Committee 1

Epidemiology of Urinary (UI) and Faecal (FI) Incontinence and Pelvic Organ Prolapse (POP)

Chairman

I. Milsom (Sweden)

Members

D. Altman (Sweden),

M.C. Lapitan (The Philippines),

R. Nelson (U.K),

U. Sillén (Sweden),

D. Thom (USA)

CONTENTS

LIST OF ABBREVIATIONS

ADHD = Attention Deficit Hyperactivity Disorders
ADL = Activity of Daily Living
AI = Anal Incontinence
BMI = Body Mass Index
CAT = Childrens Apperception Test
CBCL = Childs Behaviour Check List
CI = Confidence Interval
DV = Dysfunctional Voiding
EEG = Electroencephalogram
FI = Faecal Incontinence
IBD = Irritable Bowel Disorder
ICCS = The International Childrens Continence Society
ICI = International Consultation on Incontinence
ICS = International Continence Society
LE = Level of Evidence
LUTS = Lower Urinary Tract Symptoms
MDS = Mininimum Data Set
MMSE = Mini Mental Status Examination
MNE = Monosymptomatic Nocturnal Enuresis
MRI = Magnetic Resonance Imaging
NE = Nocturnal Enuresis
NME = Non-Monosymptomatic Nocturnal Enuresis
OAB = Overactive Bladder
OR = Odds Ratio
POP = Pelvic Organ Prolapse
POPQ = The ICS Pelvic Organ Prolapse Quantification Examination
PSA = Prostate Specific Antigen
RR = Relative Risk
SUI = Stress Urinary Incontinence
TAH = Transabdominal Hysterectomy
TURP = Trans Urethral Resection of Prostate
UI = Urinary incontinence
UTI = Urinary Tract Infection
VD = Voiding postponement
VH = Vaginal hysterectomy

Epidemiology of Urinary (UI) and Faecal (FI) Incontinence and Pelvic Organ Prolapse (POP)

I. Milsom,
D. Altman, M.C. Lapitan, R. Nelson, U. Sillén, D. Thom

A. INTRODUCTION

In this report we focus on the epidemiology (distribution and determinants) of urinary incontinence (UI), faecal incontinence (FI), and pelvic organ prolapse (POP). We also discuss important topics such as differences between epidemiological and clinical approaches to health problems, help seeking behaviour, and methodological issues for this research.

Overactive bladder (OAB) is an evolving concept with close relationship to UI, and we have included a section on this topic. A worldwide estimation of the current and future number of individuals with lower urinary tract symptoms (LUTS)[1] including urinary incontinence and overactive bladder is also included at the end of this chapter.

The epidemiological population under study for this review will mainly be community dwelling non-institutionalized persons. The review will include discussion of the prevalence, incidence, natural history, and presence of racial and ethnic differences. We also review correlates and potential risk factors that have been revealed in epidemiological studies. Progress has clearly been made during the 4 years since our previous report when the 3rd International Consultation on Incontinence (3rd ICI) was published [2]. We are however greatly indebted to the authors of the previous chapter on epidemiology from the 3rd ICI who contributed important information which has in some instances been utilised in this chapter (S. Hunskaar, K Burgio and A Clark). Some new important areas have been studied with increasing regularity and quality. We have searched the literature for relevant new articles, thus reviewing a large number of high-quality and population based studies, as well as clinical trials that might include relevant epidemiological data. Because of an abundant number of studies, only a small fraction can be presented in a text like this. Other studies not presented here may have equally useful information, but lack of space precluded their inclusion.

Summary points:

- This review includes discussion of the prevalence, incidence, natural history, and presence of racial and ethnic differences in the epidemiology of UI, OAB, FI and POP.
- Correlates and potential risk factors that have been revealed in epidemiological studies are also reviewed.

B. BASIC EPIDEMIOLOGICAL CONSIDERATIONS

Epidemiology is the scientific study of the distribution and determinants of disease in people. **Descriptive epidemiology** is the description of disease prevalence, incidence, (and mortality) by persons, place and time, while the term **analytical epidemiology** describes the search for determinants of disease risk. The discovery of risk factors and protective factors may then in turn lead to primary or secondary prevention.

In order to collect knowledge about risk factors or natural history, observational studies are needed. Cohort studies and case-control studies are the most common. However, caution is always needed when interpreting the results from such studies, as associations found in epidemiological studies may not be the same as causes. Longitudinal study designs and appropriate control for confounding factors are preferred, as these increase the validity of epidemiologic studies. For practical and ethical reasons, experimental designs are seldom used.

Recommendations and conclusions should always be based on the best available evidence. Studies of interventions, and studies of risk factors generally cannot be randomised because they relate to inherent human characteristics or practices, and exposing subjects to harmful risk factors is unethical. No uniform guidelines for assessing the results of observational studies exist, and the level of evidence for risk factors

from observational studies should be judged on the soundness of the exclusion of alternative explanations by statistical and other controls. But some initiatives for how to report meta-analyses of observational studies have been taken.[3]

Studies of disease frequency should rely on a very specific definition of the condition under investigation. The absence of unifying definitions for the conditions reviewed here is a fundamental problem which has not been resolved. Definitions used and problems associated with them are discussed in the subsections for the particular populations below.

Prevalence is defined as the probability of experiencing a symptom or having a condition or a disease within a defined population and at a defined time point. The concept is important for establishing the distribution of the condition in the population and for projecting the need for health and medical services.

Incidence is defined as the probability of developing the condition under study during a defined time period. Incidence is usually reported for one-, two- or five-year time interval.

Even in many of the recent studies reviewed analyses are very simple. Often only proportions or percentages are used to describe differences in different subgroups. Many analyses do not control for confounders (by stratification or multivariate analysis techniques). There is an obvious need for more advanced epidemiological analyses of risk factors and comorbidity, and strength of associations should be determined by relative risks and odds ratios.

The relative risk (RR) estimates the magnitude of an association between exposure and a condition, and indicates the likelihood of having the condition in the exposed group relative to those who are not exposed (e.g. do not have the risk factor). A RR of 1.0 indicates that the rates in the exposed and non-exposed groups are identical and thus that there is no association between the exposure and the condition in that specific dataset. A value greater than 1.0 indicates a positive association or an increased risk. A RR of 2.5 for UI indicates that there is a 2.5 times increased risk or that the persons in question are 150 percent more likely to have incontinence than those without the risk factor.

The odds ratio (OR) is the odds for having a risk factor in persons with a condition divided by the odds among those without the condition. An OR of 2.5 for UI my be interpreted as meaning that in this sample the odds in favour of having incontinence are 2.5 times higher among those with the risk factor than among those without.

For a condition with high prevalence, like UI or POP, OR and RR will not be identical, but in practice the results can be interpreted similarly. Results should always be given with a 95% confidence interval (CI). Words like well established and established may be used about risk factors and findings with a high level of evidence in the literature. For less documented findings words like " indications of" or " data are suggestive" may be used.

Summary points:

- Descriptive epidemiology reports disease incidence, prevalence (and mortality) by persons, place and time.
- Analytical epidemiology searches for determinants of disease risk. There is a need for good longitudinal cohort studies.
- Variations in definitions and measurement issues are fundamental, and lead to problems with assessing the findings in epidemiological studies.
- There is a need for more advanced epidemiological analyses of risk factors and comorbidity using multivariable techniques, and strength of associations should be determined by relative risks and odds ratios.

C. EPIDEMIOLOGY OF ENURESIS AND UI IN CHILDREN

I. GENERAL COMMENTS AND DEFINITIONS

The International Children's Continence Society (ICCS) has issued new recommendations regarding terminology of bedwetting or **nocturnal enuresis** (NE) [4]. NE is now the term for all urinary incontinence during sleep taking place in discrete episodes, regardless of the presence or absence of concomitant daytime symptoms. **Monosymptomatic nocturnal enuresis** (MNE) denotes bedwetting without any other lower urinary tract (LUT) symptoms, and **non-monosymptomatic nocturnal enuresis** (NMNE) should be used for those with any concomitant LUT symptom.

NE is caused by relative nocturnal polyuria [5] and/or nocturnal bladder over-activity [6], combined with the lack of arousal at the time when the bladder needs to be emptied. The most important cause is, of course, the lack of arousal, otherwise the child would have had nocturia.

Any other leakage of urine in children during both the day and night is referred to as **urinary incontinence** (UI), just as it is in the adult population. UI with no obvious cause, i.e. without neurological or congenital anatomic alterations, is often seen together with other

urinary symptoms such as frequency, urgency and infections. Altogether these symptoms are referred to as functional LUT dysfunction, which is the term used to describe the entire spectrum of functional filling-voiding disturbances. Several sub-classifications have been used for children who present with varying degrees of "functional" urinary symptoms. Some are based on urodynamic patterns, others on clinical presentation.

According to recent definitions by the ICCS [1], based on symptoms and flow-residual studies rather than invasive urodynamic investigations, incontinence as a result of a filling-phase dysfunction, is in most cases due to an **overactive bladder** (OAB), which can also be referred to as "urge syndrome" and "urge incontinence". Children with OAB usually have detrusor overactivity, but this label cannot be applied to them without cystometric evaluation. When incontinence is the result of a voiding-phase dysfunction, the diagnosis is often **dysfunctional voiding** (DV), which is induced by increased activity in the sphincter and pelvic floor during voiding. It is subdivided into staccato and fractionated voiding, and the terms cannot be applied unless repeat uroflow measurements have been performed. **Voiding postponement** (VD) is another common LUT dysfunction causing UI in children, but differs from the other since it is induced by habitual postponement of voiding and not a LUT dysfunction per se.

NE and UI due to functional LUT dysfunction are the wetting problems addressed in this paper. Both can be either primary (the child has not been dry for more than six months) or secondary (the wetting has recurred after a dry period lasting more than six months). If the complaints are secondary, they may signify psychological, neurological or even structural anomalies and therefore require careful consideration.

The healthy infant is socially incontinent but physiologically continent, because micturitions (about once every hour) are discrete and there is no leakage of urine between micturitions [7]. Bladder control develops during the first four to six years of life and is a highly complex process, which is still not fully understood. Most children are toilet trained by the age of three years, although there is huge social and cultural variation. By the age of five years, the child is normally able to void at will and to postpone voiding in a socially acceptable manner [8]. By this age, night-time and daytime involuntary wetting becomes a social problem and a cause for therapeutic intervention.

II. PREVALENCE OF NOCTURNAL ENURESIS (NE)

As bladder control is something that develops over time, longitudinal studies are the best way of defining the dynamics of this process. Studies giving us the prevalence for all children between five and 15 years of age, for example, are not appropriate, as all the developmental stages are clustered together. It is therefore better to give the prevalence for an age cohort, such as seven-year-olds. Furthermore, random sampling should preferably be used in order to be able to say anything about the population. These problems associated with understanding epidemiology were summarised by Krantz [9], who also reviewed the epidemiological studies that had been published by 1993.

One explanation for the variation in prevalence in different studies is the fact that some studies include only monosymptomatic enuresis (MNE), whereas others also include what is defined as nonmono-symptomatic enuresis (NMNE). Another explanatory factor is that the frequency of enuretic episodes differs or is not taken into account in some studies. Moreover, most epidemiological studies link primary and secondary enuresis together.

1. PREVALENCE OF ALL NIGHT WETTING (MNE+ NMNE) ACCORDING TO AGE

Longitudinal cohort studies should be the ideal when analysing epidemiology in childhood NE, as there is a successive reduction in prevalence. Only a few of these studies are available[10-14] and cross-sectional studies at different ages therefore have to be used.

Most studies investigate cohorts of children in an age span of six to 12 years of age, for example, and give the prevalence for the entire group. Some of them also give the age-related prevalence [12, 15-24] which is summarised in Table I. Cross-sectional studies of a specific age are also included [25-29] in Table 1**.**

In most studies (Table 1), the prevalence for seven-year-olds was between 7% and 10%. In two studies, the prevalence was higher; 15.1% and 16.4% for Turkish [19] and Korean [20] children respectively, despite the fact that the inclusion criteria were very similar in all the studies dealing with seven-year-olds (NE=night wetting once/month or more), apart from the studies by Hellström [25] (once/3 months or more) and Järvelin [26] (once/6 months or more). The prevalence of more frequent wetting (once/week or more) was lower compared to the prevalence for all wetting (once/month or more) by age, which have been illustrated in **Figure 1**.

In nine studies at age seven years [13, 15, 18, 20-23, 25, 26] (**Table 1**) the numbers of both non-enuretic and enuretic children were given and the definitions for enuresis were similar (MNE and NMNE, wetting once/1-3 months or more). A prevalence of 10% was obtained by meta-analyses of these studies (cohort of 14372 seven-year-old children, of whom 1422 were enuretic). Only four studies included groups of children that were chosen at random from the population [15, 20, 23, 26].

Table 1. Prevalence of nocturnal enuresis (NE) (= Monosymptomatic nocturnal enuresis (MNE) + Non-monosymptomatic nocturnal enuresis (NMNE) together) according to age

Author and year	Prevalence of NE (%)		
	7 years	11-12 years	16-17 years
Chiozza [15]	6.8	2	
Järvelin [26]	8		
Spee-van der Wekke [17]	8	4.6	
Cher [18]	9.3	1.7	
Hellström [25, 27]	9.5		0.5
Ferguson [12]	10.3		
Kanaheswari [24]	10.3	3.3	
Serel [19]	15.1	4	
Lee [20]	16.4	4.5	
Swithinbank [28, 29]		4.7	1.1
Soderstrom [22]	7.0	2.6	
Kajiwara [21]	10.1	3.7	
Yeung [23]	10.1	2.0	1.7

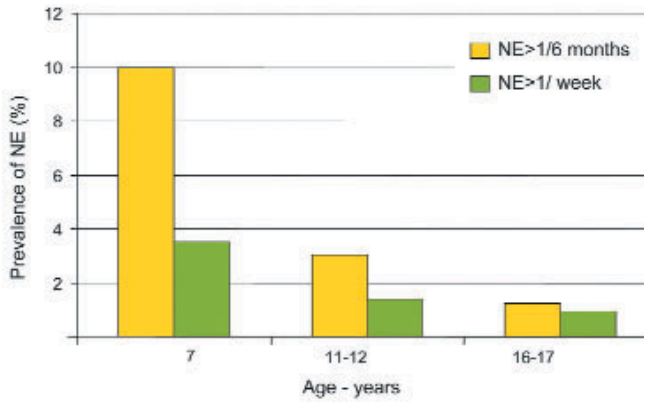

Figure 1 : Prevalence of nocturnal enuresis (NE) by frequency of enuretic episodes and age. The data were obtained from metaanalyses of the epidemiological studies included in table III.1. NE>1 episode/6months: at 7 years [15, 18, 20-26], 11-12 years [15, 18, 20-23, 28] and 16-17 years [23, 27, 29]. NE>1 episode/week: at age 7 years [22, 23, 25, 26], 11-12 years [22, 23, 28] and 16-17 years [23, 27].

At age 11-12 years, the prevalence of NE had decreased and from the studies shown in Table I the prevalence varied between 1.7% and 4.7%. In seven of the studies, the number of non-enuretics and enuretics were available and the definition of NE was similar (once/month or more), apart from Swithinbank's [28] study (once/3 months or more). In these studies, the total number of children included was 8947, while the number of children with NE was 278, giving a prevalence of 3.1%. So, of those children with NE at age seven years, almost 15% spontaneously grow out of the wetting every year. In a recent Japanese study a higher resolution rate was reported in children with MNE compared to NMNE in children 7 to 12 years of age (21% and 15%, respectively) [21]. Similar results was found in a study from Hong Kong in which the proportion of children with NMNE was significantly greater in adolescent boys than in boys aged 5-10 years (32% vs 14.6%), even if the total prevalence of NE was decreasing as in other studies [23].

The variation in the prevalence of NE at 11-12 years between the studies is less than that seen at age seven years. The highest prevalence is no longer found in Turkey or Korea, as was the case at age seven years, but instead comes from a non-randomised cross-sectional study of 11- to 12-year-old schoolchildren (n=1145) in the UK (4.7%). It can therefore be suggested that the high prevalence seen in the studies from Turkey and Korea at age seven is not due to differences in genetic predisposition, but rather to phenotypic differences, such as the age of toilet training and the subsequent attainment of bladder control, socio-economic status, or cultural differences.

At age 16-17, three cross-sectional studies show a further reduction in prevalence to 0.5-1.7%. Two of the studies re-investigate children who had previously been studied; at age seven years [27] and 11-12 years [28] respectively. The prevalence when the cohorts were added together was 1.3% (cohort=3819, NE=51) [23, 27, 29], which gives a spontaneous cure rate of 11% a year among those who wet at age 11-12 years.

In a study of 13,081 adults randomly sampled in the Netherlands [30], an overall prevalence of NE of 0.5% was found. There was no significant difference between age groups. Primary NE was reported by 50% of the men and 19% of the women, indicating that a small group of the enuretic children remain enuretic as adults.

2. PREVALENCE OF MONOSYMPTOMATIC ENURESIS (MNE)

Very few studies make a distinction between MNE and NMNE and it is therefore difficult to obtain relevant figures for MNE (**Table 2**). In two studies from Scandinavia dealing exclusively with seven-year-olds, there was agreement between the studies; 6.4% [26] and 7.4% [25]. Recently, a Japanese study gave similar figures for MNE; 6.2% at age 7 years. In this latter study MNE corresponded to approximately 60% of all NE in ages from 7 to 12 years [21]. When it comes to studies in which all ages were mixed (5-12 years), four studies were identified in which those without daytime voiding problems could be identified. However, the difference in prevalence of MNE varied in these studies; 3.5% [16], 6.9% [31], 9.4% [20] and 15% [32].

3. PREVALENCE OF NE VERSUS GENDER

Almost all epidemiological studies of NE report a higher prevalence in boys than in girls, with a ratio of 2:1 in western countries [15-22, 25, 26, 28, 31-33] . It appears that the gender difference diminishes with age and becomes less visible and less proven among older children [27, 29, 34] (**Figure 2**).

4. PREVALENCE OF NE VERSUS ETHNICITY

In a study from The Netherlands [17], a higher prevalence was reported in the Turkish/Moroccan group (14%) than in the Dutch children (6%) (OR 3.76 (95%CI 1.98-7.12)). An equally high prevalence was found in a Turkish study of children with NE [19] at age seven years (15.1%). In a study from Korea [20], the same high prevalence at age 7 years was identified (16.4%). However, other studies from South-East Asia had comparable [18, 21, 23] or even lower levels of prevalence to those in western countries. In fact, two Chinese studies have shown a low prevalence of nocturnal enuresis [16, 35], 3.6% and 4.3% for children aged 4-12 and 6-16 respectively, which they attribute to earlier nocturnal urinary control in Chinese children, due to earlier toilet training.

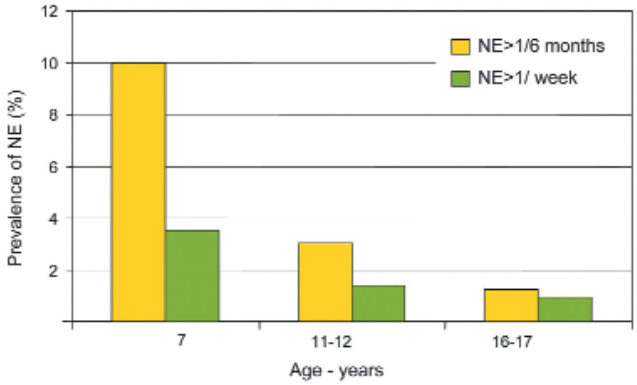

Figure 2 : Prevalence of nocturnal enuresis (NE) >1 episode/6months, by gender and age. The prevalence data were obtained from metaanalyses of the following epidemiological studies: at age 7 [15, 21-23, 25, 26], age 11-12 [15, 21-23, 28] and age 16-17 [23, 27, 29].

5. PREVALENCE OF NE VERSUS FREQUENCY OF WET NIGHTS AND AGE

Yeung et al [23]showed in a large epidemiological study that that the relative proportion of subjects with frequent bed-wetting increased with age. Overall 82% of the adolescence had >3 wet nights/week versus enuretic children aged 5-10 (42%) (**Figure 3**).

III. POTENTIAL RISK FACTORS FOR NE

Several risk factors have been established or suggested by epidemiological studies and the most important ones will be discussed here.

Table 2. Prevalence of Monosymptomatic nocturnal enuresis (MNE) at age seven years and overall (including all ages)

Author	Prevalence of MNE (%)	
	Age 7 years	All ages included
Järvelin [26]	6.4	
Hellström [25]	7.4	
Kanaheswari [24]	9.0	6.2
Lee [20]	13.6	9.4
Yeung [16]		3.5
Neveus [31]		6.9
Bower [32]		15.0
Kajiwara [21]	6.2	3.5

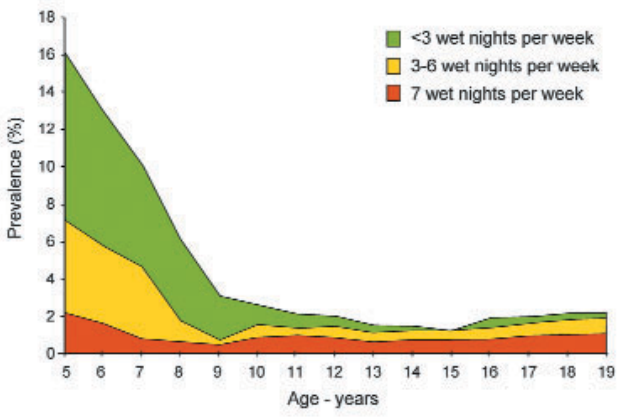

Figure 3 : Prevalence of nocturnal enuresis by frequency of enuretic episodes and age. Data from [23].

1. FAMILY HISTORY

NE is a hereditary disorder and this has been demonstrated in many studies (for example, [15, 16, 19, 26, 31, 32, 36]). The mode of inheritance appears to be autosomal dominant. Järvelin [26] showed that, if both parents were enuretics as children, the RR (95% CI) for the child to have NE was 16 (6.3-20.1), while if only one was enuretic, the RR was 7.8 (5.1-9.8). Using molecular genetic methods, foci have been found on chromosomes 13, 12, 8 and 22 [37, 38]. A picture of pronounced heterogeneity for both genotype and phenotype emerges [39].

2. PSYCHOPATHOLOGY

There are evident connections between childhood enuresis and mental well-being [13, 14, 35, 36, 40-42]. Evidence is accumulating to show that psychological consequences are probably caused by the enuresis and not a cause of primary NE, which has been thought for a long time [41]. The findings presented by Feehan [14] support this latter statement, as he only found an association between psychopathology and secondary NE, while children with NE did not display a connection of this kind.

3. DEVELOPMENTAL DELAY AND MENTAL RETARDATION

Children with developmental delay and mental retardation have been shown to have a higher prevalence of NE [12, 17, 26, 43]. Spee-van der Wekke [17] found that children who were given special education, including both those with and without mental retardation, had an OR of 3.74 (95%CI 2.32-6.03) for NE.

Perinatal events such as toxaemia (pre-eclampsia) and low birth weight, possibly involving an increased risk of minor neurological dysfunction, have also been shown to be associated with NE [12, 26, 36]. A connection between NE and minor neurological dysfunction of this kind has also been shown by Lunsing [44] in 12-year-old enuretic children. Furthermore, children with attention deficit hyperactivity disorders (ADHD) are more likely to have enuresis than the general child population [42, 45, 46].

4. SLEEP AND AROUSAL

The main pathology behind NE in children is the inability to wake up to the sensation of a full bladder. Parents often say that their enuretic child " sleeps very deeply". Some recent studies support this view. By using auditory signals [47], computerised EEG [48] or questionnaires [31], a defect in arousal has been largely validated. In the study by Neveus [31], the odds ratios were significantly higher for a high arousal threshold (2.7), pavour nocturnus (2.4) and confusion when awoken from sleep (3.4). Computerised EEG energy analysis has indicated both greater depth of sleep and impaired arousal in enuretics [49]. Difficulty in arousal from sleep has also been shown in children with NE compared to children with isolated day-wetting problems and controls, by using a scoring system in a questionnaire [50]

5. SOCIO-CULTURAL FACTORS

Differences in the prevalence of NE [16, 19, 20, 35, 51] at early ages in different parts of the world are probably partly due to socio-cultural differences and not to differences in genetic predisposition [17]. It has been suggested that socio-economic status correlates with NE in some studies [15, 42], whereas in others no correlation was found [12].

6. OTHER RISK FACTORS

Sleep apnoea has been associated with enuresis in some patients [52]. **Upper airway obstruction** due to large adenoids or tonsils also appears to be a cause of NE, as it has been reported that the removal of these obstructions significantly reduced or cured NE [53]. **Constipation** may cause secondary NE or make primary NE persist [54]. **Sexual abuse** must also be included among the factors that may lead to NE [55]. **Organic conditions** such as infravesical obstruction and neuropathic bladder may also present as NE. In most cases, however, additional symptoms are present to make detection possible. **Type1 diabetes** was reported to be a risk factor for secondary MNE due to the polyuria seen at presentation [56]

IV. PREVALENCE OF FUNCTIONAL INCONTINENCE IN CHILDREN

In children with functional LUT dysfunction, the OAB is far more common than dysfunctional voiding. In a urodynamic study of 1,000 patients with functional LUT dysfunction, approximately two-thirds had an

overactive bladder and one-third had dysfunctional voiding [57]. Based on clinical information, another study comprising 226 children revealed that 76% were considered to have an overactive bladder and only 1% dysfunctional voiding. The difference illustrates that different inclusion criteria influence the rate of prevalence [58]. When considering the total prevalence of UI (all frequencies of UI included) (Table 3), there was a variation between 3.2% and 9% in different studies at the age of seven years. In the earliest studies the prevalence was lower (3.2%-5.0%), whereas in the studies performed later in the 2000 [20, 22, 59, 60], the prevalence was higher 6.3%-9.0%. One explanation for the difference was probably an increased recognition of the problem in the population through information via media etc. At 11-13 years the reported prevalence varied between 1.1% and 12.5%. Swithinbank's study [28] showed a very high prevalence (12.5%) and differed most from the rest (1.1%-4.2%). The difference could probably partly be explained by different limits for frequency of UI (occasionally [28] vs once/month or more). The fact that the studies were performed in different parts of the world was also a possible explanatory factor (UK and Korea).

The frequency of UI decreased with age (Table 3), which was clearly demonstrated in the subjects with frequent episodes of UI (>1/week) (**Figure 4**). The prevalence at 7 years, 11-13 years and 15-17 years was 2.6%, 1.1% and 0.3% respectively. There were only two authors who investigated the same cohort of children on two occasions; Hellström [25, 27] in Sweden and Swithinbank [28, 29] in the UK. According to the studies by Hellström, the reduction from seven years to 17 years was 0.2% per year in those with wetting at least once a week and 0.3% when including all kinds of wetting. Swithinbank reported a far higher frequency for all kinds of wetting at age 11-12 years but not at 15-16 years and the reduction in her cohort of children was therefore approximately 2% per year.

