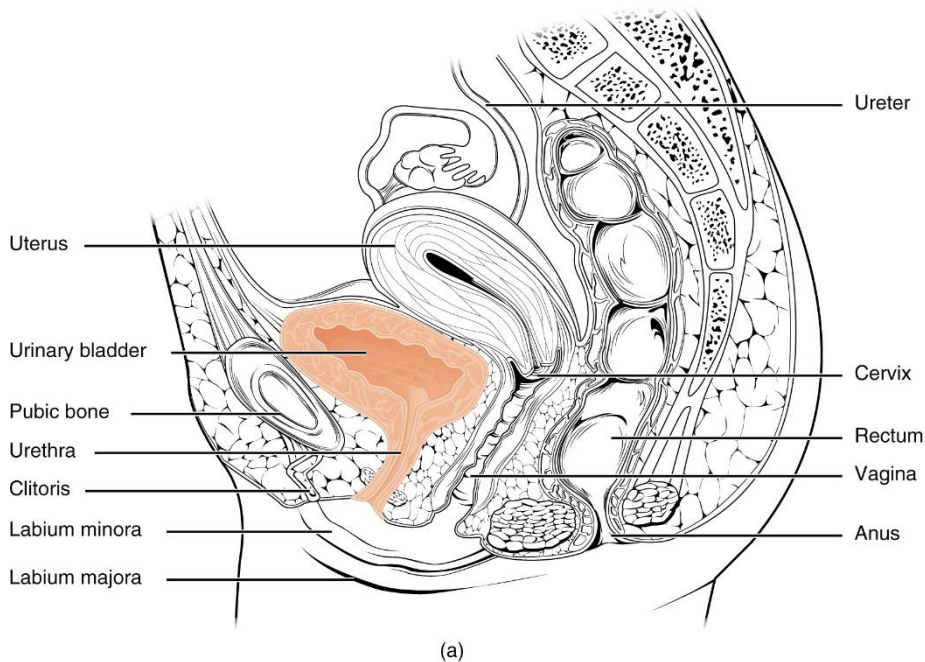


Figure 6. "OpenStax AnatPhys fig.25.3(a) - Female Urethra - English labels " at AnatomyTOOL.org by OpenStax, license: CC BY 4.0. Source: book 'Anatomy and Physiology', <https://openstax.org/details/books/anatomy-and-physiology>.

The EAS is directly attached to the puborectalis of the LAM, and the IAS is a continuation of the circular muscles of the rectum (Figure 5) (46,48). The IAS, EAS, and the puborectal muscle all have a constant, tonic activation that closes the anal canal (3,53). During rest, the IAS is responsible for 50-60% of the tonus (54). The EAS contributes to the anal resting tone, but its main function is to contract as stool or flatus fills the rectum or when intra-abdominal pressure increases, for example from coughing. The contraction at raised intra-abdominal pressure could be voluntary or reflexive (55). The puborectalis muscle is responsible for the closure of the first part of the anal canal as it runs in a U-shape from the pubic bone to encircle the anorectal junction, giving it an almost 90-degree angle (56). At defecation, all three muscles relax which not only opens the anal canal but also increases the angle at the anorectal junction to ease evacuation (3).

The uterus and vagina are held in place by the endopelvic fascia, suspended to the pelvis by the arcus tendineus, and the LAM on which they rest (Figures 2 and 3). The connective tissue support is divided into three levels according to DeLancey (Figure 3); *Level I*, at the bottom of the uterus, the tissues along the vagina (the paracoplium) form the uterosacral ligaments, which attach to the pelvis. At *Level II*, the vaginal walls are stretched laterally between the bladder and the rectum by the paracolpium, attaching to the endopelvic and the LAM fascia. At *Level III*, the vagina attaches to the urethra, the perineal membrane and the perineal body (48,57,58).

The urethra (Figures 1 and 6) first runs through the wall of the bladder, surrounded by smooth muscle and a loop of the bladder muscle – called the internal urethral sphincter. Hereafter, it consists of three muscle layers and a vascular plexus. Distally, the urethra runs under an arch of striated muscle, referred to as the external urethral sphincter. The last section is a fibrous outlet that lacks muscles (59).

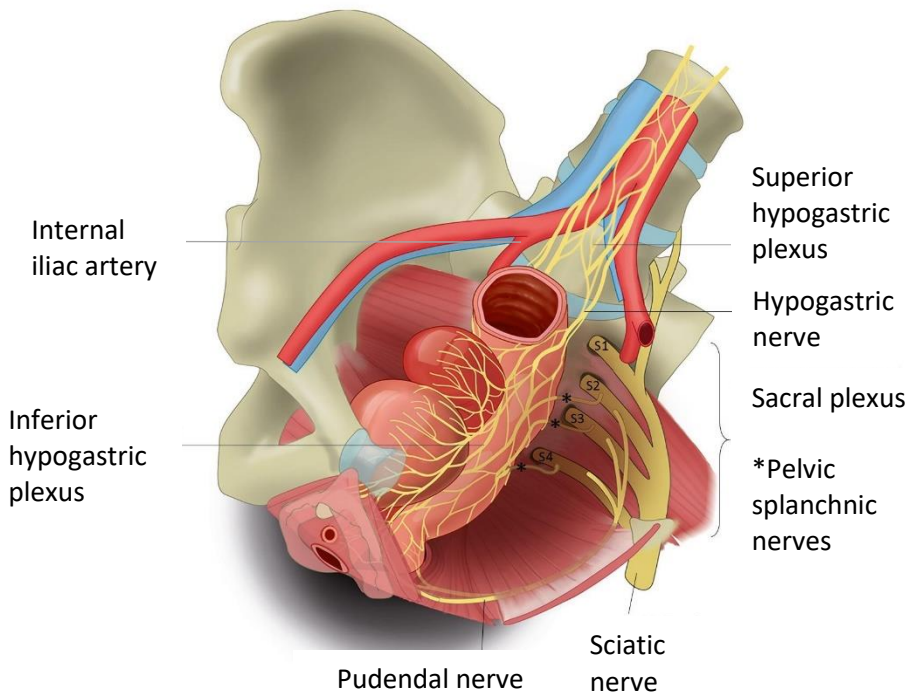


Figure 7. "Anterior view of female pelvis; internal organs and innervation - Latin and English labels" at AnatomyTOOL.org by Ron Slagter, LUMC, Marco DeRuiter, LUMC and O. Paul Gobée, LUMC, license: CC BY-NC-SA 4.0. Simplified by IEK Nilsson 2022.

Branches of the sacral plexus; the pudendal nerve, the levator ani nerve, and the parasympathetic pelvic splanchnic nerves innervate the pelvic floor and its organs, as well as the hypogastric nerve, which constitutes

its sympathetic nerve supply (Figure 7) (46,48). The main blood supply comes from branches of the internal iliac artery (Figure 7) (47).

Hence, this highly complex muscle, ligament, and connective tissue entity must be firm enough to support the inner organs and maintain continence but still permit urination, defecation, intercourse, and vaginal birth.

1.2 THE EFFECT OF VAGINAL DELIVERY

The biomechanics of a vaginal delivery (VD) includes the forces from the uterine contractions and voluntary pushes, the size of the foetal head and its potential to remodel through the birth canal, and the birth canal itself – the pelvis and the pelvic floor and its ability to remodel (60). Female physiology changes during pregnancy (61), including the pelvic floor (60). A magnetic resonance imaging (MRI) study comparing nulliparous, pregnant, and post-partum women showed that the pelvic floor relaxed in pregnant patients, creating more space in the anterior parts of the pelvis, most probably to aid vaginal delivery (62). MRI studies have been performed on women giving birth, but because of its inherent difficulties, foremost computational models have been used to get information on the biomechanical process of VD. A geometric mathematical model showed that the muscle portions of the levator ani that stretched the most during vaginal birth were the pubococcygeus (more than 320%) and the iliococcygeus (more than 270%) (60), figures consistent with MR images of a live birth (63). These findings are

supported by post-partum MRI studies, where the pubococcygeus muscle is most often injured at a vaginal birth, followed by the iliococcygeus muscle (60). Injuries of the LAM are not visible to the naked eye, but 3D and 4D ultrasound examinations of the pelvic floor after VD have shown an incidence of 15-40% post-partum and up to 9 months (3). Also, MRI studies 6-12 months post-partum showed LAM injuries in approximately 19% of VDs (3). Forceps delivery (but not vacuum extraction), OASI, and episiotomy have been found to significantly increase the risk of injury to the LAM (64).

In addition to muscles tearing from over-stretching, there are other theories on the causes of birth-related injuries to the pelvic floor. For example, ischemia is proposed, which would be due to the compression of the pelvic floor between the foetal head and the bony pelvis (65). Another theory is that pelvic floor injuries result from nerve damage during delivery (66-68). On MRI, muscle tearing is evident immediately (60,69), and muscle dysfunction due to nerve injury probably develop over time (70-73).

1.3 PATHOGENESIS AND PATOPHYSIOLOGY

1.3.1 OBSTETRICAL ANAL SPHINCTER INJURY

The OASI is typically a continuation of a perineal laceration, running from the vaginal mucosa, separating the bulbocavernosus muscles, tearing the transverse perineal muscle, and finally involving muscle

fibres of either or both the IAS and the EAS, and in the worst case scenario, the rectal mucosa (Figure 2 and 4) (17). In the short term, women with OASI are at a high risk of wound infection and dehiscence (wound breakdown), in one study reported as high as 20% and 25% respectively (74). In the prospective cohort study by Gommesen et al, 3% of women with OASI had wound infection, 13% had wound dehiscence, and a protective effect of antibiotics was found (75). A protective effect of antibiotics has also been suggested by a Cochrane review (76). In a meta-analysis of 10 observational studies, the overall incidence of wound infection was 4% and wound dehiscence was 7% (77). Sexual dysfunction and dyspareunia 1-4 years postpartum occur in 14-53% of women with OASI, compared to 6-41% in controls (8,78). The prevalence of faecal and anal incontinence is increased in the first 12 months after an OASI, compared to those without OASI (16,79,80). However, many women with anal incontinence (AI) symptoms after delivery will recover within the first year (19-22). Reconstructive surgery has been shown to have unsatisfactory long-term results on bowel incontinence (31).

1.3.2 FAECAL AND ANAL INCONTINENCE

The bowel continence function is complex. It involves the anal sphincter muscles, a functioning pelvic floor entity, an intact sensory function of the lower rectum, and the ability of the rectum to relax and accommodate storage (3,5). Faecal incontinence (FI) is often multifactorial, and more than one factor can be found in up to 80% of patients

with FI (3). The most important risk factors for FI in community-dwelling individuals are any disease causing diarrhoea, ageing, rectal urgency (a sudden, strong, need to defecate), multiple chronic illnesses, anal sphincter trauma, obesity, and smoking (3,5,81).

Ageing is a well-documented risk factor for FI, but the pathophysiology behind it is less clear (3,5,82). Theories include debilitating effects on the anal sphincter complex including increasing fibrosis and atrophy (83-85), decreased contractility (86), decreased anal squeeze pressures (70,87), decreased rectal sensitivity (87), and possibly progressing neuropathy (70-72).

Diarrhoea adds to FI due to the fluid texture of the stool (88). Regarding urgency, it is still unknown whether it originates from hypersensitivity of the nerves, or a fast passage of bowel content overflowing the reservoir function of the rectum, or a combination of both (3,89,90). Changes in the reservoir function can arise from radiation therapy, surgical resection, or diseases in the rectum (91). Whether obesity causes FI by a chronically increased intra-abdominal pressure, or if obesity is a mere confounding factor since diarrhoea is more common among the obese, is unclear (92).

1.3.3 URINARY INCONTINENCE

The two main types of urinary incontinence (UI) are stress UI (SUI), caused by physical strain such as sporting, a sudden laugh, cough, or

sneeze, and urgency UI (UUI), caused by a sudden urge to urinate (93). Currently, three main reasons for UI are proposed. In SUI, loss of urethral support to the underlying connective tissue and its connection to pelvic floor muscles has been proposed. In UUI, the proposed mechanisms are bladder muscle and/or urethral overactivity, or decreased capacity of the brain to handle incoming nerve signals from the urethra and the bladder. Finally, for both SUI and UUI, urethral closing pressure failure is emerging as an important element (3,59). Several factors are needed for the urethra to remain closed during rest and strain. The striated muscles of the external urethral sphincter (Figure 6) need to be intact and to have functioning innervation by the pudendal nerve (Figure 7). The urethral mucosa needs an effective circulation, the urethral smooth muscles needs to be active, and finally, the support by the vaginal wall needs to be intact (3,59).

1.3.4 PELVIC ORGAN PROLAPSE

Pelvic organ prolapse (POP) constitutes the loss of support of the bladder, uterus, colon, or rectum, leading to the descent of one or several of these organs into, and in severe cases, out of the vagina (1). Women are usually asymptomatic if the prolapse is located above the hymeneal ring of the vulva (94). Symptoms may be aggravated by straining, such as long periods of standing, exercise, or defecation, and are relieved by rest (93).

Injuries to the connective tissue at *Level I* may cause prolapse of the uterus or upper vagina, at *Level II* prolapse of the bladder or rectum, and at *Level III* prolapse of the rectum or descent of the perineal body (Figure 3) (3). Pelvic organ prolapse develops through the LAM hiatus; injuries to or irreversible stretching of the muscles leads to a decreased support of the organs and increased strain on *Level I* connective tissue (Figure 2 and 3) (3,48). In an ultrasound study comprising 781 women, prolapse was seen in 83% (150/181) of the women with LAM avulsion, and in 44% (265/600) of women without avulsion (relative risk (RR) 1.9, 95% CI 1.7–2.1) (95).

1.4 EPIDEMIOLOGY

1.4.1 OBSTETRICAL ANAL SPHINCTER INJURY

The incidence of OASI is internationally estimated to be about 0.6-11%, and it differs between and within countries and with time (17,33,40,96-98). The risk of a recurrent OASI is probably higher (99). Andrews et al performed a prospective study where women having a first vaginal delivery were re-examined after delivery by an experienced research fellow, resulting in an increase of clinically diagnosed OASI from 11% to 25%. The study showed that 87% of midwives and 27% of junior doctors failed to diagnose an OASI (100). These results were confirmed in a study by Groom et al., where re-examination of all second-degree tears revealed that 15% were OASIs (101). Common risk factors for OASI are nulliparity, instrumental delivery (vacuum and forceps

assisted delivery), high infant birth weight, the first vaginal delivery after caesarean section (VBAC), and midline episiotomy (32-40).

1.4.2 FAECAL AND ANAL INCONTINENCE

In the meta-analysis by Ng et al, the prevalence of FI in both men and women was reported to be 8% (ranging from 2% to 21%), increasing with age (highest in institutionalized patients), and dependent on definition (2). The prevalence of AI was 16% (ranging from 2 to 47%). Twenty-two of the studies reported sex-specific rates of FI, and in all but four, women presented a higher prevalence of FI (median 9%). Qualitative studies have mapped the many debilitating effects that accidental bowel leakage (ABL) may have on afflicted persons, for example feelings of shame, lowered self-esteem, social limitations and isolation, sexual dysfunction, workplace and private economy difficulties, etcetera (27,28,102). In two large studies from the USA, FI posed a grave negative impact on the quality of life in 23–39% of women afflicted (29,30).

1.4.3 URINARY INCONTINENCE

Epidemiological studies have demonstrated that UI is more common among women than in men (103,104). Prevalence varies highly between studies due to the definition used, different populations, cultural differences and the willingness to report symptoms, possibly racial differences, and methodological differences such as the wording of

questionnaires and the collection of data. However, prevalence is most often reported to be 25% to 45% (1,105).

There is robust epidemiological evidence linking VD and parity to the development of UI (3,59,105). Current evidence indicates that women who have a poor urethral function to start with, also will leak during pregnancy as the mechanical load on the bladder increases, and in addition, these women are more prone to UI post-partum and in the long term (59,105). Compared to women with caesarean section (CS), women with VD have been reported to have a significantly increased risk of UI, both in the short- and long-term, and a higher risk of UI surgery (106-108).

Age is a well-documented risk factor for UI both in parous and nulliparous women (3,59,105). Urethral closing pressure decreases with advancing age, most probably due to a decreasing number and density of striated muscle cells and nerve loss (3,59). UI is more common in obese women, and prevalence increases with increasing BMI (59,109). A urodynamic case-control study on 103 women with SUI and 108 controls (matched for age, race, parity, and hysterectomy), suggested that obese women had stronger urethras, but still not strong enough to hold back urinary leakage caused by their increased intra-abdominal pressure (110).

1.4.4 PELVIC ORGAN PROLAPSE

About 9% of all women worldwide have symptomatic POP (sPOP) (111). Currently, the pathogenesis of pelvic organ prolapse is assumed to be due to both environmental and genetic factors, the most important environmental risk factors being age and VD, especially with increasing parity (3,111). At 3-6 months after the first vaginal delivery, prolapse is found in 18-56% of clinical examinations (3). Among women aged ≥ 45 years who underwent prolapse surgery in Sweden from 2010 to 2017, 98% had ≥ 1 VDs (112), and 27 years after ≥ 3 deliveries, the rate of sPOP surgery was 8% in women with VDs and $< 1\%$ in women with CSs (108).

It is well-known that the incidence and prevalence of sPOP increase with age (3,111). Twenty percent of women aged 80 will have had at least one operation for sPOP or UI, procedures that are uncommon in women < 30 years (6,7).

Regarding genetic factors, sPOP is more common in women with mothers and sisters with sPOP, regardless of eventual parity and delivery modes, with the greatest similarities in monozygotic twins (3,111). Other factors with an association to sPOP include ethnicity, connective tissue disorders (especially among young women with sPOP), obesity, employment with heavy lifting, and a wider transverse inlet of the bony pelvis (3,111).

2 AIM OF THE THESIS

The overall objective of this thesis was to investigate the long-term effects of one and two obstetrical anal sphincter injuries and vacuum extraction on pelvic floor function. In addition, temporal trends of OASI incidence in primiparous women were compared in countries with comparable high-quality national medical birth registers and state-funded healthcare systems.

2.1 THE SPECIFIC AIMS WERE

Paper I To describe the prevalence of obstetrical anal sphincter injury and the prevalence, severity, and subjective impact of pelvic floor disorders (urinary incontinence, symptomatic pelvic organ prolapse, and faecal incontinence) two decades after one vaginal delivery with or without a vacuum extraction, compared to one spontaneous vaginal delivery or one acute caesarean section.

Paper II To determine the age-related prevalence of faecal incontinence two decades after two consecutive vaginal deliveries with no, one or two obstetrical anal sphincter injuries.

Paper III To compare the incidence of obstetrical anal sphincter injury in the first vaginal delivery in four countries with national medical birth registers and comparable state-funded health care systems. Primiparous women with spontaneous or instrumental delivery (vacuum

and forceps) and women with a first vaginal birth after caesarean section were surveyed.

Paper IV To describe the severity and impact of anal incontinence two decades after birth and to analyse the relative effect of one and two obstetrical anal sphincter injuries and the possible influence of sphincter injury on other pelvic floor disorders.

3 METHODS

The studies in this thesis were based on national birth registers. In Papers I, II, and IV, register data were linked to information from a questionnaire survey.

3.1 ETHICS

The ethical approvals for Papers I, II, and IV were obtained from the Regional Ethical Review Board of Gothenburg University (Paper I; reference number 381-07, September 21, 2007. Paper II and IV; reference number 776-13, November 18, 2013). All responders gave their written consent to participate. For Paper III, no ethical consent was required since aggregated data were used as authorized by the national birth registers.

3.2 THE REGISTERS

The Swedish Medical Birth Register (MBR) was founded in 1973 and is administered by the Swedish National Board of Health and Welfare. The register includes information on 95-99% of all live birth, and from 2008 all stillbirths from the 22nd gestational week (113,114). It is mandatory for every healthcare provider to report to the register. Data are collected prospectively, starting at the first antenatal visit, and comprise maternal information such as; height, weight, smoking habits, concomitant diseases, socio-demographic factors, parity, and

complications during pregnancy, and obstetrical and infant information such as; gestational age at delivery, birth weight and head circumference, perineal tears, induction of labour, and mode of delivery (114).

The validity of the MBR data has been assessed and published by the National Board of Health and Welfare (115). Information on parity in the MBR was incorrect in approximately 2% of Swedish-born women and approximately 9% of women born abroad in 1973-1998 (115). In Papers I, II, and IV, parity was checked with information about parity in the Total Population Register (TPR). Women with conflicting information about parity were not included in the study population. In addition, parity was controlled by a separate question in the questionnaire.

Data validation was performed by comparing register data with patient records. Among 440 patient records, there were 1264 obstetrical diagnoses (e.g. spontaneous delivery, premature birth, caesarean section, induction, pre-eclampsia, perineal laceration) of which 43 (3.4%) were incorrect, uncertain, or should have been changed to a more appropriate diagnosis. For 11 (2.5%) of the 440 records, obstetrical diagnoses were completely missing in the MBR. The delivery mode was not among the 25 most frequently missed diagnoses. Of 498 patient records, 440 (90.6%) contained information on 'maternal height', of which MBR data were incorrect in two (0.4%) of the cases and missing in 11 (2.4%). Infant birth weight was recorded in 526 (97.6%) of 539

patient records, of which MBR data was incorrect in one case (0.02%), and missing in three (0.06%). Three percent (4/133) of the epidurals stated in the MBR were incorrect compared to patient records (115). For the other MBR variables used in Paper I, II and IV, the mean maximum rate of missing values were as follows in 1980-2000: age (birth date) of mothers 0.2%, and gestational age (weeks) 4%. Regarding “time from first delivery”, social security numbers are registered for all infants, hence no delivery dates are missing (114).

Maternal weight at delivery and weight gain during pregnancy have been collected since 1982, and weight in early pregnancy (at the registration to antenatal care) since 1992. Before 1990, the weight could only be recorded with two digits; consequently, 99 kg was the maximum weight (115). In SWEPOP-1, the research group reviewed the patient records of all women with a registered body weight of 99 kg (n=300) to obtain their correct weights. For 1990-1991, weight, both at the registration in antenatal care and the delivery ward, is missing in the MBR (115). However, only the responders’ current weight, as stated in the questionnaire, was used in Paper II and IV. Between 1983 and 1989, 21% of all CSs lacked planned versus acute CS information (115). Only those women with a stated acute CS were included in Paper I.

The TPR is a nationwide, compulsory register administered by Statistics Sweden. It includes all persons registered in Sweden and contains data on the size of the population and changes within the population, such as civil status, citizenship, ancestral homeland, residence permit,

relationships (marital status, biological or adoptive parents, registered caregivers, etcetera), change of address, emigration, and immigration (116). Using the mandatory 10 digits personal identity numbers, Statistics Sweden serves researchers with data by linking the TPR data to other registers (117). Comparing responders and non-responders in Papers II and IV, the TPR was further linked to the Income and Taxation Register and the Swedish Register of Education. As the TPR, the Swedish Income and taxation register is a compulsory register, including nearly 100% of the registered population. Data regarding income from employment, investment incomes, pensions, taxes, and the populations educational level are collected from different authorities (118,119).

In Paper III, aggregated, non-individual data from the MBR and three comparable, quality-controlled national registers from Austria, Canada, and Norway were used (120-122). The Austrian Perinatal Registry was founded in 2008 and collects data from all maternity units in Austria, and its data validity is checked regularly (122). The Canadian Institute for Health Information Register (the Canadian Institute for Health Information's Discharge Abstract Database, CIHI DAD), was developed in 1963 and has a high accuracy for perineal lacerations, 99.9% specificity for both 3rd and 4th-degree lacerations, and 97.1 and 94.7% sensitivity, respectively (123). The Medical Birth Registry of Norway (MBRN) was established in 1967 and includes information on pregnant women from all antenatal clinics, all delivery units, and all paediatric examinations during the child's first month of life. The

MBRN served as a template for the Swedish MBR, and the two registers have an equally high quality (124,125).

3.3 THE STUDY COHORTS

Paper I is part of the Swedish Pregnancy, Obesity, and Pelvic floor survey (SWEPOP-1) on women with exclusively one VD or one CS. The inclusion criteria for SWEPOP-1 were primiparity with one singleton birth between 1985 and 1988, no previous or further births, or ongoing pregnancy. The study population were obtained from the MBR by the Epidemiology Centre of the National Board of Health and Welfare in Sweden. The addresses of 10,117 eligible women were obtained from the Swedish state Personal Address Register (SPAR), which includes all residents of Sweden (126). After excluding 694 women with unknown addresses, hidden personal IDs, and newly deceased, 9,423 women were contacted. A letter was sent in 2008, including information on the study, and asking for written, informed consent to complete an enclosed questionnaire on current pelvic floor disorders (PFDs). After four months, including three mailing cycles, 6,060 women had completed the questionnaire. Of these, 824 women were excluded as they did not state parity ($n=59$), affirmed multiparity ($n=716$), had a multifetal ($n=43$), or an ongoing pregnancy ($n=6$) resulting in 5,236 women in the final, total study population (Figure 8). The questionnaires were linked to individual data from the MBR. In Paper I, the study cohort included 3,061 women with one spontaneous VD (SVD), 704 with a vacuum extraction (VE), and 438 with one acute

CS (ACS). The ACS group included 28 women with a failed VE. 1037 women were excluded from the study population; 766 had an elective CS (ECS), 208 unclassified CS, 26 forceps delivery, and 37 women had missing information on the mode of delivery.

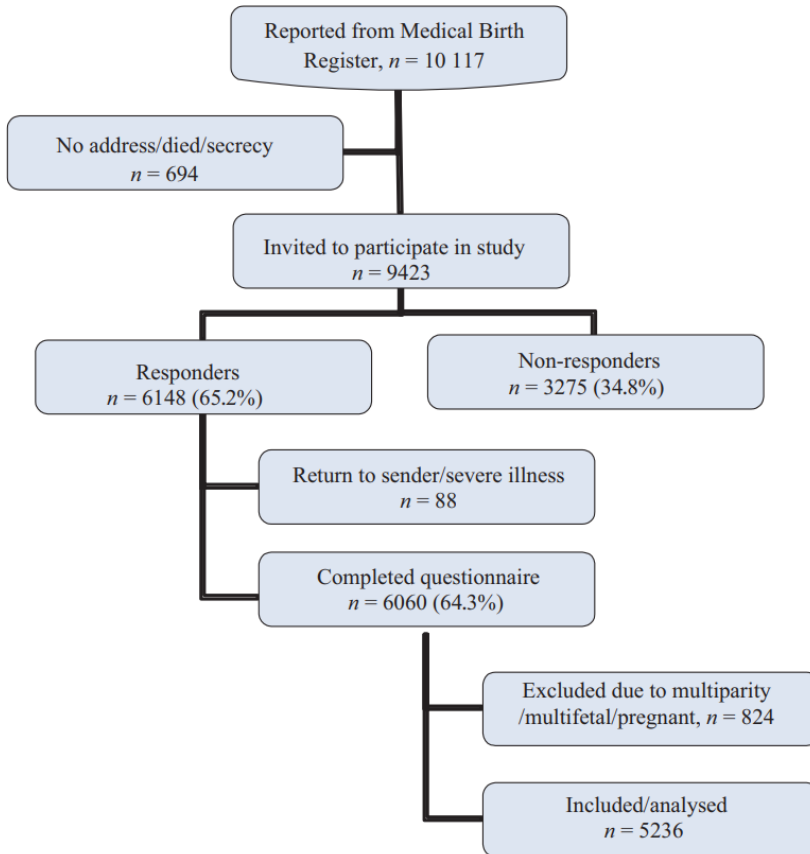


Figure 8. The study population in Paper I. Reproduced from Gyhagen *et al.* The prevalence of urinary incontinence 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. *BJOG*. 2013 Jan;120(2):144-151.

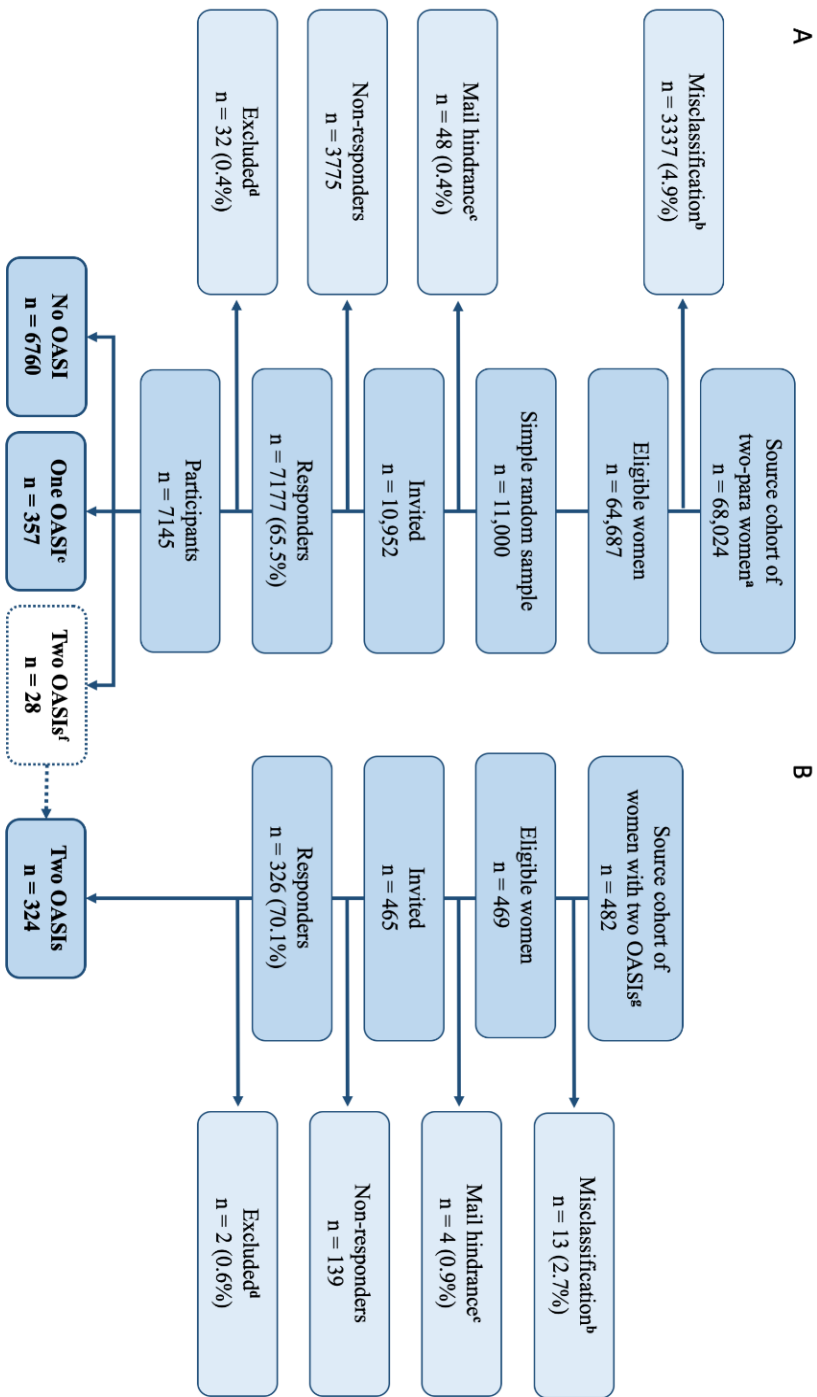
Non-responders in SWEPOP-1 were 1.6 years younger ($P<.001$), more often overweight or obese (37% versus 27%, $P<.001$), and more often gave birth to a child of ≥ 4000 g (48% versus 42%, $P<.003$) compared to responders (127).

Papers II and IV are parts of the SWEPOP-2 survey on women with two VD. The inclusion criteria were two-para women, with two singleton VDs in 1992–1998 and no further births or ongoing pregnancy. After a check for oversubscription performed by Statistics Sweden in 2014, we excluded deceased, emigrated, and persons who had changed personal numbers, 64,687 women were eligible. A simple random sample of 11,000 women was drawn.

Figure 9. Study population flow chart, SWEPOP-2. **A**, Cohort 1, a random sample of 11,000 women from 1992 to 1998. **B**, Cohort 2, all women with repeat (two) OASIs from 1987 to 2000. ^aThe source cohort denotes all women who had two vaginal deliveries, registered in Sweden and with two singleton births 1992–1998. ^bMisclassification for deceased, emigrated, and change of social security number (January 12, 2014), this information is updated every fourth week. ^cMail hindrance denotes mail returned to sender, addressee not found, or emigrated. ^dExcluded denotes declined participation, and a blank or an unusable form. ^eAmong women with one OASI, 253 had an OASI at the first birth and 104 had an OASI at the second birth. ^fThese 28 women occurred both in the random sample and in the cohort with two OASIs. ^gAll women who had two vaginal deliveries and had sustained two consecutive OASIs 1987–2000.

ID, identification; OASI, obstetrical anal sphincter injury.

Reproduced from Nilsson *et al.* *Symptoms of fecal incontinence two decades after no, one, or two obstetrical anal sphincter injuries.* *Am J Obstet Gynecol* 2021 Mar;224:276.e1–e23.



Of these, 39 women had two consecutive OASIs. To yield equal numbers of women with one and two OASIs the recruitment period of women with two OASIs was extended to 1987–2000. A postal and web-based questionnaire survey on symptoms of current PFDs was sent to the study populations by Statistics Sweden in 2015, and answers were collected throughout three mailing cycles for three months. Questionnaire data were linked to obstetrical data from the MBR. Of the final study population, 6,760 had no OASI, 357 had one OASI (253 at the first and 104 at the second delivery), and 324 had two OASIs (28 in the randomly selected cohort) (Figure 9). The response rate was 65.5% among the randomly selected women with VDs and 70.1% in the cohort with two OASIs. With the size of the study cohorts, an alpha level of 0.05, a power value of 80%, and using the Fisher exact test for the analysis, power calculations were completed to evaluate the minimum significant difference in the prevalence of PFDs between cohorts.

In SWEPOP-2, non-responders were younger (57.8% of women aged 30-39 compared to 23.9% of women aged 60-69), more often had foreign citizenship (42.1% compared to 34.1% of women with Swedish citizenship), and were more often born abroad (48.0% compared to 34.1% of women born in Sweden) compared to responders. They also had a lower income (50.6% of those with an income of $\leq 124,999$ Swedish krona (SEK)/year, compared to 28.4% of those with an income of $\geq 370,000$ SEK/year), and lower education (57.6% with maximum compulsory school, compared to 28.0% with ≥ 3 years in upper secondary school) than responders.

In Paper III, aggregated data were used to compare the incidence of OASI in the first vaginal delivery in four countries with comparable high-quality national medical birth registers and state-funded healthcare systems. In 2016, all countries presented a similar maternal age at the first delivery according to the Organisation for Economic Co-operation and Development (OECD) (128); 29.1 years in Canada and 29.2 years in Austria, Norway, and Sweden. In all four countries, home deliveries are unusual. The aim of this study was to survey the prevalence of OASI in four main risk scenarios: the first vaginal birth, spontaneous or instrumental (vacuum or forceps), and in women with a first vaginal birth after caesarean section (VBAC). Hence, the inclusion criteria were an in-hospital first VD that was instrumental or spontaneous, with or without previous CS, in gestational week ≥ 37 , a singleton foetus in cephalic presentation, and information on OASI (“yes” or “no”). The study population all had available information according to the inclusion criteria. The aggregated data were commissioned from each country’s register via their data controller and statistician and were available 2007–2016 for Austria (n=258 828), 2004–2016 for Canada outside Quebec, and Sweden (n=923 357, and n=502 315), and 2004–2015 for Norway (n=249 430).

3.4 DEFINITION OF OUTCOMES

According to the classification of Sultan, a first-degree perineal laceration involves the vaginal mucosa and/or the perineal skin, a second-degree laceration involves the perineal muscles but not the anal

sphincters, a third-degree laceration (OASI) is defined as a laceration in the EAS and/or the IAS, and a fourth-degree OASI involves the rectal mucosa (17). OASI was presented as one group, regardless of degree, and were identified from the MBR by codes 658.1 and 658.2 in the International Classification of Diseases 8th revision (ICD-8) (1969-1986), codes 664.2 and 664.3 in the ICD-9 (1987-1996), and by codes O70.2 and O70.3 in the ICD10 (1997-). From the period 1997 to 2000, OASI was also identified using the surgical code MBC33.

Primiparous (one-para) women were defined as having delivered one infant only, secundiparous (two-para) women as having delivered two infants only, and multiparous were women defined as having delivered two or more infants.

According to the International Urogynecological Association (IUGA) and the International Continence Society (ICS), FI was defined as the involuntary loss of solid and/or liquid stool, with and without concomitant leakage of gas, and AI was defined as FI *or* isolated gas incontinence (IGI) (93,129). Hence, the concept of FI always includes leakage of stool, whereas AI can describe either leakage of stool or gas incontinence only. The term accidental bowel leakage (ABL) is an umbrella term for FI and AI. High frequency of leakage was defined as leakage several times a month or more often, and low frequency of leakage as less than once a month. Severe FI or AI was defined as having bothersome symptoms several times a month or more often.

UI and subtypes were defined according to the IUGA/ICS as a complaint of involuntary loss of urine. SUI was further specified as UI occurring on effort, physical straining, sneezing, or coughing, UUI was UI associated with urgency, and mixed UI was a combination of SUI and UUI (93). Overactive bladder (OAB) was defined according to the IUGA/ICS as urinary urgency, but a more restricted definition of nocturia (≥ 2 times/night) was used (93).

Symptomatic pelvic organ prolapse was defined according to the IUGA/ICS as a sensation of tissue protrusion from the vagina or a vaginal bulge (93,130).

Bothersome FI, AI, IGI, UI, and sPOP were defined as symptoms causing “Some bother”, “Much bother”, or “A major problem”.