UI was more common in girls in most studies, especially in the older age groups (Table 3, fig.4). From the prevalence found in the different studies, daytime UI could be suggested to be 1.5 times more common in girls than in boys at age seven years, whereas at age 16 years the difference was even more pronounced: 5-10 times more common in girls than in boys (**Table 3**).

1. PREVALENCE OF OVERACTIVE BLADDER (OAB)

In a Japanese study [59], the prevalence in children between 7 and 12 years of age, OAB was seen in 17.8%, with no significant difference between boys and girls. There was a gradual decrease in prevalence from 19.8% at the age of 7 years to 12.8% at 12 years.

2. COMORBIDITY: PREVALENCE OF BOWEL PROBLEMS

Urinary and faecal incontinence often coexist in different combinations. Constipation in childhood is a very common condition and when functional faecal incontinence is seen, constipation is often the cause. The term encopresis can be used synonymously with functional faecal incontinence. Soiling, on the other hand, is a confusing term that is poorly defined and should therefore not be used [4].

An increasing number of epidemiological studies reporting the frequency of bowel problems are accumulating, either in terms of constipation or functional faecal incontinence, in children with daytime wetting. Table 4 shows that the prevalence of bowel problems in day-wetting children approximately corresponded to a third of the children (24%-35%) [22, 50, 59, 60, 62], with even higher prevalence in the subgroup with dysfunctional voiding (43%) [62].

A significant association between day-wetting and bowel problems was shown [22]. These results support the new treatment concept of day-wetting children, with treatment of bowel problems as the first step. Children with MNE, on the other hand, seldom have bowel problems (0%-1%), whereas in NMNE it is more

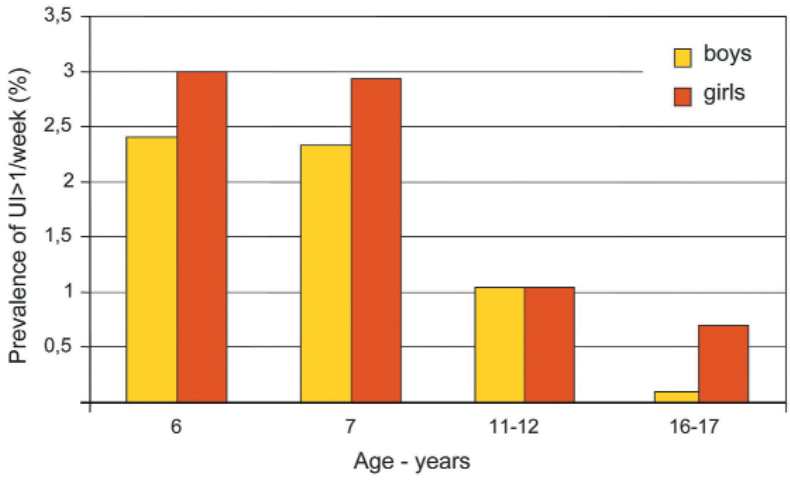

Figure 4 : Prevalence of day UI (including mixed day/night) >1 episode/week by age and gender. Data are from: at age 6 years [63], 7 years [22, 25], 11-12 years [22, 28] and 16-17 years [27]

Table 3. Day urinary incontinence (UI) (including mixed day/night)

Author (ref)	Sample size	Prevalence (%)			
		<1/week	>1/week	Total day+night	day only
Children 7 years:					
Järvelin [26]	Total: 2,892			**3.2**[1]	1.8
	Boys – 1,444			2.7	1.3
	Girls –1,445			3.7	2.3
Hellström [25]	Total: 3,555	2.3	2.5	**4.9**[2]	2.7
	Boys – 1,834	1.7	2.1	3.8	1.7
	Girls – 1,721	2.9	3.1	6.0	3.7
Bloom [61]	Total: 101			**5.0**[4]	
Lee [20]	Total: 1,325			**6.7**[3]	3.9
Kajiwara [59]	Total: 984			**9.0**[3]	9.0
	Boys: 532			9.2	9.2
	Girls: 452			8.9	8.9
Söderstrom [22]	Total: 715			**6.3**[3]	
	Boys: 367	3.0	3.8	6.8	
Joinson [60]	Girls: 348	3.2	2.6	5.8	
	Total: 8213			**7.8**[3]	
	Boys: 4222			6.9	
	Girls: 3991			8.8	
Children aged 11-13 years:					
Bloom [61]	Total: 165			**1.2**[4]	
Swithinbank [28]	Total: 1,171	11.9	0.6	**12.5**[5]	
	Boys – 510	7.0	0.2	7.2	
	Girls – 661	15.7	0.9	16.6	
Lee [20]	Total: 913			**1.1**[3]	0.9
Kajiwara [59]	Total: 761			**2.5**[3]	
	Boys: 366			1.0	
	Girls: 395			3.9	
Söderstrom [22]	Total: 763			**4.2**[3]	
	Boys: 398	1.8	2.3	4.1	
	Girls: 365	3.0	1.3	4.3	
Children aged 15-17 years:					
Bloom [61]	Total: 81			**1.2**[4]	
Hellström [27]	Total: 651	1.5	0.3	**1.8**[2]	1.8
	Boys – 344	0.3	0.0	0.3	0.3
	Girls – 307	2.9	0.7	3.6	3.6
Swithinbank [29]	Total: 940			**3.0**[2]	
	Boys – 411			0.9	
	Girls – 529			4.7	

Episodes of UI: [1] >1/6 months, [2] >1/3 months, [3]> 1/ month, [4] >1/2 weeks, [5] occasionally

Table 4. Comobidities. The prevalence of concommittant bowel problems in children with day-wetting and nocturnal enuresis

Author	Number children bowel problems in day-wetting group	OR (95%CI)	Number children bowel problems in NE	OR (95%CI)
Söderstrom 2004 [22]	35%	7.2 (4.1-12.7)		1.2 (0.6-2.5)[2] 2.0 (0.6-6.3)[3]
Kajiwara 2004 [59]	33%			
Von Gontard 2004 [62]	25%[4]		0%-16%[1]	
Chandra 2004 [50]	24%		1%-24%[1]	
Joinson 2006 [60]	33%			

1 low value represents MNE, high value NMNE, 2OR for fecal incontinence, 3 OR for constipation, 4 Subgrouping of daywetting in OAB, VP and DV the prevalences are: 18%, 25% and 43%, respectively.

common (16%-24%) [50, 62]. In an epidemiological study from Japan including 5282 children, ages between 7-12 years, 81.5% were reported to have daily bowel movements. A significant higher prevalence of NMNE was found in those with constipation, compared to among those with regular daily bowel movements (3.4% vs 2.2%) [21].

V. POTENTIAL RISK FACTORS FOR DAY WETTING

1. FAMILY HISTORY

Day wetting, also including those subjects with mixed day and night wetting, has been shown to be correlated to hereditary factors, in parallel to what is known about children with NE. However, the number of studies are limited (**Table 5**).

2. PSYCHOPATHOLOGY

Children under stress as a result of marital separation, for example, have a higher incidence of diurnal or mixed UI, according to some authors [15, 36, 63]. Moreover, psychopathology investigated by Järvelin [36] using the Children's Apperception Test (CAT) revealed a significant increase in the signs of repression, including an inability to express one's emotions and feelings (p=0.027), when comparing day wetting children with controls. Neveus [31] found that day-wetting children had more difficulty falling asleep (OR 2.4, CI 1.4-4) and he interpreted them as "anxious children". Lettgren [64] found a significant increase in attention problems and delinquent behaviour in a certain form of day-wetting children (voiding postponement) using the Child Behaviour Check List (CBCL, Achenbach). In a recent paper [65] similar results was found with the highest rate of psychiatric comorbidity in children with UI due to voiding postponement and the lowest in children with MNE. In the group with encopresis 65% were considered to have severe behavioural problems [62], meaning that children with both wetting and bowel problems are at the highest risk for psychopathology. In a population-based study investigating psychological problems associated with day UI, 8213 children were included of whom 643 suffered from daytime wetting at median age 7.5 years [60]. Overall the results indicated a rate of psychological problems that was twice the rate reported for children with no daytime wetting, particularly notable was the increase in externalising problems. After adjustment for developmental delay, gender, stressful life events, variables associated to family sociodemographic background and soiling, there was still an independent association of daytime wetting and behaviour problems (OR 2.04, CI 1.67-2.51). It is not clear whether the behavioural problems described are a cause or a consequence of daytime wetting.

3. MINOR NEUROLOGICAL DYSFUNCTION AND DEVELOP-MENTAL DELAY

Children with minor neurological dysfunction have also been shown to have an increased rate of day wetting. Duel [45] found that children with ADHD are three times more likely to have day UI than controls (p<0.0005). Also in children with delayed maturation or with mental retardation, the risk of day wetting is increased (OR 1.9 and 4 respectively), according to studies by Järvelin [26]. Perinatal events, which can also be suggestive of minimal brain dysfunction, have also been shown to be over-represented in day-wetting children. For example, Järvelin [36] found that the children of mothers who had suffered from toxaemia had an RR of 8.5 (CI1.4-51.9) for day UI.

4. OTHER RISK FACTORS FOR DAY UI

Sometimes, functional day UI is difficult to distinguish from UI due to organic anomalies. The most prominent examples are the adolescent form of posterior urethral

Table 5. Day wetting vs family history (including mixed day/night wetting)

Author	RR (95%CI)	OR (95%CI)	Positive history (%)
Järvelin [36]			
-enuresis in mother	10.1 (3.4-29.3)		
-enuresis in father	5.9 (1.9-17.8)		
Sureshkumar [63]			
daytime wetting in			
-male sibling		5.3 (1.6-18.2)	
-paternal lineage		9.3 (3.2-27.3)	
Chiozza [15]*			
-enuresis in parents		12.3	
Bower [29]			
-family history of enuresis			70**
Neveus [28]			
-family history		2.0 (1.1-3.7)	

*Only children with mixed day and night wetting, **compared with 45% in dry children

valves in boys and epispadias in girls. In many papers, UTI is regarded as a risk factor for day UI. Järvelin [36] found an RR of 8.6 (2.3-32.3) for UTI in day UI children. Neveus [31] was able to demonstrate similar connections; OR 2.3 (1.3-3.9). However, these infections should probably be regarded as a consequence of the functional bladder disturbance with UI and not the other way round as a cause of the UI.

VI. SUMMARY POINTS

Nocturnal enuresis (NE)

- The prevalence of NE at age 7 seems to be around 10% for most countries, at age 11-12 years around 3% and at age 16 around 1.3%.
- The spontaneous cure rate seems to be around 15% annually between 7 and 12 years, and between 12 and 17 years 11%.
- In an adult population the prevalence of NE seems to be 0.5%. The prevalence was 0.1% when including only those with a history of NE during childhood. Thus the risk for NE as adult if having the condition at 7 years of age can be calculated to 1%.
- Potential risk factors for NE in children include OAB, polyuria, family history, psychopathology, developmental delay, mental retardation, socio-cultural factors, sleep and arousal problems, sleep apnoea, constipation, sexual abuse and organic conditions such as infravesical obstruction.

Functional incontinence

- Children who are and remain dry in the day seem to attain their diurnal continence already between age 4 and 5 years
- Diurnal UI, or combined diurnal and nocturnal UI, in children is caused by overactive bladder in the great majority of cases.
- Prevalence for functional UI decrease with age. At age 7 years prevalence figures varies between 3.2% and 9%, with the highest prevalence in recent studies. At age 15-17 years the corresponding prevalence is 1.2-3%.
- Variation in prevalence figures is mainly dependant on differences in frequency of incontinence episodes in the studies.
- Potential risk factors for diurnal UI in children include bowel problems such as constipation and functional faecal incontinence, family history, psychopathology, socio-cultural factors, minor neurological dysfunction, developmental delay, organic anomalies such as infravesical obstruction in boys and sexual abuse.

D. EPIDEMIOLOGY OF UI IN WOMEN

I. GENERAL COMMENTS AND DEFINITIONS

UI is a common symptom that may affect women of all ages, and there is a wide range of severity and nature of symptoms. UI is not a life-threatening disease, but the symptoms may seriously influence the physical, psychological, and social well being of the affected individuals.

Virtually all epidemiologic studies rely on participant self-report of UI. Multiple studies have examined the association between self-reported incontinence and clinically demonstrable incontinence, and by type of incontinence based on self-reported compared to type based on clinical diagnosis. While self-report and clinical diagnosis are highly correlated, they are inherently different. For example, Sandvik and coauthors [66] validated diagnostic questions used in a survey against a final diagnosis made by a gynaecologist after urodynamic evaluation. The percentage of stress only incontinence increased from 51% to 77%, mixed incontinence was reduced from 39% to 11% while the proportion with urge only UI remained virtually the same (10% vs. 12%). For public health purposes, self-report has the advantage of reflecting the woman's experience and allows further characterisation of the incontinence by frequency and quantity of urine loss. Despite the uncertainty in determining type of incontinence, the differences in the epidemiology of urge and stress incontinence are potentially important given the presumed differences in underlying pathophysiology. Differences in the association of stress and urge UI (based on self-report) with respect to age, race/ethnicity, and risk factors suggest that questionnaire-based determination of UI type is useful and should be included in all epidemiologic studies of UI [67].

Usually incontinence is defined by the episode frequency, for example one or more episodes in the past year or past month, or typically leaking at least once a week or daily. UI can also be defined by severity (a combination of frequency and quantity). Probably the most widely used measure of severity is the Sandvik Severity index [68-69] which is calculated by multiplying the reported frequency (four levels) by the amount of leakage (three levels). Incontinence can also be characterized by impact, with is usually assessed either in terms of bothersomeness, or by the degree to which it restricts a woman's activity (for example, the Incontinence Impact Questionnaire) [70].

The vast majority of epidemiological studies of female UI have been conducted in the U.S, Canada, Europe,

Australia or Japan. A few additional studies have been reported from other Asian countries. Thus we know very little about the prevalence, incidence and risk factors for UI in Africa or other parts of the developing world.

II. PREVALENCE

Estimates of UI prevalence differ for a variety of reasons other than variation in true prevalence:

1. Sampling frame. Representative samples drawn from a large, usually geographically defined, population (e.g. via random digit dialling) are thought to provide prevalence estimates that are the most generalisable. In contrast, studies of hospitalized patients or patients seen in specialty clinics are likely to provide estimates biased by self-selection and local referral patterns. In this chapter, we will cite population-based studies unless otherwise stated.

2. Response rates. Even population-based studies can provide biased prevalence estimates due to poor response rates [71]. Women with incontinence may be more predisposed to enroll in studies of incontinence or women's health. One study that asked about continence status among women declining to enroll did find a higher proportion of enrollment by women who were incontinent compared to those who were continent, particularly among minority women [72]. While the degree of bias is often unknown, as a general rule response rates above 80% are considered good, while those between 60 and 80% are marginal.

3. Threshold definition of UI. Usually incontinence is defined by a threshold frequency, for example one or more episodes in the past year or past month, or typically leaking at least once a week or daily. Severity of degree of bother may also be used. The threshold chosen to define UI obviously affects the prevalence and incidence.

4. Types of UI. UI in women is typically classified as stress, urge or mixed. Stress UI is urine leakage resulting from increased intra-abdominal pressure (e.g., coughing, standing up) while urge UI is leakage immediately preceded and accompanied by a strong physical 'urge' to urinate, while mixed UI is a combination of the two. Other UI – neither stress or urge – is uncommon in women. Prevalence estimates for each type of UI are sensitive to the wording of the questions and to definitions of stress and urge UI, with some definitions requiring all UI episodes to be of one type to be classified as stress or urge, while other definitions may require only that the majority of episodes be of that type.

5. Survey methods. While there are few data comparing differences in prevalence based on method of ascertainment [73-74], variation it ascertainment (e.g., in person interviews, phone interviews, mailed questionnaires, or diaries) have been shown to affect prevalence estimates for other socially sensitive conditions.

6. Culture and language. While this area has not been investigated, it is likely that there are cultural differences in the perception of and terms used to describe UI that may affect prevalence estimates. Similarly, differences in language may lead to differences in ascertainment.

In the following sections, we attempt to account for variation from the above sources by using population-based estimates with high response rates whenever possible and examining prevalence by age, by type of incontinence, and among major subgroups of interest (pregnant and post-partum women, women in long term care, and by race/ethnicity). Even so, there remain substantial variations in prevalence estimates likely related to remaining differences in study methods or sample characteristics.

1. PREVALENCE IN GENERAL POPULATION OF ADULT WOMEN

A review of 36 general population studies from 17 countries in the 2004 3rd ICI edition found that the prevalence estimates for the most inclusive definitions of UI ('ever' 'any' or 'at least once in the past 12 months') ranged from 5% [75] to 69% [76], with most studies reporting a prevalence of any UI in the range of 25% to 45%. For daily incontinence, prevalence estimates typically range between 5 and 15% for middle-aged and older women [77-82], The following section summarizes prevalence estimates from general population studies since 2004, grouped roughly by age, for two extremes of frequency, 'any' (including 'ever' or 'any in past 12 months or any in past month) and daily. Because of the large number of U.S. studies, only those with a response rate > 60% are included.

As seen in **Table 6**, studies since 2004 have added important information on prevalence of incontinence in women younger than 30 and older than 80 years of age, particularly for prevalence of incontinence by type. These studies are consistent with previous studies reporting that older women are more likely to have mixed and urge incontinence [103-106] while young and middle-aged women generally report stress incontinence [78-79, 107-109]. Overall, approximately half of all incontinent women are classified as stress incontinent. A smaller proportion are classified as mixed incontinent and the smallest fraction as urge incontinent. A recent study which included the entire adult age range by Hannested et al.[79] demonstrated a fairly regular increase in prevalence of mixed incontinence across the age range, and a decrease in prevalence of stress incontinence from the 40-49 year old age group through the 60-69 year old group

Table 6. Prevalence of "any' 'current' or 'ever' incontinent UI, and of daily UI, in the general population by age of women and type of incontinence

Reference	Country	Size	Survey Method	Ages (yrs)	Prevalence of 'any' UI (%)	Prevalence of daily UI (%)
Irwin [83]	Multiple	9605	Inteview	18+	13 (all)	-
					6 (stress)	
					2 (urge)	
					2 (mixed)	
				<39	7 (all)	
					4 (stress)	
					1 (urge)	
					1 (mixed)	
				40-59	14 (all)	
					8 (stress)	
					1 (urge)	
					2 (mixed)	
				60+	19 (all)	
					8 (stress)	
					3 (urge)	
					4 (mixed)	
Zhu [84]	China	5221	Interview	20+	49 (all)	21 (all)
					24 (stress)	9 (stress)
					10 (urge)	4 (urge)
					15 (mixed)	8 (mixed)
				20-29	25.5 (all)	6 (all)
					14 (stress)	5 (stress)
					6 (urge)	1 (urge)
					3 (mixed)	1 (mixed) 11
				30-39	37 (all)	(all)
					23 (stress)	7 (stress)
					5 (urge)	1 (urge)
					7 (mixed)	2 (mixed)
				40-49	59 (all)	20 (all)
					34 (stress)	11 (stress)
					11 (urge)	4 (urge)
					13 (mixed)	5 (mixed)
				50-59	61 (all)	25 (all)
					36 (stress)	14(stress)
					8 (urge)	3 (urge)
					16 (mixed)	8 (mixed)
				60-69	55 (all)	28 (all)
					23 (stress)	10 (stress)
					10 (urge)	5 (urge)
					21 (mixed)	14 (mixed)
				70-79	61 (all)	30 (all)
					22 (stress)	9(stress)
					15 (urge)	4 (urge)
					23 (mixed)	16 (mixed)
				80+	63 (all)	39 (all)
					16 (stress)	7 (stress)
					18 (urge)	10 (urge)
					28 (mixed)	20 (mixed)
Minassian [85]	USA	2577	Interview	20+	49 (all)	21 (all)
					24 (stress)	9 (stress)
					10 (urge)	4 (urge)
					15 (mixed)	8 (mixed)
				20-29	25.5 (all)	6 (all)
					14 (stress)	5 (stress)
					6 (urge)	1 (urge)
					3 (mixed)	1 (mixed) 11
				30-39	37 (all)	(all)
					23 (stress)	7 (stress)
					5 (urge)	1 (urge)
					7 (mixed)	2 (mixed)
				40-49	59 (all)	20 (all)

Table 6. (Ctd) Prevalence of "any' 'current' or 'ever' incontinent UI, and of daily UI, in the general population by age of women and type of incontinence (To be continued)

Reference	Country	Size	Survey Method	Ages (yrs)	Prevalence of 'any' UI (%)	Prevalence of daily UI (%)
					34 (stress)	11 (stress)
					11 (urge)	4 (urge)
					13 (mixed)	5 (mixed)
				50-59	61 (all)	25 (all)
					36 (stress)	14(stress)
					8 (urge)	3 (urge)
					16 (mixed)	8 (mixed)
				60-69	55 (all)	28 (all)
					23 (stress)	10 (stress)
					10 (urge)	5 (urge)
					21 (mixed)	14 (mixed)
				70-79	61 (all)	30 (all)
					22 (stress)	9(stress)
					15 (urge)	4 (urge)
					23 (mixed)	16 (mixed)
				80+	63 (all)	39 (all)
					16 (stress)	7 (stress)
					18 (urge)	10 (urge)
					28 (mixed)	20 (mixed)
Tegerstedt [86]	Sweden	5489	Postal	30-79	63 (stress)	-
					51 (urge)	
				30-33	50 (stress)	
					33 (urge)	
				40-49	65 (stress)	
					43 (urge)	
				50-59	70 (stress)	
					58 (urge)	
				60-69	68 (stress)	
					62 (urge)	
				70-79	65 (stress)	
					71 (urge)	
Melville [87]	USA	3536	Postal	30-80	42 (all)	11 (all)
					14 (stress)	
					5 (urge)	
					21 (mixed)	
				30-39	28 (all)	
					13 (stress)	
					3 (urge)	
					11(mixed)	
				40-49	41 (all)	
					17 (stress)	
					4 (urge)	
					18 (mixed)	
				50-59	48 (all)	
					16 (stress)	
					5 (urge)	
					26 (mixed)	
				60-69	51 (all)	
					15 (stress)	
					6 (urge)	
					29 (mixed)	
				70-79	55 (all)	
					12 (stress)	
					11 (urge)	
					30 (mixed)	
				80-90	54 (all)	
					8 (stress)	
					11 (urge)	
					29 (mixed)	
Corcos [88]	Canada	3249	Interview	35+	13.6 (stress)	-
					6.5 (urge)	
Danforth [89]	USA	83,355	Postal	37-54	43 (all)	-
Homma [90]	Japan	5406	Postal	40+	-	12 (any)
						6 (stress)
						6 (urge)
Thom [72]	USA	2109	Interview	40-69	70 (all)	10 (all)

Table 6. (Ctd) Prevalence of "any' 'current' or 'ever' incontinent UI, and of daily UI, in the general population by age of women and type of incontinence.

Reference	Country	Size	Survey Method	Ages (yrs)	Prevalence of 'any' UI (%)	Prevalence of daily UI (%)
Waetjen [91]	USA	3302	Interview	42-52	47 (any) 31 (stress) 9 (urge) 14 (mixed)	-
Rohr [92]	Denmark	5795	Interview	46+ 46-59 60-80 80+	33 (all) 24 (stress) 20 (urge) 20 (all) 16 (stress) 9 (urge) 30 (all) 22 (stress) 16 (urge) 44 (all) 32 (stress) 30 (urge)	-
Moghaddas [93]	Sweden	6642	Postal	50-64	31 (any)	
Lewis [94]	USA	10,678	Interview	50-90	22 (all)	9 (all)
Mardon [95]	USA	144,265	Postal	65+	44 (all)	-
Goode [96]	USA	490	Interview	65+`	41 (all)	-
Anger [97]	USA	Not reported	Interview	60+ 60-69 70-79 80+	38 (all) 37 (all) 40 (all) 37 (all)	14 (all) 11 (all) 15 (all) 19 (all)
Hsieh [98]	Taiwan	1517	Interview	60-69 70+ 60+	25 (all) 36 (all) 30 (all)	13 (all) 9 (all) 17 (all)
Ostbye [99]	Canada	5133	Interview	65+ 65-69 70-74 75-79 80-84 85-89 90+	19 (any) 14 (any) 16 (any) 20 (any) 25 (any) 25 (any) 29 (any)	-
Adelman [100]	USA	747	Interview	65+ 65-74 75-84 85+	45 (all) 37 (all) 48 (all) 62 (all)	-
Holroyd-Leduc [101]	USA	4099	Interview	70+	19 (all)	-
Stenzelius [102]	Sweden	2636	Postal	75+ 75-79 80-84 85-89 90+	42 (all) 33 (all) 40 (all) 45 (all) 56 (all)	-

The prevalences shown in Table 6 generally fall into ranges previous reported. There is no hard evidence for different prevalences of UI among Western countries. Comparing prevalence between countries based on separate studies is difficult due to differences in methods and definitions, as well as language, cultural and social differences. One of the few studies to estimate the prevalence of UI in more than one country found similar prevalences of any UI (41% to 44%) in 3 of the 4 countries examined (France, Germany and UK) but a lower prevalence (23%) in the fourth country (Spain) [110]. There was no apparent reason for the lower prevalence in Spain. Virtually no population based studies of UI prevalence have been done in developing countries.

Only 3 population-based studies were located which reported prevalence of UI based on the Sandvik severity score. In one US study of over 3300 women age 42 to 52, the prevalence of severe UI was 10% [111], while in another US study of over 3500 women age 30 to 90, the prevalence was 18% [87]. In the Norwegian EPINCONT study, the prevalence of UI which was moderate to severe by the Sandvik score and of at least some bother was 7%. Unfortunately, while there is wide recognition that 'any incontinence' which has a prevalence of 40 to 70 percent among middle aged and older women, is overly broad for many purposes, including estimating economic and health burden, there is no generally agreed upon criteria for what might be called "significant incontinence." Frequency, severity, impact and bother all have their own drawbacks as the sole basis for defining significant incontinence. Reporting UI prevalence using different threshold definitions, including severity, bothersomeness, impact, and desire for treatment or treatment seeking allows for flexibility, but by itself does not move us toward consensus.

2. PREVALENCE IN SPECIFIC POPULATIONS

The following section reviews UI prevalences among women living in long term care facilities, during pregnancy and post-partum period, and among women of different race/ethnicity.

3. PREVALENCE IN LONG TERM CARE FACILITIES

Several studies have documented a higher prevalence of UI in women residing in long-term care facilities compared to community dwelling women [112-116]. Because UI is associated with dementia, limited mobility and other co-morbid conditions, prevalence of UI is higher in facilities with residents requiring a higher level of care. In addition to being more frequent, UI in long term care residents tends to be more severe, costly, and have greater burden on caregivers compared to UI in the community [117].

Estimating the prevalence of UI women in institutions is complicated by the variety of definitions of long term care, ranging from supervised residential living facilities to nursing homes specialising in patients with advanced dementia. Some facilities offer a mixture of levels of care. In addition, differences in methods used to ascertain incontinence in nursing home residents resulting in wide differences in prevalence estimates. For example, a US study found a prevalence of just 1.4% based on medical record diagnosis, compared to 56% of patients with UI documented by the nursing staff [118]. Another study which analysed nearly 30,000 nursing home residents in US found that 30% had UI recorded (usually at admission) in the required 'minimum data set' (MDS) [119], similar to the prevalence of 34% found in smaller, study in Switzerland [113]. Studies that directly survey patients, family and/or staff consistently generally find higher prevalences of 50% to 80% among current residents [100, 112, 114].

4. PREVALENCE DURING PREGNANCY AND POST-PARTUM

Studies of UI during pregnancy have reported period prevalences of 32% to 64% for all UI and 40% to 59% for stress (including mixed) UI as documented in the 3rd ICI edition. Period prevalence is higher in parous than in nulliparous women [120-123], whereas new onset of UI (cumulative incidence) during pregnancy is higher in primagravida [121-123]. Point prevalence of UI is low in the first trimester, rising rapidly in the second trimester and increasing further, though less rapidly, in the 3rdtrimester [121-122]. Severity of incontinence appears to increase during pregnancy as well [124]. Data recently reported from the Norwegian Mother and Child Cohort Study, a large (N=43,279) population-based study, confirms these observations [125]. In this study, the prevalence of stress incontinence from before to during pregnancy, rose from 9% to 31% in nulliparous women, and from 24% to 42% in parous women. In contrast, mixed incontinence showed a similar rise in both groups (from 6% to 16% and from 8% to 20%, respectively). Urge incontinence remained virtually unchanged in both groups at less 5%.

Estimating the post-partum prevalence of urinary incontinence is complex because in addition to differences in study design, method of ascertainment and choices of definition of incontinence, prevalence may also depend on number of previous births, type of delivery, and history of previous incontinence. The table below summarizes studies which report the prevalence of UI in the first year post-partum for primiparous women by mode of delivery. These studies were chosen because they enrolled consecutive women delivered at one or more large hospitals serving a defined population, and were able to recruit and evaluate over 50% of women.

As shown in **Table 7**, nearly all post-partum UI is stress related. Most studies did not provide information

Table 7. Prevalence of urinary incontinence in the first post-partum year among primiparous women by type of delivery.

						Prevalence (%) by months post-partum		
Reference	**Country**	**Type of delivey**	**N**	**Type of UI**	**Freq. of UI**	**1 to 3 months**	**4 to 6 months**	**7 to 12 months**
Altman [128]	Sweden	VD	287	Stress	Any ≥Weekly Daily		20 4 1	24 6 1
Chaliha [129]	UK	VD	289	All Stress	Any	15 13		
Eason [130]	Canada	VD	467	All	Any ≥weekly Daily	31 10 3		
Eftekhar [131]	Iran	VD	357	Stress	Any		16*	
Ekstrom [132]	Sweden	VD	197	Stress Urge	Any	20 4		15 6
Pregazzi [133]	Italy	VD	379	Stress Urge	Any	8 6		
Farrell [134]	Canada	SVD	313	All	Any	23*	22*	
Groutz [135]	Israel	SVD	145	Stress	2+/mo.			10*
Pregazzi [136]	Italy	SVD	218	Stress Urge	Any	16 1		
Wijma [137]	Netherlands	SVD	62	All	Any	16	15	
Arya [138]	USA	SVD IVD	150	Stress	Any	7* 12*		3 6
Glazener [126]	New Zealand, Scotland, England	VD SVD IVD	2805 19548 51	All	Any	32, 29* 31, 28* 33, 30*		
Schytt [139]	Sweden	VD SVD IVD	750 617 133	SUI	Any			20 19 22
Chaliha [129]	UK	CS	131	All Stress	Any	9 8		
Eason [130]	Canada	CS	104	All	Any ≥weekly Daily	12 2 1		
Glazener [126]	New Zealand, Scotland, England	CS	569	All	Any	16, 12*		
Ekstrom [132]	Sweden	CS	192	Stress Urge	Any	4 3		5 5
Groutz [135]	Israel	ECS CSL	118 100	Stress	2+/mo.			3* 12*
Eftekhar [131]	Iran	CS ECS CSL	345	Stress	Any		12* 11* 25*	
Farrell [134]	Canada	CS ECS CSL	125 27 98	All	Any	8* 4* 9*	10* 5* 12*	
Schytt [139]	Sweden	CS ECS CSL	165 43 122	Stress	Any			9 0 11
Borello-France [127]	USA	ECS	116	All Stress Urge Mixed	Any	25 11 4 10	23 14 1 8	

* Restricted to women with no UI prior to pregnancy
VD=all vaginal deliveries; SVD=spontaneous (non-instrumental) vaginal delivery; IVD=instrumental vaginal delivery (forceps and/or vacuum); CS=all Cesarean sections; ECS=elective Cesarean section (prior to labor); CSL=Cesarean section after onset of labor

on frequency of leakage or degree of bother. Overall, it appears that any incontinence is reported by 15% to 30% women during the post-partum year, while weekly or greater incontinence is reported by 5% to 10% and daily UI by less than 5% of women. Excluding women with UI prior to pregnancy reduced the prevalence of post-partum incontinence by 3% to 4% in the one study reporting separate prevalences for each group [126].

Studies which included women delivered by Caesarean section and delivered vaginally consistently found a lower prevalence in those delivered by Caesarean section. Studies reporting prevalence in women delivered by elective Caesarean section and by Caesarean section after initiation of labor showed a substantially lower prevalence of post-partum UI in the elective Caesarean section group. The relatively high prevalence of post-partum incontinence among women having had an elective Caesarean section in the study reported by Borello-France is not consistent with other studies [127].

5. PREVALENCE IN DIFFERENT RACIAL AND ETHNIC GROUPS

While most epidemiological studies of UI have been conducted on Caucasian populations in the US, Canada and Europe, there are now a substantial number of studies in Asian countries [73, 91, 118-126], and among other racial/ethnic groups in the US [72, 89, 91, 97, 108, 111, 140-147]. Because of the substantial variation in prevalence of incontinence between studies, it is best to examine differences by race and ethnicity in the same study. Several population-based studies comparing the prevalence of UI among white women and women from one or more racial or ethnic groups are summarised in the table below. Most striking is the lower prevalence of stress UI in Black and Asian compared to White women. In most studies, Blacks have half to a quarter the prevalence of stress UI compared to White women but the same or slightly higher prevalence of urge UI and a slightly lower prevalence of mixed UI. Thus the difference in prevalence of UI between White and Black women appears to be almost entirely the result of a markedly lower prevalence of stress UI symptoms in Black women. The consistency of this difference across all studies and different frequencies and severities of UI suggest this is a real difference and not simply reporting bias. In contrast, the lower prevalence of UI seen in Asian women compared to White women reflects a lower prevalence of both stress and urge UI. The comparison of prevalences between Hispanic and non-Hispanic White women is complex in that some studies have found a lower prevalence of UI in Hispanic women while others have reported the prevalence to be higher in this group. This heterogeneity may be explained, at least in part, by differences in prevalence among sub-population of

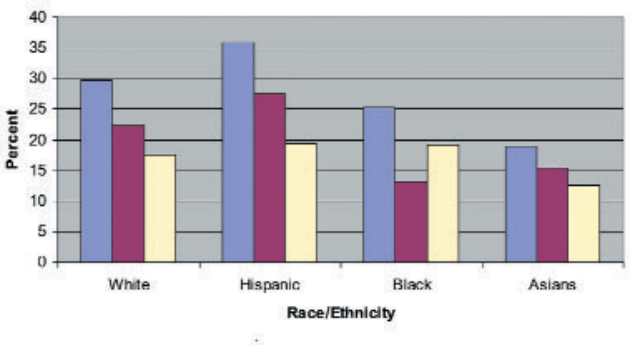

Figure 5 : Prevalence of weekly urinary incontinence by type in each racial/ethnic group, adjusted for age. Dark blue indicates any incontinence, red indicates stress incontinence and yellow indicates urge incontinence.

Hispanic women. Studies, such as the SWAN study, [46] that include Hispanic women who are primarily of Caribbean origin have reported lower UI prevalence, while those enrolling primarily Mexican-American women have reported higher UI prevalences [72, 144, 148]. This relationship is further supported by the NHANES data reported by Anger et al which found prevalences of any UI to be 36% in Mexican-American women and 30% in other Hispanic women [97, 148] (**Figure 5**).

Most studies used multivariate analysis to adjust for differences in parity, age, BMI, hysterectomy, oestrogen use, and other variables potentially related to both race and UI and have found that White women continue to have a significantly elevated risk of UI compared to Blacks or Asians [72, 89, 91, 140-145]. For studies in which Hispanic women report a higher prevalence of UI, adjusting for these variables reduces the difference with White women [72, 144, 148] (**Table 8**).

A few studies have compared physiological parameters between Black and White women to try to explain the lower prevalence of stress incontinence in Black women. These studies have reported Black women to have higher urethral closure pressures, greater urethral length and pubococcygeal muscle strength, larger urethral volumes and, paradoxically, greater vesical mobility [150-151]. The extent to which these differences, if confirmed, can explain differences in the prevalence of stress incontinence is not yet clear.

III. INCIDENCE AND REMISSION

In summarizing rate data, several factors should be borne in mind. Incontinence is a dynamic condition and both remission and reoccurrence are possible. As a practical matter, incidence is usually defined as the percent of continent women who later report incontinence. Some women who are continent at the time of enrollment may have been incontinent in the

Table 8. Prevalence (%) of UI by Race/Ethnicity in Population-based Studies

Reference/ Study name	Age	Size	% white	Definition of UI	White	Hispanic	Black	Asian
Fultz [140] AHEAD	70+	3991	85	any in past year	23	-	16	-
Nygaard [141] HRS	50-69	5701	72	any ≥1/year any ≥15 days/mo.	17 5	10 2	10 3	-
Burgio [108]	42-50	541	91	any ≥1/month	32	-	18	-
Grodstein [142] NHS	50-75	82,936	77	any ≥ 1/month any ≥1/week	35 18	28 16	21 10	26 13
Danforth [89] NHS II	37-54	85,670	92	any ≥ 1/month any ≥1/week	44 26	45 26	36 22	32 18
Sampselle [111] SWAN	42-52	3258	47	any in past year any mild* any moderate* any severe*	66 37 17 12	42 22 10 9	50 26 14 9	52# 34* 12# 6#
Anger [97] NHANES	60+	23,477,726*	80	any in past year any ≥1/month any ≥1/week any ≥1/day	41 35 25 15	31& 27& 25& 8&	20 17 15 11	-
Jackson [143] Health ABC	70-79	1558	54	any ≥1/week stress ≥1/week urge ≥1/week	27 12 11	-	14 5 7	-
Waetjen [91] SWAN	42-52	3002	47	any ≥1/month any ≥1/week stress ≥ 1/month urge ≥ 1/month mixed ≥ 1/month	57 20 32 8 16	28 11 21 1 5	39 13 13 12 13	39# 9# 27# 4# 7*
Dooley [144] NHANES	20+	4229	58	any ≥1/year stress ≥1/year urge ≥1/year mixed ≥1/year	53 27 8 19	50 26 8 17	38 12 11 15	-
Fenner [145] EPIS	35-64	2814	68	any ≥ 1/month any ≥ 1/week stress ≥ 1/month urge ≥ 1/month mixed ≥ 1/month	33 21 13 4 7	-	15 9 4 4 4	-
Thom [72] RRISK	40-69	2109	48	any ≥ 1/month any ≥ 1/week any ≥ 1/day stress ≥ 1/week urge ≥ 1/week mixed ≥ 1/week	45 30 12 15 9 3	51 36 17 18 10 5	37 25 12 8 14 2	34 19 9 8 7 3
Markland [146] SALSA	65+	421	45	"Difficulty holding urine" at least some of the time	45	29	-	-
Markland [147] UABSA	65+	490	50	any ≥ 1/month	41		25	

HRS = Health and Retirement Study, AHEAD = Asset and health Dynamics Among the Oldest Old Study, Health ABC = Health, Aging, and Body Composition Study, SWAN = Study of Women's Health Across the Nation, NHS = Nurses' Health Study, NHANES = National Health and Nutrition, EPIS = EPI Study, RRISK = Reproductive Risks for Incontinence Study at Kaiser, UABSA=University of Alabama at Birmingham Study on Aging; SALSA=San Antonio Longitudinal Study of Aging;
# Chinese and Japanese combined

past. Also, women who are continent at baseline may develop incontinence which then remits (either spontaneously or due to treatment) prior to the follow-up survey. Depending on the study design, such women may or may not be counted as incident cases. For follow-up periods of a few years, the impact of such development and remission on estimates of new incontinence, particularly incontinence at least weekly, is probably negligible. The one study of more then 5 years duration included cases where new incontinence remitted as a part of the cumulative incidence [152]. As with prevalence, incidence will depend on the exact definition of UI used. It is worth noting that remission rates are usually higher than incidence rates even if the overall prevalence of incontinence is increasing, because the denominator for remission, the number of incontinent women, is usually smaller than the denominator for incidence of new incontinence (continent women).

Table 9 summarises population based studies reporting incidence and remission; 6 are new since 2004. Several studies report rates by age groups, frequency of UI, and type of UI. Annual incidence of any new UI ranged from 3 to 11% and increased with age. Rates of complete remission ranged from 0 to 13% per year and tended to increase with age, though not as consistently as did incidence.

IV. POTENTIAL RISK FACTORS

Many epidemiological studies have investigated potential risk factors for UI. The design of most of these studies has been cross-sectional using prevalent UI. In cross-sectional studies, the temporal relationship between some potential risk factors such as diet, physical activity, medications or body weight is ambiguous as the development of UI can lead to changes in behaviour. Since 2004, there have been several prospective studies looking at risk factors for incident UI which have alloweds for evaluation of the temporal relationship. Associations in all observational studies are subject to potential confounding, necessitating multivariate analysis to estimate the independent association between risk factors and UI. The risk factors of oestrogen use and body mass (obesity) have been examined in randomised controlled trials. With the exception of age, the following risk factors are considered to be at least potentially modifiable.

1. AGE

Virtually all studies have found an increase of UI prevalence with age [66, 77, 79, 81, 112, 159-168]. While incontinence is not considered to be an inevitable part of aging; there are changes in the bladder and the pelvic structures that occur with age and which can contribute to UI [169-170]. Further, UI is often attributable to medical problems or diseases that can disrupt the mechanisms of continence (e.g., diabetes mellitus, cognitive impairment, physical disability), many of which are more common among older adults. Several investigators have noted that the prevalence of any UI appears to increase up to middle age, with a leveling off between ages 50 and 70, followed by a steady increase among the aged [79, 110, 160]. Cross-sectional studies suggest that this pattern reflects an increase in the prevalence of stress incontinence during middle age followed by little increase or even a decline after age 50. In contrast, urge and mixed UI continue to rise after age 50. As a result, while stress incontinence predominates in younger and middle aged women, urge and mixed incontinence are more common in older women [79] .

2. PREGNANCY, PARITY AND PARTURITION EVENTS

Stress UI is reported by most women at some time during pregnancy, usually in the third trimester, but is generally self-limited and resolves after delivery. For some women, UI that began during pregnancy persists after delivery as chronic UI [171-173]. In addition, women who are incontinent during pregnancy, even if they become continent after delivery, are more likely to develop incontinence later [128, 174]. The interpretation of this association is not clear. It may be that physiological changes during pregnancy cause incontinence later in life, but it is also possible that the temporary physiological changes during pregnancy may 'uncover' women with a predisposition to incontinence who are destined to become incontinent later in life regardless of the effects of pregnancy.

The vast majority of studies have found an association between parity and later UI [78, 124, 160, 167-168, 175-187]. Some studies have reported a threshold effect at one delivery and little or no additional risk with increasing parity [78, 185-186, 188]. However, other studies suggest that increasing parity increases the risk of UI [179, 185, 187,189-190].

The effect of parturition on risk of incontinence is difficult to separate from the effect of pregnancy. Possible mechanisms by which vaginal birth could lead to incontinence include stretching damage to pudendal and other nerves and tearing of connective tissue reducing pelvic floor support for urethral competence [191]. One approach to estimating the impact of vaginal delivery separately from the impact of pregnancy itself is to compare the risk of incontinence in women who deliver vaginally compared to those who have a Caesarean delivery. Most such studies have found vaginal delivery to be a risk factor for both post-partum incontinence and incontinence in later life, particularly for stress incontinence, compared to delivery by Cesarean section [134, 167-168, 171-172, 192-200]. For example, in a large study of over 15,000 women under the age of 65 years,

Table 9. Incidence of new UI in community-dwelling women from 11 population-based prospective studies since 2000

Study	Length of study (yrs)	Number without UI at baseline	Number with UI at baseline	Definition of UI	Age Group (yrs)	Average annual incidence (%)	Average annual remission (%)
Samuelsson [153]	5	292	90	any	20-59	3	6
Hagglund [154]	4	135	113	any stress	22-55	4	4
Wehrberger [152]	6.5	300	141	any 1+/4 weeks	20+	4	3
					20-39	2	0
					40-49	4	4
					50-59	4	1
					60-69	4	3
					70+	7	5
				any 2+/week	20+	2	-
					20-39	0.6	-
					40-49	2	-
					50-59	2	-
					60-69	2	-
					70+	5	-
Townsend [155] NHS	2	33,952	30,698	any 1+/month	36-55	7	7
					36-45	6	9
					46-55	7	6
				any 1-3/month	36-55	5	4
					36-45	5	4
					46-55	5	4
				any 1+/week	36-55	2	9
					36-45	2	12
					46-55	2	8
				severe*	36-55	0.6	-
					36-45	0.5	-
					46-55	0.7	-
Dallosso [156] MRC	1	6,224	-	stress "at least several times per month"	40+	8	-
					40-49	9	
					50-59	7	
					60-69	8	
					70+	9	
McGrother [157] MRC	1	NR	NR	any	40+	9	25
					40-49	8	27
					50-59	8	23
					60-69	9	26
					70-79	9	22
					80+	15	25
Wactjen [91] SWAN	5	1439	-	any 1+/month	42-52	11	-
				any 1+/week		1	
				stress 1+/month		5	
				urge 1+/month		3	
				mixed 1+/month		2	
Liu [158]	2	733	300	any stess	65+	11	5
		604	429	any urge		11	5
Goode [96] USMB	3	290	156	any 1+/month	65+	10	13
Ostbye [99] CSHA	5	4291	1020	any in past year	65+	4	6
					65-74	3	-
					75-84	4	-
					85+	5	-

CSHA =Canadian Study of Health and Aging, SWAN = Study of Women's Health Across the Nation, NHS = Nurses' Health Study. MRC=Leicestershire MRC Incontinence Study, USMB=US Medicare Beneficiaries

* 1+/month of sufficient quantity to at least soak underwear

Rortveit et al demonstrated that women who had delivered only by cesarean section were at increased risk of stress or mixed UI compared to nulliparous women (OR = 1.5) [198]. Further, those who had delivered vaginally only were at even greater risk of stress UI compared to those who had a Caesarean section only (OR = 2.4). Other studies of primiparous women have shown similar results with odds ratios (or relative risks) for UI in later life ranging from 1.7 to 2.8 [134, 167-168, 171, 201] for vaginal delivery compared to Caesarean section only.

Studies of maternal and fetal factors have found conflicting evidence regarding older maternal age at delivery and birth weight to be independent risk factors for UI. Foldspang et al found increased risk of UI with increasing age at the last childbirth for women aged 30-44 years [182]. Other studies have reported that older age at first birth is associated with an increased risk of later UI [183, 202-203]. Greater birth weight may also predispose the mother to UI [165, 193].

Studies have also examined the association between specific parturition factors during vaginal delivery and the risk of both post-partum UI and UI in later life. While several studies have reported forceps delivery [134, 171, 204], induced labour [190, 205], and episiotomy [206] to increase the risk of incontinence, these associations tend to become non-significant in multivariate analyses [138, 171-172, 183, 187, 189-190, 193, 205, 207-208] suggesting that risk of UI associated with vaginal delivery is not primarily the result of any single aspect of delivery or limited to any subset of women. Virtually all these studies have been conducted in developed countries. For women in developing countries with limited access to modern obstetrical care, traumatic vaginal delivery is a major cause of UI, including vesico-vaginal fistula [209].

Importantly, the association between parity, as well as between mode of delivery and specific parturition variables, appears to diminish and even disappear over time [160, 182, 185, 200]. A large study in Australia found that parity was strongly associated with UI for young women (18-23 years). For women 45-50 years there was only a modest association, and for older women (70-75 years) the association disappeared [160]. In a Norwegian study [185], the relationship between UI and parity was significant for younger women (age 20-34 years), weaker in middle-aged women, (35-64) and absent in older women (> 65 years).

3. OBESITY AND BODY MASS

Obesity is well established as a factor that can cause UI or contribute to the severity of the condition. It is believed that the added weight of obesity, like pregnancy, may bear down on pelvic tissues causing chronic strain, stretching and weakening of the muscles, nerves, and other structures of the pelvic floor. Some studies have found central obesity, measured by waist to hip ratio, to be more strongly associated with UI then is the traditional body mass index (BMI) [91, 210]. Because obesity has also been associated with urge UI it is thought that obesity may also act via pathways other than increased intra-abdominal pressure.

Data from numerous cross-sectional studies indicate that UI in women is associated with higher body mass index and greater weight [85, 87, 89, 92-94, 108, 111,140, 160, 162, 165-166, 168, 171, 178, 180, 183-184, 187-188, 204-205, 211-222]. The relationship between BMI and UI has been reported in studies of younger women [160], middle aged women [111, 160,184,204], older women [160], and both parous and nulliparous women [171, 216]. Several studies have reported an association between body mass and all 3 major incontinence types (stress, urge and mixed) [188, 213, 218]. The association appears to increase with increasing body mass with women in the highest body mass quartile being 2 to 4 times more likely to have UI compared to women in the lowest quartile. One large cross-sectional study estimated that the risk of UI increased by 60% for every 5 unit increase in body mass index (BMI) [143]. In another study, risk of UI increased by about 50% and risk of severe UI increased by approximately 100% for each 5 unit increase in body mass [218].

In addition to cross-sectional data, several longitudinal studies have found that weight at baseline predicts later incontinence, and that an increase in body weight is associated with an increased risk of new incontinence or progression of existing incontinence [91, 223]. In one study, a weight gain of 5-10 kg increased the risk of incontinence by nearly 50% while in another, a gain of 5 units on the body mass index (BMI) was associated with a 30% increase in risk of stress UI and a 15% increase in risk of urge UI [91].

Finally, intervention studies for weight reduction have reported that loss of weight is associated with partial remission or complete resolution of UI. Several studies of incontinent women who sustained substantial weight loss from bariatric surgery found that a majority experienced resolution of stress and urge incontinence with the probability of resolution strongly correlated to the degree of weight loss [224-226]. Weight reduction has also resulted in a significant reduction in UI frequency in a randomized controlled trial of non-surgical weight reduction for obese women [227] and in a large intervention trial for women with type 2 diabetes [228]. Thus, there is strong evidence to support the causal role of excess weight in the development of UI.

4. HORMONES

Oral oestrogen replacement therapy has been widely used to treat urinary incontinence during or after menopause for many years, despite equivocal data from clinical trials. The positive association between

oestrogen use and incontinence noted in cross-sectional studies [205, 229-230] was generally assumed to be the result of women with incontinence receiving estrogen for treatment of their incontinence. In 2001, Grady et al published data from the Heart Estrogen/Progestin Replacement Study (HERS), a large placebo-controlled randomized trial of estrogen replacement therapy which showed that post-menopausal women randomized to conjugated oral estrogen plus medroxyprogesterone were more likely then women taking the placebo to experience worsening of their incontinence over 4 years (39% vs 27%, p<.001) [231-232] A similar association was later found in women age 50-79 enrolled in the Womens' Health Initiative (WHI) Hormone Replacement Trial [233]. In this large randomized controlled trial, continent women receiving estrogen, with or without progestogen, were approximately twice as likely to have developed stress incontinence at 1 year (16% vs 9%, p<.0001). The risks of mixed and urge incontinence were also significantly increased, though more modestly. The effect was seen primarily in women 55 and older. Thus, while oral estrogen as a risk factor for stress UI may be unexpected, the evidence from these 2 large randomized controlled trials supports this conclusion for stress UI, at least in women age 55 and older. The effect of estrogen on urge incontinence, and on incontinence in women less 55 years of age, is less clear.

While relatively little is known about the effects of the newer class of selective estrogen receptor modulators (SERMs) on incontinence, two SERMs, levormeloxifene and idoxifene have been associated with an increased risk of pelvic organ prolapse surgery in clinical trials [234], and women taking levormeloxifene have experienced a 4-fold increase in urinary incontinence [235]. In contrast, no increase in UI was seen an a randomized controlled trial of raloxifene [236].

5. DIABETES

Many, though not all, cross sectional studies have reported urinary incontinence to be more common in women with type 2 diabetes than among women with normal glucose levels even after adjusting for common risk factors, including age and obesity [87, 89, 91, 111, 143, 228, 237-238]. In the analysis of the large, prospective Nurses' Health Study cohort, diabetes was a significant predictor for new incontinence [237], and the strength of the association increased after 5 years of diabetes and by severity of incontinence. Women with diabetes of more than 5 years duration had approximately 50% more risk of severe UI and twice the risk of very severe UI. Data from another prospective cohort study did not find an association between new diagnosis of diabetes and UI up to 1 year later [239], suggesting that diabetes needs to have been present for more than a year to substantially increase the risk of UI.

6. HYSTERECTOMY

Approximately 600,000 women have hysterectomies in the U.S. each year, usually for non-cancerous conditions. There has been concern that hysterectomy may be associated with development of urinary incontinence via damage to the pelvic nerves and pelvic supportive structures [240-242]. However, most studies have not found an increase in reported incontinence in the first two years following hysterectomy [243-247] and at least two studies have reported a statistically significant decrease in incontinence following hysterectomy [248-249]. Most of these studies have been uncontrolled and limited by small sample sizes (fewer than 35 patients per study). However, a larger (n=158) prospective study that did include a control group (women undergoing a dilatation and curettage), also found no increase in incontinence 3 to 21 months after hysterectomy [250]. A 2-year, randomized controlled trial of 160 women who had undergone either hysterectomy or endometrial ablation for menorrhagia found no difference in incontinence or in cystometric abnormalities between the two groups [251]. A third, large, retrospective study, of 415 hysterectomised women and 335 women who had undergone laparoscopic sterilization, also found no difference in the prevalence of urinary incontinence between the two groups 2 to 8 years after hysterectomy [252].