3.5 QUESTIONNAIRE

In the survey of SWEPOP-1, the questionnaire included 31 questions (Paper I) and in SWEPOP-2 40 questions by adding questions about lower urinary tract symptoms other than urinary incontinence (Paper II, IV) (Appendix). The questionnaire was divided into three different sections serving both as a symptom inventory and a measure of the degree of bother and distress caused by UI, FI and AI, and sPOP. The introductory section dealt with demographics such as current age, height and weight, menstrual status, hysterectomy, menopause, hormone treatment, treatment for any of the PFDs, and a control question about

parity. Body mass index (BMI; kg/m^2) was calculated from weight and height given in the questionnaire in Papers I, II, and IV, and additionally from MBR data (“BMI early pregnancy”) in Paper I. The second section asked validated questions to estimate the prevalence, severity, and subjective impact of PFDs from the Jorge-Wexner anal incontinence score (131), the Sandvik severity index for UI (132,133), and the Tegestedt short-form questionnaire for sPOP (130).

Prevalences of ABL were explored by the questions “Do you leak solid faeces involuntarily?”, “Do you leak liquid faeces involuntarily?”, and “Do you leak flatus/gas involuntarily?”. Any solid or liquid leakage referred to isolated symptoms, or any combination, in women with FI. The frequency of each symptom was reported as “Never”, “Less than once a month”, “Several times a month but less than once a week”, “Once a week or more”, or “Once a day or more”. Pad use was enquired by the question “Do you use a protective product/pad because of involuntary leakage from the back passage?” and the subjective impact of AI by the question “Is your daily lifestyle affected by involuntary leakage from your back passage?”, with frequency alternatives as for ABL. The Jorge-Wexner score, ranging from 0 (continent) to 20 (complete incontinence) by cross-tabulating frequencies and the five items, was further divided into mild (1-3), moderate (4-8), and severe (≥ 9). The mental impact of AI was explored by the question “How do your bowel symptoms affect you?” (“No problem”, “A minor nuisance”, “Some bother”, “Much bother”, or “A major problem”).

The prevalence of UI was surveyed by the question “Do you have involuntary loss of urine?”. UI subtypes were specified with further questions, for SUI “Do you have involuntary loss of urine in connection with coughing, sneezing, laughing or lifting heavy items?”, for UUI “Do you have involuntary loss of urine in connection with a sudden and strong urge to void?”, and for MUI positive answers to all three questions were required. In addition, in Paper I, UI symptoms for >10 years were reported. The frequency of urinary leakage was reported as “Less than once a month,” “Once or more per month”, “Once or more per week” and “Every day and/or night”. The amount was reported as “A few drops”, “Small amounts” and “Large amounts”. The mental impact of UI was explored by the question “How does your urinary leakage affect you?” (“No problem”, “A minor nuisance”, “Some bother”, “Much bother”, or “A major problem”). OAB was affirmed by a positive answer to the question “Do you have urinary urgency with a sudden and strong urge to void which is hard to postpone?”, and nocturia by answering the question “Do you have to urinate during the night?” ≥ 2 times/night.

Symptomatic POP was regarded as present if the woman affirmed the question “Do you have a sensation of tissue protrusion (a vaginal bulge) from your vagina”, although different frequencies were regarded as an affirmative answer in Paper I (often/sometimes/infrequently = yes, never = no) and Paper IV (often/sometimes = yes, infrequently/never = no). The subjective impact of sPOP was assessed by the question “How

do these vaginal symptoms affect you?” (“No problem”, “A minor nuisance”, “Some bother”, “Much bother”, or “A major problem”).

Having one or more complaints of FI, UI, or sPOP was defined as having “any PFD”. In Paper IV, “Other PFDs” refers to UI and sPOP, and lower urinary tract symptoms (LUTS) refer to OAB and nocturia.

3.6 MISSING DATA

The overall rate of missing data for outcomes was low. In SWEPOP-1, the amount of missing data varied between 0% (age) and 15.9% (hysterectomy) (127). In Swepop-2 the rate of missing data ranged from 0.3% (maternal height) to 3.8% (bothersome anal incontinence). In each analysis, missing data were excluded, except for in the calculation of the Jorge-Wexner score, where a missing answer in single questions was regarded as “never”/“no problem”.

3.7 STATISTICS

For categorical data, number, percent, and 95% CI were presented. The mean and standard deviation (SD), and the median and interquartile range (Q1-Q3) were presented for continuous variables. When comparing two groups, Student’s t-test for independent samples (Paper I) and Mann–Whitney *U* test (Papers II and IV) were used for continuous variables. Furthermore, Chi-square test (Paper I) and Fisher’s exact test

(Papers II and IV) was used for dichotomous variables, and Mantel–Haenszel chi-square test was used for ordered categorical variables. In addition, the results for continuous variables were presented as the mean difference, and for categorical variables as the difference in percentage, with 95% CI and *P* value.

When analysing PFDs between VE and SVD, and VE and ACS, adjusted odds ratios (aOR) with 95 % CI were calculated using logistic regression, adjusting for BMI, maternal age, and infant birth weight. When analysing independent risk factors for any PFD and ≥ 2 PFDs, multivariable logistic regression was used including maternal age at delivery, infant birth weight, episiotomy, epidural anaesthesia, VE, OASI, and current BMI as potential risk factors, presenting OR with 95 % CI.

Trends for women with no, one, and two OASIs were analysed using Mantel–Haenszel statistics for dichotomous variables and the Spearman rank correlation test for continuous variables. The Pearson chi-squared test was used for the distributions of FI symptoms among non-ordered groups. The order of the cohorts presumed that women with no OASI were least affected, followed by those with one, and lastly that women with two OASIs were most affected.

Logistic regression models were used when comparing the prevalence of FI symptoms between two groups (no, one, or two OASIs) adjusting for age and current BMI. Three independent logistic regression models,

weighted with the year 2000 as reference, obtained the estimated age-related probability of FI with OR/10 years, 95% CI, and *P* value. For the groups with one or two OASIs, piecewise logistic regressions were performed with 52 years of age as the cut-off. Results were presented as OR/10 years, 95% CI, and *P* value.

When analysing OASI rates by year from 2004 to 2016, linear regression models were used to show the trend. The average, yearly, change in percentage units were used as the β -value of the regression model. Based on the number of women at risk, the trend for each country was analysed using Mantel–Haenszel chi-squared statistics.

To estimate the degree of bother according to the Jorge-Wexner score, four separate logistic regression models were made. The cumulative degree of bother (“A major problem”, “A major problem + Much bother”, “A major problem + Much bother + Some bother”, and “A major problem + Much bother + Some bother + A minor nuisance”) was the dependent variable, and Jorge-Wexner anal incontinence score the independent variable. Results were presented as the observed prevalence of the degree of bother and the estimated percent of the degree of bother.

Statistical testing was two-sided and the significance level was set to $P < .05$. Statistical analysis system (SAS) version 9.1 (Paper I) and 9.4 (Paper II, III, and IV) was used for the statistical analysis (SAS Institute, Cay, USA).

4 RESULTS

4.1 PAPER I

Compared with SVD, women with a VE delivery were older ($P<.001$), shorter ($P<.001$), had a higher BMI in early pregnancy ($P<.001$), gave birth to an infant with higher birth weight ($P<.001$), more often had an episiotomy ($P=.001$), and epidural anaesthesia ($P<.001$). OASI occurred in 6.3% after VE and 2.4% after SVDs ($P<.001$). The prevalence of FI, sPOP, UI, UI>10 years, and bothersome UI was similar in both groups; the rate of any PFD was 49.9 % after VE and 49.0 % after SVD (Table 1).

When comparing VE deliveries with and without OASI, an OASI doubled the rate of FI (15.4% to 30.2 %, OR 2.55; 95 % CI 1.26–5.15). A similar increase in the rate of FI was seen in SVD complicated by an OASI (30.2 vs 27.8 %, OR 1.12; 95 % CI 0.49–2.56). In addition, OASI (regardless of delivery mode) increased the rate of UI by over 60%, but no difference was found in sPOP prevalences after OASI. The prevalence of most PFDs was overall approximately halved in women with an ACS compared with a SVD or a VE delivery (Table 1).

The multivariate logistic regression analysis showed that maternal age, current BMI unit, infant birth weight, and OASI were risk factors for any PFD (one or more PFD symptoms). The risk factors for two or more PFDs were maternal age, current BMI, epidural anaesthesia, and OASI.

Table 1. Crude prevalence and adjusted odds ratio and 95% confidence interval of pelvic floor disorders grouped according to mode of delivery.

	VE <i>n</i> =704 %	SVD <i>n</i> =3061 %	ACS <i>n</i> =438 %	VE vs SVD adjOR (95% CI)	VE vs ACS adjOR (95% CI)
sPOP	15.1	15.2	6.2	0.90 (0.71-1.14)	2.71 (1.73-4.25)
sPOP+UI	8.8	9.7	2.7	0.79 (0.59-1.07)	3.74 (1.96-7.16)
Treatment for sPOP/UI	4.8	5.0	2.5	0.74 (0.50-1.10)	1.99 (0.98-4.04)
FI	16.3	13.6	12.5	1.16 (0.92-1.46)	1.59 (1.10-2.28)
Any PFD	49.9	49.0	38.8	1.09 (0.91-1.28)	1.68 (1.30-2.16)
≥2 PFDs	17.9	16.5	9.8	1.02 (0.82-1.27)	2.39 (1.62-3.52)
UI	40.5	41.0	31.2	0.89 (0.75-1.06)	1.65 (1.26-2.14)
UI>10 years	9.0	10.5	5.1	0.78 (0.58-1.05)	1.90 (1.14-3.17)
Bothersome UI	13.6	11.0	5.9	1.20 (0.94-1.55)	2.69 (1.68-4.29)
Sought doctor	6.3	5.1	3.5	1.04 (0.73-1.49)	2.03 (1.09-3.77)

ACS, acute caesarean section; *aOR*, adjusted odds ratio; *FI*, faecal incontinence; *PFD*, pelvic floor disorder; *sPOP*, Symptomatic pelvic organ prolapse; *SVD*, spontaneous vaginal delivery; *VE*, vaginal delivery assisted by vacuum extraction. Odds ratios were adjusted for BMI, maternal age, and infant birth weight. Reproduced from Nilsson I, Åkervall S, Milsom I, Gyhagen M. Long-term effects of vacuum extraction on pelvic floor function: a cohort study in primipara. *Int Urogynecol J* 2016;27:1051-6

4.2 PAPER II

At the first delivery, the risk of sphincter injury was 3.9%, and the risk of a repeat sphincter injury was 10.0% (OR 2.70, 95%CI 1.80-4.07). Demographics showed that women with OASI were somewhat older than women without OASI. The mean current age was similar (~50 years) in women with no, one, or two OASIs, respectively. Due to the extended inclusion period, the follow-up time from first birth was somewhat longer (~0.6 years) in women with two OASIs. The time interval between the first and second delivery was somewhat longer in women with OASI at the first delivery (~0.2 years). In women with one and two OASIs, infant birth weight $\geq 4\text{kg}$ occurred twice as often as in those without (Trend $P < .0001$). The rate of VE was more than doubled in the OASI groups (Trend $P < .0001$).

Two decades after the first delivery, 11.7% of the women without OASI had FI, whereas the prevalence in women with one or two OASIs was 23.7% and 36.1% (Trend $P < .0001$) (Figure 10). In women with no, one, and two OASIs, leakage of any solid stool increased from 3.4% to 7.3% and 11.7%, and leakage of any liquid stool increased from 10.8% to 21.7% and 34.9 %, respectively (Trend $P < .0001$) (Figure 10). Concomitant gas incontinence and any liquid stool incontinence occurred at similar frequencies. Severe FI increased from 1.8% to 5.4% and 9.0% in women with no, one, and two OASIs (Trend $P < .0001$). The most common combinations of FI, liquid stool and gas, and the triple combination of solid and liquid stools and gas, increased with one and two OASIs. There was no difference in the prevalence of isolated gas

incontinence between the three groups. The age-related prevalence of FI increased after 52 years of age in women with sphincter injuries. At the age of 60 years, less than one in five women with OASI was continent.

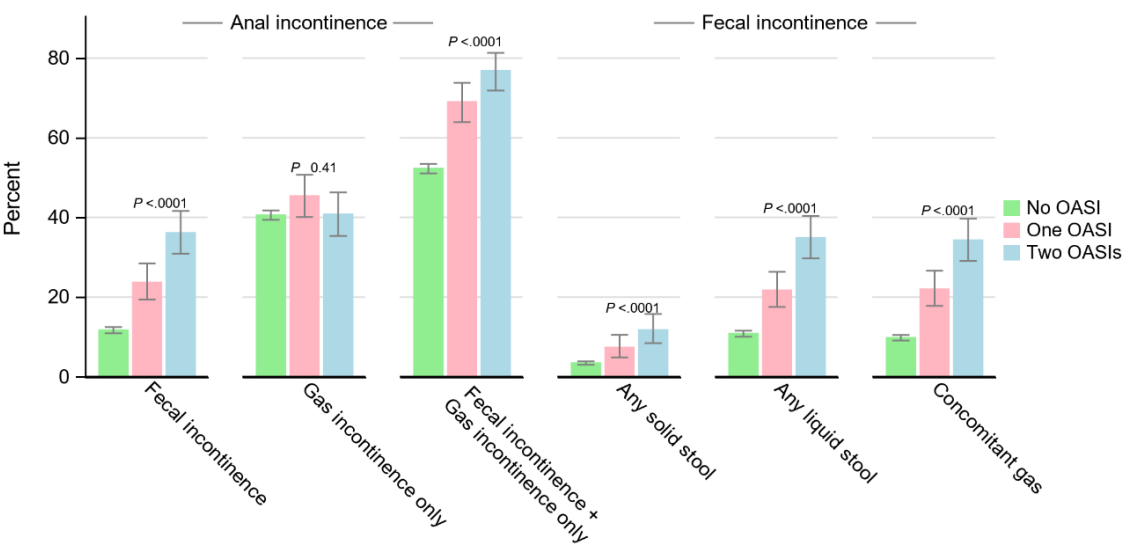
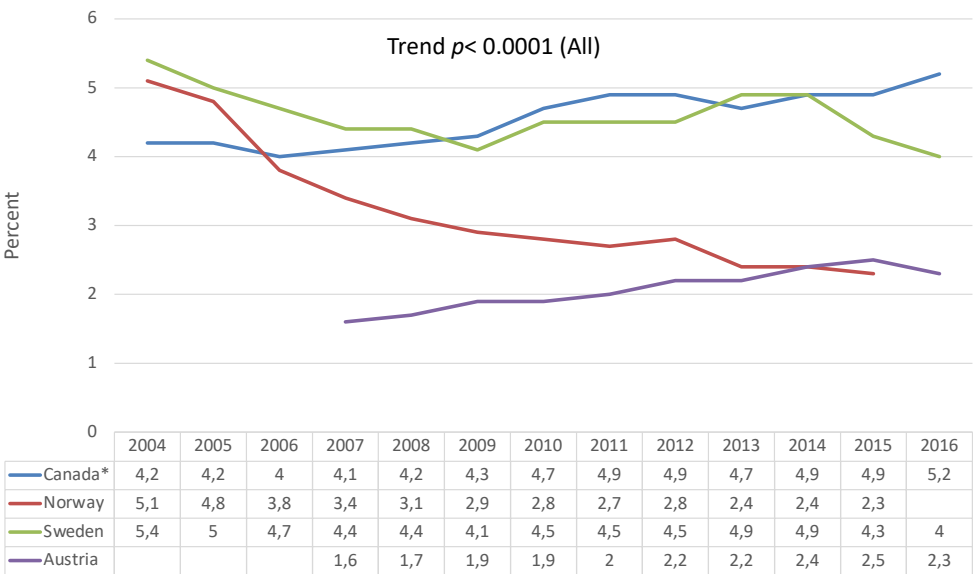


Figure 10. Components of accidental bowel leakage. CI, confidence interval; OASI, obstetrical anal sphincter injury. Reproduced from Nilsson et al. Symptoms of fecal incontinence two decades after no, one, or two obstetrical anal sphincter injuries. *Am J Obstet Gynecol* 2021 Mar;224:276.e1-.e23.

4.3 PAPER III

The total study population consisted of 1 933 930 women with a first vaginal delivery in Austria, Canada, Norway, and Sweden. Overall, 115 070 OASI were identified; 62 197 in women with SVD, 35 258 in women with VE (n=312 317), and 17 615 in women with a forceps delivery (n=86 123). In all countries, the annual number of births increased during the study period. The annual prevalence of CS was

rather similar within the four countries during 2004-2016, Austria (31%) and Canada (27%) had the highest rates, and Norway (18%) and Sweden (16%) had the lowest. Almost every fourth primipara in Canada and Norway was delivered instrumentally, and forceps-assisted delivery was performed in ~8% of primiparous deliveries in Canada and in ~3% of primiparous deliveries in Norway. The rate of operative VD was somewhat lower in Austria and Sweden, compared with Canada and Norway, and forceps were rarely used (<0.5%).

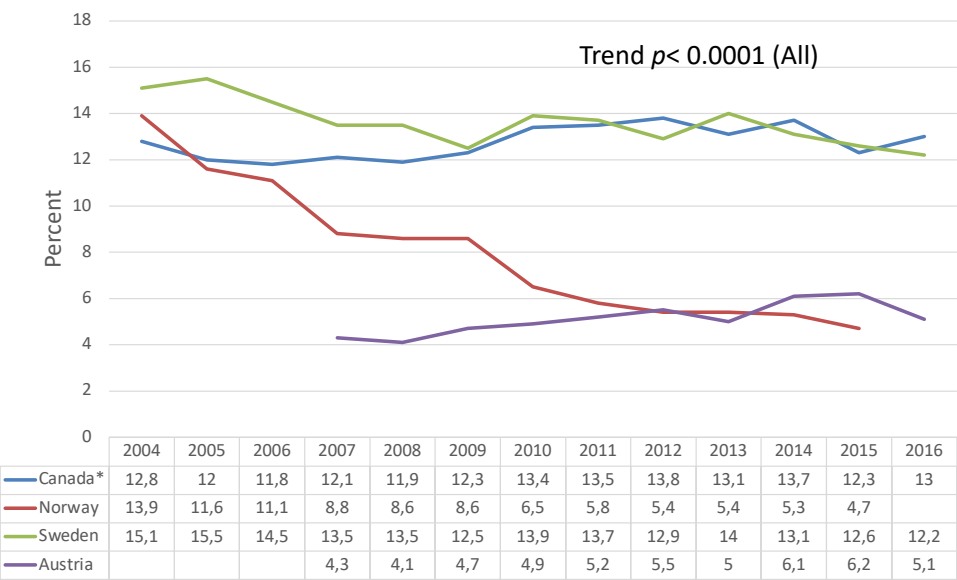


* Quebec not included

Figure 11. Incidence of sphincter injury at first birth by spontaneous vaginal delivery.