While most prospective studies have failed to find an association between hysterectomy and subsequent incontinence, several epidemiologic studies have reported an association between a history of hysterectomy and current UI, with relative risks ranging from 1.2 to 2.1 after controlling for other variables using multivariate analysis [87, 89, 93, 145, 205, 229]. In two studies, the association between hysterectomy and incontinence was found to be stronger if the hysterectomy was performed before age 50 [81,205].

Some studies have reported an increased prevalence of UI in women receiving a subtotal, compared to a total, hysterectomy [253-254] while others found no difference by techniques [255]. Additional factors, such as indication for hysterectomy (fibroids, endometriosis, or prolapse) and concomitant oophorectomy, have not been well studied.

Prospective studies of the association between a history of hysterectomy on incident UI have provided conflicting results. One prospective study reported a significant association between history of hysterectomy at baseline and subsequent incidence of new urge, but not stress, UI [221]. In contrast, two other studies failed to find a significant association between baseline hysterectomy status and UI [153, 256].

Based on the available data, hysterectomy does not appear to increase the risk of UI in the short term. While several cross-sectional studies have reported an association between a history of hysterectomy and

current UI, the strength of the adjusted association has been weak to moderate. The few prospective studies have given mixed results on whether a history of hysterectomy is an independent risk factor for new onset UI. While two studies have suggested that hysterectomy at a younger age may be a stronger risk factor, this needs to be confirmed in future studies.

7. URINARY TRACT INFECTIONS AND LOWER URINARY TRACT SYMPTOMS

Urinary tract infection (UTI) has long been considered to be a cause of transient UI. The role UTIs with respect to the development of chronic UI is less clear. While several cross-sectional epidemiologic studies have found that women with UI are more likely to report having had one or more UTIs [73, 145, 159, 164, 211-213, 219, 221, 257-265] these results should be treated cautiously. UTIs are often diagnosed and treated based on symptoms without culture confirmation and therefore in some cases women may received a diagnosis of a UTI for what is actually urge incontinence. In addition, lower urinary tract symptoms, including burning on micturition, frequency and nocturia may increase the likelihood of being diagnosed with a UTI in addition to being associated with UI. UI may also be a risk factor for UTI. A recent prospective 2 year study of 913 women age 55-75 found that baseline prevalence of UI was nearly twice as high in women who subsequently developed a UTI compared to those who did not [266]. Without additional data, it is not possible to say if UTIs actually increase the risk of later UI, if UI increases the risk of being diagnosed with a UTI, or if both UI and UTIs are manifestations of a common underlying process.

8. IMPAIRED PHYSICAL FUNCTION

Functional impairments, particularly mobility limitations, a history of falls, arthritis, dizziness, need to use walking aid, and poor lower extremity strength, have been correlated with UI in many community-based and nursing home studies [102-103, 112, 115, 140-141, 146-147, 159, 161, 165, 219, 221, 262, 267-274]. Odds ratios for UI from a study of 839 nursing home residents demonstrated the increasing likelihood of incontinence with an ORs=1.8, 5.6 and 7.4 for needing partial assistance, needing full assistance, or being wheelchair or bed bound, respectively [112]. A study of 2025 older women indicated that several measures of mobility impairment were associated with UI including inability to walk 0.5 mile (OR=2.2), inability to move heavy objects (OR=1.9) and inability to climb stairs (OR=1.7) [161]. Improvement in ADLs was associated with remission of urge UI at 3 years. A longitudinal study of 6346 women found that decline in physical function over 4 years, as measured by walking speed or chair stand time greater than 1 standard deviation beyond the mean, was associated with significantly greater risk of weekly incontinence at 4 years in multivariate analysis (OR = 1.3, 95% CI = 1.1-1.6, and OR = 1.4, 95% CI = 1.2-1.6, respectively) [275]. Similarly, a prospective study of older women found that slower chair stand time predicted the development of UI over the next 3 years [96].

The exact interpretation of the relationship between functional impairment and UI is still being debated. At issue is whether UI is a direct consequence of difficulties in getting to the bathroom and removing clothing, or whether mobility limitations and UI may both be consequences of general frailty in older age or of an underlying systemic illness. UI is more common in stroke patients and in patients with a variety of other neurological diseases that can also impair physical function [140, 159-161, 270]. It seems likely that at least for some patients, improving functional ability could reduce incontinence. Studies aimed at improving functional ability should evaluate the impact of improving physical function on incontinence.

9. COGNITIVE IMPAIRMENT

Research on UI in nursing home residents has consistently supported an association between UI and dementia [99,112,267,270, 275-279]. In studies using multivariate analyses, patients lacking mental orientation had a 3.6 times greater risk of being incontinent than those with normal mental status [112] and the presence of dementia increased the odds of UI by 1.5 to 2.3 [267, 270]. A link between incontinence and mental status has also been demonstrated in the acute care setting. In a sample of women admitted to the hospital for hip fracture, presence of confusion increased the odds of developing UI during the hospitalization (OR = 3.4) [272]. In the Canadian Study of Health and Aging a strong association was found between severity of dementia and UI in elderly women [115]. Odds ratios were 1.2, 4.0, and 12.6 for mild, moderate and severe dementia, respectively, after controlling for age, residence, and ambulatory function. However, in a study of community dwelling women, no relationship was found between mental status and difficulty holding urine [280]. Another, more recent community-based study found an association between cognitive impairment, defined as a score of < 24 on the mini mental status exam (MMSE), and prevalent UI, though after adjusting for physical functioning and other variables the association became substantially weaker and of borderline statistical significant (OR=1.3, 95% CI-1.0-1.6) [279]. A recent longitudinal study of 6349 community women found that a decrease in mental functioning more than 1 standard deviation, as measured by the modified mini mental status exam (MMSE) and by Trails making A and B, was not associated with increased risk of UI; however it did predict a greater impact of UI [275]. Another study of over 5000 women age 65 and older found no significant association between moderate or severe cognitive impairment, defined by the modified

MMSE, and incident UI over 10 years [99]. Together these studies suggest that mild dementia in community dwelling women is at most weakly associated with UI, while moderate to severe dementia has a consistent moderate to strong association. No studies were found which investigated whether an intervention to maintain or improve mental functioning would also improve or prevent UI.

10. DEPRESSION

Several cross-sectional studies have documented an association between depression and incontinence with estimates of the relative risk ranging from 1.4 to 2.7 [87, 91, 93,141, 143, 147, 259, 261]. The cross-sectional and observational nature of studies linking depression and incontinence means that while depression may predispose a woman to developing UI, it is also possible that UI leads to depression (for example by reducing a woman's social network), that depression and UI share one or more common neurochemical or hormonal pathways [281], or that women with depression are more likely than non-depressed women to be bothered by, and hence report, having incontinence. One prospective cohort study found no association between baseline depression or new depression and subsequent UI [91, 239, 281]. A second prospective study reported a weak association between baseline depression and subsequent UI over 3 years (OR=1.2, 95% CI=1.0-1.4) [96].

11. MENOPAUSE

Historically, menopause has been considered a risk factor for UI. However, establishing a relationship between menopause and UI has been difficult, probably in part because changes in endogenous hormone levels vary over a period of at least several years and only approximately correspond to amenorrhea or vasomotor symptoms used to define clinical menopause. Many cross-sectional studies have found that the prevalence of incontinence does not increase with natural menopause [81, 177, 179, 188, 145, 164, 282] and some studies have found a significantly lower prevalence of UI among postmenopausal women than among premenopausal women [108, 177,179, 188]. Two recent longitudinal studies have confirmed this neutral or protective effect of the natural menopausal transition (without hormone replacement therapy) on UI [222, 239]. There is evidence that menopause may have a different association with stress incontinence than with urge incontinence [76, 175]. In one study, the prevalence of urodynamic stress incontinence decreased from 21% to 12% after menopause while the prevalence of detrusor overactivity increased from 9% to 19% [283].

12. PHYSICAL ACTIVITY

Evaluating physical activity as a potential risk factor or protective factor for incontinence has been difficult. Incontinence, particularly stress UI, can be a barrier to exercise [284], which could lead to the spurious appearance of a protective effect of physical activity on UI. Alternatively, engaging in physical activity could 'unmask' an existing propensity toward stress incontinence by increasing intra-abdominal pressure, resulting in physical activity being identified as a risk factor in the short term even if it is protective in the long term. Some cross-section studies have found a protective association between exercise and UI [84, 229, 262]. However establishing physical activity as a risk factor for incontinence using observational data is challenging due to difficulties in measuring and classifying physical activity prospectively over sufficient time to see an effect. In a longitudinal study of 4291 women age 65+ "regular exercise" at baseline was not associated with either prevalent UI or new UI 10 years later in multivariate analysis [99]. In contrast, a study of over 80,000 US nurses age 54-79 found that a higher level of physical activity, averaged from 7 reports over 14 years, was associated with a reduced risk of new UI [285]. Specifically, the investigators found that that being in the top quintile for overall physical activity reduced the risk of new UI by about 20% (OR=0.81, 95% C=0.71-0.93) compared to the bottom quintile. For walking, being in the top quintile reduced the risk of UI by about 25% (OR=0.74, 95% CI=0.63-0.88). Both total activity and walking significantly reduced stress UI, but not urge or mixed UI.

13. SMOKING, COUGH AND CHRONIC LUNG DISEASE

Smoking has been reported to be an independent risk factor for incontinence in women in some cross-sectional studies [85, 89, 109, 111, 141, 171, 189, 204, 218, 222, 285-287] but not in others [87, 93, 165-166, 186, 207, 211-213, 262, 288] possible mechanisms by which smoking could be associated with UI is by increased intra-abdominal pressure due to increased coughing among smokers. Other mechanisms, such as inhibition of collagen formation [286], also have been suggested.

Recent data from the Nurses' Health Study II, with over 83,000 US women age 37 to 54 years old, supports smoking as a weak risk factor for UI, with the odds ratio for current smoking vs. never smoking increasing 0.91 to 1.20 to 1.34 for occasional, frequent and severe incontinence respectively, by multivariate analysis [89]. Former smokers had a risk intermediate between never and current smokers. In contrast, four prospective cohort studies failed to find a significant association between either past or current smoking and incident UI in multivariate analysis [99, 153, 239, 256].

The conflicting data from cross-sectional studies and lack of association between smoking and incident UI

in prospective studies suggests that smoking per se is probably not an independent risk factor for UI.

14. DIET

Studying diet as a risk factor for UI is challenging. While some dietary constituents such as coffee, alcohol or carbonated beverages have been suspected as worsening UI, there is little reason to suspect other dietary constituents as causing, or protecting from, UI. Studies of dietary factors must adjust for confounders, notably age and body mass, and should consider the tradeoffs made with other constituents. Dietary data are difficult to obtain reliably. Finally, women may change dietary intake in response to UI, making cross-sectional studies of diet and UI difficult to interpret.

Several studies have examined the consumption of alcohol, coffee as risk factors for UI. While a few studies have reported coffee drinking to be associated with an increased risk of UI [99, 289]. others have not found an association [211, 218, 229, 262]. Similarly, a positive association between alcohol consumption and UI has been reported by some [166, 290] but not others [211, 218, 222, 229]. Drinking 1 or more carbonated beverage daily compared to less then 1 per week was associated with a higher incidence of stress UI in one study (adjusted OR= 1.6, 95% CI=1.2-2.2) [291]. Tea drinkers were found to be at slightly higher risk of all types of incontinence in another study [218].

15. FAMILY HISTORY AND GENETICS

Since 2004 several family and twin studies of UI have been published. One twin study of 161 monozygotic (identical) and 249 dizygotic (fraternal) twins age 75+ found evidence for significant inheritability for urge, but not for stress UI [292]. Another twin study estimated that genetics accounted for nearly 60% of the variation in bladder neck descent as measured by ultrasound [293]. Several family history studies have found a two to three fold greater prevalence of stress UI among first degree relatives of women with stress UI compared to first degree relatives of continent women [294-296]. In one study, relative risks for stress UI was 2.8, 2.9, and 2.3, respectively, for mothers, sisters, and daughters of women with stress incontinence compared to women without first degree relatives with stress UI [294]. A large study of sisters, daughters, mothers and grandmothers of women with UI found an association between incontinence status in first-degree relatives with an increased risk for stress and mixed UI after adjustment for body mass index and parity [297]. In general the risk was somewhat higher for sisters of a woman with UI than for daughters.

16. ISCHEMIC HEART DISEASE

A recent community-based study of urinary tract symptoms reported an adjusted association between UI and ischemic heart disease in Black, but not White or Hispanic women [298] an association previously reported, though unadjusted [94]. However, at least 3 other cross-sectional studies have not found an association between coronary heart disease and UI [143, 299-300]. In a prospective cohort study, “heart attack/angina” was associated with stress UI cross-sectionally but not longitudinally in age-adjusted analysis, and became non-significant after adjusting for other variables [273]. In contrast, a prospective analysis of the Nurses’ Health Study found that coronary heart disease was associated with frequent UI (OR=1.5, 95% CI=1.2-1.8) and severe UI (OR=1.8, 95% CI=1.3-2.4) [256]. Given these mixed associations and the difficulty of completely controlling for common risk factors for heart disease and UI, it is not clear if heart disease is an independent risk factor for UI.

17. OTHER FACTORS

Additional variables reported as associated with UI include constipation [84, 160, 184, 187, 270], faecal incontinence [103, 112], genital prolapse [107, 189], congestive heart failure [301], use of diuretics [138, 187, 262], benzodiazepines [302], and other drugs [161, 303-304], and childhood enuresis [188, 215, 305-306]. In a review of medical records of 5986 members aged 65+ of a large health maintenance organization in California an increased risk of UI was associated with Parkinson’s disease, dementia, stroke, depression, and congestive heart failure [307].

V. IMPACT AND CONSEQUENCES

The impact of urinary incontinence is typically assessed by degree of bother or by scales that measure the impact of incontinence on functional status and quality of life. A single question asking patients to report the degree of bother they experience from their incontinence provides an important dimension to characterising UI in addition to frequency and amount of urine lost. Typically incontinence that is more severe, of longer duration, and is urge or mixed, is also more bothersome [308]. There are validated scales that measure the impact of UI. One of the first scales, the Incontinence Impact Questionnaire [70], uses 30 items asking whether incontinence limits activities in 4 domains. There are several other similar scales, some specific for urge or stress incontinence or for overactive bladder [70, 309-314]. In addition to severity of UI, interference with sexual activities and incontinence at night are associated with greater reported impact on quality of life [315].

Incontinence has been identified as a potential risk factor for falls and nursing home admissions. In a prospective study of over 6000 older women, women with weekly urge, but not stress, incontinence were more likely to fall during the 3 year follow-up, adjusting

for multiple other factors (adjusted OR=1.3, 95% CI=1.1-1.4) [316]. Similarly, in a study of 472 long term care residents in Germany, UI at baseline predicted falls over the next 12 months after adjusting for a variety of patient factors and co-morbid conditions [317]. UI was the second strongest predictor of falls (a history of falls being first). However, it is possible that both falls and UI are markers for frailty that cannot completely be adjusted for. Whether treatment of UI would reduce the number of falls has not been established.

UI has also been reported as a risk factor for nursing home admissions even after adjusting for multiple co-morbid conditions [301,326]. However, another study that also adjusted for additional measures of frailty, including activities of daily living, found that the association became weaker and non-significant [101].

UI has substantial, well documented, economic consequences as well. Annual costs of urinary incontinence are estimated to be over $20 billion in direct costs (primarily costs of routine care, including out of pocket costs) [318] which is greater than the annual costs for breast, cervical, uterine and ovarian cancer combined [319]. There are also substantial indirect costs from work loss and missed volunteer work as well [320].

VI. TREATMENT SEEKING AND TREATMENT

Since 2004, at least 8 papers have been published which report the proportion of women with incontinence who have sought treatment [84, 95, 98, 110, 321-325] (**Table 10**). Combined with previously published studies [78, 81, 105, 111, 308, 322, 326-328], the proportion of women with any UI in the past 12 months who report having sought medical treatment ranges from 12% [46] to 53% [30] with about half the studies showing 25% to 30% [81, 98, 322, 326]. All studies that have examined treatment seeking by type of incontinence have found that a women with urge or

Table 10. Percent of women with UI in the general population who have sought help from a physician

Reference	Country	Age (years)	Definition of UI	N	Sought help (%)
Hannestad [322]	Norway	20+	In past 12 mos		
			Any	6625	26
			Stress	3300	18
			Urge or mixed	3085	35
			Daily	1201	61
			Severe	1538	54
			Bothersome	2201	50
Roberts [105)	USA	50+	Any in past 12 mos	356	13
Hagglund [326)	Sweden	23-51	Persistently incontinent for ≥4 years	78	26
Rekers [81)	Belgium	35-79	Any	344	28
			Minor*	242	22
			Serious*	97	44
Kinchen [327) Diokno [329]	USA	18+	Any in past 30 days	1970	45
			4+ days/week	632	56
			Moderate/severe	453	73
			Bothersome	877	62
Holst [78]	New Zealand	18+	Any in past 12 mos	142	35
Van der Vaart [308]	Netherlands	20-45	Any stress	365	9
			Any urge	143	11
Mardon [95]	USA	65+	Any in past 6 mos	37,805	53
Sampselle [111]	USA	42-52	Any in past 12 mos	1854	12
Hsieh [98]	Taiwan	60+	Any	485	30
Gasquet [325]	France	18-70	Stress in past 30 days	980	
			Mild severity#	729	44
			Moderate severity#	198	62
			High severity#	53	81
Harris [324]	USA	30-79	Any 1+/week	331	45
Zhu [84]	China	20+	Any in past month	2110	12.8
			Any SUI in past month	1196	7.4
Hunskaar [110]	France, Germany, Spain, UK	18+	Any in past 30 days	5976	31
			France		33
			Germany		40
			Spain		24
			UK		25

* Serious defined as >weekly and > few drops; minor defined as other then serious

low severity=minor amount of urine loss less than daily; moderate=either more then a minor amount of urine loss or daily urine loss; severe=daily loss of greater than a minor amount of urine

mixed incontinence are more likely to seek treatment then women with stress incontinence [84, 89, 322]. As expected, the proportion of women seeking treatment is strongly associated with frequency, severity and bothersomeness, with several studies reporting proportions of 50% to 80% of women with daily, moderate to severe, or bothersome incontinence [321-322, 325, 327].

Reasons given by people for not seeking help include: not regarding incontinence as abnormal or serious [78, 330-331], embarrassment [332-334], considering incontinence to be a normal part of ageing [331, 335], having low expectations of treatment [78, 330] and thinking they should cope on their own [332, 335]. Several studies have identified factors associated with seeking care for UI, including severity, duration and type of UI, nocturnal symptoms, and the bother or impact of UI [78, 105, 322, 326-327, 330-331]. Other characteristics such as age, general health, or attitude toward health care may also play a role [327].

Even when women seek care for incontinence, treatment is often limited or not provided at all [336-337]. For those receiving medical treatment, most discontinue within 12 months. Probably less than 20% of women with mild to moderate incontinence are actively being treated for incontinence [325]. In a study of management of incontinence in general practice, 30% of the women who had told their doctor about their symptoms perceived that they were offered no help [335]. It is probable that many primary health care providers lack confidence in managing UI, and that this contributes to under treatment in those seeking help [338].

Only a small proportion of incontinent community-residing women have had surgery, medication, or exercise regimens [110, 327, 339-341]. In addition to seeking help from the formal health care system, common responses to symptoms of illness are self-management and self-treatment behaviour. The major method of actively managing UI among community residents is the use of absorbent products [339, 342-345]. For many women with very mild or occasional UI it is probably adequate not to seek help from the health care system. Others are satisfied with information and understanding about the causes and in many cases self-care may be quite appropriate. A Danish study has shown that simple information and advice was adequate " treatment" for 23% of the women seeking an open access incontinence clinic [346]. A Swedish study found that among 136 women with UI, 36% wanted clinical evaluation, and only 24% subsequently started treatment [109].

Both epidemiological and qualitative research in this field should be encouraged in order to understand cultural, religious, and personal factors including attitudes and perceptions of health care, for help seeking behaviour world wide [331, 333, 337].

VII. SUMMARY POINTS

1. The estimated prevalence of UI in middle-aged and older women in the general population appears to be in the range of 30% to 60% (increasing with age); while the prevalence of daily UI ranges from 5% to 15%, rising to over 15% in women over age 70 who are institutionalized. Some studies have found prevalences outside theses ranges, demonstrating that there remains a large variation in the estimated prevalence of urinary incontinence in women, even after taking into account differences in definitions, ascertainment, and demographic characteristics. At least part of this variation is likely to be due to the sensitivity of the subject and subtle differences in the conduct of studies. (LE 1)

2. Multiple observational studies have confirmed that White, non-Hispanic women have a substantially higher prevalence of stress UI then Black or Asian women that is not explained by differences in known risk factors for UI. (LE 1)

3. Pregnancy, labour and vaginal delivery (vs Caesarean section) are significant risk factors for later UI, but the strength of this association diminishes substantially with age. (LE 1)

4. While several specific parturition factors such as instrumental delivery and birth weight are risk factors for UI in the post-partum period, their association with UI in later life is weak or non-existent, suggesting that changes in birthing practices in developed countries are unlikely to affect UI in older age. (LE 2)

5. Additional evidence has now established body mass as an important, modifiable risk factors for UI. (LE 1)

6. Physical function also appears to be an independent risk factor for UI in older women. Whether improvement in physical function leads to a reduction in UI remains to be established. (LE 2)

7. Evidence from 2 blinded, randomised controlled trials indicate that oral oestrogen, with or without progestogen, is a significant risk factor for UI in women age 55 and older (LE 1).

8. Diabetes is a risk factor for UI in most studies. While diabetic neuropathy and/or vasculopathy are possible mechanisms by which diabetes could lead to UI, no mechanism has been established, nor is it clear whether prevention or treatment of diabetes, separate from weight reduction, will reduce the risk of UI. (LE 2)

9. Menopause, as generally defined, does not appear to be an independent risk factor for stress UI. (LE 2)

10. Hysterectomy remains a possible risk factor for later UI, but the evidence is inconsistent. (LE 2)

11. Moderate to severe dementia in older women is a moderate to strong independent risk factor for UI (LE 2). Whether interventions to maintain or improve cognitive functioning also reduce UI has not been evaluated.

12. Mild loss of cognitive function in community-dwelling women, separated from physical function and other factors, increases the risk of UI slightly if at all, but may increase the impact of UI. (LE 2)

13. Data from twin studies suggests that there is a substantial genetic component to UI. (LE 1)

14. Other potential risk factors, including smoking, diet, depression, constipation, UTIs, and exercise, while associated with UI, have not been established as etiologic risk factors and are in fact difficult to study with observational data because of the potential for unmeasured confounding and questions of direction of the association. (LE 3).

VIII. FUTURE DIRECTIONS

Since the 3rd ICI in 2004, the quantity and quality of epidemiologic studies of UI has continue to increase. Most notable are the availability of prospective data from several studies that can examine risk factors for incident incontinence and a growing number of studies comparing the prevalence and incidence of UI among Caucasian, Black, Asian and Hispanic women using population-based samples and multivariate analysis. Below are several suggestions for research over the next 5 years.

- Obesity is now an established, modifiable risk factor for UI. Investigation and dissemination of strategies to reduce the risk of UI through weight control or reduction should be a priority.
- Poor physical function is a consistent risk factor for incontinence, particularly in the elderly. Whether or not it is modifiable is not clear. Intervention studies are needed to assess the impact of improvement of physical function on prevention or reduction of UI in frail elderly.
- Moderate to severe dementia is also a consistent risk factor for incontinence. Studies aimed to maintain or improve cognitive functioning should assess change in UI as an outcome variable.
- The higher prevalence and incidence of UI, particularly stress UI, among Caucasian women, compared to Black or Asian women remains unexplained. Further studies are needed to identify additional exposures or biological factors that could explain these differences.
- Determining the role of genes and identifying specific genes that increase the risk of UI is a daunting challenge. Nonetheless, laboratory and epidemiologic studies are needed to investigate this area.
- While the role of oestrogen in the aetiology of UI has been rendered largely moot due to the move away from estrogen use due to concerns about increased risk of breast cancer and cardiovascular disease, the use of other medications for control of menopausal symptoms and the introduction of selective estrogen receptor modulators (SERMs) is growing. Their impact on UI needs to be studied.
- More is now understood about the prevalence of and factors affecting treatment seeking among women with UI. However, more work needs to be done to identify women who would benefit from treatment, but who do not seek, or who do not receive, treatment and to develop interventions to help these women.

E. EPIDEMIOLOGY OF UI IN MEN

I. GENERAL COMMENTS

The epidemiology of UI in men has not been investigated to the same extent as for females. However, progress has been made during recent years, particularly in the reporting of population-based studies of urinary incontinence among men and more specifically, of urinary incontinence associated with prostatectomy. In addition, more reports have been published on the risk factors for the development of UI in men.

In almost all community based studies, the prevalence rates of UI continue to be reported to be less in men than in women by a 1:2 ratio. The type and age distribution of UI appear to be different between the sexes, and risk factors, although less investigated in men, seem to be different from women. It is also important not to consider UI as an isolated problem in men, but rather as a component of a multifactorial problem. Often other urogenital symptoms (LUTS) such as weak stream, hesitancy, and dribbling, or erectile dysfunction, exist.

Post-prostatectomy incontinence has been studied and reported with increasing regularity in the last few years. Since radical prostatectomy is being performed with increased frequency, and incontinence is one of the main complications of the procedure, a specific review of UI in the postprostatectomy patient population is presented in this section. In addition to epidemiological studies, we included clinical trial data on postprostectomy incontinence.

II. PREVALENCE

Several surveys from the general population have been conducted to determine the prevalence of UI in men (**Table 11**). Prevalences ranging from 1 – 39% have been published. The wide span of results may be explained by the variation in the population studied, the definition of incontinence used and the methods used in the surveys. A systematic review of 21 studies reported a prevalence of UI in older men ranging from 11-34% (median = 17, pooled mean = 22%). In the same review, the prevalence of daily UI in men ranged from 2-11% (median = 4%, pooled mean = 5%) [347]. A wide definition of UI, older age, inclusion of institutionalized men, and the use of self-reporting methods tend to result in higher prevalence rates [347-348].

For any definition of UI, there is a steady increase in prevalence with increasing age (**Table 12**).

1. INCONTINENCE SUBTYPES

Due to differences in pathological anatomy and pathophysiology of UI in men and women, there is a different distribution in incontinence subtypes. Recent studies confirmed our previous reports of the predominance of urge incontinence (40-80%), followed by mixed forms of UI (10-30%), and stress incontinence (<10%) [376] (**Table 13**).