In Norway, the rate of OASI in SVD decreased from 5.1% to 2.3% (Trend $\beta -0.23$, $P<.001$) during the study period, and in Sweden a similar but weaker trend was seen (5.4% to 4.0%, Trend $\beta -0.05$,

$P<.001$). The opposite trend was noticed in Canada (4.2% to 5.2%, Trend β 0.09, $P<.001$) and Austria (1.6% to 2.3%, Trend β 0.09, $P<.001$) (Figure 11).



*Quebec not included

Figure 12. Incidence of sphincter injury at first birth by vacuum delivery.

The incidence of OASI after operative VD varied with the mode of delivery, between countries, and during the study period; in VE deliveries from 4.1% (Austria) to 15.5% (Sweden) (Figure 12), and in forceps deliveries from 4.0% (Austria) to 26.6% (Sweden). The mean rate of OASI was 2.7 times higher after a VE delivery, and 4.3 times higher after a forceps delivery, compared to SVD. The rate of OASI after VE delivery in 2015 was twice as high in Canada and Sweden (12.3% and 12.6%), compared to Norway and Austria (4.7% and 6.2%) (Figure 12). During the study period, the incidence of OASI in VE deliveries

decreased from 13.9% to 4.7% in Norway (Trend $\beta -0.79$, $P<.001$). The rate of OASI in forceps delivery was similar in Canada (18.6%) and Norway (20.4%) in 2004, but decreased to 6.2% in Norway (Trend $\beta -1.15$, $P<.001$) and increased to 24.3% in Canada (Trend $\beta 0.49$, $P<.001$) at 2015.

Compared to having an SVD at first birth, the risk of OASI in VD after a prior caesarean section (VBAC) was increased in all countries (about 10-60% at the end of the study period). There was a decreasing trend in both Norway and Sweden (Trend $\beta -0.20$, $P=.017$, and Trend $\beta -0.17$, $P<.001$), however in 2014, Sweden had the highest rate of OASI in spontaneous VBAC (8.1%), corresponding to a difference of ~5 percentage units between Norway and Sweden. The rate of OASI in vacuum-assisted VBAC varied markedly between years and countries. Overall, the incidence rates were approximately doubled in vacuum-assisted VBAC in all countries, compared to spontaneous VBAC (2014-2016). Norway showed a downward trend in the incidence of OASI also in VE-assisted VBAC, from 16% in 2004 to 6.7% in 2015 (Trend $\beta -0.71$, $P<.001$). The registers in Austria and Sweden denied information on forceps-assisted VBAC since there were so few cases and hence a risk of identifying these women.

4.4 PAPER IV

The study cohorts were identical to those in Paper II. The mean rate of missing data was 1.6%, lower in women with two OASIs (0.8%).

Bothersome FI increased 3- and 5-fold, from 3.3% in women without OASI, to 10.4% (aOR, 3.25; 95% CI, 2.23-4.73) in women with one OASI and 16.5 % (aOR, 5.16; 95% CI, 3.69-7.22) in women with two OASIs (Figure 13). The prevalence of IGI was about 40% in each group, but bothersome IGI was higher after one and two OASIs (Trend $P=0.0024$) (Figure 13). 28.2% of women without OASI perceived their FI as bothersome, compared to 43.9% and 46.0% in women with one or two OASIs (Trend $P<.0001$). The frequency of leakage of solid and liquid stool and IGI increased in women with one or two OASIs compared with those without (Trend all $P<.0001$). For example, the frequency of any leakage of liquid stool was 10.8% in women with no OASI, 21.7% in women with one, and 34.9% in women with two OASIs (Trend $P<.0001$). The use of pads was higher in the one and two OASIs groups compared with no OASI, from 2.3% in women without OASI to 7.1% and 8.4% in those with one and two OASIs (Trend $P<.0001$) (Figure 13). An effect on daily life was reported by 8.6% of women without OASI and 19.7% and 29.6% in women with one and two OASIs (Trend $P<.0001$) (Figure 13). The mean Jorge-Wexner score was 1.27, 2.25, and 2.98 with 0, 1, and 2 OASIs, respectively (Trend $P<.0001$). In women with a Jorge-Wexner score of six, more than 50% had bothersome AI, and in those with a score of 9, almost 80% had bothersome AI. Few women had received treatment for bowel leakage, there were however significantly more women with one and two OASIs (3.4% and 4.1%) compared with women without OASI (1.3%) (Trend $P<.0001$) who had received treatment (Figure 13).

Neither one nor two OASIs was associated with an increased risk for other PFDs and lower urinary tract symptoms (LUTS).

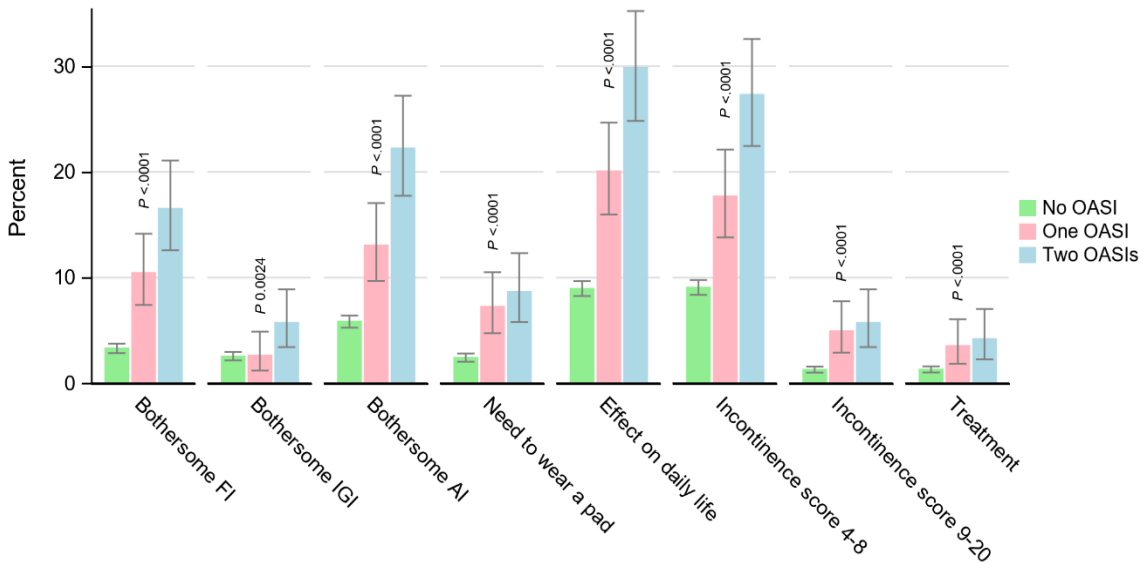


Figure 13. Impact of incontinence.

AI, anal incontinence; FI, fecal incontinence; IGI, isolated gas incontinence; Incontinence score, the Jorge-Wexner anal incontinence score; OASI, obstetrical anal sphincter injury; Treatment, refers to any kind of treatment (surgery, physiotherapy, medical, etcetera). The trend was analyzed using Mantel-Haenszel statistics for categorical variables, and the Spearman's rank correlation test for continuous variables. Reproduced from Nilsson *et al. Severity and impact of accidental bowel leakage two decades after no, one, or two sphincter injuries. Am J Obstet Gynecol* 2023; doi: <https://doi.org/10.1016/j.ajog.2022.11.1312> Online ahead of print.

5 GENERAL DISCUSSION

5.1 STUDY DESIGN

This thesis was based on national, high-quality, birth registers with either national total cohorts or national large simple random samples of women with restricted parity and mode of delivery. Restricting the inclusion criteria is an effective method to prevent confounding of known risk factors (134), in our case regional differences, multiparity, and mode of delivery. In Papers I, II, and IV, birth register data from comprehensive cohorts were combined with a self-administered, validated questionnaire on current symptoms of PFDs, controlling for recall bias. Questionnaire surveys are considered to yield more accurate data on sensitive or embarrassing topics (135) and gave the opportunity to further control for parity. The long-term follow-up from the first delivery in Papers I, II, and IV was approximately two decades.

5.2 METHODOLOGICAL CONSIDERATIONS

5.2.1 INTERNAL VALIDITY

Internal validity describes to what extent an observation or symptom is correct for the specific group studied, so that the effect is measured accurately in the specific group. Three general categories compromise internal validity: selection bias, information bias, and confounding (136).

5.2.1.1 SELECTION BIAS

Selection bias arises from systematic differences in the studied group and the source cohort. For example, even if questionnaire surveys are considered the most valid measure of the presence, severity, and impact of sensitive issues such as PFDs (137), the willingness to participate might be higher in women with symptoms, hence risking overestimating symptom frequency (130,138). A response rate of under 60% has been correlated to a high risk of response bias, which is why the response rates of the SWEPOP-1 and -2 surveys could be regarded as acceptable (139). In Paper I (SWEPOP-1), the non-responders were younger, more often overweight or obese, and more often gave birth to a child of ≥ 4 kg compared to responders, therefore having risk factors acting both ways on the risk of PFDs (lower age versus higher BMI and birth weight). In Papers II and IV (SWEPOP-2), the response rate was somewhat higher in the cohort of women with two OASIs compared to the randomly selected women with VDs. This may support the notion that women with symptoms are more willing to participate. Compared to responders in SWEPOP-2, the non-responders were younger, more often born abroad, and more often non-Swedish citizens with a lower education level and income (MBR data was not offered for non-responders). A lower age implies a lower prevalence of PFDs, but the possible impact of the other factors on the risk for PFDs is more uncertain – as they are all associated with a lower socioeconomic status. This group might also have a higher prevalence of various illnesses (140,141).

5.2.1.2 INFORMATION BIAS

Information bias occurs when there are measurement errors in the information about the participants, which is also called misclassification. In this thesis, obstetrical data of mothers and infants, and data for the analysis of responders and non-responders, were collected from high-quality national medical birth registers, as well as the TPR, the Income and Taxation Register, and the Register of Education. In the MBR, the information on parity is missing in approximately 2-9% of cases, leading to an incorrect parity (115). Importantly, we used a separate control question in the questionnaire about parity. Those who stated a higher parity than in the MBR and women with missing information in the questionnaire were excluded.

5.2.1.3 CONFOUNDING

Confounding arises when the effect of the studied exposure is mixed with the effect of an unknown exposure linked to the studied exposure, hence a mixing of effects. A confounder may lead to both over- and underestimation of results. Epidemiological studies must therefore try to control (adjust) for known and unknown confounders in the cohorts (142).

In paper I, we considered it relevant to adjust for current maternal weight and age, and infant birth weight. Crude ORs and adjusted ORs were almost similar, and crude ORs were not published. In Papers II and IV adjustments were made for known risk factors for FI (age and BMI).

5.2.2 EXTERNAL VALIDITY

External validity refers to the generalizability of the study results to populations outside the study (136). SWEPOP-1 aimed to study the effect of one vaginal or one caesarean delivery on future PFDs. As such, it ought to be highly generalizable to similar populations. With the same reservations, this applies to the 2-para women in SWEPOP-2. Due to the large, random sample of national data, the external validity is high. Paper III was predominantly a descriptive study, highlighting the differences in OASI incidence in countries with presumed medical and socioeconomic similarities.

In the SWEPOP surveys, we deliberately chose to include all VDs of primiparous or secundiparous women, regardless of gestational week, foetal presentation, instrumental delivery, etcetera, since the aim of this study was to compare vaginal delivery with or without VE, and with or without OASI, in total. In addition, women were included irrespective of health status and obstetrical complications, to better be able to generalise results and to get a more realistic basis for consultation.

5.2.3 RANDOM ERROR

Precision is essential for the accuracy and validity of the study (136). In epidemiology, random error is mainly about the process of collecting the study population and, therefore, a sampling error. The precision can be improved by increasing the sample size of the study and by dividing data into strata for comparisons. SWEPOP-1 and -2 both consisted

simple random samples of ~10,000 women each, and in Paper III, national, total, cohorts were used. In all papers, study participants were further divided into strata based on the mode of delivery, or delivery with or without OASI.

5.3 REFLECTION REGARDING ALTERNATIVE STUDY DESIGNS

Papers I, II, and IV are cohort studies establishing selection criteria and exposure factors in advance from a national register, e.g., parity, delivery mode, diagnosis, etcetera. In agreement with the recommendation by Rothman, Greenland, and Lash, they are prospective studies, as the studied disease outcomes (PFDs) could not influence the exposure information (143). Information about exposure was obtained from a national register and not from information obtained from the participants. Recall bias regarding the degree of perineal tears has been found to be high 5-10 years after delivery (144). The SWEPOP surveys report on long-term (20 years) symptoms of PFDs after childbirth. Prospective studies with long-term follow-up are time- and money-consuming and are afflicted with a progressively increasing number of drop-outs and consequently decreasing the number of participants, which often has led to underpowered analyses. Paper III is an ecological study, as no individual data were available (145).

5.3.1 RANDOMISED CONTROLLED TRIALS

The most robust evidence regarding causality is obtained through randomised controlled trials (RCT) (146). However, an RCT to analyse the association between obstetrical events and future PFDs, will not be approved due to the ethical considerations of randomising women to a set parity or a fixed delivery mode. Furthermore, RCTs with long-term follow-up will exhibit similar shortcomings as prospective long-term follow-up cohort studies.

The incidence of OASI is however a short-term outcome. In a recent RCT that was published in *The Lancet*, women with a first vaginal delivery were randomised to either standard care with one midwife present at delivery or to an intervention group with an extra midwife assisting from the second stage of labour to the delivery of the infant (147). The primary outcome was the prevalence of OASI in the intervention group compared to the standard care group. The result showed that assistance from a second midwife during the active second stage of labour reduced the risk of OASI (OR 0.68, 95% CI 0.49–0.95).

There are many studies analysing risk factors for OASI (32-40), and the next step would be to identify the individual risk of OASI using logistic prediction models developed from large databases (45,148,149).

5.3.2 CASE-CONTROL STUDIES

Initially, a retrospective case-control study was considered less reliable than a cohort study. This is based on the early experiences from case-control studies, in which exposure was identified by asking study participants to recall their exposure history (143). The case-control study typically investigates rare diseases, and the cases are chosen based on their disease status (in our case PFDs) and not exposure (VD, CS, VE, and OASI) (145). If we had conducted a case-control study, the exposure status could have been identified without the risk of recall bias. Even though the rate of AI and FI was not established among parous women when designing the SWEPOP-1 and -2 studies, it was unlikely that AI and FI would be rare (2). Furthermore, without a large random sample, we would have missed valuable information on the prevalence of OASI and repeat OASI (Figure 9) (150). Hence, a case-control study design was not considered for the SWEPOP surveys.

5.3.3 A QUALITATIVE APPROACH

The qualitative approach is founded on theories about human experience and human interpretation. It aims to explore the meaning of social and cultural phenomena as the afflicted person perceives them in their habitual context. The results of qualitative studies are often used to create hypotheses for quantitative studies (151). Qualitative studies have been crucial in stressing the relevance of epidemiological studies of FI and AI as they have revealed a range of severe consequences due to the

symptoms, such as reduced self-esteem, anxiety, unwillingness to leave home, as well as relationship and work difficulties (27,28,102).

5.3.4 SYSTEMATIC REVIEW AND META-ANALYSIS

The explosion of scientific articles, and the need for informed decision-making in clinical practice and public health, has led to the need for research results to be synthesised in systematic reviews and meta-analyses. Meta-analyses may further help to understand and quantify the variability of the results between studies (152). The systematic review and meta-analysis by LaCross et al. highlighted that high-quality studies on the long-term (>10 years) prevalence of PFDs after OASI was missing (43), which is why the SWEPOP surveys were conducted. Regarding the incidence of OASI, rates seem to differ considerably between countries, and over time (40,96-98,153), and no meta-analysis has yet been performed on the overall incidence. Still, one has affirmed the increased risk of OASI in VBAC (154).

5.3.5 ANIMAL MODELS

Changes in female physiology and changes to the pelvic floor with pregnancy are still essentially unknown and pose a largely unexplored research field (60,61). Using animal models is one alternative to spread light on these unresolved questions. A study on rats found that an increased load on the pelvic floor induced plasticity of the muscles, resulting in a protective effect against mechanical injury. The effect

increased if the rat was gravid (155). Crush injury to the pudendal nerve in rats, simulating a birth injury, induced a recoverable SUI where the leak point pressure was lowest after four days but recovered to almost normal levels two weeks after injury (156). However, there may be ethical considerations of subjecting animals to potentially painful experiments, especially since the results of animal models may not apply to humans. For example, the fact that rats are quadrupeds and humans are bipeds may influence comparability.

5.4 STRENGTHS AND LIMITATIONS

The national birth registers and the study design were the main strengths of this thesis. Because of their completeness, the quality of the birth registers and the TPR is considered high. The study cohorts were large, from countries with similar socio-economic and demographic conditions, and surveyed during limited periods. Response rates were high in the questionnaire surveys compared with earlier studies in this research field (157,158). The design strategy of restriction controlled for confounding factors. The study populations of Papers I, II, and IV were retrieved from a high-quality national register, and the control groups were the theoretically most appropriate (159). The questionnaire used validated questions about PFDs and LUTS. In large populations, a questionnaire survey is considered the most appropriate tool to collect information about sensitive issues such as FI (135), and numerous studies have shown that self-reporting is consistent (160) and valid when the symptoms exist concurrently (160-162).

However, the prevalence of PFDs in Papers I, II, and IV might be overestimated as the willingness to participate in questionnaire studies is higher in symptomatic women. In addition, the participants reported their current height, weight, and PFD symptoms in the questionnaire. The accuracy of this information is dependent on the participants' willingness and ability to provide it. Responders may misinterpret questions and feel uncomfortable answering sensitive questions, which may lead to under- or overestimation of the rates. We presumed that FI was more bothersome than IGI. It is a limitation that SWEPOP-1 and -2 lacked information about PFDs before or during pregnancy.

Underreporting of OASI is recognised as a substantial hindrance in epidemiologic research of perineal lacerations and the aetiology of FI (17). In Sweden, the incidence of sphincter injury was less than 1% in nullipara during the mid-80s, which most likely reflects an underreporting in MBR. Tears involving only a minor part of the sphincter muscle may be unreported. This may have led to a selection bias of more severe lacerations in the OASI cohorts and, most probably, an overestimation of FI. Conversely, it would also entail that less severe OASIs were present in women allocated to the control groups. In Paper III, differences in the diagnoses and reporting of OASI and the exact technique for perineal protection during delivery of the foetus were not available. Misclassification of perineal injuries may explain the differences to some extent. There is, however, no reason to assume that the trends in OASI rates within and between countries vary by chance.

5.5 CONSIDERATIONS OF DEFINITIONS AND CLASSIFICATIONS

5.5.1 OBSTETRICAL ANAL SPHINCTER INJURY

OASI was analysed as one entity with no distinction between 3rd and 4th-degree injury. Until 1997, there was no information in the MBR about the subgroups of 3rd-degree OASIs, and 4th-degree injuries were rare (approximately 7% of all OASIs in SWEPOP-2).

5.5.2 QUESTIONNAIRE

In 2007, when the SWEPOP-1 survey was designed, no validated questionnaire for all three main PFDs (UI, sPOP, and FI) had been constructed and used. We, therefore, used validated questions from Jorge and Wexner (FI), Tegerstedt et al. (sPOP), and Sandvik et al. (UI) (130-132). In our questionnaire, the number and order of the questions might have affected the answers, but in that case, the groups that were compared would have been equally afflicted by this bias.

5.5.3 FAECAL INCONTINENCE

Qualitative studies have revealed numerous debilitating effects due to FI, such as feelings of shame, social limitations and isolation, sexual difficulties, and not being able to work full time (27,28). The results of qualitative studies are, however, limited by small numbers of study

participants, and participants were often recruited among patients seeking healthcare, or awaiting surgical treatment, for FI. Questionnaires are used to quantitatively estimate the prevalence and subjective impact of FI in larger populations. At the time of the construction of our questionnaire, the Jorge-Wexner incontinence score was the most widely used form and was considered easy to understand and sensitive to changes over time with a limited number of questions (163). The score has been validated in several studies (164-167), but it has been unclear how the women's perception of their incontinence compares to the Jorge-Wexner score. A Jorge-Wexner score of ≥ 9 has indicated a significant impairment in quality of life (166). In one study, even a lower score was shown to affect the quality of life for women still employed or having active physical and social lives (168). It has also been shown that the degree of bother is strongly associated with coping behaviours, such as the use of pads (169).

5.5.4 URINARY INCONTINENCE

The question about UI was specified by questions about subtypes of UI. UI subtypes have been shown to be associated with different degrees of severity and bother, and with age (1,170-173). As UI is a frequent symptom in epidemiological surveys, with a prevalence of 25-45%, the severity and impact of UI are clinically more relevant. The question about the subjective impact (bothersomeness) was also used in the EPICONT survey of almost 30 000 Norwegian women (172).

5.5.5 PELVIC ORGAN PROLAPSE

In 2005, Tegerstedt et al. validated a short-form questionnaire for sPOP, the question “Do you have a sense of tissue protrusion (vaginal bulge) from your vagina” had almost all the predictive capacity of the test (130), which has been verified by others (94,127,174). The short form was developed on women with and without confirmed prolapse to the introitus, and when tested on a random sample of 8000 women (29-79 years old), the questionnaire had 66.5% sensitivity and 94.2% specificity (175). When the question was used in the SWEPOP-0 project, a positive answer was set to “often, sometimes, or infrequently” and nulliparous women aged 25-34 years presented a higher prevalence (9.6%) of sPOP than women aged 55-64 (6.4%) (176). However, bothersome sPOP and symptoms aggravated by heavy lifting were about twice as common in older women. When using the cutoff “sometimes or more often”, the prevalence decreased to 2.9%, compared to 2.4% in the study of Tegerstedt et al. (175). The high prevalence of a “vaginal bulge” among the younger nullipara was thought to reflect coexisting conditions (such as “a chafing feeling”, OAB, and FI) common in the group, indicating a spectrum bias (174,176,177). Hence, the frequency for affirming sPOP was changed from often/sometimes/infrequently in Paper I, to often/sometimes in Paper IV.

5.6 MAIN FINDINGS AND COMMENTS

5.6.1 PAPER I

OASI occurred three times more often in vacuum-assisted vaginal delivery than in spontaneous delivery. An OASI doubled the long-term prevalence of faecal incontinence irrespective of whether the OASI occurred after a spontaneous or vacuum-assisted delivery. The long-term prevalence of other PFDs was similar after SVD and VE but consistently lower after an ACS. Further, OASI was associated with an increased risk of UI and FI rates but not sPOP.

The increased incidence of sphincter injury after VE in our cohort is consistent with previous reports on primipara. In the study by Robinson et al., the incidence of OASI was 8.7% after SVD and 29.8% after VE (178). In addition, the risk of sustaining an OASI was 2.9 in VE compared with SVD in a study on primiparas by Ekeus et al. (179). According to the MBR, the mean incidence of sphincter injury in primipara 2009-2013 was 13.4% after VE and 16.9% after VE in a VBAC (180). In the present study, the prevalence of FI was 30.2% two decades after OASI, considering the 17 years longer follow-up, this is consistent with the mean FI rate of 21.3% found in 13 studies from 1993 to 2004 (OASI n=936, median follow-up 31 months, range 10–72) (181).

There was scarce information about long-term PFDs after VE compared with spontaneous delivery. In a study from the US, the prevalence of bothersome PFDs 5-10 years after delivery was similar after VE (n= 49) and SVD (n=324) conforming with our results (182). In a Norwegian study with 15-23 years of follow-up, the prevalence of PFDs was higher after operative VD (VE n = 299, forceps n=335) compared to SVD (n = 692): FI 12 vs. 6%, UI 48 vs. 49%, sPOP 15 vs. 9% (183). However, in that study, the cohorts were mixed regarding mode of delivery and parity (mean parity 2.2); hence, the comparison with our results is uncertain.

In a study with a fictive VE setting, clinicians were urged to achieve a minimum, average, and excessive traction force, and they generally misjudged and underestimated the pull force applied at the tests (184). Furthermore, in a clinical setting, the employed extraction force was even higher (184). In the Cochrane review from 2010 by O'Mahony et al., forceps and VE delivery were compared in ten randomized prospective studies. The overall OASI rate was 14.0% after forceps and 7.5% after VE. However, the OASI rate varied from 0 to 25%, significantly related to the OASI rates after forceps (Kendall's $\tau=0.81$, $p<0.004$), which indicates that the setting for instrumental delivery was a risk factor for OASI (185).

5.6.2 PAPER II

The risk for a repeat sphincter injury nearly tripled after having an OASI at first birth.

Twenty years after birth, the prevalence of all symptoms of FI was doubled in women with one OASI and tripled after two OASIs compared to women with two VD's without OASI. After one and two OASIs, severe FI increased 3- and 5-fold. Sphincter injury was associated with increased occurrence of the triple combination of solid and liquid stools and concomitant gas. The incidence of FI was relatively constant at ages 40 to 60 years in women without OASI but increased after 52 years in those with OASI. Less than one in five women with OASI were continent for stool and gas at the age of 60 years.

The risk of a repeat sphincter injury at the subsequent birth was 6.3% (range 2.0-13.4%) in a review by Jha and Parker on 99,042 women (99). The wide range was thought to be due to a skewed distribution of predictors of sphincter injury and obstetrical practice. Due to the higher response rate after two OASIs (70.1% vs 65.5%), our result (10.0%) may be somewhat inflated.

Currently, convenience samples from single hospitals (11,23-25,144,186) with follow-up periods of <10 years (11,24,144) have been used in most studies about long-term FI after sphincter injury. In two studies, only women with "complete" OASI were included (11,186), and six studies used uncertain definitions of FI or presented solid and liquid stool leakage separately, which most likely add up to an overestimated rate of FI when summarized (11,23-25,144,186). The mode and number of previous and subsequent births have not been fully controlled for in any of the studies. In women without OASI, the

prevalence of FI ranged from 5.3% (23) to 12.2% (24), and in women with one OASI, FI rates ranged from 13.5% (23) to 28.8% (25). The mean increase of FI from no OASI to one OASI in four studies was 13.5% (24,25,144,186). In Denmark and Sweden, there are currently two large projects on FI after no, one, or two OASIs, both based on validated national registers and questionnaire surveys and with follow-up periods of 12, respectively, 20 years (26,187,188). The prevalence of FI among women without OASI was 5.6% in the Danish study (188) and 13.7% in the Swedish study (187). In women with one OASI, the prevalence was 13.2% and 16.5% in the Danish cohort with two VDs (26,189) and 28.4% in the Swedish cohort with one VD (187). After two sphincter injuries, the prevalence was 23.6% (26) and 36.1% in this study. On average, the difference in FI rates was about 10% in the national cohorts with no, one, and two OASIs, ~10% higher in the Swedish compared to the Danish study. The difference between the two national cohorts may be explained by the difference in follow-up time and age when answering the questionnaires. A longitudinal project from the Netherlands supports this assumption as it reported an increasing prevalence of FI (once a week or more often) from 8.8% (11/125) at 14 years after OASI to 15.1% (18/119) 25 years after (190,191).

5.6.3 PAPER III

The incidence of sphincter injury in primiparous women varied widely over time and between countries despite similar socio-economic conditions. Canada reported the highest incidence of OASI, ~25%, in

forceps delivered primipara in 2016. Austria had the lowest rate of OASI, but the rate increased in all scenarios. Starting in 2004, only Norway reported a consistent and significant decrease in OASI incidence, which more than halved during the study period.

Due to the lack of high-quality birth registers, the number of international comparisons on OASI rates is limited (96,192). Laine et al. compared the incidence of OASI in Denmark, Finland, Norway, and Sweden and found a significant increasing trend from 1986 to 2004 (192). From 2004 to 2010, there was a decreasing rate of OASI in Norway but not in any of the other three countries (193). The present study confirmed this trend in Norway.

The present study showed some apparent dissimilarities in clinical practices among the countries. The incidence of OASIs may be influenced by the overall national rate of caesarean sections, assisted vaginal births (i.e., vacuum and forceps deliveries), and the episiotomy rate. The rate of caesarean sections was significantly higher in Canada and Austria (27% and 31%) compared to Sweden and Norway (16% and 18%). In 2010, the rate of episiotomies differed widely. In Sweden, the episiotomy rate was 5% in SVD and 26% in VE. In Canada, the episiotomy rate was much higher, 18% in SVD and 45% in VE (39,96). In the present study, the OASI rates were similar in Sweden and Canada in both scenarios, indicating a complex association between episiotomy and OASI. In a study of 20 European countries, Blondel et al. came to

the same conclusion and urged the need for better evidence to define an optimal rate of episiotomies (96).

Midwives attend SVDs in Austria, Norway, and Sweden, whereas physicians perform vacuum and forceps deliveries. In Canada, physicians attend a majority of even low-risk births. Factors such as financial motives and concerns regarding lawsuits could influence obstetricians' choice of delivery mode, consequently influencing the rates of instrumental delivery and possibly increasing the rate of OASI (194). In Austria and Sweden, obstetricians managed VD almost entirely without forceps in contrast to Canada, where every third instrumental birth was a forceps delivery, which was associated with a high and increasing rate of OASI (194). In a Cochrane review from 2010 by O'Mahony et al., forceps delivery almost doubled the risk of OASI compared with VE (relative risk 1.89, 95% CI 1.51–2.37) (185). Pergialiotis et al. found similar results in a meta-analysis of 22 studies ($n = 651\ 934$); a forceps delivery increased the risk for OASI by an OR of 5.50, and VE increased the risk of OASI by an OR of 3.98 (195). Hence, women should be entitled to this information antenatally, especially in countries where both delivery modes are in use. In the Cochrane review from 2010, the rate of OASI after VE was significantly related to the rate after forceps, indicating that the setting highly influences the risk of sphincter injury (185). Hence, to reduce the rate of OASI, appropriate training of obstetricians is most probably necessary (196).

Women are generally recommended a trial of labour after a CS in the first birth (197,198). However, they are often uninformed of the increased risk of OASI (especially if an instrumental delivery is required) and this must be considered in the overall risk assessment (33,38,96,197-199).

A significant problem is the misdiagnosis of OASIs. Differences in skills and formalized training might explain why recognition and classification sometimes fail (33,193,200,201) and why women are withheld treatment and information (200). Postpartum perineal ultrasound has been suggested to improve the detection of an OASI (202).

Austrian obstetric practice has for many years focused on pelvic floor protection during childbirth, both in education and clinical work, which is thought to explain the low rates of OASI. However, since 2007, the trend of the OASI rate has increased significantly.

In 2004, The National Board of Health and Supervision in Norway requested a national action plan for obstetricians and midwives to reduce the rate of sphincter injury. A structured training program was initiated that included: to manually slow down the crowning of the infant's head at the second stage of delivery, avoiding pushing during crowning, having appropriate visualization and manual support of the perineum, and performing a lateral or mediolateral episiotomy on indication (203,204). The interventions should be used in SVD and operative

vaginal deliveries (193,203). After implementation, there was a consistent decrease in OASI incidence by more than 50% in all subgroups. OASI rates were reduced by 70% for forceps deliveries in 2015, although the rate of operative vaginal deliveries has increased by four percentage points. When a comparable program was introduced at a UK general hospital, OASI rates were reduced from 4.8% to 3.1% ($P=0.008$) after a year (205), and at the Croydon University Hospital, the OASI incidence decreased from 4.7% to 4.1%, with a statistically significant decrease in grade 3c and grade 4 OASI ($P<0.001$) (206). In a large teaching hospital in the Netherlands, implementing a similar program was associated with reducing the OASI incidence by more than 50%, but after the initial improvement, the OASI rate relapsed. Non-adherence to the program among specialists was considered the main obstacle to upholding the lower rates (207).

5.6.4 PAPER IV

Two decades after two vaginal births with one or two OASIs, there was a significant trend of more frequent leakage, more severe grades of all outcomes, and an increasing impact of anal incontinence, compared to controls. The first and the second sphincter injury showed an equal cumulative effect on multiple self-reported severity and impact outcome measures. However, one or two sphincter injuries did not affect the prevalence of urinary incontinence, symptomatic pelvic organ prolapse, or lower urinary tract symptoms.

Studies on the long-term severity and impact of anal incontinence after OASI are contradictory (23-26,144,186,190,208,209), and currently, there is only one study investigating the consequences of two consecutive OASIs (26). In a study by de Leeuw et al., 125 matched pairs of women with and without OASI were followed for about 14 years. In women with OASI, symptoms of AI had a more rapid onset (within 3 months postnatally, $P.003$) and a higher frequency than in women without OASI ($P.004$). In conformity with our results, no association between OASI and SUI or UUI was found (190).

Fifty-three women with one OASI, of which ~60% were verified with ultrasound, were followed by Samarasekera et al. and compared with 69 women without OASI and 53 women delivered by ECS. Ten years (or more) after delivery, there was a significant increase in the mean Jorge-Wexner score and a significant decrease in the QoL among women with OASI compared to controls (208).

In a hospital-based study by Baud et al., an OASI was sustained in 1.5% of the deliveries in a population where 70% were multiparous (1996-2006). OASI cases were matched with three times as many VD controls and were invited to a questionnaire survey ~6 years after delivery, 37.5% (66/176) of the cases and 36% (192/534) of the controls responded. The Jorge-Wexner scores corresponded with our study, as women with OASI had a mean score of 2.3, compared to 1.2 in women without ($P.004$). Furthermore, 15.4% of women with OASI reported severe anal incontinence (a Jorge-Wexner score ≥ 5) compared with

6.6% of controls (RR 2.6, 95%CI 1.1-6.3), numbers in agreement with the mean total effect size of severe AI after one OASI in our study (OR 2.2) (24).

The hospital-based study of Soerensen et al. had a mean follow-up of 22 years after delivery and revealed conflicting results. This case-control study compared 125 women with a complete sphincter rupture with 238 matched controls without OASI. Even though both the Jorge-Wexner and the St Mark's scores were increased in women with a complete sphincter rupture compared to the controls ($P=0.02$ and $P<0.001$), bothersome AI and impact on QoL due to AI did not differ between cases and controls (186). In a national register and questionnaire study, Jangö et al. surveyed all women with two deliveries from 1997-2005 (no previous or further births), of which an OASI complicated the first delivery. Of the responders, 1384 had one OASI, and 106 women had a recurrent OASI. About 11 years after the first delivery, 34.9% (37/106) of the women with two OASIs had affected QoL (yes = "some" and "a lot of bother") due to AI, compared to 24.2% (335/1384) in women with one OASI. However, the difference was not considered significant when adjusting for degree of OASI, age, infant birth weight, years since second delivery, and if AI was present between the two deliveries (aOR 1.53, 95%CI 0.92-2.56, $P=0.10$) (26).

6 CONCLUSION

6.1 PAPER I

The incidence of OASI in primiparous women was three times higher after a VE-assisted delivery than an SVD. OASI doubled the long-term risk of faecal incontinence regardless of whether the injury occurred at a VE or an SVD. Antenatally, women should be entitled to information about these risks. Vacuum-assisted delivery is a procedure that requires training, experience, and that the physician adheres to established guidelines regarding indications as well as knowing when to abstain from the procedure.

6.2 PAPER II

The risk of a repeat OASI tripled after an OASI at the first delivery. The prevalence of faecal incontinence doubled and tripled after one and two OASIs. Avoiding an OASI may significantly reduce the risk of and postpone the onset of age-related FI. This study supports the hypothesis that there is a causal link between OASI and an increased risk of FI in the long term.

6.3 PAPER III

Despite similar demographics, Austria, Canada, Norway, and Sweden had varying incidences of OASI and obstetric practices over time and between the countries. There appeared to be considerable potential for lowering the incidence of OASI in countries with high rates. Limiting the use of forceps in favour of vacuum delivery seems like a reasonable first step in reducing the rate of OASI. The perineal protection program implemented by Norway in 2004 appeared to be advantageous. Each country should critically analyse its current rates of OASI and clinical practices, and consider the best preventive strategy – a national, recurring action plan for structured education and training of all staff members appears to be essential.

6.4 PAPER IV

One and two OASIs showed a consistent cumulative effect on the severity and impact of anal incontinence two decades after two vaginal births, compared to women without OASI. This information is essential for healthcare economics, healthcare professionals, clinical practice, and policy. This study showed that an OASI did not increase the risk for other pelvic floor disorders or lower urinary tract symptoms. The severity and impact of long-term AI would most probably be significantly reduced by avoiding OASI at delivery. This calls for further research to elucidate the best strategy for preventing OASI and a critical attitude toward current clinical practice.

7 FUTURE PERSPECTIVES

The results of the four papers of this thesis show that OASI was a potent risk factor for long-term faecal incontinence in vaginally parous women based on prevalence, severity, and impact, irrespective of what caused the injury. Instrumental delivery was still the leading risk factor for OASI, forceps delivery being the dominant obstetrical adverse intervention. Perineal protection, when systematically and persistently applied, may lower the rate of OASI. In the future, prevention may be further advanced selectively, using prediction algorithms for individual risk assessment of OASI in different scenarios, offering women a tool for their autonomous decision about their preferred mode of delivery.