The higher percentages of the urge and mixed types of incontinence are more significant in studies involving older people. In fact, the increasing prevalence of any UI by age in men is largely due to the contribution of urge incontinence rather than stress incontinence. One study demonstrated an increasing rate of urge UI from 0.7% between age 50-59, 2.7% between 60-69 and 3.4% for 70 years and older respondents. Stress UI was steady at 0.5%, 0.5% and 0.1% for the above groups respectively [356]. On the other hand, Maral and coworkers reported increasing prevalence also of SUI with age, from 0.9% between age 35-44, to 1.2% between 45-54, 3.8% between 55-64, and 4.9% at age 65 and older [353].

Most studies report a significant fraction of other/unclassified type of urinary incontinence. One study reported that a majority of men with UI had overflow and functional types of incontinence [354], while another found constant dribbling in 7% of their respondents [373]. Terminal dribbling or postvoid dribbling is another type of leakage in men that is difficult to assign to the conventional subtypes of UI. In an Australian survey, 12% of respondents reported frequent terminal dribbling [387].

2. SEVERITY

When it comes to severity, the distribution in men follows that of the women. Estimates for severe UI in older women tend to be about twice as high as for older men [376].

3. RACE-ETHNICITY

Very few studies have looked at the impact of race or ethnicity on the prevalence of UI among men. A four-country study presented lower prevalences of reported UI among men from Korea (4%) and France (7%) than in men from Britain (14%) and Denmark (16%) [349]. On the other hand, unpublished data from the MESA study did not indicate differences in prevalence among white male respondents compared to African American respondents.

4. INCIDENCE AND REMISSION

Literature on the incidence of male UI is very scarce. The MESA study [376] found a one-year incidence rate for men older than 60 years of 9-10%. In a population-based survey in the UK among men at least 40 years of age, the one-year incidence of UI was noted to be 3.8% [364]. A review of a health organization database of males at least 65 years old revealed an UI incidence of 23.8 per 1000 person years. Malmsten [360] analysed the age of onset of UI for each age cohort. Mean starting age for all men was 63 years. The mean duration was about 8-10 years in the cohorts.

Substantial remission rates for UI in males were noted by the MESA study, higher among men (27-32%) than women (11-13%) [376]. A similarly high one-year remission rate of 39.6% was noted among British males [364].

One possible explanation for the difference in the published incidence and remission rates in men compared to women, is the predominance of urge type incontinence among men, and its close relation to overactive bladder with and without incontinence. Another factor is the close association between urge UI and prostate gland disease, infections, or bowel dysfunction, all of which are relatively amenable to treatment or may improve even without treatment.

III. POTENTIAL RISK FACTORS FOR UI

There is relatively little research concerning conditions and factors that may be associated with UI in men, and clear risk factors are more seldom scientifically documented. However, a few available studies have identified potential risk factors, which are described below.

1. AGE

As in women, increasing age is correlated with increasing prevalence of UI (Table 12). Multivariate analysis in several studies has shown that age is an independent risk factor for incontinence [349, 378,

Table 11. Examples of prevalence studies of UI among men

A. General Population Sampling, all adult age groups

Author and year [ref]	N	Response rate (%)	Population (age)	Definition of UI used	Method of assessment	Prevalence (%)
Boyle 2003 [349]	4 979	28-72%	40-79	Lack of control over bladder function which caused urine leakage at times	Self-administered questionnaire	7 (France), 16 (The Netherlands), 14 (UK), 4 (Korea)
Engstrom 2003 [350]	?	86	40-80		Self-administered questionnaire	2 (SUI)
Van Oyen 2002 [351]	7 266	-	> =15			1.4
Schmidbauer 2001 [352]	1 236	-	Mean 49			5
Maral 2001 [353]	1 000	90	> = 15			1 (SUI), 3 (UUI)
Bortolotti 2000 [354]	2 721	-	≥ 50	Any urine loss in the last year	Telephone interview	3 32 (last year), 14 (weekly)
Smoger 2000 [355]	840	85	25-93, VA clinic	Incontinence in the past 12 months	Self administered questionnaire	32.3
Ueda 2000 [356]	3 500	52.5	> 40		Mailed self-administered questionnaire	10.5 (UUI)
Roberts 1999 [357]	778	-	≥ 50			25.6 (95CI 22.5-28.8)
Roberts 1998 [358]	2 150	-	≥ 40	Urinary leakage in the previous 12 months	self	18
Schulman 1997 [359]	2 499	-	≥ 30			5.2
Malmsten 1997 [360]	10 458	74	≥ 45			9
Brocklehurst 2003 [361]	1883	-	>=30	Ever suffered from bladder problems such as leaking, wet pants, damp pants	Interview	6.6% overall, 3.8% incontinent in the previous year, 2.8% in the previous 2 months
Irwin 2006 [362]	19165	33%	>=18	ICS 2002 definition	Telephone interview	5.4 (1.9-5.9)
Legace 1993 [363]	2830	86%	>-20	Any urine loss in the past 12 months	Self-administered questionnaire	11 (9-13)
McGrother 2004 [364]	92491	60.2	>=40	In the last year, did you ever leak urine when you don't mean to?	Postal questionnaire	14.2
OBrien 1991 [365]	2496	79			Self administered questionnaire	7.4 (95Ci 6.4 – 8.4)
Parrazzini 2002 [366]	9613	97.5	>=50	Involuntarily leaked in the past 3 months		8.3 (7.7-8.9)
Roc 1999 [367]	12529	53				5.3
Diokno 1986 [368]	805	65.1	60 older			

Table 11. Examples of prevalence studies of UI among men (Continued)

B. General Population Sampling, Older Group

Author and year [ref]	N	Response rate (%)	Population (age)	Definition of UI used	Method of assessment	Prevalence (%)
Dios-Diz 2003 [369]	350	-	> 64	-	-	? (95CI: 15-28)
Stoddart 2001 [370]	1 000	79	> 65	Incontinence in the previous month		23
Aggazzotti 2000 [371]	893	90	> 65, Community and residential homes	Involuntary loss of urine at least 2x/month	Questionnaire, review of clinical record	39.2
Gavira-Iglesias 2000 [372]	827	-	≥ 65	-	-	29 (25-38 95CI)
Smoger 2000 [355]	840	85	25-93, VA clinic	Incontinence in the past 12 months	Self administered questionnaire	32.3
Damian 1998 [373]	589 (including women)	78	≥ 65	Current experience of difficulty in controlling urine or urine escaping involuntarily	Interview	15
Umlauf 1996 [374]	1 490	53	Elderly	Uncontrolled urinary leakage of any amount the month before	Mailed self administered questionnaire	29
Nuotio 2003 [375]	171	-	≥ 70			24 (UUI)
Herzog 1990 (MESA study) [376]		66% - 72%	>=60	In the past 12 months about on how many days have you lost any urine, even a small amount beyond control	Interview	18.9%
Janssen 2007 [377]		57%	>= 65	Leaked or lost control of urine in the past year	Interview	13.1%
Landi 2003 [378]	5372		>= 85	MDS urinary incontinence scale of >=1	Health care professional assessment	49%
Thom 1997 [379]	1420	NA	>=65		Review of database	5.3

SUI: Stress UI, UUI: Urge UI, MUI: Mixed UI

Table 12. Examples of prevalence of UI across age spectrum in men

Author and year [ref]	N	Distribution by age	Prevalence (%)
Yarnell, 1979 [380]	169	65	9
		70 – 80	8
		80+	22
Thomas, 1980 [381]	?	45 – 54	5
		55 – 64	9
		65 – 74	15
		75+	18
Diokno,1986 [103]	805	60+	19
Malmsten, 1997 [360]	10458	45	3.6
		50	4.1
		55	3.3
		60	5.1
		65	6.1
		70	7.3
		75	9.6
		80	19.7
		85	21.8
		90+	28.2
Schulman, 1997 [342]	2499	50 – 54	5
		60 – 64	6
		70+	14
Bortolotti, 2000 [211]	2721	51 – 60	2
		61 – 70	3
		70+	7
Ueda, 2000 [356]	3500	40 – 59	2
		60 – 69	4
		70+	4
Aggazzotti, 2000 [371]	839	<65	19
		65 – 74	23
		75 – 84	52
		85+	53
		>= 95	57
Temml, 2000 [382]	1236	20 – 39	2
		40 – 59	4
		60 – 69	8
		70+	12
Smoger, 2000 [355]	840	≤40	25.4
		41 – 50	30.9
		51 – 60	31.4
		61 – 70	36.3
		71 – 80	33.2
		>80	20.0
Mariappan 2006 [383]	353	40-49	6.6
		50-59	7.9
		60-69	10.6
		>=70	10.3
McGrother 2004 [157]	92491	40-49	7.4
		50-59	11.1
		60-69	16.8
		70-79	23.2
		>=80	30.5
Obrien 1991 [365]	2496	35-44	2.4
		45-54	5.5
		55-64	5.7
		65-74	12.1
		>=75	15.4
Thom 1997 [379]	1420	65-74	2.8
		75-79	5.6
		>=80	7.6

Table 13. Relative proportion of types of urinary incontinence in men

CITATION	Population	Age group	UUI	SUI	MUI	Others
Diokno 1986 [368]		>= 60 y	34.9	7.9	28.9	28.3
Damian 1998 [373]	589	>=65 y	52.2	10.6	16.1	21.1
Chaojie 2002 [384]	2087 (total)	>=70 y	17.4	11.9	30.4	20.7
Nuotio 2003 [106]	171	>70	70.8	8.3	25.0	
Irwin 2006 [362]	19,165	>=18 y	22.2	11.1	11.1	53.7
Herschorn 2007 [386]	482	>=18 y	58	27	15	-

388-390] Compared to women, however, there seems to be a more steady increase in prevalence in men with increasing age.

2. LOWER URINARY TRACT SYMPTOMS (LUTS) AND INFECTIONS

In postal and telephone surveys of community-living incontinent men, a majority has experienced a variety of other medical conditions, many of which may cause or aggravate UI. LUTS like urgency, nocturia, feeling of incomplete voiding and reduced flow are typically associated with UI [352,368, 394]. In one study, UI was reported by 15% of men without voiding symptoms, frequency or urgency and by 34% of those with such symptoms [368].

Studies have also reported that urinary tract infections and cystitis are strongly associated with male UI [356, 373], with an odds ratio of 3.7 for UI in men reporting cystitis [356] and an odds ratio of 12.5 among men with recurrent infections [354]. It should be noted that reports indicating a positive association between UTI and incontinence involved men aged older than 60 years.

3. FUNCTIONAL AND COGNITIVE IMPAIRMENT, PHYSICAL ACTIVITY

Mobility problems such as use of a wheelchair or aids to walking, as well as diagnosed arthritis or rheumatism or having a fall during the last year, were significantly greater among incontinent than continent men [373, 395]. A Japanese study of community dwelling men noted that UI is more likely among men whose activities of daily living (ADL) are impaired, specifically those who are unable to change clothes and unable to walk outside, with odds ratio of 17.4 and 4.36 respectively [356]. A Canadian study found odds ratios of 1.8 and 6.4 for partially and totally immobile men aged 65+, respectively, for daily UI compared to those with normal ambulatory function. Similarly, the Silver Network Home Care project among the frail older persons in Italy showed that those with higher ADL scores (i.e., greater functional impairment) had 2-4x higher odds of having UI. A survey of nursing home residents in Wisconsin identified dementia and poor ADL as risk factors for the occurrence of UI [390]. In general, most studies find similarities between men and women (see subsection on women) for functional and cognitive impairment as risk factors for UI.

Corollary to this, the association between physical activity and UI has been studied by Kikuchi and co workers among the elderly, community-based population in Japan [391]. They found that men with middle level physical activity was associated with a lower UI prevalence compared to those with low level physical activity, with as odds ratio of 0.38 (0.17-0.78). High level physical activity showed similar relations but was not statistically significant.

4. NEUROLOGICAL DISORDERS

Many specific neurological diseases may lead to UI [392]. Neurogenic detrusor overactivity is seen commonly in mengingo-myelocele patients and in spinal injuries, Parkinson's disease and multiple sclerosis. Underactive bladder dysfunction due to a cauda equina lesion or diabetes might cause overflow or a paralysed pelvic floor and hence stress incontinence. Men who have suffered a stroke were at increased risk for incontinence with an odds ratio of 7.1 [356]. A case control study of age-matched long-term stroke male survivors with controls showed a higher prevalence of UI among stroke survivors compared to controls (17% vs 9%). In addition, among UI sufferers, the stroke survivors were found to have higher frequency and more leakage than controls. In a study of 235 stroke patients, the occurrence of UI correlated with motor weakness (OR 5.4), visual defects (OR 4.8, and dysphagia (OR 4.0).

5. PROSTATECTOMY

A well known iatrogenic cause of male incontinence is prostatectomy, but we do not know the attributable risk for this factor in the population of men with UI. In a Norwegian survey of elderly men with UI almost a third had undergone prostatectomy. [394].

In a cross sectional study among men in Vienna, Schmidbauer and associates identified previous prostatectomy to be associated with UI [352].

TURP seems to be followed by an incidence of stress incontinence of about 1%. A randomized controlled trial

comparing TURP, laser prostatectomy and evaporization of the prostate for benign disease showed comparable incontinence rates immediately and up to 12 months postoperatively [396].

Radical prostatectomy seems to induce UI at a much higher rate than TURP. The overall prevalence of post- radical prostatectomy incontinence ranges from 2 to nearly 60% (**Table 14**). This wide range may be explained by many factors, including differences in study characteristics, population characteristics, study site, the definition used, and the timing of assessment of continence in relation to the surgery. The era in development of the procedure has also been found to be associated to the prevalence rates [411], as well as the various procedural modifications of the surgery (see below).

Post-prostatectomy incontinence rates elicited from symptoms reported by patients are generally 2-3x higher than those from physicians' observations. Studies that have performed both assessments in the same population confirm this observation that doctors underestimate postprostatectomy incontinence by as much as 75% [419,422-424].

Incontinence rates after prostatectomy seem to steadily decline with time and plateaus 1 - 2 years after surgery [400, 404,407-410] (**Table 15)**. This emphasizes the need of a long follow-up period to establish continence status postprostatectomy. An actuarial study among 647 postprostatectomy men estimated UI rate of 13% at one year and 7% at two years postsurgery [425].

The **technique of radical prostatectomy** impacts on UI rates. Modifications associated with lower UI rates include the perineal approach [416, 418] and preservation of neurovascular bundle [431-433]. Bladder neck preservation affords earlier return to continence compared with bladder neck resection, but with similar UI rates after one year [405, 434]. One study showed earlier recovery of UI after tennis racquet reconstruction and bladder neck preservation compared with bladder neck resection with puboprostatic ligament preservation, but with similar UI rates at one year [410,435]. However, continence status assessed more than 12 months after surgery showed even lower rates of UI after bladder neck preservation (11.6%) compared to resection (4.9%) in one study [410]. UI after laparoscopic procedures seems to be higher initially but approximates that of open techniques by one year [427].

Older age at time of surgery has been found to be associated with a higher prevalence of post-prostatectomy UI [398, 408, 418, 43-433], one study showed a doubled risk for every 10 years of age beginning at age 40 [406]. Another study suggested that rather than absolutely affecting final continence prevalence, elderly men need a longer time to achieve continence after surgery [407]. Two studies found no relation between age at surgery and UI [423, 436].

Other factors have been found to be associated with a higher prevalence of post-prostatectomy UI, although not consistently. Such factors include prior TURP, preoperative lower urinary tract symptoms, obesity, clinical stage, PSA, prostate volume and Gleason score [398, 432-433, 437-438]. A retrospective analysis of 156 patients who had undergone preoperative MRI of the prostate and were followed up post-prostatectomy showed that time to return to continence was associated with the variation in the shape of the prostatic apex. The prostatic apex that do not overlap with the membranous urethra was found to be significantly associated with an early return of continence [438 The 5-year cohort study of the Prostate Cancer Outcomes Study found that among prostatectomy patients, race and ethnic differences was related to urinary incontinence, with African-Americans having better recovery compared to non-hispanic whites and Hispanics [439].

Adjuvant radiotherapy has not been found to affect post-prostatectomy incontinence rates when assessed beyond 1 year [Formenti 2000, Hofmann 2003, Kundu 2004].

IV. FACTORS OF UNCLEAR ASSOCIATION WITH UI IN MEN

A 9 year study of Janssen (377) showed increasing rates of UI with increasing BMI among the older men and women. However, multivariate analysis failed to show increased BMI (overweight and obese levels) as an independent risk factor for the development of UI.

In a study including a younger population in Australia, obesity was noted to be associated with UI with an odds ratio of 3.2 (1.2-9.0) [389]. In this study, however, being merely overweight was not associated with UI.

Several studies in the older persons have shown an association between physical activity and UI among women that is not seen among men [349, 391].

Table 14. Examples of studies on the prevalence of post-prostatectomy incontinence

Author/Ref	Procedure	N	Follow up (months)	Definition	Prevalence (%)
Demirkesen 2007 [397]	RRP	72	>12	More than once a day leakage	8
Kundu 2004 [398]	RRP	2737	>= 18	Use of pads	7
Salomon 2003 [399]	RRP	205	12	Use of pads	34
Moinzadeh 2003 [400]	RRP	200	12-15	Use of pads	2 1
Maffezzini 2003 [401]	RRP	300			9
Deliveliotis 2002 [402]	RRP	149	12		6-8
Benoit 2000 [403]	RRP	25 651	12		8
Walsh 2000 [404]	RRP	64	12-18		7
Poon 2000 [405]	RRP	220	Mean >12		3-7
Catalona 1999 [406]	RRP	1 870	>12		8
Horie 1999 [407]	RRP	104	12	Use of pads	22
Goluboff 1998 [408]	RRP	480	12	Any UI Daily or pad use Continuous	57 7 1
Weldon 1997 [409]	RRP	220	18		5
Lowe 1996 [410]	RRP	180	12	Any protection	12.
Hu 2003 [411]	RP	12 079	> 36		4-20
Augustin 2002 [412]	RP		12	Any protection	27
Sebesta 2002 [413]	RP	674	> 24	Use of pads	32
Potosky 2000 [414]	RP		24		10
Arai 1999 [415]	RP	60	12	Use of pads	3-19
Bishoff 1998 [416]	RP	907			
Egawa 1997 [417]	RP	94	18	Use of pads	27
Gray 1999 [418]	RRP/RPP	209	Median 32		25
Olsson 2001 [419]	Lap RP	228	12	Use of pads	21.6
La Fontaine 2000 [420]	Lap RP	522	Mean 31	Use of pads	15
Galli 2006 [421]	Lap RP	150	12	Use of pads	8.3

RRP: radical retropubic prostatectomy
RPP : radical perineal prostatectomy
RP: radical prostatectomy, unspecified or combined
Lap RRP: laparoscopic retropubic prostatectomy

Table 15. Examples of studies on postprostatectomy UI rates at different times of assessment

			UI Rates by Time of Assessment of Continence					
CITATION	Population	N	1 mo	3 mo	6 mo	12 mo	24 mo	
Harris 2007 [426]		210	48	29	15	6	-	
Jacobsen 2007 [427]	RRP, Lap RP		-	42 (RRP), 70.4 (lapRRP1), 60 (lapRRP2)	-	12.8 (RRP), 20.7 (lapRRP1) 14.5 (lapRRP2)	-	
Galli 2006 [421]	Lap RRP	150	45	26.2	12.1	8.3	-	
Link 2005 [428]	Lap RRP	122	-	83.0[1] 49.0[2]	47.8[1] 10.1[2]	33.3[1] 6.6[2]	-	
Moinzadeh 2003 [400]	RRP		-	18	9	1.5	-	
Jonler 1996 [429]	RRP	24	87	67	63	-	-	
Penson 2005 [430]	RP	1213	-	-	33	18	15	

[1] – continence defined as 0 pads
[2] – continence defined as <= 1 pad daily

V. SUMMARY POINTS

- The epidemiology of UI in men has not been investigated to the same extent as for females. But it appears that UI is at least twice as prevalent in women as compared with men. There seems to be a more steady increase in prevalence with increasing age than for women (LE1).
- Most studies find a predominance of urge incontinence, followed by mixed forms of UI and stress incontinence the least. Most studies have a large fraction of other/unclassified types (LE 2).
- Literature on incidence and remission of male UI is still very scarce.
- Clear risk factors are more seldom scientifically documented, but several medical correlates have been reported. Established risk factors predisposing men to UI include increasing age, presence of lower urinary tract symptoms (LUTS), urinary tract infections, functional and cognitive impairment, neurological disorders, and prostatectomy (LE 2).
- UI after radical prostatectomy is frequent, ranging from 2-57%. Rates steadily decline from the time of surgery and plateaus at 1 to 2 years (LE 1).
- Factors affecting post-prostatectomy UI include the age at surgery, type of prostatectomy, and the modifications in the technique involving nerve and bladder neck preservation (LE 2).
- Comparative studies of surgical procedures and their various modifications should be performed to better assess their impact on postoperative continence rates.
- Substantial gains have been achieved on the study of the epidemiology of UI in men compared to the previous years. The conduct of more community-based prevalence studies and the closer analyses of post-prostatectomy incontinence permitted a better understanding of the problem of UI among men.

F. EPIDEMIOLOGY OF OAB

Overactive bladder (OAB) is defined as the presence of urgency and frequency (either daytime or nighttime), with or without urinary incontinence(UI) [1]. OAB is often divided into OAB without UI (OAB_{dry}) and those with OAB and UI (OAB_{wet}). To find studies looking for the prevalence of OAB a search was carried out on Medline with the strategy (((Overactive bladder.mp) or (OAB$.mp)) and ((prevalence.mp) or (incidence.mp))). The studies identified in the search were then scanned to be sure they were population based (i.e. not based on doctor attendances or similar). The references to the studies found were searched for further applicable studies. The studies often used different populations and ways of selecting samples, and the definition of OAB changed slightly, although this was standardised after the 2002 ICS standardization report. The results of these studies are summarized in the table.

The prevalence of OAB in adult males varies from 10.2% to 17.4% and in females from 7.7 to 31.3 (**Table 16**). Clearly different definitions, the different age groups studied and maybe cultural differences affect the rates (LE 2). OAB_{wet} was less prevalent than OAB_{dry} except for one implausible looking result. The response rate was quite low in many of these studies, and sometimes it was impossible to know the response rate (e.g. when quota sampling was used).

A common finding was that the rates increased with age, but this was not universal (LE 2). Some studies found that after adjusting for state of health then the relationship with age went away, and some found that it only applied to OAB_{wet}. Usually OAB was more prevalent in females, and this was very much more pronounced for OAB_{wet} which sometimes meant that OAB_{dry} was the same for males and females. OAB was related to general health problems and in particular diabetes. It is likely to be related to urinary problems in childhood.

Table 16. Prevalence (%) of OAB in different studies

First Author	Year	Age	N	OAB_{total}	Males OAB_{dry}	OAB_{wet}	OAB_{total}	Females OAB_{dry}	OAB_{wet}
Milsom [452]	2001	≥40	16776	15.6			17.4		
Chen [258]	2003	≥20	1253				18.6		
Stewart [453]	2003	≥18	5204	16.0	13.4	2.6	16.9	7.6	9.3
Corcos [88]	2004	≥35	3249	14.8	11.7	2.0	21.2	15.6	2.6
Castro [442]	2005	≥40	1669	17.4	—	—	25.6	—	—
Homma [446]	2005	≥40	4570	14	8	6	11	4	7
Temml [205]	2005	20–91	2418	10.2	8.4	1.8	16.8	10.3	6.5
Irwin [447]	2006	≥18	8139	10.8	6.9	3.0	12.8	6.2	6.3
Kajiwara [21]	2006	13,15	624	10.1	—	—	20.4	—	—
McGrother [157]	2006	≥40	19241	—	—	—	7.7	—	—
Teloken [206]	2006	15 55	848	14.0			23.2		
Zhang [456]	2006	≥20	4745	—	—	—	8.0	2.4	5.6
Choo [443]	2007	40-89	2005	20.8	13.3	7.5	31.3	16.3	15.0
Herschorn [445]	2007	≥18	1000	13.1	9.8	3.3	14.7	7.5	7.1
Lawrence [450]	2007	25-84	3877	—	—	—	13.4	—	—
Coyne [662]	2008	≥40	30 000	25.9 (≥ sometimes 14.9 (≥ often)			42.3 (≥ sometimes 32.1 (≥ often)		

G. EPIDEMIOLOGY OF FAECAL INCONTINENCE

I. GENERAL COMMENTS AND DEFINITIONS

Faecal Incontinence (FI) is the involuntary loss of faeces – solid or liquid. Anal Incontinence (AI) includes these events as well as the involuntary loss of flatus, which is felt by many patients to be an equally disabling disorder. The discussion below will therefore focus on the broader definition: AI. A third cause of soiling or embarrassment is anal mucoid seepage, a condition that cannot be deferred by an able sphincter and intact cognition, most often caused by an organic colonic disease or dietary sensitivity, and more rarely by faecal impaction. This is the loss of fluid, sometimes feculent, often following a normal continent defecation. This is an important condition to distinguish from true incontinence because authors that report very high prevalence rates of AI include leakage in their questionnaires and thus may include these individuals with this very common symptom. However in this case there is no detectable sphincter abnormality [457] and it is not treatable by any of the standard therapies for true incontinence: sphincter repair, neuromuscular re-education or even faecal diversion. It is in fact why we wear underclothes.

Older reports of AI prevalence have come from single institutions, and the patients described therein have been subject to referral bias when demographics and aetiology are discussed. The accuracy of AI prevalence estimates may also be diminished by difficulty in ascertaining those figures due to the common underreporting of AI and patients' reluctance to report symptoms or to seek treatment [458-459]. It has been shown that women are more willing to report AI than men [460]. In addition, the character (incontinence of solid faeces, diarrhoea, or flatus, or merely anal seepage) and frequency (daily versus episodic) of reported AI varies greatly in each report, and indeed between individuals. So, prevalence depends heavily on the definition of AI.

• Data Sources and Level of Evidence

Since ICI 3, New studies were sought using Medline and EMBASE using the search terms faecal, faecal, anal, incontinence, epidemiology. In addition systematic reviews were specifically sought in Medline, EMBASE and the Cochrane Library.

Because therapeutic interventions are not the subject of this chapter, and so the epidemiology is descriptive and not derived from randomised clinical trials, the level of evidence will be at best 2, and the strongest evidence will come from systematic reviews in which there was a predefined search strategy and application of quality assessment tools that were designed specifically to minimise bias in referral or ascertainment.