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REFERENCES

1. Milsom I, Altman D, Cartwright R, et al. Epidemiology of urinary incontinence (UI) and other lower urinary tract symptoms (LUTS), pelvic organ prolapse (POP) and anal incontinence (AI). In: Abrams P, Cardozo L, Wagg A, Wein A, eds. *Incontinence* 6ed. Bristol, UK: ICI-ICS. International Continence Society; 2017:4-93.
2. Ng KS, Sivakumaran Y, Nassar N, Gladman MA. Fecal Incontinence: Community Prevalence and Associated Factors - A Systematic Review. *Dis Colon Rectum* 2015;58:1194-209.
3. Salvatore S, Delancey J, Igawa Y, et al. Pathophysiology of Urinary Incontinence, Faecal Incontinence and Pelvic Organ Prolapse. In: Abrams P, Cardozo L, Wagg A, Wein A, eds. *Incontinence* 6ed. Bristol, UK: ICI-ICS. International Continence Society; 2017:413-31.
4. DeLancey JO. The hidden epidemic of pelvic floor dysfunction: achievable goals for improved prevention and treatment. *Am J Obstet Gynecol* 2005;192:1488-95.
5. Freeman A, Menees S. Fecal Incontinence and Pelvic Floor Dysfunction in Women: A Review. *Gastroenterol Clin North Am* 2016;45:217-37.
6. de Boer TA, Sliker-Ten Hove MC, Burger CW, Kluivers KB, Vierhout ME. The prevalence and factors associated with previous surgery for pelvic organ prolapse and/or urinary incontinence in a cross-sectional study in The Netherlands. *Eur J Obstet Gynecol Reprod Biol* 2011;158:343-9.
7. Wu JM, Matthews CA, Conover MM, Pate V, Jonsson Funk M. Lifetime risk of stress urinary incontinence or pelvic organ prolapse surgery. *Obstet Gynecol* 2014;123:1201-6.
8. Wagenius J, Laurin J. Clinical symptoms after anal sphincter rupture: a retrospective study. *Acta Obstet Gynecol Scand* 2003;82:246-50.
9. Otero M, Boulvain M, Bianchi-Demicheli F, et al. Women's health 18 years after rupture of the anal sphincter during childbirth: II. Urinary incontinence, sexual function, and physical and mental health. *Am J Obstet Gynecol* 2006;194:1260-5.
10. Heilbrun ME, Nygaard IE, Lockhart ME, et al. Correlation between levator ani muscle injuries on magnetic resonance imaging and fecal incontinence, pelvic organ prolapse, and urinary incontinence in primiparous women. *Am J Obstet Gynecol* 2010;202:488.e1-6.
11. Wegnelius G, Hammarström M. Complete rupture of anal sphincter in primiparas: long-term effects and subsequent delivery. *Acta Obstet Gynecol Scand* 2011;90:258-63.
12. Siahal SF, Irvani M, Mohaghegh Z, Sharifipour F, Zahedian M. Maternal, obstetrical and neonatal risk factors' impact on female urinary incontinence: a systematic review. *Int Urogynecol J* 2020.

13. Lord Neuberger. Montgomery (Appellant) v Lanarkshire Health Board (Respondent) (Scotland). United Kingdom Supreme Court <http://www.bailii.org/uk/cases/UKSC/2015/11.html>. Published 2015. Accessed 10 October, 2022.
14. Bols EMJ, Hendriks EJM, Berghmans BCM, Baeten CGMI, Nijhuis JG, de Bie RA. A systematic review of etiological factors for postpartum fecal incontinence. *Acta Obstet Gynecol Scand* 2010;89:302-14.
15. Waldenström U, Ekéus C. Risk of obstetric anal sphincter injury increases with maternal age irrespective of parity: a population-based register study. *BMC Pregnancy Childbirth* 2017;17:306-.
16. Pollack J, Nordenstam J, Brismar S, Lopez A, Altman D, Zetterstrom J. Anal incontinence after vaginal delivery: a five-year prospective cohort study. *Obstet Gynecol* 2004;104:1397-402.
17. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Engl J Med* 1993;329:1905-11.
18. Kamm MA. Obstetric damage and faecal incontinence. *Lancet* 1994;344:730-3.
19. Zetterström JP, López A, Anzén B, Dolk A, Norman M, Mellgren A. Anal incontinence after vaginal delivery: a prospective study in primiparous women. *Br J Obstet Gynaecol* 1999;106:324-30.
20. Groutz A, Fait G, Lessing JB, et al. Incidence and obstetric risk factors of postpartum anal incontinence. *Scand J Gastroenterol* 1999;34:315-8.
21. Nazir M, Stien R, Carlsen E, Jacobsen AF, Nesheim BI. Early evaluation of bowel symptoms after primary repair of obstetric perineal rupture is misleading: an observational cohort study. *Dis Colon Rectum* 2003;46:1245-50.
22. Tilak M, Mann GK, Gong M, Koenig NA, Lee T, Geoffrion R. Pelvic floor healing milestones after obstetric anal sphincter injury: a prospective case control feasibility study. *Int Urogynecol J* 2022;1-9.
23. Faltin DL, Otero M, Petignat P, et al. Women's health 18 years after rupture of the anal sphincter during childbirth: I. Fecal incontinence. *Am J Obstet Gynecol* 2006;194:1255-9.
24. Baud D, Meyer S, Vial Y, Hohlfeld P, Achtari C. Pelvic floor dysfunction 6 years post-anal sphincter tear at the time of vaginal delivery. *Int Urogynecol J* 2011;22:1127-34.
25. Halle TK, Salvesen K, Volløyhaug I. Obstetric anal sphincter injury and incontinence 15-23 years after vaginal delivery. *Acta Obstet Gynecol Scand* 2016;95:941-7.
26. Jangö H, Langhoff-Roos J, Rosthøj S, Sakse A. Recurrent obstetric anal sphincter injury and the risk of long-term anal incontinence. *Am J Obstet Gynecol* 2017;216:610.e1-.e8.

27. Rasmussen JL, Ringsberg KC. Being involved in an everlasting fight - a life with postnatal faecal incontinence. A qualitative study. *Scand J Caring Sci* 2010;24:108-15.
28. Olsson F, Berterö C. Living with faecal incontinence: trying to control the daily life that is out of control. *J Clin Nurs* 2015;24:141-50.
29. Bharucha AE, Zinsmeister AR, Locke GR, Schleck C, Mckeon K, Melton LJ. Symptoms and quality of life in community women with fecal incontinence. *Clin Gastroenterol Hepatol* 2006;4:1004-9.
30. Brown HW, Wexner SD, Segall MM, Brezoczky KL, Lukacz ES. Quality of life impact in women with accidental bowel leakage. *Int J Clin Pract* 2012;66:1109-16.
31. Barbosa M, Glavind-Kristensen M, Moller Soerensen M, Christensen P. Secondary sphincter repair for anal incontinence following obstetric sphincter injury: functional outcome and quality of life at 18 years of follow-up. *Colorectal Dis* 2020;22:71-9.
32. Jangö H, Langhoff-Roos J, Rosthøj S, Sakse A. Modifiable risk factors of obstetric anal sphincter injury in primiparous women: a population-based cohort study. *Am J Obstet Gynecol* 2014;210:59.e1-6.
33. Practice Bulletin No. 198: Prevention and Management of Obstetric Lacerations at Vaginal Delivery. The American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2018;132:e87-e102.
34. Gurol-Urganci I, Cromwell DA, Edozien LC, et al. Third- and fourth-degree perineal tears among primiparous women in England between 2000 and 2012: time trends and risk factors. *BJOG* 2013;120:1516-25.
35. Richter HE, Brumfield CG, Cliver SP, Burgio KL, Neely CL, Varner RE. Risk factors associated with anal sphincter tear: a comparison of primiparous patients, vaginal births after cesarean deliveries, and patients with previous vaginal delivery. *Am J Obstet Gynecol* 2002;187:1194-8.
36. Marschalek ML, Worda C, Kuessel L, et al. Risk and protective factors for obstetric anal sphincter injuries: A retrospective nationwide study. *Birth* 2018;45:409-15.
37. Fernando R, Sultan A, Freeman R, Williams A, Adams E. Third- and Fourth-degree Perineal Tears, Management. Green-top Guideline No. 29. Royal College of Obstetricians & Gynaecologists. <https://www.rcog.org.uk/guidance/browse-all-guidance/green-top-guidelines/third-and-fourth-degree-perineal-tears-management-green-top-guideline-no-29/>. Published 2015. Accessed 10 October, 2022.
38. Jardine JE, Knight HE, Carroll FE, Gurol-Urganci I. Risk of obstetric anal sphincter injury in women having a vaginal birth after a previous caesarean section: A population-based cohort study. *Eur J Obstet Gynecol Reprod Biol* 2019;236:7-13.

39. Muraca GM, Liu S, Sabr Y, et al. Episiotomy use among vaginal deliveries and the association with anal sphincter injury: a population-based retrospective cohort study. *Cmaj* 2019;191:E1149-e58.
40. Safe acute care – obstetric trauma. OECD. <https://www.oecd-ilibrary.org/content/component/c2a6b2da-en>. Published 2019. Accessed 10 October, 2022.
41. Pergialiotis V, Bellos I, Fanaki M, Vrachnis N, Doumouchtsis SK. Risk factors for severe perineal trauma during childbirth: An updated meta-analysis. *Eur J Obstet Gynecol Reprod Biol* 2020;247:94-100.
42. Statistics database for pregnancies, births and newborns. The Swedish National Board of Health and Welfare. https://sdb.socialstyrelsen.se/if_mfr_004/val.aspx Published 2022. Accessed June 7, 2022.
43. Lacross A, Groff M, Smaldone A. Obstetric anal sphincter injury and anal incontinence following vaginal birth: a systematic review and meta-analysis. *J Midwifery Womens Health* 2015;60:37-47.
44. Webb SS, Hemming K, Khalfaoui MY, et al. An obstetric sphincter injury risk identification system (OSIRIS): is this a clinically useful tool? *Int Urogynecol J* 2017;28:367-74.
45. Luchristt D, Meekins AR, Zhao C, et al. Risk of obstetric anal sphincter injuries at the time of admission for delivery: A clinical prediction model. *BJOG* 2022.
46. Stoker J. Anorectal and pelvic floor anatomy. *Best Pract Res Clin Gastroenterol* 2009;23:463-75.
47. Grimes WR, Stratton M. Pelvic Floor Dysfunction. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing Copyright © 2022, StatPearls Publishing LLC.; 2022.
48. Shobeiri SA, DeLancey JOL. Pelvic floor anatomy. In: Santoro GA, Wiczorek AP, Sultan AH, eds. *Pelvic floor disorders, a multidisciplinary textbook*. 2 ed. Cham, Swiss Confederation: Springer Nature Switzerland AG 2021; 2021:3-24.
49. Hodges PW, Sapsford R, Pengel LH. Postural and respiratory functions of the pelvic floor muscles. *Neurol Urodyn* 2007;26:362-71.
50. Szczygieł E, Blaut J, Zielonka-Pycka K, et al. The Impact of Deep Muscle Training on the Quality of Posture and Breathing. *J Mot Behav* 2018;50:219-27.
51. Koelbl H, Halpern-Elenskaia K. Biochemical properties and hormonal receptors of pelvic floor tissues. In: Santoro GA, Wiczorek AP, Sultan AH, eds. *Pelvic floor disorders, a multidisciplinary textbook*. 2 ed. Cham, Swiss Confederation: Springer Nature Switzerland AG 2021; 2021:25-30.
52. Kearney R, Sawhney R, DeLancey JO. Levator ani muscle anatomy evaluated by origin-insertion pairs. *Obstet Gynecol* 2004;104:168-73.

53. Shuster M. Treatment: Biofeedback. In: Henry M, Swash M, eds. *Coloproctology and the pelvic floor*. 2 ed. London: Butterworth-Heinemann; 1991.
54. Lestar B, Penninckx F, Kerremans R. The composition of anal basal pressure. An in vivo and in vitro study in man. *Int J Colorectal Dis* 1989;4:118-22.
55. Meagher AP, Lubowski DZ, King DW. The cough response of the anal sphincter. *Int J Colorectal Dis* 1993;8:217-9.
56. Liu J, Guaderrama N, Nager CW, Pretorius DH, Master S, Mittal RK. Functional correlates of anal canal anatomy: puborectalis muscle and anal canal pressure. *Am J Gastroenterol* 2006;101:1092-7.
57. DeLancey JO. Anatomic aspects of vaginal eversion after hysterectomy. *Am J Obstet Gynecol* 1992;166:1717-24; discussion 24-8.
58. DeLancey JO. The anatomy of the pelvic floor. *Curr Opin Obstet Gynecol* 1994;6:313-6.
59. Pipitone F, Sadeghi Z, DeLancey JOL. Urethral function and failure: A review of current knowledge of urethral closure mechanisms, how they vary, and how they are affected by life events. *Neurourol Urodyn* 2021;40:1869-79.
60. Ashton-Miller JA, DeLancey JOL. Mechanisms of pelvic floor trauma during vaginal delivery. In: Santoro GA, Wieczorek AP, Sultan AH, eds. *Pelvic floor disorders, a multidisciplinary textbook*. 2 ed. Cham, Swiss Confederation: Springer Nature Switzerland AG 2021; 2021:189-208.
61. Clapp JF, 3rd, Seaward BL, Sleamaker RH, Hiser J. Maternal physiologic adaptations to early human pregnancy. *Am J Obstet Gynecol* 1988;159:1456-60.
62. Routzong MR, Rostaminia G, Moalli PA, Abramowitch SD. Pelvic floor shape variations during pregnancy and after vaginal delivery. *Comput Methods Programs Biomed* 2020;194:105516.
63. Sindhwani N, Bamberg C, Famaey N, et al. In vivo evidence of significant levator ani muscle stretch on MR images of a live childbirth. *Am J Obstet Gynecol* 2017;217:194.e1-.e8.
64. 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.
65. Griffin KM, O'Herlihy C, O'Connell PR, Jones JF. Combined ischemic and neuropathic insult to the anal canal in an animal model of obstetric-related trauma. *Dis Colon Rectum* 2012;55:32-41.
66. 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.

67. Tetzschner T, Sørensen M, Jønsson L, Lose G, Christiansen J. Delivery and pudendal nerve function. *Acta Obstet Gynecol Scand* 1997;76:324-31.
68. Dolan LM, Hosker GL, Mallett VT, Allen RE, Smith AR. Stress incontinence and pelvic floor neurophysiology 15 years after the first delivery. *BJOG* 2003;110:1107-14.
69. Miller JM, Low LK, Zielinski R, Smith AR, DeLancey JO, Brandon C. Evaluating maternal recovery from labor and delivery: bone and levator ani injuries. *Am J Obstet Gynecol* 2015;213:188.e1-.e11.
70. Laurberg S, Swash M. Effects of aging on the anorectal sphincters and their innervation. *Dis Colon Rectum* 1989;32:737-42.
71. Healy CF, O'Herlihy C, O'Brien C, O'Connell PR, Jones JF. Experimental models of neuropathic fecal incontinence: an animal model of childbirth injury to the pudendal nerve and external anal sphincter. *Dis Colon Rectum* 2008;51:1619-26; discussion 26.
72. Kaneko T, Nemoto T, Funahashi K, Koike J, Shibuya K, Kaneko H. Differences in innervated neurons of the internal anal sphincter based on age and sex: A histological study. *Geriatr Gerontol Int* 2018;18:495-500.
73. Rieger M, Duran P, Cook M, et al. Quantifying the Effects of Aging on Morphological and Cellular Properties of Human Female Pelvic Floor Muscles. *Ann Biomed Eng* 2021;49:1836-47.
74. Lewicky-Gaupp C, Leader-Cramer A, Johnson LL, Kenton K, Gossett DR. Wound complications after obstetric anal sphincter injuries. *Obstet Gynecol* 2015;125:1088-93.
75. Gommesen D, Nohr EA, Drue HC, Qvist N, Rasch V. Obstetric perineal tears: risk factors, wound infection and dehiscence: a prospective cohort study. *Arch Gynecol Obstet* 2019;300:67-77.
76. Buppasiri P, Lumbiganon P, Thinkhamrop J, Thinkhamrop B. Antibiotic prophylaxis for third- and fourth-degree perineal tear during vaginal birth. *Cochrane Database Syst Rev* 2014:Cd005125.
77. Okeahialam NA, Wong KW, Thakar R, Sultan AH. The incidence of wound complications following primary repair of obstetric anal sphincter injury: a systematic review and meta-analysis. *Am J Obstet Gynecol* 2022;227:182-91.
78. Gommesen D, Nøhr E, Qvist N, Rasch V. Obstetric perineal tears, sexual function and dyspareunia among primiparous women 12 months postpartum: a prospective cohort study. *BMJ Open* 2019;9:e032368.
79. Gommesen D, Nohr EA, Qvist N, Rasch V. Obstetric perineal ruptures-risk of anal incontinence among primiparous women 12 months postpartum: a prospective cohort study. *Am J Obstet Gynecol* 2020;222:165.e1-.e11.
80. Eason E, Labrecque M, Marcoux S, Mondor M. Anal incontinence after childbirth. *Cmaj* 2002;166:326-30.

81. Bharucha AE, Dunivan G, Goode PS, et al. Epidemiology, pathophysiology, and classification of fecal incontinence: state of the science summary for the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) workshop. *Am J Gastroenterol* 2015;110:127-36.
82. Al-Mukhtar Othman J, Åkervall S, Nilsson IEK, Molin M, Milsom I, Gyhagen M. Fecal incontinence in nonpregnant nulliparous women aged 25 to 64 years-a randomly selected national cohort prevalence study. *Am J Obstet Gynecol* 2022;226:706.e1-.e23.
83. Lewicky-Gaupp C, Hamilton Q, Ashton-Miller J, Huebner M, DeLancey JO, Fenner DE. Anal sphincter structure and function relationships in aging and fecal incontinence. *Am J Obstet Gynecol* 2009;200:559.e1-5.
84. Huebner M, Margulies RU, Fenner DE, Ashton-Miller JA, Bitar KN, DeLancey JO. Age effects on internal anal sphincter thickness and diameter in nulliparous females. *Dis Colon Rectum* 2007;50:1405-11.
85. Osterberg A, Graf W, Edebol Eeg-Olofsson K, Hynninen P, Pålman L. Results of neurophysiologic evaluation in fecal incontinence. *Dis Colon Rectum* 2000;43:1256-61.
86. Burnett LA, Cook M, Shah S, Michelle Wong M, Kado DM, Alperin M. Age-associated changes in the mechanical properties of human cadaveric pelvic floor muscles. *J Biomech* 2020;98:109436.
87. Åkervall S, Nordgren S, Fasth S, Oresland T, Pettersson K, Hultén L. The effects of age, gender, and parity on rectoanal functions in adults. *Scand J Gastroenterol* 1990;25:1247-56.
88. Bharucha AE, Zinsmeister AR, Schleck CD, Melton LJ, 3rd. Bowel disturbances are the most important risk factors for late onset fecal incontinence: a population-based case-control study in women. *Gastroenterology* 2010;139:1559-66.
89. Chan CL, Scott SM, Williams NS, Lunniss PJ. Rectal hypersensitivity worsens stool frequency, urgency, and lifestyle in patients with urge fecal incontinence. *Dis Colon Rectum* 2005;48:134-40.
90. Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors; an underdiagnosed problem in the Australian community? *Med J Aust* 2002;176:54-7.
91. Yu SW, Rao SS. Anorectal physiology and pathophysiology in the elderly. *Clin Geriatr Med* 2014;30:95-106.
92. Poylin V, Serrot FJ, Madoff RD, et al. Obesity and bariatric surgery: a systematic review of associations with defecatory dysfunction. *Colorectal Dis* 2011;13:e92-103.
93. Haylen BT, De Ridder D, Freeman RM, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. *Neurourol Urodyn* 2010;29:4-20.