II. PREVALENCE

1. ADULTS

In an effort to resolve the widely varying reported prevalence figures (**Table 17**), two systematic reviews of the published frequencies have been done of community dwelling adults (above age 15 in the second). A summary frequency was not calculated in the first because of the marked clinical heterogeneity between reports (**Table 18**). The three reports that the authors judged most free of potential biases had frequencies between 11% and 15%, though only one of these three used a validated assessment instrument [461]. The degree of disability present in these 11%-15% is not known, nor even if a portion of them had only anal seepage. These high prevalences were obtained in surveys that employed anonymous self-administered questionnaires, which may not allow objective confirmation of AI or assessment of degree of disability associated with AI. The second systematic review found a range of solid and liquid anal incontinence of 0-15.2% for solid and liquid faeces, with an average across both genders and all age groups of 4.3% [462].

2. CHILDREN

The reported prevalence of AI in children can be broadly divided into two facets: in those children born with congenital anomalies of the anus and rectum – either congenital aganglionosis (Hirschprung's Disease) or imperforate anus – and those children without congenital anomalies. Among those children and adults who were born with defects, despite surgical correction of the defect, life long defaecation difficulties are common, occurring in roughly half of affected children [463-465]. Problems with psychological health and development because of the defaecation disorder is also common in this group, as is a generally depressed quality of life [466]. These disorders are not horribly rare, occurring in 3 to 5 per 10,000 live births [467].

Among children without congenital defects of the anal canal, bowel control has been found to be complete in one Swiss cohort in 33% by age 1 year, 75% by age two and 97% by age three. Nevertheless in this longitudinal study, a quarter of the boys and one tenth of the girls had a major period of incomplete bowel or bladder control between the ages of 6 and 18. At least annual encopresis occurred in 2-3% of these children, boys more frequently than girls [468]. In the Wisconsin Family Health Survey the prevalence of AI in children from the ages of 5 and 16 years was 12/1367 (0.88%) with the gender distribution being 7 boys and 5 girls (Wisconsin Family Health Survey: unpublished data). The common disorder for all children and then adults in this discussion is faecal retention with overflow and seepage.

III. INCIDENCE

Clinical trials have provided incidence data after a therapeutic interventions, but usually without a preliminary continence assessment. This is best seen in two Cochrane reviews of therapy for anal fissure [469-470]. AI incidence rates varied widely from 0% to 30%, to flatus only, and the duration was unspecified in the trials. Medical therapy was less likely than surgery to cause AI (0.23, 0.02-2.1), and certain operations (anal stretch) were more likely to cause AI than others (sphincterotomy) (4.2, 1.9-9.4).

IV. RISK FACTORS

1. AGE

Two systematic reviews have analyses of the association of age and anal incontinence and found age to be the most significant of all assessed associations [462,471].

2. GENDER

Most discussions of the aetiology of AI have been based upon the assumption that women, particularly for individuals under the age of 65 years, are far more at risk for AI than men. Injury to the pudendal nerve or sphincter muscle from prior obstetric trauma is described as the primary risk factor [472-474], followed by irritable bowel syndrome (a disease thought to be more prevalent in women) [475], and other aetiologies such as diabetes a distant third [476]. Yet each population based-survey of the prevalence of AI has shown a surprisingly high prevalence in males (Table 17) [459-460, 477-508]. Of the two systematic reviews that looked specifically at prevalence, only one assessed the role gender played and in that review gender was not associated with incontinence in any age group [462]. Clearly, aetiologies other than childbirth must be sought.

3. OBESITY

Three recent reports have demonstrated an increased risk of AI in obese women, a Kaiser cohort, a cross sectional survey in a specialty clinic and a case control study [508-510]. One longitudinal study found a reduction in anal leakage (again not necessarily a

Table 17. Population-based Surveys of Prevalence of Anal Incontinence

COUNTRY (ref)	POPULATION	N	PREVALENCE
U.K. [459]	Community Service	4 844	1.9%
France [478]	All >45 years	1 100	11%, 6% to faeces, 60% are women
U.S.A. [476]	Market mailing	5 430	7% soiling, 0.7% to faeces
U.S.A. [460]	Wisconsin households	6 959	2.2%, 63% women
Australia [475] 15	Household survey	3 010	6.8% in men, 10.9% in women >age
Germany [481]	>18 years	500	4.4%-6.7% (by health)
Australia [480]	>18 years	618	11-20% (gender M>F)
Australia [482]	>18 years	651	11.3%
New Zealand [484]	>18 years old	717	8.1% for solid and higher for gas
U.K. [485]	>40 years	10 116	1.4%
U.K. [486]	Postpartum women	549	5.5%
Canada [487]	Postpartum women	949	3.1% solid, 25.5% flatus
Denmark [488]	Postpartum women	1 726	8.6% in past year, 0.6% to solid stool
Nigeria [489]	Gynecology patients	3 963	6.9%, 2.3% to solid stool
United Arab Emirates [490]	Women multips	450	11.3%, 5.5% to solid stool
Canada [491]	Teenage females	228	3.5% flatus, 3% FI
Czech Republic [492] community	Gynecology patients	2 212	5.6%, 4.4% in the
Japan [493] ureterosigmoidostomy	Cystectomy patients	28	60.7% post
Sweden [494]	Prostate cancer	864	RR 1.3-4.5
Australia [495]	Diabetics	8 657	Increased risk
Holland [496]	Women >60 years	719	4.2% to 16.9% with rising age
U.S.A. [497]	>65 years at home	328	3.7% (M >F)
Japan [498]	>65 years at home	1 405	6.6-8.7% (by age).
U.S.A. [499]	>50 years	1 440	11.1 – 15.2% (F > M)
U.K. [500]	>65 years at home	2 818	3%
Holland [501]	>60 years	3 345	6%, (M = F)
Czech Republic [502]	Nursing homes	1 162	54.4%
U.S.A. [503]	Nursing homes	18 170	47% FI
Canada [504]	Nursing homes	447	46% FI, 44% both UI and FI
France [505]	>18 years	713	30% response rate. 11% gas,0.4% feces, Women>men.
U.S.A. [506] onset 55	Women >20 years	2 800	53% response rate. Median age years. Urgency.
France [507] includes	Women >50 years	2 640	85% response rate. 9.5% FI, but leakage.
U.S.A. [508] Obesity.	Women >25 years	4 103	37% response rate. 25% AI.

Table 18. Systematic Reviews Related to Anal Incontinence

Population	Question	Quality Assessment	Findings
Community Dwelling Adults [461]	Prevalence	Yes	11-15% among 3 highest quality reports
Community Dwelling Adults > 15 [462]	Prevalence and Age and Gender Assessment	Yes	4.3% Overall; 0.8% in men under 60 years to 6.2% in women over 60 years. Age only significant predictor of risk. Gender not significant. See Figure 10
Post Partum Women [471]	Differential risk of Incontinence in Women having Cesarean Delivery and Women having Vaginal Delivery	Yes	Odds Ratio and 95% Confidence Intervals for Fecal Incontinence: 0.98, 0.79-1.22. For Flatus; 0.99, 0.86-1.14. For Emergent vs. Elective CD; 0.98, 0.68-1.51. For Elective CD vs. VD; 0.73, 0.52-1.03 See Figures 7,8,9
Decision Analysis [540]	Elective Cesarean Delivery to Prevent Anal Incontinence in Macrosomia	No	Cost Saving of Elective CD estimated to be $32/CD. NNT 539, CDs to prevent a single case of AI

direct correlate with incontinence) in women after bariatric surgery and weight loss, though other factors including dietary and activity change may have been responsible for the improvement [511].

4. CHILDBIRTH AND MODE OF DELIVERY

A meta-analysis of published reports that assessed anal sphincter integrity after vaginal delivery and correlated this with continence stated that 77%-83% (depending on parity) of anal incontinence in parous women was due to sphincter disruption [512]. Three things are implied by this conclusion: first, that incontinence in men, children, of elderly onset (or even in middle aged women) and in nulliparous women, or women having Caesarean section has a completely different cause than in women who have ever delivered vaginally. There is scant epidemiologic evidence that this is the case [513]. Second, it is implied that sphincter repair would be effective treatment for anal incontinence in almost all parous women. Yet repair of disrupted sphincter has less than a perfect track record. Even more importantly, there is a reported rapid decay in function after repair that is far too great to be explained by age alone [515-522]. Third, if direct trauma to the anal sphincter (and not intra-pelvic nerves) were the major cause of anal incontinence, then Caesarean section should be effective in preventing incontinence (**Figs 6 & 7**).

However a systematic review has shown that this is not the case [471]. Seventeen reports have been found eligible for inclusion in the review, encompassing 16,036 women having had 3,101 Caesarean deliveries and 12,935 vaginal births as the index event prior to anal continence assessment . None of these reports demonstrated a significant benefit of Caesarean section in the preservation of anal continence. The greater the quality of the report, the closer its Odds ration approached 1.0. There was no difference in continence preservation in women have emergency versus elective Caesarean section. Among the seven best studies, the NNT is 339, i.e. 339 Caesarean section would have to be performed to prevent a single case of fecal incontinence. Surprisingly, in the only reported cohort that included nulliparous women, when compared to nulliparous women, there is an increased risk of anal incontinence in women who have had Caesarean section [480] that approaches that of women having vaginal delivery (**Figures 6,7,8**). Forrest plots demonstrate the meta-analyses of the 7 best studies providing data on post partum fecal incontinence (quality in this instance being defined as age and parity adjustment of data, no prior vaginal delivery in the Caesarean section cohort, and assessment a long enough time after parturition to allow for perineal healing), comparisons of elective versus emergency Caesarean section and elective Caesarean section versus vaginal delivery. Summary data are presented in **Table 18.**

But why doesn't Caesarean section prevent anal incontinence, especially when associating perineal trauma with loss of bowel control is not just intuitive, but sometimes visibly obvious? Certain aspects of vaginal delivery are clearly causally related to anal incontinence: significant laceration, forceps, and some episiotomies [523-524]. However this review demonstrates that other factors need to be explored. So one must look to pregnancy and not just labour and delivery as an initiating factor. Further evidence in

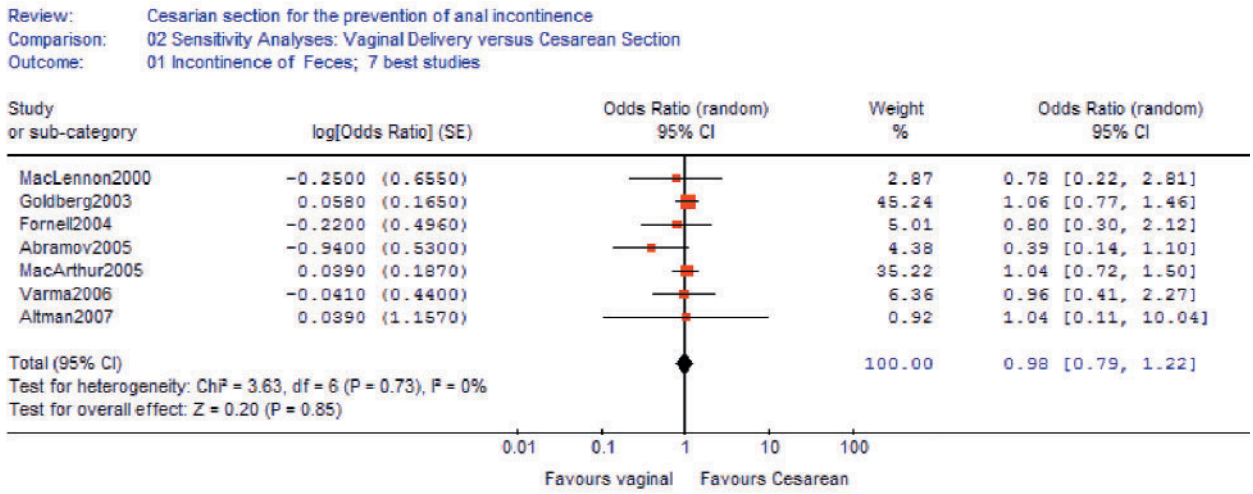

Figure 6 : Seven of the published studies that compare fecal incontinence rates after C.section compared to vaginal delivery that are adjusted for age, parity, primipara or prior C.section only and have adequate follow up to allow for perineal healing.

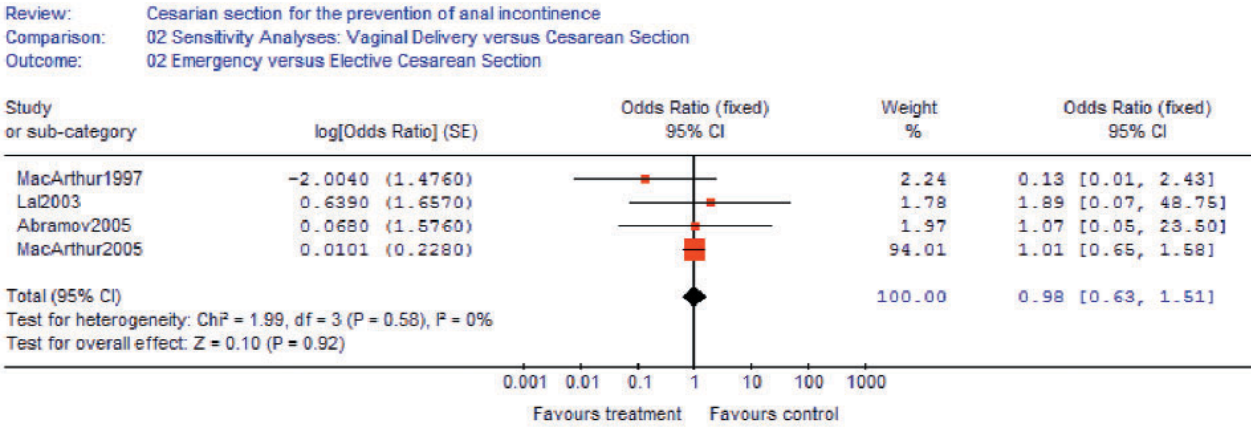

Figure 7 : Studies that provide data allowing comparison of fecal incontinence rates in women having elective versus emergent C. section.

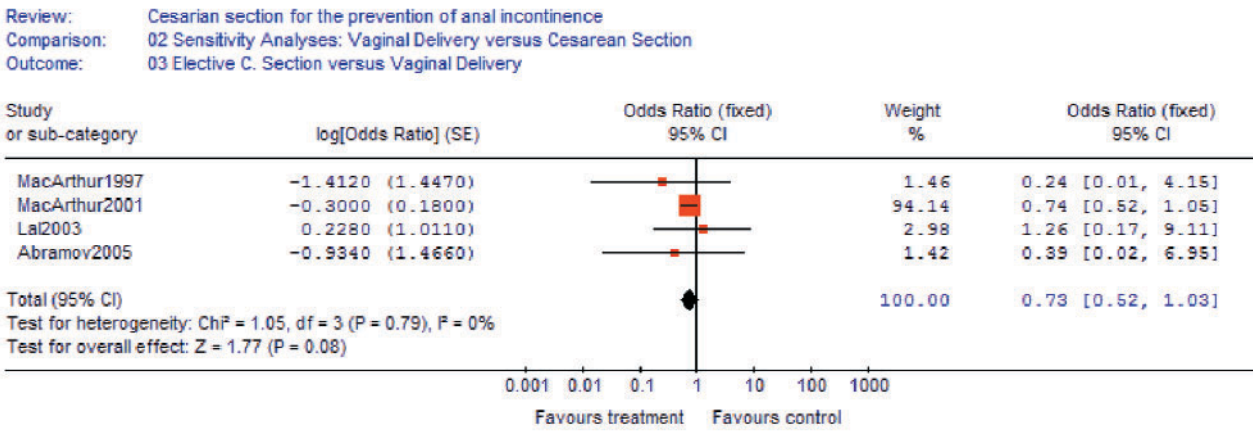

Figure 8 : Studies with data that allowed comparisons of fecal incontinence rates in women have only elective C.section versus vaginal delivery.

favour of this comes from the sphincter repair literature cited above. The rapid decay in function suggests that another defect is present besides a gap in the sphincter that remains after the early effects of sphincter repair wear off. What this is is not yet known, though trauma at the pelvic inlet during pregnancy or in early labour [525] seems likely.

Indirect evidence for the possibility that injury higher in the pelvis may be related to AI in pregnant women can be found in the association of hysterectomy with AI, an association seen more prominently with abdominal hysterectomy (TAH) than vaginal hysterectomy (VH), and for flatus only [526] (Odds Ratio of TAH vs. VH for faeces: 1.2, 0.3-4.7, Odds Ratio for gas: 18.9, 1.1-327). Pelvic nerve injury during surgery is the postulated reason for this difference.

5. NURSING HOME RESIDENCE

The most prominent association with AI by far is nursing home residence. Whereas the prevalence of AI is probably around 2% to 5% for community-dwelling persons, and may rise with increasing age to greater than 10%, among nursing home residents the prevalence approaches 50% [502-504]. This is partly explained by FI being one of the most common reasons for nursing home admission. In a large survey of 18,000 Wisconsin nursing home residents, risk factors for fecal incontinence (FI) were directly observed by nursing home personnel [503]. Urinary incontinence (UI) was the greatest risk factor for FI (OR = 12.6, 11.5-13.7), followed by the loss of ability to perform daily living activities (6.0, 4.7-7.7), tube feeding (7.6, 5.6-10.4), physical restraints (3.2, 4.7-7.7), diarrhoea (3.3, 2.7-4.2), dementia (1.5, 1.4-1.7), impaired vision (1.5, 1.4-1.7), constipation (1.4, 1.3-1.6), fecal impaction (1.5, 1.1-2.1), stroke (1.3, 1.2-1.5) male gender (1.2, 1.1-1.3), age and body mass index. Inverse associations were noted with heart disease, arthritis and depression.

6. DIARRHOEA

The importance of diarrhoea of liquid stool in FI cannot be overemphasized. One case series noted that 51% of individuals with chronic diarrhoea were incontinent [458]. In the Wisconsin Family Health Survey of AI [560], 10 of the 25 subjects with FI lived in Milwaukee when the city experienced an outbreak of waterborne disease [527]. Non-infectious causes of diarrhoea must also be considered, including those initiated by leisure activities such as running [528].

7. SURGERY

AI originating from surgery would seem fairly insignificant in the general population, since prior anal surgery has not been an apparent risk factor in the larger surveys. Several operations nonetheless frequently can result in AI. Examples are midline internal sphincterotomy, lateral internal sphincterotomy, fistulectomy, fistulotomy, ileo-anal reservoir reconstruction, low anterior rectal resection, total abdominal colectomy, and ureterosigmoidostomy. The risk of lateral internal sphincterotomy for anal fissure causing AI was previously thought to be insignificant when compared to midline sphincterotomy, but a recent reappraisal of this operation has shown an AI risk may be 8% [529]. The risk of AI after fistulotomy has been reported to be as high as 18% to 52% [530]. New approaches to fissure and fistula have recently been developed specifically to lower this risk [530-531]. However incontinence after haemorrhoidectomy has also been reported to be as high as 33%, an operation in which no sphincter is divided [532]. This suggests either that division of the anoderm, not the sphincter may be affecting continence, or that the method of ascertainment used in published surveys is not accurate. Mixing urine and stool has been found to have a predictable effect on anal sphincter control, as does diarrhoea, in patients having uretero-sigmoidostomy after urinary bladder resection [493].

8. SPECIFIC NEUROLOGICAL AND OTHER DISEASES

Several specific diseases have been anecdotally associated with AI in case series, and mechanisms to explain the associations have been investigated [533]. Examples are diabetes, stroke [534-535], multiple sclerosis, Parkinson's disease, systemic sclerosis, myotonic dystrophy, amyloidosis, spinal cord injury, imperforate anus, Hirschsprung's disease, retarded or interrupted toilet training, procidentia, and any illness causing diarrhoea (HIV, IBD, radiation, infection). Many of these conditions directly affect patient mobility and ability to perform daily living activities or they cause diarrhoea or faecal impaction.

9. OTHER FACTORS AND PROSPECTIVE ASSESSMENT OF RISK FACTORS

Because of a paucity of clinical trials that specifically address risk factors and prevention of AI, the strongest available data to identify risk come from cohorts that collected data on potential risk factors prior to the onset of incontinence. The only prospectively collected risk assessments for FI have occurred in three nursing home cohorts. Porell combined UI and FI into a single outcome variable and found many positive associations in a cohort of 60,000 nursing home residents in Massachusetts [536]. Age, African American race, cognitive and ADL impairments predicted the outcome, though specific relative risks for incidence are not presented. Chassange followed 234 previously non-FI residents in France for 10 months, during which 20% had FI episodes, but only 7.5% developed long lasting FI [537]. The others had acute episodes due to diarrhoea or impaction. The factors associated with the development of long lasting FI were urinary incontinence (UI) (2.9, 1.8-4.6), decreased mobility (1.8, 1.1-3.0), and cognitive defects:

either as seen in an MMSE score <15 (2.5,1.4-4.4) of history of dementia (2.1, 1.2-3.5). Neither gender nor age were risk factors. Nelson reported, in a cohort of 18,000 nursing home residents in Wisconsin, a subgroup of 3,850 continent of both urine and faeces in 1992 and were assessed one year later [538]. 15% developed FI. Positive associations were seen for ADL loss (3.4, 2.4-4.5), trunk restraints (2.5, 1.7-3.6), dementia (1.7, 1.4-2.0), African American race (2.1, 1.3-3.4) and age (1.02, 1.0-1.0). UI was not investigated as a risk factor because it was felt to be a co-morbid condition.

Lastly, in a broadly based cross sectional survey, it was apparent that factors that affect an individual's general health or physical capabilities, independent of age and gender, place that individual at greatest risk for AI [475], though all four are significantly associated with AI [460]. Among obstetrical patients age has also been a consistent association, with less consistent associations noted for chronic bronchitis (OR =6.5, 1.1-38), symptoms of pelvic prolapse (5.0, 3.0-8.7) and obesity (3.0, 1.0-3.4) [539].

V. PREVENTION

This discussion is by necessity descriptive, so preventive measures are only relevant insofar as they provide insight into aetiology of incontinence. By far the most frequently applied preventive measure is Caesarean delivery, discussed above. Its lack of effectiveness in preventing anal incontinence provides a valuable insight into the relationship of pregnancy and AI – that the focus may need to be more on the pregnancy rather than the delivery and how it effects defecation afterwards. A decision analysis study suggests specific obstetrical indication for elective C. section that may be cost effective [540]. Another study related to birth trauma randomized mothers to immediate post-partum anal ultrasound with repair of occult defects in the sphincter and continence assessed in follow-up, demonstrating an improved outcome with this intervention [541].

The AHRQ recently published a monograph on prevention of incontinence, though the strategies listed for AI were therapies for existing AI, such as pelvic floor exercises and retraining, rather than established mechanisms for prevention [542].

VI. SUMMARY POINTS

- Anal and urinary incontinence commonly coexist, particularly in the elderly and in nursing home residents (LE 1).
- The prevalence of anal incontinence increases with age, but is present in all age groups and both genders varying from 1.5% in children to more than 50% in nursing home residents (LE 1).
- AI is almost as common in men as in women (LE 2).
- Mode of delivery does not seem to be a significant factor in the development of obstetric anal incontinence, i.e., AI develops after Caesarean delivery as often as after vaginal delivery (LE 2).
- Obesity is perhaps the most modifiable risk factor for AI (LE 2).
- As populations age, co-morbid disease becomes a significant component of fecal incontinence risk. Surgery, neurological diseases, and stroke are examples.
- Cognitive and ADL impairment are associated with fecal incontinence.
- More population based prevalence surveys have been published.
- More analyses comparing AI after Caesarean section and vaginal delivery have been published.
- Systematic reviews of prevalence, including the role of age and gender, Caesarean delivery and decision analyses for the application of Caesarean delivery in macrosomia have been published, providing needed aggregation of data with quality assessment of existing literature.

VII. FUTURE NEEDS

- Risk factors for AI in each age group are still poorly defined
- Prevention research, much less policy, are therefore still a great distance away.

H. EPIDEMIOLOGY OF POP

I. GENERAL COMMENTS AND DEFINITIONS

Pelvic organ prolapse (POP) refers to loss of support for uterus, bladder, colon or rectum leading to prolapse of one or more of these organs into the vagina. Prolapse is thus a continuous condition when measured by visual inspection of the vaginal wall during valsalva. For clinical purposes, the degree of POP is commonly described as above the introitus, at the introitus, or beyond the introitus with or without valsalva. The International Continence Society first developed a standardised definition for the condition of POP in 1996 [543]. The ICS Pelvic Organ Prolapse Quantification (POPQ) examination defines prolapse by measuring the descent of specific segments of the reproductive tract during valsalva strain relative to a fixed point, the hymen. The POPQ system describes the anatomic findings of pelvic organ prolapse without consideration for symptoms and bother perceived by the woman. Validation of this system has shown it to be highly reliable [544]. The stages of prolapse severity are arbitrarily defined, and there is no clear differentiation between normal anatomic variation and mild POP. For research purposes there is consensus for use of the POPQ system until further evidence might clarify the distinction between normal variation and mild prolapse [545].

Determining POP based on self-reported symptoms is difficult because of the lack of specificity and sensitivity of most symptoms attributed to pelvic organ prolapse [546] and the fact that prolapse above the level of the hymeneal ring is usually asymptomatic [547]. The only exception appears to be a sensation of bulging into the vaginal [6]which is most strongly associated with prolapse at or below the hymeneal ring [549-550]. A recent study of 110 women found that a question asking about a feeling of something bulging in or dropping out of their vagina had a sensitivity of 84% and a specificity of 94% for POP at or beyond the hymeneal ring on examination [547]. Seeing prolapse would presumably be even more specific, but is too uncommon to be useful as a definition.

II. PREVALENCE OF POP

Since the 3rd ICI, several additional studies have reported the prevalence of POP in a general population [551-556]. Reports from the Women's Health initiative (WHI) Oestrogen Plus Progestin Trial, and randomized controlled trial, have been included [551,553-554]. While not actually population-based, the women in the trial were recruited from the community rather then from women seeking gynaecological care, and provide important information on the prevalence of POP based on pelvic examination.

The prevalence of POP based on a sensation of a mass bulging into the vagina was remarkably consistent, ranging between 5 and 10 percent. The study by Eva et al., which reported a substantially higher prevalence included in the definition of POP, pelvic heaviness or digital pressure on the perineum or in the vagina to aid with defaecation [557]. The prevalence of observed prolapse in women enrolled in the WHI trial is similar to the prevalence found in the one population-based study that also used pelvic examination [558], although the prevalence of each type of prolapse was higher in the WHI study [550]. In both studies, prolapse occurs most frequently in the anterior compartment, next most frequently in the posterior compartment, and least in the apical compartment (**Table 19**).

Two studies that examined prolapse by race found the that Black women had the lowest prevalence and Hispanic women the highest after controlling for multiple other factors in multivariate analysis [550,556]. The study reported by Rortveit et al based on symptoms found adjusted odds ratios of 0.4 (95% CI=0.2-0.8) for Black and 1.3 (95% CI=0.8-2.2) for Hispanic women, with White women as the referent group [556] [20]. Hendrix et al reported adjusted odds ratios of 0.6 (95% CI=0.5-0.8) for Black and 1.2 (95% CI=1.0-1.5) for Hispanic women compared to White women for POP based on genital examination [551].

III. INCIDENCE

Only two studies could be located that reported the incidence of new POP. Both studies were done on sub-groups of women enrolled in the WHI Oestrogen Plus Progestin Trial. The first study of 412 women enrolled at the University of California, Davis site, used a standardised pelvic examination repeated every 2 years over 8 years [551]. The incidence of new cystocoele, rectocoele and uterine prolapse was 9%, 6% and 2%, respectively. Annual rates of remission from grade 1 (prolapse to above introitus) was relatively common for each type of POP (24%, 22% and 48%, respectively) but less common from grade 2 or 3 (prolapse to or beyond introitus) (9%, 3% and 0%, respectively). In a second study of 259 women post-menopausal women with a uterus were examined using the POP-Q at baseline and annually for 3 years. POP was defined as prolapse to or beyond the hymeneal ring. The incidence of new POP was 26% at 1 year and 40% at 3 years, with remission rates of 21% at 1 year and 19% at 3 years [553].

Table 19. Prevalence of pelvic organ prolapse (POP) defined by symptoms or observed on pelvic examination in the general population

First author	Country	Definition of POP	Ages (years)	N	Prevalence Subgroup: %
Kumari [559]	India	”a mass of flesh in the vagina” or equivalent using local terminology	15+	2990	15-24: 5 25-34: 10 35-44: 8 45-54: 6 55-64: 9 65+: 3
McLennan (560)	Australia	A feeling of something coming down in the vagina	15-97	1546	8
Tegerstedt (555)	Sweden	Validated 5 item quetionnaire	30-79	5489	8
Eva [557]	Sweden	Any symptom of pelvic heaviness, genital bulge, or use of fingers in vagina or on perineum for defecation	40 60	641 663	23 28
Samuelsson [558]	Sweden	Standardized pelvic examination	20-59 (mean=39)	487	Any prolapse: 31 To introitus: 2 Cystocele: 16 Rectocele: 14 Uterocele*: 5
Rortveit [556]	USA	Feeling of bulging, pressure or protrusion or visible bulge or protrusion	40-73 (mean=56)	2109	6
Lawrence [552]	USA	Sensation of bulge in vagina or something falling out of vagina with a degree of bother of at least 33 on a 1-100 visual analogue scale (validated)	25-84 (mean= 57)	4103	6
Hendrix [550]	USA	Standardized pelvic examination	50-79 (mean=63)	27,342	Any prolapse: 40 Cystocele: 34 Rectocele: 19 Uterocele*: 14
Handa [551]	USA	Standardized pelvic examination	50-79 (mean=63)	412	Any prolapse: 32 Cystocele any: 25 Cystocele grade 1: 14 Cystocele grade 2: 10 Rectocele any: 13 Rectocele grade 1: 8 Rectocele grade 2: 5 Uterocele any: 4 Uterocele grade 1: 3 Uterocele grade 2: 1
Nygaard [554]	USA	POP-Q**	50-79 (mean=68)	270	Stage 0: 2 Stage 1: 33 Stage 2: 63 Stage 3: 2 Stage 4: 0 ≥ hymeneal ring: 26
Bradley [553]	USA	POP-Q	50-79 (mean=68)	270	≥ hymeneal ring: 24

* Denominator is women with a uterus

** Stages defined as 0: no prolapse, 1: prolapse to 1 cm above hymen, 2: prolapse to between 1 cm above and 1 cm below hymen, 3: prolapse between 1 cm below hymen and 2 cm above introitus, 4: prolapse beyond 2 cm above introitus.

Note: Studies reported by Handa, Nygaard and Bradley are all subsets from study reported by Hendrix.

Several studies have reported the annual incidence of surgery for POP in the US and at least one in the UK. A longitudinal study of over 17,000 women in the U K, age 25 to 39 at baseline, reported an annual rate of prolapse surgery of 0.16% [561]. This rate is consistent with the rate of approximately 0.2% per year reported in the US [562-563]. One US study reported an annual incidence rising with age from 0.05% in women age 30-39 to 0.5% in women age 70-79 with an estimated lifetime cumulative risk of surgery from prolapse of 7% to 11% [564]. A recent US study reported similar surgical rates: 0.07% for women 18-39, 0.24% for women age 40-59, and 0.31% for women age 60-79 [565]. Surgical rates drop substantially after age 80 [564-565]. Estimating rates of prolapse surgery has the advantage of use of hospital discharge data on procedures, which is highly accurate for the procedure performed, but less accurate for the indications for the procedures, particularly when a procedure may have more then one indication.

IV. POTENTIAL RISK FACTORS

1. BOWEL DYSFUNCTION AND PELVIC ORGAN PROLAPSE

Bowel dysfunction is highly prevalent in women and comprises a wide variety of symptoms including constipation, rectal emptying difficulties, incomplete defecation, manually assisted defecation and faecal urgency. It has been estimated that up to 27 percent of the population in industrialised countries is affected by constipation. The overall prevalence of constipation and associated symptoms in women with pelvic organ prolapse range between 20-53% depending on definition of disorders[566-568]. Neurophysiological assessments have shown that damage to the pelvic floor musculature and nerve supply can occur in women with chronic constipation [569]. Although definitions of disease differ between studies, it is widely acknowledged that bowel dysfunction is a complex condition with a multifactorial aetiology. Predisposing factors comprise low socio-economic status, pelvic floor surgery, depressive disorders, thyroid dysfunction, physical disability and inactivity, and food habits [570].

Current epidemiological evidence on the association between bowel dysfunction and pelvic organ prolapse are at odds. A number of studies suggest that women with pelvic organ prolapse are significantly more likely to experience constipation and other symptoms of bowel dysfunction [568, 571-574], whereas others show a weak or non-existent association [567, 575-577]. In a case-control study, manually assisted defaecation was present in 19.7% of women with prolapse compared to 4.4% of control subjects ($p<0.001$) [568]. In a randomly selected populaton based study, irritable bowel syndrome and constipation were both strongly associated with pelvic organ prolapse (OR 2.8 95% CI 1.7-4.6, and OR 2.5 95% CI 1.7-3.7 respectively) [573]. Varma et al. [574], suggested that among randomly selected women, having symptomatic pelvic organ prolapse more than doubled the risk of obstructed defecation (OR 2.3 95% CI 1.5-3.7). A retrospective questionnaire based survey of women with and without prolapse concluded that constipation as a young adult was an important factor in the development of uterovaginal prolapse [571]. In a case-control study, women with prolapse were at increased risk of constipation also after adjustment for dietary fibre intake (OR 2.9, 95% CI 1.1-13.5). when compared to women without prolapse [572].

In the cross-sectional Women´s Health Initiative (WHI), cystocoele and rectocoele were only weakly associated with constipation (OR 1.1 95% CI 1.0-1.2) [575]. Similar weak associations between prolapse and bowel dysfunction have been observed in other large cross-sectional studies [567,576-577]. Overall severity and prevalence of bowel dysfunction has shown poor correlation with findings of pelvic organ prolapse on radiological imaging [578-580]. Also at clinical examination, increasing vaginal descent and prolapse severity, show a generally weak (or absent) association with symptoms related to bowel dysfunction [566, 581-584]. In a substudy of the WHI, no specific bowel symptom was associated with increasing loss of pelvic organ support in any vaginal compartment [577]. When considering compartment-specific pelvic floor defects, most studies suggest that increasing posterior vaginal wall prolapse and perineal descent are correlated more to symptoms of obstructive defecation [567, 580, 582].

2. PELVIC SURGERY AND POP

Even though the notion that hysterectomy increases the risk of pelvic organ prolapse has wide acceptance, longitudinal studies confirming a temporal association are few and previous studies often do not differentiate between various types of hysterectomy. A number of cross-sectional and retrospective studies implicate hysterectomy as an independent risk factor for pelvic organ prolapse. However, due to a delay of onset, large population samples and a sufficiently long duration of follow-up are required to determine an association with adequate statistical certainty.

In a nationwide prospective cohort study, Altman et al. [585] reported that 3.2% of women with hysterectomy had pelvic organ prolapse surgery, compared with 2.0% in non-hysterectomised controls, corresponding to a risk of 1.7 (95% CI, 1.6-1.7). In this study, vaginal hysterectomy had the highest risk for subsequent prolapse surgery (HR 3.8, 95% CI, 3.1 to 4.8) in comparison to non-hysterectomised controls. These data are largely in agreement with the longitudinal

Oxford Family Planning Association study by Mant et al. [586] reporting increased overall incidence rates for prolapse surgery following hysterectomy. Although not separating various hysterectomy techniques, Mant el al. determined that the risk of prolapse following hysterectomy was 5.5 times higher (95% CI 3.1-9.7) in women whose hysterectomy was performed for prolapse as opposed to other benign conditions. A history of hysterectomy has also been shown to increase the risk of prolapse in several cross-sectional and retrospective studies [587-588].

Specific risk factors for posthysterectomy prolapse have been assessed in two case-control studies. Both Dällenbach et al. [589] and Forsgren et al. [590] showed that pelvic floor surgery before hysterectomy was the strongest risk factor for developing posthysterectomy pelvic organ prolapse (OR 7.9, 95% CI 1.3-48.2 and OR 2.8, 95% CI 1.0-7.7 respectively). The risk of prolapse repair was 4.7 times higher in women whose initial hysterectomy was indicated for prolapse [589]. Vaginal vault prolapse involves the loss of vaginal apical support and can only occur after hysterectomy [591]. Marchionni et al. reported a 4.4% overall incidence of vaginal vault prolapse after hysterectomy but in women where uterine prolapse was the indication for hysterectomy the incidence was 11.6% [592].

It has also been suggested that other pelvic surgery may predispose women to subsequent genital prolapse including: rectopexy for rectal prolapse (OR 3.1; 95% CI 1.4-6.9) [593] ; gynaecological surgery in general (OR = 3.9, 95% CI 1.8-8.8) [594], and retropubic colposuspension procedures are associated with a near 30% risk of subsequent vaginal vault and posterior vaginal prolapse at long-term evaluation [595-596]. In a prospective cohort study of 374 women, the 10-year re-operation rate was 17% after traditional prolapse or incontinence surgery [597]. Having undergone pelvic organ prolapse or incontinence surgery prior to the index operation increased the risk of re-operation to 17% compared with 12% for women who underwent a first procedure (p=.04) [597].

3. GENETIC EPIDEMIOLOGY OF UI AND POP

The aetiology of female pelvic floor disorders is widely recognised to be multifactorial, yet the complex interaction between genetic predisposition and environmental influences is poorly understood. Evidence in support of a genetic influence on pelvic floor disorders derives from studies on familial transmission of disease, studies on ethnic group diversity and twin studies.

4. FAMILIAL TRANSMISSION

A large number of studies suggest that the risk for urinary incontinence “runs in the family” [598-603]. In the Norwegian Nord-Trøndelag health survey (EPINCONT), daughters of mothers with urinary incontinence had an increased risk of stress incontinence (RR 1.5, 95% CI 1.3 to 1.8), mixed incontinence (RR 1.6, 95% CI 1.2-2.0), and urgency incontinence (RR 1.8 95% CI 0.8-3.9) [600]. Although study methodology and the magnitude of the risk estimates vary, studies on familial transmission of incontinence are in agreement: having a first degree female family member with stress urinary incontinence increases the risk for an individual becoming afflicted by the same disorder [604].

There is far less evidence to support the familial transmission of pelvic organ prolapse. In a case-control study, Chiaffarino et al. [605] showed that in comparison with women whose mother or sisters reported no prolapse, the risk for prolapse was higher in women with mothers (OR 3.2 95% CI 1.1-7.6) or sisters (OR 2.4 95% CI 1.0-5.6) reporting the condition. In young women (≤45 years of age) who had been operated on for prolapse, the familial incidence of genital prolapse was about 30% [606].

It is, however, a common misunderstanding that familial aggregation of any pelvic floor disorder invariably is a result of genetic factors. Risk estimates derived from family members in most cases cannot distinguish between heritability and non-inherited (environmental) factors in the family environment. Familial environmental influences which may have a direct effect on transmission of risk for stress urinary incontinence and pelvic organ prolapse includes smoking habits, socio-economic status, care seeking behaviour, attitudes towards physical exercise, dietary and drinking habits, and toilet training.

5. ETHNIC AND RACIAL INFLUENCES

The variation of disease occurrence in groups of different racial origin yet similar environmental exposures, lend support to the presumed genetic influence on the causation of benign pelvic floor disorders. This again provides circumstantial evidence for a genetic contribution to pelvic floor disorders since most of these studies have been unable to control for heritability in relation to the complex interaction of environmental factors.

There is consistent evidence to suggest that Caucasian women are at increased risk of stress urinary incontinence when compared to African-American women [607-610]. In a consecutive analysis of women referred for gynaecological care, Caucasian race increased the risk for stress urinary incontinence by two-fold (OR 2.2. 95% CI 1.3-3.7) when compared to African-Americans [611]. Three studies report higher risk of symptoms associated with overactive bladder in African-American women when compared to Caucasians [608-609, 611].

In cross-sectional studies from the US, African-American ethnicity conferred a significantly lower risk (OR 0.4 95% CI 0.2-0.8) [573] and Asian-Americans

had a higher risk of pelvic organ prolapse when compared to Caucasians (OR 1.4 95% CI 1.1-1.9 [612]. In women presenting for routine gynaecological examination 67% of Asian-American patients had stage 2 or greater prolapse as compared to 26% of African-American and 28% of Caucasian patients [612]. In the WHI, Hispanic women had the highest risk for uterine prolapse (OR 1.2 95% CI 1.0-1.5) and African-American (OR 0.6 95% CI 0.5-0.8) the lowest when compared to Caucasians [575]. Also within racial groups, the risk may differ as shown when comparing prevalence of pelvic organ prolapse between tribes in rural Gambia [613].

6. TWIN STUDIES

By comparing monozygotic female twins with identical genotype, and dizogytic female twins who on average share 50 percent of their segregating genes, the relative proportions of phenotypic variance resulting from genetic and environmental factors can be estimated. A genetic influence is suggested if monozygotic twins are more concordant for the disease than dizygotic twins whereas evidence for environmental effects comes from monozygotic twins who are discordant for the disease.

Recent studies in twins have indeed suggested a genetic influence on the phenotype for pelvic floor disorders [614-616]. However, studies based on volunteers are liable to bias since pairs who are concordant for the disease, are more likely to participate [617]. Only recently have large-scale population based genetic epidemiological studies in twins become available which clearly suggest a genetic contribution to pelvic floor disorders while controlling for both shared and non-shared environmental risk factors [618]. For both stress urinary incontinence and pelvic organ prolapse the genetic variation in liability to develop surgically managed disease after adjusting for age and parity was estimated to be about 40%. Recognising the considerable environmental effects and that heritability estimates in twins are likely to represent the upper limit of the genetic effects, [619-620] the influence of genetic factors should not be overstated. The role of gene-by-environment interactions, i.e. that certain genes may increase the risk for pelvic floor disorders only when the individual is exposed to a specific risk factor, remains to be decided and further complicates attempts to determine the genetic effects [621].

7. CANDIDATE GENES

A number of candidate genes which may be involved in the heritability of pelvic floor disorders have been investigated. Polymorphisms of the collagen type I gene have been shown to increase the risk of stress urinary incontinence, [622] and knock-out of the nitric-oxide-syntase gene in mice results in voiding disorders [623]. Mutations in lysyl-oxidase-like-1 gene is associated with pelvic organ prolapse [624] ; ATP receptor $P2X_3$-deletion in mice increases bladder capacity and reduces urinary frequency; and polymorphisms of the endothelin-1 gene may be involved in pelvic organ prolapse [625-626]. Polymorphisms in the promoter segment of laminin gamma 1 gene increases the susceptibility for early-onset pelvic organ prolapse [627]. Other candidate genes are almost certainly waiting to be discovered and results are so far of a preliminary nature.

8. OBSTETRICAL FACTORS AND POP

The debate on the importance of mode of delivery and obstetrical events to the development of pelvic floor disorders later in life is largely unresolved. For ethical and practical reasons, randomised controlled trials to study the causal effects of vaginal versus caesarean delivery will never be performed. Observational studies will therefore most likely remain the main source of knowledge on this subject. Nonetheless it is widely accepted that childbirth is a significant risk factor for pelvic organ prolapse, presumably due to overt or occult pelvic floor tissue trauma. Controversy does, however, remain with regard to the protective effects of Caesarean section and if specific obstetrical events should be considered as risk modifiers. Due to a delayed onset of pelvic organ prolapse in relation to giving birth, studies on the subject need a long duration of follow-up as well as large study populations to be able to elucidate the possible causative events. Therefore, the majority of studies on the subject are typically designed as cross-sectional surveys or retrospective cohort or case-control studies.

Pregnancy in itself has been identified as a risk factor for stress urinary incontinence. With regard to pelvic organ prolapse, the association is less well substantiated. In a clinical case-control study, all 21 nulliparous non-pregnant women had POP-Q stage 0 or 1, whereas 47.6% of 21 nulliparous pregnant women had pelvic organ descent corresponding to stage II ($p<0.001$) [628]. Overall POP-Q stage was higher in the third trimester than in the first ($p=0.001$). Also Sze et al. [629] found that in 94 nulliparous women evaluated at the 36 week antepartum visit and six weeks postpartum, POP-Q staging increased.

A large number of studies identify childbirth as one of the strongest predictors for developing pelvic organ prolapse later in life [573, 575, 586, 588 629-633]. It is also a recurrent observation that number of deliveries is associated with the risk for prolapse. In the prospective Oxford Family Planning Association study [586], childbirth was the single strongest risk factor for developing prolapse in women under 59 years of age and the risk increased by every delivery. Similar findings derived from the WHI [575], where a parity of one conveyed an overall two-fold risk increase for prolapse compared to having no children, after which

each additional birth added a 10-20% risk increase. In a case-control study, Tegerstedt et al. [633] } found that the risk for symptomatic pelvic organ prolapse increased with number of deliveries and were 3.3-times higher among mothers of four than among mothers of one. Similarly Rortveit et al. [573] found that the risk for prolapse increased in women with one (OR 2.8 95% CI 1.1-7.2), two (OR 4.1, 95% CI 1.8-9.5), and three or more (OR 5.3, 95% CI 2.3-12.3) vaginal deliveries compared with nulliparous women.

Whether or not caesarean section provides sufficient protection to the pelvic floor, thereby preventing loss of pelvic organ support, is controversial. Several studies suggest that elective caesarean does indeed protect women from developing pelvic organ prolapse later in life [629-633]. In 4,458 randomly selected women, vaginal childbirth increased the risk for prolapse by 1.82 (95% CI 1.04-3.19) [631]. In a nested case-control study, Uma et al. [632] found that Caesarean section was associated with a significantly reduced risk of pelvic floor surgery compared with spontaneous vaginal delivery (OR 0.16, 95% CI 0.05-0.55). In a case-control study, Chiaffarino et al. [605] found that women who were delivered by Caesarean section were at significantly lower risk for prolapse (OR 0.3 95% CI 0.1-1.0). Other investigators suggest that in the long term, caesarean delivery does not provide a significant risk reduction in pelvic floor morbidity compared with vaginal delivery [480, 634].

A number of specific obstetric events and interventions have been implicated as risk factors for the development of pelvic organ prolapse. In one study maternal age and use of epidural analgesia was associated with an increased need for pelvic organ prolapse surgery [630]. A Swedish case-control study found no significant association with maternal age nor instrumental delivery (forceps or vacuum) or length of delivery when comparing women with prolapse to randomly selected controls [633]. In a case-control study, Chiaffarino et al. [605] found that after forceps delivery women had an OR of 3.6 (95% CI 1.0-13.5) for developing pelvic organ prolapse, but after adjustment for vaginal delivery the odds were no longer significant (OR 1.3 95% CI 0.6-3.1). Also Moalli et al. [594] concluded that forceps delivery posed a risk for prolapse. Uma et al. [632] found no significant association between pelvic organ prolapse and forceps delivery (OR 0.9, 95% CI 0.7-1.2); infant birthweight >4.0 kg (OR 0.9, 95% CI 0.5-1.7); episiotomy (OR 1.46, 95% CI 1.0-2.10); and prolonged labour >12 hours (OR 1.51, 95% CI 1.00, 2.27). The inconsistency and wide variety in the magnitude of the risk estimates suggest that studies so far lack sufficient statistical power for valid conclusions.

9. OTHER FACTORS AND POP

A wide variety of risk factors for pelvic organ prolapse, others than those addressed above, have been identified in the literature. Most of these have been investigated as part of larger multivariate analyses based on cross-sectional surveys or retrospective case-control studies. Overall, the evidence for these associations are largely of level III-IV and further research is needed to disentangle the effects and interactions of environmental risk factors for prolapse.

Several somatic risk factors for pelvic organ prolapse have been identified. Generalised connective tissue disorders such as Ehlers-Danhlos disease and Marfans syndrome [636-637] have been linked to an increased risk of pelvic organ prolapse. In a community-based study of prolapse in rural West Africa, chronic anaemia was the strongest risk factor for prolapse after parity and age (OR 2.1 95% CI 1.1-3.4) [613] and chronic obstructive pulmonary disorders. Skeletal abnormalities such as thoracic kyphosis, lumbar lordosis and pelvic dimension changes have been associated with an increased risk for prolapse [638-639]. Women with joint hypermobility have a significantly higher prevalence of genital (and rectal) prolapse in comparison to women with normal mobility [640-641]. Weak associations have also been shown for osteoporosis and rheumatoid arthritis [634].Variables that did not demonstrate any significant linkage to prolapse includes body mass index [642-643], the presence of chronic obstructive pulmonary disease and diabetes mellitus [587, 643]. Pulmonary impairment has been shown to be more common in women with loss of pelvic organ support compared to those without [644].

A low educational level (OR 2.16, 95% CI 1.10-4.24) [645] and low annual income [646], are socio-economic factors which have been associated with an increased risk for pelvic organ prolapse.

In 21,449 non-hysterectomised Italian women, higher education was associated was a protective factor for uterine prolapse [635].However, despite significant differences in educational level, smoking habits, alcohol con-sumption, and socio-economic indices, the prevalence of pelvic organ prolapse did not differ between Kroatian urban and rural women [647].

Physically strenuous occupation have also been shown to influence the risk for pelvic organ prolapse. In a register based study of 28,000 Danish assistant nurses exposed to repetitive heavy lifting, the risk for prolapse was higher among the nurses compared to controls (OR 1.6 95% CI 1.2-2.2) [648].

Women who were labourers/factory workers had significantly more severe prolapse than other job categories ($p < 0.001$) in a cross-sectional study of women presenting for routine gynaecological care [646]. Also, hard physical training may increase the risk for prolapse as women attending paratrooper training, were more likely to present stage II prolapse compared to controls (RR=2.7 95% CI 1.4-5.4) [649].

V. SUMMARY POINTS

- Most studies have used a cross-sectional design and there is limited longitudinal data to suggest a causal relationship between symptoms of obstructed defaecation and pelvic organ prolapse or vice versa.
- Posterior vaginal wall prolapse and perineal descent are the compartment specific defects most clearly associated with symptoms of obstructive defecation.
- A number of studies suggest that hysterectomy and other pelvic surgery may increase the risk for pelvic organ prolapse (LE 2).
- Hysterectomy due to pelvic organ prolapse and other pelvic floor surgery are the strongest predictors of secondary pelvic floor surgery (LE 2).
- Stress urinary incontinence and pelvic organ prolapse have familial transmission patterns mediated by either genetic or environmental factors (LE 2).
- Stress urinary incontinence is more common in Caucasian women, and pelvic organ prolapse in Caucasian and Hispanic women when compared to African.American women (LE 2).
- Twin studies suggest that heritability contributes to the liability of developing pelvic floor disorders but environmental effects are substantial.
- Childbirth is associated with an increased risk for pelvic organ prolapse later in life. Current evidence also suggests that increasing number of childbirths increases the risk (LE 2).
- It remains undecided if caesarean section prevents the development of pelvic organ prolapse but most studies indicate that caesarean is associated with a decreased risk for subsequent pelvic floor morbidity in comparison to giving vaginal birth (LE 2).
- There is a dearth in the understanding of how specific obstetrical events and the process of labour and delivery affects the risk for pelvic organ prolapse.
- Life style factors and socio-economic indices appear to be associated with the risk for pelvic organ prolapse.
- A number of somatic diseases and conditions have been linked to the occurrence of prolapse but the cause-effect relationship is undetermined.

I. WHY DO PREVALENCE ESTIMATES DIFFER?

The discussion here relates to UI only, as data and literature for FI and POP are very scarce. However, many of the principal arguments will be relevant to these conditions as well.

I. GENERAL PROBLEMS IN SURVEY RESEARCH

The well documented variation in prevalence estimates is thought to result at least in part from several confounders common to survey and epidemiological research. Herzog and Fultz,[73] in a review of the prevalence and incidence of UI in community-dwelling populations, proposed that past investigations were plagued by sampling and non-response issues, by self selection and attrition, by definitional, conceptual, and measurement issues. Comprehensive reviews about measurements and methodological aspects of investigating UI are provided.[361] It is clear that there are large methodological challenges to rigorous research in this field. In general, quality of recent large studies has undoubtedly improved, but the scientific community must continue to deal with methodological challenges in order to achieve progress.

II. DIFFERENT DEFINITIONS AND MEASUREMENT

A major problem in research on UI has been the use of different definitions and measurements, and this might contribute to the wide range of reported prevalence estimates. The former ICS definition of UI – as a condition in which involuntary loss of urine is a social or hygienic problem and is objectively demonstrable - included objective demonstration of urine loss as one critical component. This aspect limited the ICS definition for community based epidemiological investigations, because objective demonstration of UI is difficult to achieve outside of the clinical setting, and studies which were able to include this aspect in their assessment might have produced different prevalences. In addition, a social or hygienic aspect of the definition was problematic in epidemiologic studies because it added a subjective aspect to an objectively defined condition and therefore confounded the investigation of prevalence, incidence, and risk factors. In our previous report[2] we argued for reconsideration of the definition of UI, and we emphasized that the core of the definition should be ”any involuntary loss of urine”. In accordance with this view, ICS changed its definition in 2001 to UI being ”the complaint of any involuntary leakage of urine” [1].