94. Lukacz ES, Lawrence JM, Buckwalter JG, Burchette RJ, Nager CW, Luber KM. Epidemiology of prolapse and incontinence questionnaire: validation of a new epidemiologic survey. *Int Urogynecol J Pelvic Floor Dysfunct* 2005;16:272-84.
95. Dietz HP, Simpson JM. Levator trauma is associated with pelvic organ prolapse. *BJOG* 2008;115:979-84.
96. Blondel B, Alexander S, Bjarnadóttir RI, et al. Variations in rates of severe perineal tears and episiotomies in 20 European countries: a study based on routine national data in Euro-Peristat Project. *Acta Obstet Gynecol Scand* 2016;95:746-54.
97. Jangö H, Westergaard HB, Kjaerbye-Thygesen A, Langhoff-Roos J, Lauenborg J. Changing incidence of obstetric anal sphincter injuries- A result of formal prevention programs? *Acta Obstet Gynecol Scand* 2019;98:1455-63.
98. Laine K, Gissler M, Pirhonen J. Changing incidence of anal sphincter tears in four Nordic countries through the last decades. *Eur J Obstet Gynecol Reprod Biol* 2009;146:71-5.
99. Jha S, Parker V. Risk factors for recurrent obstetric anal sphincter injury (rOASI): a systematic review and meta-analysis. *Int Urogynecol J* 2016;27:849-57.
100. Andrews V, Sultan AH, Thakar R, Jones PW. Occult anal sphincter injuries - myth or reality? *BJOG* 2006;113:195-200.
101. Groom KM, Paterson-Brown S. Can we improve on the diagnosis of third degree tears? *Eur J Obstet Gynecol Reprod Biol* 2002;101:19-21.
102. Meyer I, Richter HE. Impact of fecal incontinence and its treatment on quality of life in women. *Womens Health (Lond)* 2015;11:225-38.
103. Milsom I, Ekelund P, Molander U, Arvidsson L, Areskoug B. The influence of age, parity, oral contraception, hysterectomy and menopause on the prevalence of urinary incontinence in women. *J Urol* 1993;149:1459-62.
104. Malmsten UG, Milsom I, Molander U, Norlén LJ. Urinary incontinence and lower urinary tract symptoms: an epidemiological study of men aged 45 to 99 years. *J Urol* 1997;158:1733-7.
105. Milsom I, Gyhagen M. Overview: Epidemiology and etiology of urinary incontinence and voiding dysfunction. In: Santoro GA, Wieczorek AP, Sultan AH, eds. *Pelvic floor disorders, a multidisciplinary textbook*. 2 ed. Cham, Swiss Confederation: Springer Nature Switzerland AG 2021; 2021:239-48.
106. Thom DH, Rortveit G. Prevalence of postpartum urinary incontinence: a systematic review. *Acta Obstet Gynecol Scand* 2010;89:1511-22.
107. Gyhagen M, Åkervall S, Molin M, Milsom I. The effect of childbirth on urinary incontinence: a matched cohort study in women aged 40-64 years. *Am J Obstet Gynecol* 2019;221:322.e1-e17.
108. Leijonhufvud A, Lundholm C, Cnattingius S, Granath F, Andolf E, Altman D. Risks of stress urinary incontinence and pelvic organ

- prolapse surgery in relation to mode of childbirth. *Am J Obstet Gynecol* 2011;204:70.e1-7.
109. Al-Mukhtar Othman J, Åkervall S, Milsom I, Gyhagen M. Urinary incontinence in nulliparous women aged 25-64 years: a national survey. *Am J Obstet Gynecol* 2017;216:149.e1-.e11.
 110. Swenson CW, Kolenic GE, Trowbridge ER, et al. Obesity and stress urinary incontinence in women: compromised continence mechanism or excess bladder pressure during cough? *Int Urogynecol J* 2017;28:1377-85.
 111. Salvatore S, De Bastiani S, Del Deo F. Epidemiology and etiology of pelvic organ prolapse. In: Santoro GA, Wiczorek AP, Sultan AH, eds. *Pelvic floor disorders, a multidisciplinary textbook*. 2 ed. Cham, Swiss Confederation: Springer Nature Switzerland AG 2021; 2021:547-54.
 112. Larsudd-Kåverud J, Gyhagen J, Åkervall S, et al. The influence of pregnancy, parity, and mode of delivery on urinary incontinence and prolapse surgery-a national register study. *Am J Obstet Gynecol* 2022:Online ahead of print.
 113. Rydberg H. Kvalitetsdeklaration: Statistik om graviditeter, förlossningar och nyfödda barn 2020. Socialstyrelsen, Avdelningen för register och statistik. https://www.scb.se/contentassets/5d1632afd0c64f08bb75e70f23bd9beb/hs0107_kd_2020_hs_20211201.pdf. Published 2021. Updated 2021, December 1. Accessed October 28, 2022.
 114. The Swedish National Medical Birth Register. <https://www.socialstyrelsen.se/en/statistics-and-data/registers/national-medical-birth-register/>. Accessed October 28, 2022.
 115. Källén B, Källén K, Edlund M, Otterblad Olausson P. Utvärdering av det svenska Medicinska födelserregistret. 2002. https://www.socialstyrelsen.se/globalassets/sharepoint-dokument/artikelkatalog/statistik/2002-112-4_20021124.pdf. Accessed 24th August 2022.
 116. TPR. Registret över totalbefolkningen. Statistics Sweden. <https://www.scb.se/vara-tjanster/bestall-data-och-statistik/bestalla-mikrodata/vilka-mikrodata-finns/individregister/registret-over-totalbefolkningen-rtb/>. Accessed 24th August, 2022.
 117. Ludvigsson JF, Otterblad-Olausson P, Pettersson BU, Ekbom A. The Swedish personal identity number: possibilities and pitfalls in healthcare and medical research. *Eur J Epidemiol* 2009;24:659-67.
 118. Inkomst- och taxeringsregistret. Statistics Sweden. <https://www.scb.se/vara-tjanster/bestall-data-och-statistik/bestalla-mikrodata/vilka-mikrodata-finns/individregister/inkomst--och-taxeringsregistret-iot/>. Published 2020. Accessed September 5th, 2022.

119. Mikrodata för Registret över befolkningens utbildning. Statistics Sweden. <https://www.scb.se/vara-tjanster/bestall-data-och-statistik/bestalla-mikrodata/vilka-mikrodata-finns/individregister/registret-over-befolkningens-utbildning/>. Published 2021. Accessed September 5th, 2022.
120. Irgens LM. The Medical Birth Registry of Norway. Epidemiological research and surveillance throughout 30 years. *Acta Obstet Gynecol Scand* 2000;79:435-9.
121. *Data Quality Study of the 2015–2016 Discharge Abstract Database: A Focus on Hospital Harm*. Ottawa, ON: Canadian Institute for Health Information. ;2016.
122. Muin DA, Helmer H, Leitner H, Neururer S. Epidemiology of Antepartum Stillbirths in Austria - A Population-Based Study between 2008 and 2020. *J Clin Med* 2021;10.
123. Joseph KS, Fahey J. Validation of perinatal data in the Discharge Abstract Database of the Canadian Institute for Health Information. *Chronic Dis Can* 2009;29:96-100.
124. Langhoff-Roos J, Krebs L, Klungsøyr K, et al. The Nordic medical birth registers - a potential goldmine for clinical research. *Acta Obstet Gynecol Scand* 2014;93:132-7.
125. Lehmann S, Baghestan E, Børdahl P, Ebbing M, Irgens L, Rasmussen S. Validation of data in the Medical Birth Registry of Norway on delivery after a previous cesarean section. *Acta Obstet Gynecol Scand* 2017;96:892-7.
126. The Swedish state personal address register. <https://www.statenspersonadressregister.se/master/start/english-summary/>. Accessed October 2022, 2022.
127. Gyhagen M. *Long-term consequences of vaginal delivery on the pelvic floor: a comparison with caesarean section in one-parae women* [Compilation thesis]. Gothenburg, Sweden: Department of Obstetrics & Gynecology, Institute of Clinical Sciences, Sahlgrenska Academy, University of Gothenburg; 2013.
128. Age of mothers at childbirth and age-specific fertility. OECD Family Database. https://www.oecd.org/els/soc/SF_2_3_Age_mothers_childbirth.pdf. Published 2022. Updated June 2022. Accessed October 28, 2022.
129. Sultan AH, Monga A, Lee J, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female anorectal dysfunction. *Int Urogynecol J* 2017;28:5-31.
130. Tegerstedt G, Miedel A, Maehle-Schmidt M, Nyren O, Hammarström M. A short-form questionnaire identified genital organ prolapse. *J Clin Epidemiol* 2005;58:41-6.
131. Jorge JM, Wexner SD. Etiology and management of fecal incontinence. *Dis Colon Rectum* 1993;36:77-97.

132. Sandvik H, Hunskaar S, Seim A, Hermstad R, Vanvik A, Bratt H. Validation of a severity index in female urinary incontinence and its implementation in an epidemiological survey. *J Epidemiol Community Health* 1993;47:497-9.
133. Sandvik H, Seim A, Vanvik A, Hunskaar S. A severity index for epidemiological surveys of female urinary incontinence: comparison with 48-hour pad-weighing tests. *Neurourol Urodyn* 2000;19:137-45.
134. Rothman KJ, Greenland S, Lash TL. Design strategies to improve study accuracy. In: Rothman K, Greenland S, Lash T, eds. *Modern Epidemiology*. 3 ed. Philadelphia: Lippincott Williams & Wilkins; 2008:168-82.
135. Hartge P, Cahill J. Field methods in epidemiology. In: Rothman K, Greenland S, Lash T, eds. *Modern Epidemiology*. 3 ed. Philadelphia: Lippincott Williams & Wilkins; 2008:492-510.
136. Rothman KJ, Greenland S, Lash TL. Validity in epidemiologic studies. In: Rothman KJ, Greenland S, Lash TL, eds. *Modern epidemiology*. 3 ed. Philadelphia, PA, USA: Wolters Kluwer, Lippincott Williams & Wilkins; 2008.
137. Maher C, Baessler K, Barber M, et al. Pelvic Organ Prolapse Surgery. In: Abrams P, Cardozo L, Wagg A, Weil A, eds. *Incontinence*. 6 ed. Bristol, UK: ICS-ICI. International Continence Society; 2017:1858-992.
138. Thom DH, van den Eeden SK, Ragins AI, et al. Differences in prevalence of urinary incontinence by race/ethnicity. *J Urol* 2006;175:259-64.
139. Elston DM. Participation bias, self-selection bias, and response bias. *J Am Acad Dermatol* 2021.
140. Mackenbach JP, Stirbu I, Roskam AJ, et al. Socioeconomic inequalities in health in 22 European countries. *N Engl J Med* 2008;358:2468-81.
141. Kivimäki M, Batty GD, Pentti J, et al. Association between socioeconomic status and the development of mental and physical health conditions in adulthood: a multi-cohort study. *Lancet Public Health* 2020;5:e140-e9.
142. Rothman KJ, Greenland S, Lash TL. Measures of Effect and Measures of Association. In: Rothman KJ, Greenland S, Lash TL, eds. *Modern epidemiology*. 3 ed. Philadelphia, PA, USA: Wolters Kluwer, Lippincott Williams & Wilkins; 2008:51-70.
143. Rothman K, Greenland S, Lash T. Types of epidemiologic studies. In: Rothman K, Greenland S, Lash T, eds. *Modern epidemiology*. 3 ed. Philadelphia: Lippincott Williams & Wilkins; 2008:87-99.
144. Evers EC, Blomquist JL, McDermott KC, Handa VL. Obstetrical anal sphincter laceration and anal incontinence 5-10 years after childbirth. *Am J Obstet Gynecol* 2012;207:425.e1-.e4256.

145. Bonita R, Beaglehole R, Kjellström T. Undersökningstyper. In: Bonita R, Beaglehole R, Kjellström T, eds. *Grundläggande epidemiologi*. 2 ed. Lund: Studentlitteratur; 2010:67-98.
146. Bonita R, Beaglehole R, Kjellström T. Kausalitet inom epidemiologin. In: Bonita R, Beaglehole R, Kjellström T, eds. *Grundläggande epidemiologi*. 2 ed. Lund: Studentlitteratur; 2010:125-46.
147. Edqvist M, Dahlen HG, Häggsgård C, et al. The effect of two midwives during the second stage of labour to reduce severe perineal trauma (Oneplus): a multicentre, randomised controlled trial in Sweden. *Lancet* 2022;399:1242-53.
148. Gyhagen M. Development and validation of a logistic regression algorithm to predict the risk of obstetric anal sphincter injury. <https://clinicaltrials.gov/ct2/show/NCT05218837>. Published 2022. Accessed December 2, 2022.
149. Sultan AH, de Leeuw JW. Episiotomy and operative vaginal delivery: do we need more evidence? *BJOG* 2021;128:1672-3.
150. Nilsson IEK, Åkervall S, Molin M, Milsom I, Gyhagen M. Symptoms of fecal incontinence two decades after no, one, or two obstetrical anal sphincter injuries. *Am J Obstet Gynecol* 2021;224:276.e1-.e23.
151. Malterud K. Vad är kvalitativa metoder? In: Malterud K, ed. Larson P, trans. *Kvalitativa metoder i medicinsk forskning: en introduktion*. 3 ed. Lund: Studentlitteratur; 2014:31-43.
152. Stroup DF, Berlin JA, Morton SC, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. *Jama* 2000;283:2008-12.
153. Gyhagen M, Ellström Engh M, Husslein H, et al. Temporal trends in obstetric anal sphincter injury from the first vaginal delivery in Austria, Canada, Norway, and Sweden. *Acta Obstet Gynecol Scand* 2021.
154. Perslev K, Mørch EJ, Jangö H. Increased risk of obstetric anal sphincter injury in women undergoing vaginal delivery after caesarean section: A systematic review and meta-analysis. *BJOG* 2022.
155. Rieger MM, Wong M, Burnett LA, Sesillo FB, Baynes BB, Alperin M. Mechanisms governing protective pregnancy-induced adaptations of the pelvic floor muscles in the rat preclinical model. *Am J Obstet Gynecol* 2022;226:708.e1-.e13.
156. Gill BC, Moore C, Damaser MS. Postpartum stress urinary incontinence: lessons from animal models. *Expert Rev Obstet Gynecol* 2010;5:567-80.
157. Handa VL, Blomquist JL, Knoepp LR, Hoskey KA, McDermott KC, Muñoz A. Pelvic floor disorders 5-10 years after vaginal or cesarean childbirth. *Obstet Gynecol* 2011;118:777-84.
158. Dolan LM, Hilton P. Obstetric risk factors and pelvic floor dysfunction 20 years after first delivery. *Int Urogynecol J* 2010;21:535-44.

159. Laugesen K, Ludvigsson JF, Schmidt M, et al. Nordic Health Registry-Based Research: A Review of Health Care Systems and Key Registries. *Clin Epidemiol* 2021;13:533-54.
160. Grodstein F, Fretts R, Lifford K, Resnick N, Curhan G. Association of age, race, and obstetric history with urinary symptoms among women in the Nurses' Health Study. *Am J Obstet Gynecol* 2003;189:428-34.
161. Diokno AC, Brown MB, Brock BM, Herzog AR, Normolle DP. Clinical and cystometric characteristics of continent and incontinent noninstitutionalized elderly. *J Urol* 1988;140:567-71.
162. Herzog AR, Fultz NH. Prevalence and incidence of urinary incontinence in community-dwelling populations. *J Am Geriatr Soc* 1990;38:273-81.
163. Mimura T. Patient-Reported Outcome Assessment in Anal Incontinence. In: Santoro GA, Wieczorek AP, Sultan AH, eds. *Pelvic Floor Disorders*. Second ed. Switzerland: Springer, Cham; 2021:399-409.
164. Vaizey CJ, Carapeti E, Cahill JA, Kamm MA. Prospective comparison of faecal incontinence grading systems. *Gut* 1999;44:77-80.
165. Sansoni J, Hawthorne G, Fleming G, Marosszeky N. The revised faecal incontinence scale: a clinical validation of a new, short measure for assessment and outcomes evaluation. *Dis Colon Rectum* 2013;56:652-9.
166. Rothbarth J, Bemelman WA, Meijerink WJ, et al. What is the impact of fecal incontinence on quality of life? *Dis Colon Rectum* 2001;44:67-71.
167. Bols EM, Hendriks HJ, Berghmans LC, Baeten CG, de Bie RA. Responsiveness and interpretability of incontinence severity scores and FIQL in patients with fecal incontinence: a secondary analysis from a randomized controlled trial. *Int Urogynecol J* 2013;24:469-78.
168. Jangö H, Langhoff-Roos J, Rosthøj S, Sakse A. Wexner score and quality of life in women with obstetric anal sphincter injury. *Int Urogynecol J* 2020;31:1115-21.
169. Devesa JM, Vicente R, Abaira V. Visual analogue scales for grading faecal incontinence and quality of life: their relationship with the Jorge-Wexner score and Rockwood scale. *Tech Coloproctol* 2013;17:67-71.
170. 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;46:473-80.
171. Legendre G, Fritel X, Panjo H, Zins M, Ringa V. Incidence and remission of stress, urge, and mixed urinary incontinence in midlife and older women: A longitudinal cohort study. *Neurourol Urodyn* 2020;39:650-7.
172. Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. A community-based epidemiological survey of female urinary incontinence: the

- Norwegian EPINCONT study. Epidemiology of Incontinence in the County of Nord-Trøndelag. *J Clin Epidemiol* 2000;53:1150-7.
173. Zhang D, Wang S, Gao L, et al. Analysis of Characteristics and Quality of Life of Elderly Women with Mild to Moderate Urinary Incontinence in Community Dwellings. *Int J Environ Res Public Health* 2022;19.
174. Barber MD, Neubauer NL, Klein-Olarte V. Can we screen for pelvic organ prolapse without a physical examination in epidemiologic studies? *Am J Obstet Gynecol* 2006;195:942-8.
175. Tegerstedt G, Maehle-Schmidt M, Nyrén O, Hammarström M. Prevalence of symptomatic pelvic organ prolapse in a Swedish population. *Int Urogynecol J Pelvic Floor Dysfunct* 2005;16:497-503.
176. Gyhagen M, Al-Mukhtar Othman J, Åkervall S, Nilsson I, Milsom I. The symptom of vaginal bulging in nulliparous women aged 25-64 years: a national cohort study. *Int Urogynecol J* 2019;30:639-47.
177. Ransohoff DF, Feinstein AR. Problems of spectrum and bias in evaluating the efficacy of diagnostic tests. *N Engl J Med* 1978;299:926-30.
178. Robinson JN, Norwitz ER, Cohen AP, McElrath TF, Lieberman ES. Episiotomy, operative vaginal delivery, and significant perinatal trauma in nulliparous women. *Am J Obstet Gynecol* 1999;181:1180-4.
179. Ekéus C, Nilsson E, Gottvall K. Increasing incidence of anal sphincter tears among primiparas in Sweden: a population-based register study. *Acta Obstet Gynecol Scand* 2008;87:564-73.
180. Lundqvist E. Medicinska födelseregistret 1973–2013, Assisterad befruktning 1991–2012. The National Board of Health and Welfare. Graviditeter, förlossningar och nyfödda barn. Web site. <https://www.socialstyrelsen.se/globalassets/sharepoint-dokument/artikelkatalog/statistik/2014-12-19.pdf>. Published 2014. Accessed November 3, 2022.
181. Sultan A, Thakar R. Third and fourth degree tears. In: Sultan A, Thakar R, Fenner D, eds. *Perineal and anal sphincter trauma*. 2nd ed. London, England: Springer-Verlag Ltd.; 2009:33-51.
182. Handa VL, Blomquist JL, McDermott KC, Friedman S, Muñoz A. Pelvic floor disorders after vaginal birth: effect of episiotomy, perineal laceration, and operative birth. *Obstet Gynecol* 2012;119:233-9.
183. Volløyhaug I, Mørkved S, Salvesen Ø, Salvesen K. Pelvic organ prolapse and incontinence 15-23 years after first delivery: a cross-sectional study. *BJOG* 2015;122:964-71.
184. Pettersson K, Ajne J, Yousaf K, Sturm D, Westgren M, Ajne G. Traction force during vacuum extraction: a prospective observational study. *BJOG* 2015;122:1809-16.
185. O'Mahony F, Hofmeyr GJ, Menon V. Choice of instruments for assisted vaginal delivery. *Cochrane Database Syst Rev* 2010:Cd005455.