The new definition makes epidemiological research easier. But three consequences should be addressed:

1. Epidemiological studies should not be based on this definition alone, and all studies should include a minimal additional data set, standard confounders, and questions specific to the aim of the study. This is discussed in the Section on Recommendations for further research.
2. The number of persons fulfilling the definition will increase. This should not be interpreted as an increase in the number potential of patients.
3. Public awareness, case finding of health care personnel, and help seeking behaviour may be affected of a new and more extensive definition.

Studies have used different severity levels and time frames for defining UI. A further factor complicating the conceptualization and measurement of UI in epidemiologic studies lies in the nature of the condition. UI is a chronic condition (or set of conditions) that often starts slowly and comes and goes for a considerable time period before it become fully established.[361] If people get used to their UI or notice it less, this can interfere with valid assessment.

Ideally self-report measures are validated by clinical evaluations. However, clinical and even urodynamic investigations should be regarded as other measures, not necessarily as gold standards, because it is known to be difficult to demonstrate all urinary symptoms in the clinical setting.

Holtedahl[212] calculated prevalence estimates using different definitions of UI for the same sample of 50 to 70 year old women. The prevalence of any self-reported leakage was 47%. Self-reported regular UI with or without objective demonstration was found for 31% of women, regular incontinence according to the former full ICS definition for 19%. Another study found prevalences of 69% and 30% for any UI and the former ICS definition, respectively [33]. The results indicate that the former ICS definition was rather restrictive.

Low response rates may further bias prevalence estimates.[361] Known differences between responders and non-responders can be compensated during the analysis. The major problems is unknown differences in response rates and other characteristics. Incontinent women may not answer (or deny UI) because of embarrassment or related handicaps. But incontinent women may also find the subject particularly relevant and therefore respond to a greater extent than continent women. At present, we do not know much about how these factors may affect the comparison between incontinent and continent women.

One paper explored the problem of underreporting incontinence and how it can be altered with the use of an introduction to the incontinence questions and probing.[79] Another paper explored the issue of selection bias in mailed surveys. The first wave had higher prevalence of incontinence than follow-up mailings, and thus individuals with UI tended to respond on the first wave.[657] In an English mailed survey on incontinence and other urinary symptoms, a sample of non-responders were traced, and those eligible were asked questions from the survey.[658] Compared with the responders, the non-responders overall showed little differences in reporting of urinary symptoms. However, non-responders >70 tended to be of poorer general health, and they reported certain urinary symptoms more frequently.

III. SUMMARY POINTS

- The lack of epidemiological data from populations underrepresented in research limits the world wide application of the present information.
- Many investigations are plagued by sampling and non response issues, by self selection and attrition. Many early studies were obtained from sampling patients seeking care.
- A major problem is the use of different definitions of incontinence. The new ICS definition makes epidemiological research easier.
- There are large methodological challenges to research in the field of UI. Unless the scientific community deals with these issues, progress will be difficult to make.

J. HELP SEEKING BEHAVIOUR

I. URINARY INCONTINENCE

A majority of people with UI have not sought help,[78, 81, 333, 340, 342] and this is confirmed also in recent publications.[322, 326, 327] Reasons given by people for not seeking help include: not regarding incontinence as abnormal or serious,[78, 330] considering incontinence to be a normal part of ageing,[335] having low expectations of treatment[78, 330] and thinking they should cope on their own.[501, 497] Some studies also confirm the notion that embarrassment may be an important reason for not seeking help.[332-334] There is an association between help seeking and condition-specific factors like duration, frequency and amount, and people's perceptions of the impact of incontinence,[78, 81, 358, 359, 322] but other more personal characteristics like individual health care behaviour and attitudes may also play a role.

In a Norwegian study 4.4 % of all women >20 years old in a community consulted their general practitioner for UI during a 3 year period.[659] But mentioning the symptoms to a physician may not be enough. There are reports of doctors not responding, either by ignoring the statement of symptoms or by providing a dismissive explanation,[336] and people interpreting a lack of response from the doctor as an indication that no treatment is available.[337] In a study of management of incontinence in general practice, 30% of the women who had told their doctor about their symptoms perceived that they were offered no help.[335] It is probable that many primary health care providers lack confidence in managing UI, and that this contributes to under treatment in those seeking help.[338]

Only a small proportion of incontinent community-residing women have had surgery, medication, or exercise regimens.[110, 340, 327, 339, 341] In addition to seeking help from the formal health care system, common responses to symptoms of illness are self-management and self-treatment behaviour. The major method of actively managing UI among community residents is the use of absorbent products.[359, 339, 343-345]

It is obvious that millions of men and women suffer from their UI, and that for many of them good treatment options are available. However, for many persons with very mild or occasional UI it is probably adequate not to seek help from the health care system. Others are satisfied with just information and understanding about the causes and in many cases self care may be quite appropriate. A Danish study has shown that simple information and advice was adequate " treatment" for 23% of the women attending an open access incontinence clinic.[346] A Swedish study found that among 136 women with UI, 36% wanted clinical evaluation, and only 24% subsequently started treatment.[109]

Both epidemiological and qualitative research in this field should be encouraged in order to understand cultural, religious, and personal factors for help seeking behaviour world wide.[333, 331, 337] Specifically, other than condition-specific factors should be further explored, e.g. persons' health care behaviour, perceptions and attitudes.

II. FAECAL INCONTINENCE AND PELVIC ORGAN PROLAPSE

There are indications of underreporting also of FI and patients' reluctance to report symptoms or to seek treatment.[458, 459, 490] It has been shown that women are more willing to report FI than men.[660] For POP we have no information.

III. SUMMARY POINTS

- Recent publications confirm that a majority of people with FI, UI, and POP have not sought help.
- Only a small proportion of urinary incontinent community-residing people have had surgery, medication, or exercise regimens.
- Increasing severity, increasing duration, and urge/mixed type of UI are related to consulting a health care provider.
- Associations other than condition-specific factors should be further explored in future research, e.g. persons' health care behaviour, perceptions and attitudes.
- Health care personnel should be encouraged to approach persons at risk for FI, UI and POP. People with such symptoms should be assessed so services and treatment can be offered and targeted. The patient's view of management, even denial, should be respected.

K. EPIDEMIOLOGY AND CLINICAL WORK: FROM RESPONDENT TO PATIENT

We have emphasised some major and important differences between epidemiology and clinical work. These differences may have several implications. A selection process is most often accomplished first by self-selection (help seeking), then a referral system, which provides specialist physicians to a patient population with higher prevalence of disease, more severe disease, and often skewed type distribution, thus obtaining test results with fewer false positives, better diagnostic accuracy, and more efficient use of resources. However, such intended and purposeful selection bias has its drawbacks. There is growing evidence that this selection process introduces bias into research and hampers our ability to generalize hospital based research back to general or primary care populations. Furthermore, it may result in recommendations and guidelines for diagnosis or therapy derived from tertiary care centres that are inappropriate at the primary care level. Often guidelines, review articles or teaching material do not take into account the varying prevalence and variation in clinical picture between community and hospital. They may also emphasise use of tests or equipment that are not appropriate or relevant for primary health care, thus leading to over utilisation of referrals. Data from hospitals or specialist level may also overestimate level of burden, costs and number of persons in need of treatment if such data are used for extrapolation

back to community level. Therefore it is important that this Consultation uses different algorithms for initial and specialised care (see other relevant chapters).

One study provides substantial empirical evidence to support the existence of selection bias for UI.[322] The analyses were based on three populations of incontinent women: Community level (epidemiological survey), primary care level (prospective study), and secondary care level (university hospital, prospective study). The general practice patients were older and the hospital patients younger than those in the community. From community via general practice to hospital, there was an increase in duration, frequency of leakage, amount of leakage, severity and perceived impact of incontinence. Help-seeking at the primary care level was associated with increasing age and severity, and with urge symptoms and impact. Referral from general practice to hospital was only associated with (lower) age and urge symptoms.

Under the subtitle Severity and impact we have given examples of how the prevalence estimates for women change dramatically when bothersomeness and severity are considered. Taken together with selection bias, this emphasises caution when epidemiological data are used in a clinical context. It concerns " level of care" in several ways; there is a large transitional zone from healthy to diseased, there is a danger of medicalisation, and there is a danger of treating patients at a higher level than necessary. Risk factors, predictors and correlates discovered in epidemiological studies are probabilistic of nature and may not be decisive in the clinical assessment of an individual patient. In addition, the attributable risk due to some known risk factors may be statistically but not clinically significant.

I. WORLDWIDE ESTIMATES OF CURRENT AND FUTURE INDIVIDUALS (≥20 YEARS) WITH LOWER URINARY TRACT SYMPTOMS INCLUDING URINARY INCONTINENCE AND OVERACTIVE BLADDER

In order to effectively plan health care resources it is necessary to estimate the prevalence and incidence of illnesses to know to what extent resources require to be allocated to a specific illness health care condition. This chapter has dealt with three major global problems, urinary and faecal incontinence as well as pelvic organ prolapse, that affect women and men throughout the world. At the ICI meeting in Paris data were presented regarding worldwide estimates of current and future individuals (≥20 years) with LUTS including urinary incontinence and overactive bladder [650].

The objective of the study was to estimate the current and future number of people with LUTS, including overactive bladder (OAB) and Urinary Incontinence (UI) utilising the current ICS definitions. Age- and gender-specific prevalence rates from the EPIC study[651] were applied to the worldwide over 20 year old population (4.2 billion) with males and females stratified into five-year age groups (20-24 to 80+). Projected population estimates for all worldwide regions were based on the United States Census Bureau International Database (IDB)[652].

Estimates were presented for 2008, 2013 and 2018 and are summarised in **Tables 20 and 21**. Table 21 summarises the estimated number of individuals with certain LUTS symptoms by year and sex in the world population and Table 22 describes the estimated number of individuals of LUTS and OAB over 10 years across the world regions.

Estimates and projections featured in this analysis were based on prevalence rates of LUTS described in the EPIC study – based primarily on a European population. The prevalence rates featured in the EPIC study are similar to other prevalence rates of LUTS that were found in others studies across other countries [653-654]

The projections in this report assume the prevalence rates of LUTS will remain throughout the year 2018 for all age and sex groups (**Figure 9**).

Prevalence of LUTS will also increase as other factors related to LUTS, such as obesity, increases. The estimated number for present and future years are not true numbers but are based on a projected population configured by the International Database (IDB). The IDB's estimates and projections are drawn by Census Bureau demographers and are based on reviewed censuses, surveys, and vital statistics provided by National Statistics Offices [9]. Data on international migration and refugee movements, public health efforts, socio-political circumstances, and historical events such as natural disasters and conflict are all considered when the IDB calculates the estimates and projections (**Figure 10**).

It is anticipated that with the overall aging of the population the prevalence of LUTS will also increase

It has been shown that LUTS are burdensome to individuals [655-656] and the likely increase in the number of individuals experiencing. LUTS has implications on healthcare resources and overall health burden. This analysis is an estimate of the number of individuals with LUTS based on a conservative prevalence rate, and so the future number of those with certain LUTS may surpass those of this report (**Table 22**).

Table 20. Estimated Number of Individuals with Certain LUTS By Year & Sex - World Population (In Millions) [650].

LUTS Symptoms Incontinence	Male 2008	Male 2013	Male 2018	Female 2008	Female 2013	Female 2018
Any Incontinence	98	109	120	250	275	301
UUI	22	25	27	27	30	33
MUI	11	12	14	43	47	52
SUI	10	12	13	127	140	153
Other**1**	55	61	66	53	58	64
Storage						
Any Storage Symptom (Noct**2** ≥1)	1,050	1,151	1,250	1,249	1,363	1,474
Any Storage Symptom (Noct ≥2)	597	655	713	760	831	901
Noct ≥1	942	1,035	1,127	1,098	1,200	1,301
Noct ≥2	388	427	467	464	509	555
Urgency	205	226	247	249	273	297
Frequency	127	139	152	161	174	186
Voiding Symptoms						
Voiding Symptoms	515	563	610	402	511	473
Intermittency	164	181	198	148	176	175
Slow Stream	156	173	193	122	161	146
Straining	132	145	157	83	120	98
Term Dribble	289	315	340	210	276	245
Post Micturition Symptoms						
Post Mic**3** Symptoms	332	365	396	297	350	348
Incomplete Emptying	263	288	314	257	290	302
Other Post Mic Incontinence	108	118	129	64	96	76
Any LUTS (Noct ≥1)						
Any LUTS (Noct ≥1)	1,260	1,377	1,490	1,379	1,460	1,623
Storage + Voiding Symptoms (Noct ≥1)	350	386	422	309	373	367
Storage + Post Mic Symptoms (Noct ≥1)	247	273	299	238	274	282
Voiding + Post Mic Symptoms (Noct ≥1)	205	226	247	158	205	187
Storage + Voiding + Post Mic Symptoms (Noct ≥1)	166	183	202	137	173	163
Any LUTS (Noct ≥2)						
Any LUTS (Noct ≥2)	933	1,020	1,104	994	1,068	1,170
Storage + Voiding Symptoms (Noct ≥2)	247	273	299	237	275	283
Storage + Post Mic Symptoms (Noct ≥2)	188	207	227	190	214	226
Voiding + Post Mic Symptoms (Noct ≥2)	205	226	247	158	205	187
Storage + Voiding + Post Mic Symptoms (Noct ≥2)	130	144	158	119	142	142

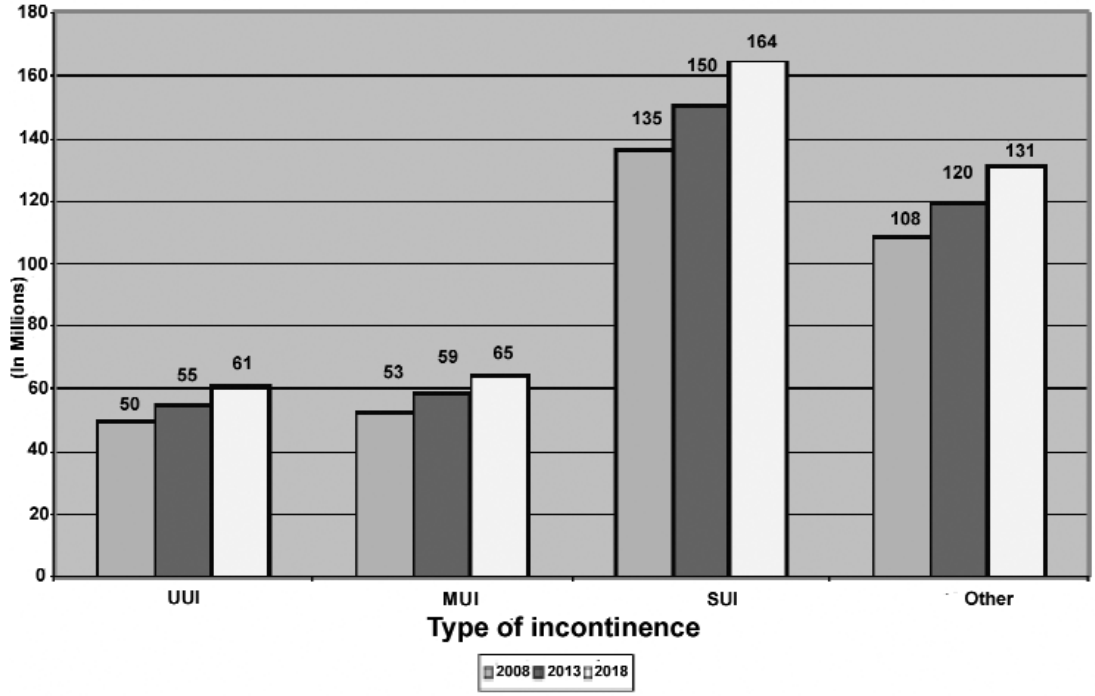

Figure 9 : Estimated number of individuals with UI 2008, 2013 and 2018 grouped according to gender [650].

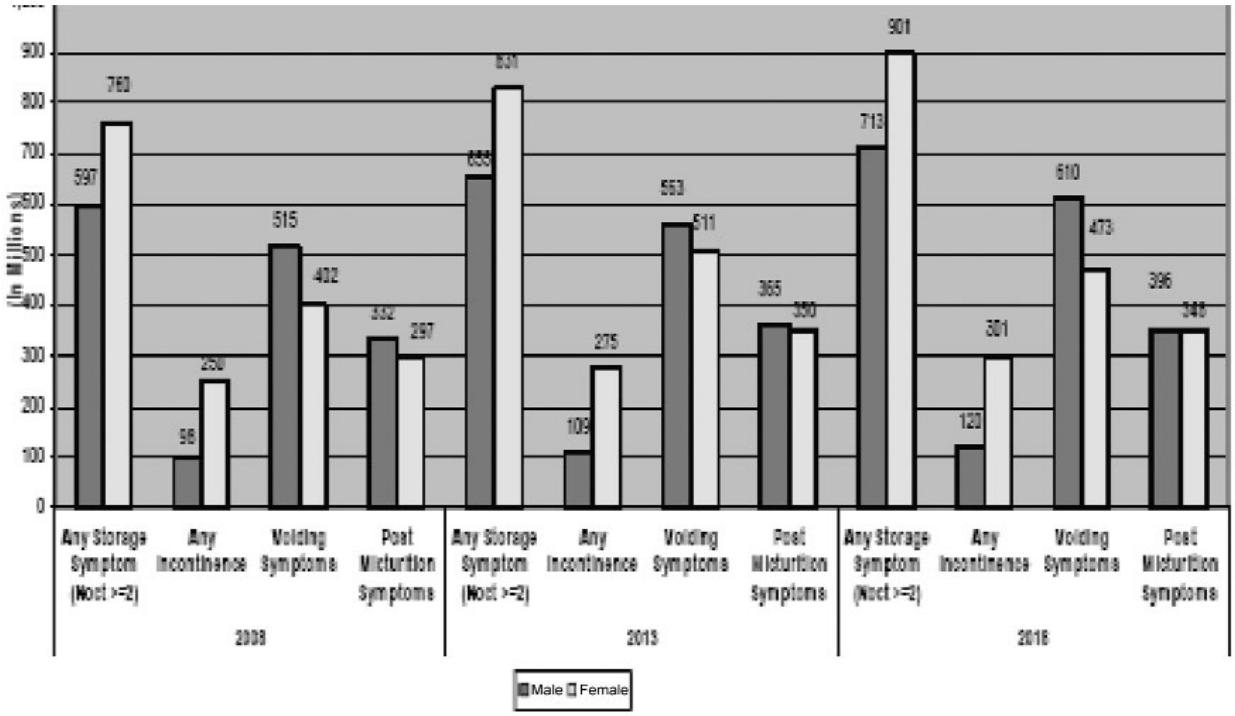

Figure 10 : Estimated number of individuals with LUTS 2008, 2013 and 2018 grouped according to gender [650].

Table 21. Estimated Worldwide Number of Individuals with LUTS including OAB and Incontinence by Region (In Millions) [650].

	Estimated Number of individuals with any LUTS			Estimated Number of Individuals with OAB			Estimated Number of Individuals with Incontinence		
Region	2008	2013	2018	2008	2013	2018	2008	2013	2018
World	1,930	2,106	2,277	455	500	545	346	383	420
Africa	203	231	263	46	53	60	33	38	43
North America	167	180	193	40	44	48	32	34	37
South America	111	122	133	26	29	32	20	22	24
Asia	1,166	1,284	1,396	272	302	332	206	231	256
Europe	273	278	280	68	70	71	54	56	57

Table 22. Summary of major findings

- 46% of the 4.2 billion of the adult world population (≥ 20 and over) experience any LUTS
- 455 million individuals or 11% of the world population estimated to experience OAB symptoms
- 346 million individuals or 8% of the world population estimated to experience some type of UI
- SUI is the most common type of incontinence in 2008 and 2018 (Fig 1.)
- 136 (3%) and 164 (4%) million individuals are estimated to experience SUI in 2008 and in 2018 respectively
- 49 (1%) and 60 (1%) million individuals are estimated to experience UUI in 2008 and in 2018 respectively
- 53(1%) and 65 (1%) million individuals are estimated to experience MUI in 2008 and in 2018 respectively
- 108 (3%) and 131 (3%) million individuals are estimated to experience Other Incontinence in 2008 and in 2018 respectively
- Assuming LUTS prevalence rates remain stable for the next ten years, 2.3 billion individuals are estimated to experience LUTS by the year 2018
- An increase of 18% from 2008
- Storage symptoms has the highest burden in both the male and female population than other LUTS (Fig 2.)
- Male: estimated 597 million in 2008, 713 million in 2018
- Female: estimated 760 million in 2008, 901 million in 2018
- Asia region is estimated to carry the highest burden of LUTS. Estimated 1.2 billion individuals in Asia regions may experience any LUTS

II. SUMMARY POINTS

- The spectrum of severity of anal and urinary incontinence, as well as pelvic organ prolapse, and the symptom profile of patients referred to specialist centres do not necessarily reflect the spectrum of disease seen in the community.
- The selection and referral process may introduce bias into research and hamper the ability to generalise hospital-based research back to primary care populations.
- One should be very careful with calculating numbers of patients in need of therapy based on epidemiological data.

L. RECOMMENDATIONS FOR FURTHER RESEARCH

Much biomedical research is observational and the reporting of such research is often inadequate which hampers the assessment of its strengths and weaknesses and of a study´s generalisability. The STROBE (Strengthening of the Reporting of OBservational studies in Epidemiology) statement was introduced [661]. It is a a checklist of items that should be addressed in articles reporting on the three main study designs of analytical epidemiology: cohort, case-control, and cross sectional studies. The use of this checklist is highly recommended.

I. URINARY INCONTINENCE

It is recommended that more sustained research on measurement of UI should be performed including, its

types and severity to move the research ahead. Longitudinal study designs are needed to estimate incidence of UI and describe the course of the condition and its different forms and to investigate its risk factors and possible protective factors.

There is still little knowledge with regard to prevalence, incidence, and other epidemiological data in developing countries. It is recommended that fundamental research regarding prevalence, incidence and other epidemiological data in developing countries should be encouraged, and tailored to the cultural, economic and social environment of the population under study.

Crude prevalence studies (descriptive epidemiology) from USA and Europe are abundant, and further studies should be done only with recommended and validated questionnaires or in order to combine data from the prevalence study with studies of co-factors and predictors (analytical epidemiology). Control for confounders, stratification, and multivariate techniques should be increasingly used because of the need for more advanced epidemiological analyses of risk factors and comorbidity. Strength of associations should be determined by relative risks and odds ratios, and confidence limits should be given. We have still very little knowledge of the absolute and relative importance of several risk factors, and almost no information about the attributable risk of the factors in the society.

Some potential risk and protective factors deserve more attention. For example, the role of pregnancy and childbirth in the development of UI must be studied in a fashion that links population-based methods to clinical assessment of pregnancy, delivery and the birth trauma and follows women over many years. Such a design is necessary because the effect of pregnancy and childbirth may become clear only years later when the woman is older and because the woman will not be able to report the exact nature of the tear or episiotomy, etc. There should be more emphasis on the associations between UI and specific diseases like stroke, diabetes, psychiatric disease and genital prolapse. Genetic components should be investigated.

Primary prevention is the main goal in the management of human disease. An important strategy would thus be to identify the individuals at risk, and then take measures to reduce the risk among those individuals or in certain risk groups. Based on current knowledge there are no well documented efforts that can be done in order to avoid the occurrence of UI in large populations. Primary prevention studies should be encouraged, but the epidemiological basis for choosing appropriate interventions is weak.

In surveys based on questionnaires or interviews symptoms can be registered. There are convincing data suggesting that the different types may reflect quite different pathologies and risk factors. Differentiating the types in future research might therefore prove very fruitful. Methodological work has still to be done in this area, but typical type descriptions should be included in new studies. Likewise, studies of risk factors should include important and known confounders such as age, parity, and weight.

Variations in definitions and measurement issues are fundamental and lead to problems with assessing the findings in epidemiological studies. We need to improve epidemiological studies by including variables that better characterise UI, so that more advanced and informative analyses may be conducted. It is therefore recommended that all epidemiological studies include a minimum data set (**Table 23**), including elements of screening question, frequency measure, quantity of urine loss, duration, type, and severity. In addition, it is recommended that validated measures of bother/quality of life and urinary symptoms other than UI should be included. We here also refer to the chapter from the committee on symptom and quality of life assessment.

In addition, it is recommended that validated measures of bother/quality of life and urinary symptoms other than UI should be included.

Table 23. Elements in a minimum data set recommended for all epidemiological studies

- Screening question for any involuntary urine loss
- Frequency measure. For example, classification into categories of none, less than once a month, one/several times a month, one/several times a week, every day/nigth, all the time
- Quantity of urine loss for a typical episode. For example, classification into categories of none, drops, small amounts, moderate amounts, much/a great deal
- Duration. For example months, years
- Type. Based on typical description; stress, urge, mixed and other
- Severity. Either by combining excisting questions or by a validated index

II. FAECAL INCONTINENCE AND PELVIC ORGAN PROLAPSE

In these areas there is a need for more epidemiological research in all areas; prevalence, incidence, and risk factors. Many of the fundamental methodological issues relevant to UI discussed above are highly relevant to the fields of FI and POP.

The committee emphasises that uniform definitions of

FI and POP should be used in studies, and there should be a move towards a standardization of measurement instruments in community surveys that can be used worldwide. Developing definitions is a scientific process requiring careful conceptualization of the condition in light of its many clinical presentations and underlying mechanisms. This will require a multi-method approach and consideration of issues such a reliability and validity.

REFERENCES

1. Abrams, P., Cardozo, L., Fall, M., Griffits, D. Rosier, P., Ulmsten, U. et al. : The standardisation of terminology in lower urinary tract function : report from the standardisation sub-committee of the International Continence Society. Urology, 61: 37m 2003.
2. Hunskaar, S., Burgio, K., Clark A, Lapitan, M, Nelson R, Sillén U, Thom D. : Epidemiology of urinary incontinence (UI) and faecal incontinence (FI) and pelvic organ prolapse (POP). In : Incontinence, 3 ed. Edited by P. Abrams, L Cardozo, S. Khoury, A. Wein. Health Publication Ltd, pp. 255, 2005.
3. Stroup, D. F., Berlin, J.A., Morton, S. C., Olkin, I., Williamson, G. D., Rennie, D. et al. : Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observtional Studies in Epidemiology (MOOSE) group. JAMA 283: 2008, 2002.
4. Nevéus, T., et al., The Standardization of Terminology of Lower Urinary Function in Children and Adolescents: Report from standardisation Committee of International Children's Continence Society. J Urol, 2006. 176: p. 314 - 324.
5. Rittig, S., et al., Abnormal diurnal rhythm of plasma vasopressin and urinary output in patients with enuresis. Am J Physiol, 1989. 25: p. 664-.
6. Yeung, C.K., Chiu, H.N., Sit, F.K., Bladder dysfunction in children with refractory monosymptomatic primary nocturnal enuresis. J Urol, 1999. 162: p. 1049-55.
7. Holmdahl, G., et al., Four-hours voiding observation in