186. Soerensen MM, Buntzen S, Bek KM, Laurberg S. Complete obstetric anal sphincter tear and risk of long-term fecal incontinence: a cohort study. *Dis Colon Rectum* 2013;56:992-1001.
187. Nilsson I, Åkervall S, Milsom I, Gyhagen M. Long-term effects of vacuum extraction on pelvic floor function: a cohort study in primipara. *Int Urogynecol J* 2016;27:1051-6.
188. Persson LKG, Sakse A, Langhoff-Roos J, Jangö H. Anal incontinence after two vaginal deliveries without obstetric anal sphincter rupture. *Arch Gynecol Obstet* 2017;295:1399-406.
189. Jangö H, Langhoff-Roos J, Rosthøj S, Sakse A. Mode of delivery after obstetric anal sphincter injury and the risk of long-term anal incontinence. *Am J Obstet Gynecol* 2016;214:733.e1-.e13.
190. De Leeuw JW, Vierhout ME, Struijk PC, Hop WC, Wallenburg HC. Anal sphincter damage after vaginal delivery: functional outcome and risk factors for fecal incontinence. *Acta Obstet Gynecol Scand* 2001;80:830-4.
191. Mous M, Muller SA, De Leeuw JW. Long-term effects of anal sphincter rupture during vaginal delivery: faecal incontinence and sexual complaints. *BJOG* 2008;115:234-8.
192. Laine K, Gissler M, Pirhonen J. Changing incidence of anal sphincter tears in four Nordic countries through the last decades. *Eur J Obstet Gynecol Reprod Biol* 2009;146:71-5.
193. Laine K, Rotvold W, Staff AC. Are obstetric anal sphincter ruptures preventable? - Large and consistent rupture rate variations between the Nordic countries and between delivery units in Norway. *Acta Obstet Gynecol Scand* 2013;92:94-100.
194. Muraca GM, Lisonkova S, Skoll A, et al. Ecological association between operative vaginal delivery and obstetric and birth trauma. *Cmaj* 2018;190:E734-e41.
195. Pergialiotis V, Vlachos D, Protopapas A, Pappa K, Vlachos G. Risk factors for severe perineal lacerations during childbirth. *Int J Gynaecol Obstet* 2014;125:6-14.
196. Bergendahl S, Lindberg P, Brismar Wendel S. Operator experience affects the risk of obstetric anal sphincter injury in vacuum extraction deliveries. *Acta Obstet Gynecol Scand* 2019;98:787-94.
197. Gupta J, Smith G, Chodankar R. Birth after Previous Caesarean Birth. Green-top Guideline No. 45. Royal College of Obstetricians and Gynaecologists. <https://www.rcog.org.uk/guidance/browse-all-guidance/green-top-guidelines/birth-after-previous-caesarean-birth-green-top-guideline-no-45/>. Published 2015. Accessed November 5, 2022.
198. Practice Bulletin No. 205 Summary: Vaginal Birth After Cesarean Delivery. The American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2019;133:393-5.

199. D'Souza JC, Monga A, Tincello DG. Risk factors for obstetric anal sphincter injuries at vaginal birth after caesarean: a retrospective cohort study. *Int Urogynecol J* 2019;30:1747-53.
200. Sultan AH. Editorial: Obstetrical Perineal Injury and Anal Incontinence. *Clinical Risk* 1999;5:193-6.
201. Fernando RJ, Sultan AH, Radley S, Jones PW, Johanson RB. Management of obstetric anal sphincter injury: a systematic review & national practice survey. *BMC Health Serv Res* 2002;2:9.
202. Dietz HP. Exoanal Imaging of the Anal Sphincters. *J Ultrasound Med* 2018;37:263-80.
203. Laine K, Pirhonen T, Rolland R, Pirhonen J. Decreasing the incidence of anal sphincter tears during delivery. *Obstet Gynecol* 2008;111:1053-7.
204. Hals E, Øian P, Pirhonen T, et al. A multicenter interventional program to reduce the incidence of anal sphincter tears. *Obstet Gynecol* 2010;116:901-8.
205. Frost J, Gundry R, Young H, Naguib A. Multidisciplinary training in perineal care during labor and delivery for the reduction of anal sphincter injuries. *Int J Gynaecol Obstet* 2016;134:177-80.
206. Naidu M, Sultan AH, Thakar R. Reducing obstetric anal sphincter injuries using perineal support: our preliminary experience. *Int Urogynecol J* 2017;28:381-9.
207. De Meutter L, A DvH, van der Woerd-Elting I, de Leeuw JW. Implementation of a perineal support programme for reduction of the incidence of obstetric anal sphincter injuries and the effect of non-compliance. *Eur J Obstet Gynecol Reprod Biol* 2018;230:119-23.
208. Samarasekera DN, Bekhit MT, Wright Y, et al. Long-term anal continence and quality of life following postpartum anal sphincter injury. *Colorectal Dis* 2008;10:793-9.
209. Palm A, Israelsson L, Bolin M, Danielsson I. Symptoms after obstetric sphincter injuries have little effect on quality of life. *Acta Obstet Gynecol Scand* 2013;92:109-15.

APPENDIX

Appendix 1: Questionnaire of SWEPOP-1

UNDERSÖKNING AV KVINNORS BÄCKENBOTTENBESVÄR EFTER BARNAFÖDANDE

Detta formulär innehåller frågor om symtom och besvär från bäckenbotten och hur det i så fall påverkar Dig i ditt dagliga liv. Sätt ett kryss i den ruta som Du anser stämmer bäst in på Dig. Även om det inte exakt beskriver hur Du upplever besvären, kryssa ändå i den ruta som känns mest riktig för Dig. Vi är tacksamma om Du fyller i frågorna även om Du inte har några besvär.

- | | | | |
|----|-----------------------------|----|-----|
| 1. | Hur lång är du? | | |
| 2. | Hur mycket väger du? | | |
| 3. | Hur många barn har du fött? | | |
| 4. | Menstruerar du fortfarande? | JA | NEJ |
| 5. | Om du inte menstruerar- | | |
| | Är du gravid? | JA | NEJ |
| | Är livmodern bortopererad? | JA | NEJ |
| | Använder du hormonspiral? | JA | NEJ |
| | Är du i klimakteriet? | JA | NEJ |
| | Använder du östrogen? | JA | NEJ |

BESVÄR IFRÅN URINVÄGARNA

- | | | | |
|----|---------------------------------|----|-----|
| 6. | Har du ofrivilligt urinläckage? | JA | NEJ |
|----|---------------------------------|----|-----|

Om du inte har besvär med urinläckage gå vidare till fråga 15.

7. Hur ofta har du urinläckage?

Mer sällan än en gång i månaden

En gång i månaden eller mer

En gång i veckan eller mer

Varje dag och/eller natt

8. Hur mycket urin läcker Du vid varje läckaetillfälle?

Några droppar eller lite

Små mängder

Stora mängder

9. Har du urinläckage när Du hostar, nyser, skrattar eller lyfter tungt?

JA NEJ

10. Har Du urinläckage i samband med plötsligt påkomna och starka urinträngningar?

JA NEJ

11. Hur länge har Du haft urinläckage?

0-5 år

5-10 år

Mer än 10 år

12. Har Du sökt läkare på grund av ditt urinläckage?

JA NEJ

13. Hur påverkas Du av ditt urinläckage?

Inget problem

Lite besvär

En del besvär

Mycket besvär

Mycket stort problem

14. Har urinläckage påverkat din(a):

- förmåga att utföra hushållsarbete?

Inte alls
Lite
Måttligt
Mycket

- fysiska aktiviteter som promenader, simning osv?

Inte alls
Lite
Måttligt
Mycket

- nöjen som att gå på bio, konsert o dyl?

Inte alls
Lite
Måttligt
Mycket

- förmåga att åka bil eller buss mer än 30 min hemifrån?

Inte alls
Lite
Måttligt
Mycket

- medverka vid sociala evenemang utanför hemmet?

Inte alls
Lite
Måttligt
Mycket

- mentala hälsa (nervositet, depression osv)

Inte alls

Lite

Måttligt

Mycket

- känsla av frustration?

Inte alls

Lite

Måttligt

Mycket

15. Har du fått någon behandling för
urinläckage?

JA NEJ

16. Har din mor besvärats av urinläckage?

JA NEJ

BESVÄR IFRÅN SLIDAN

17. Har Du en känsla av att något buktar fram ur slidan?

Ja ofta

Ibland

Någon gång

Nej aldrig

18. Händer det att Du har skavningsbesvär i underlivet?

Ja ofta

Ibland

Någon gång

Nej aldrig

19. Händer det att Du behöver lyfta fram främre slidväggen
för att kunna kissa?

Ja ofta
Ibland
Någon gång
Nej aldrig

Besvara endast fråga 20-21 om Du har besvär från slidan, om inte, gå till fråga 22

20. Om Du anstränger dig med tunga lyft blir dina besvär:

Oförändrade
Bättre
Sämre

21. Har framfall påverkat din(a):

- förmåga att utföra hushållsarbete?

Inte alls
Lite
Måttligt
Mycket

- fysiska aktiviteter som promenader, simning osv?

Inte alls
Lite
Måttligt
Mycket

- nöjen som att gå på bio, konsert o dyl?

Inte alls
Lite
Måttligt
Mycket

- förmåga att åka bil eller buss mer än 30 min hemifrån?

Inte alls

Lite

Måttligt

Mycket

- medverka vid sociala evenemang utanför hemmet?

Inte alls

Lite

Måttligt

Mycket

- mentala hälsa (nervositet, depression osv)

Inte alls

Lite

Måttligt

Mycket

- känsla av frustration?

Inte alls

Lite

Måttligt

Mycket

22. Har du fått någon behandling för framfall?

JA

NEJ

23. Har din mor besvärats av framfall?

JA

NEJ

BESVÄR IFRÅN ÄNDTARMEN

24. Läcker Du fast avföring ofrivilligt?

Aldrig

Mer sällan än 1 ggr i månaden

Flera ggr i månaden, men inte varje vecka

Alltid, varje dag

25. Läcker Du lös avföring ofrivilligt?

Aldrig

Mer sällan än 1 ggr i månaden

Flera ggr i månaden, men inte varje vecka

Alltid, varje dag

26. Läcker Du gas ofrivilligt?

Aldrig

Mer sällan än 1 ggr i månaden

Flera ggr i månaden, men inte varje vecka

Alltid, varje dag

Om Du inte läcker gas eller avföring gå vidare till fråga 30

27. Använder Du skydd pga ofrivilligt läckage ifrån tarmen?

Aldrig

Mer sällan än 1 ggr i månaden

Flera ggr i månaden, men inte varje vecka

Alltid, varje dag

28. Påverkas din dagliga livsföring pga ofrivilligt läckage från tarmen?

Aldrig

Mer sällan än 1 ggr i månaden

Flera ggr i månaden, men inte varje vecka

Alltid, varje dag

29. Har gas eller avföringsläckage påverkat din(a):

- förmåga att utföra hushållsarbete?

Inte alls

Lite

Måttligt

Mycket

- fysiska aktiviteter som promenader, simning osv?

Inte alls

Lite

Måttligt

Mycket

- nöjen som att gå på bio, konsert o dyl?

Inte alls

Lite

Måttligt

Mycket

- förmåga att åka bil eller buss mer än 30 min hemifrån?

Inte alls

Lite

Måttligt

Mycket

- medverkan vid sociala evenemang utanför hemmet?

Inte alls

Lite

Måttligt

Mycket

- mentala hälsa (nervositet, depression osv)

Inte alls

Lite

Måttligt

Mycket

- känsla av frustration?

Inte alls

Lite

Måttligt

Mycket

30. Har du fått någon behandling för gas
eller avföringsläckage?

JA

NEJ

31. Har din mor besvärats av gas eller
avföringsläckage?

JA

NEJ

Nedan finns det utrymme för Dina egna funderingar beträffande detta
formulär:

Appendix 2: Questionnaire of SWEPOP-2

Formuläret innehåller frågor om symtom och besvär från bäckenbotten och hur det i så fall påverkar dig. Sätt kryss i rutan för det som stämmer bäst in på dig. Även om det inte exakt beskriver dina besvär, kryssa ändå i den ruta som känns mest rätt. Vi är tacksamma om du besvarar frågorna även om du inte har några besvär.

1. Hur lång är du? _____ cm

2. Hur mycket väger du? _____ kg

3. Har du fött barn?

Ja → Hur många barn har du fött ?
_____ barn

Nej

4. Menstruerar du fortfarande?

Ja → Gå till fråga 6

Nej

5. Om du inte menstruerar, vad beror det på?

Du kan markera mer än ett alternativ!

Graviditet

Livmodern är bortopererad

Använder hormonspiral

Är i klimakteriet

Använder östrogen

Annan anledning

BESVÄR FRÅN URINVÄGARNA

6. Hur många gånger kissar du under dagen på ett ungefär?

__ __ gånger

7. Behöver du kissa på natten?

Ja → Ungefär hur många gånger går du upp och
kissar?

__ __ gånger

Nej

8. Har du urinträngningar, dvs trängningar som kommer hastigt på och som känns svåra att motstå?

Ja

Nej → Gå till fråga 10

9. Hur påverkas du av dina urinträngningar?

Inget besvär

Lite besvär

En del besvär

Mycket besvär

Mycket stora besvär

10. Tar du någon medicin mot urinträngningar?

Ja

Nej

11. Var du sängvätare som barn, dvs hade ofrivillig tömning av urinblåsan i sömnen?

Ja →

Ungefär vid vilken ålder slutade det?

Vid __ års ålder

Nej

12. Har du urinläckage?

Ja

Nej →

Gå till fråga 21

13. Hur ofta har du urinläckage?

Mindre än en gång i månaden

En gång i månaden eller mer

En gång i veckan eller mer

Varje dag och/eller natt

14. Hur mycket urin läcker du vid varje läckaetillfälle?

Några droppar

Små mängder

Stora mängder

15. Har du urinläckage när du hostar, nyser, skrattar eller lyfter tungt?

Ja

Nej

16. Har du urinläckage i samband med plötsligt påkomna och starka urinträngningar?

Ja

Nej

17. Ungefär hur länge har du haft urinläckage?

0 – 5 år

5 – 10 år

Mer än 10 år

18. Har du sökt läkare på grund av ditt urinläckage?

Ja

Nej

19. Hur påverkas du av ditt urinläckage?

Inget besvär

Lite besvär

En del besvär

Mycket besvär

Mycket stora besvär

20. Om du fött barn – hade du urinläckage redan före första graviditeten?

Ja

Nej

21. Har du opererats för urinläckage?

Ja

Nej

22. Tar du någon medicin mot urinläckage?

Ja

Nej

23. Har din mor besvärats av urinläckage?

Ja

Nej

Vet inte

BESVÄR FRÅN SLIDAN

24. Har du en känsla av att något buktar fram ur slidan?

Ofta

Ibland

Någon gång

Aldrig

25. Händer det att du har skavningsbesvär i underlivet?

Ofta

Ibland

Någon gång

Aldrig

26. Händer det att du behöver lyfta fram främre slidväggen för att kunna kissa?

Ofta

Ibland

Någon gång

Aldrig

Gå direkt till fråga 29 om du inte har besvär från slidan.

27. Om du anstränger dig med tunga lyft blir dina besvär:

Oförändrade

Bättre

Sämre

28. Hur påverkas du av dina besvär från slidan?

Inget besvär

Lite besvär

En del besvär

Mycket besvär

Mycket stora besvär

29. Har du fått någon behandling för framfall?

Ja

Nej

30. Har du opererats för framfall?

Ja

Nej

31. Har din mor besvärats av framfall?

Ja

Nej

Vet inte

BESVÄR FRÅN ÄNDTARMEN

32. Läcker du fast avföring?

Aldrig

Mindre än en gång i månaden

Flera gånger i månaden, men inte varje vecka

Flera gånger i veckan, men inte varje dag

Alltid, varje dag

33. Läcker du lös avföring?

Aldrig

Mindre än en gång i månaden

Flera gånger i månaden, men inte varje vecka

Flera gånger i veckan, men inte varje dag

Alltid, varje dag

34. Läcker du gas ofrivilligt?

Aldrig

Mindre än en gång i månaden

Flera gånger i månaden, men inte varje vecka

Flera gånger i veckan, men inte varje dag

Alltid, varje dag

Gå direkt till fråga 38 om du inte läcker gas (ofrivilligt) eller avföring.

35. Använder du skydd på grund av läckage från tarmen?

Aldrig

Mindre än en gång i månaden

Flera gånger i månaden, men inte varje vecka

Flera gånger i veckan, men inte varje dag

Alltid, varje dag

36. Hur ofta påverkas ditt dagliga liv av läckage från tarmen?

Aldrig

Mindre än en gång i månaden

Flera gånger i månaden, men inte varje vecka

Flera gånger i veckan, men inte varje dag

Alltid, varje dag

37. Hur påverkas du av dina besvär från tarmen?

Inget besvär

Lite besvär

En del besvär

Mycket besvär

Mycket stora besvär

38. Har du fått någon behandling för gas eller avföringsläckage?

Ja

Nej

39. Har din mor besvärats av gas eller avföringsläckage?

Ja

Nej

Vet inte

40. Har du några synpunkter på frågeformuläret, ser vi gärna att du skriver det här nedan: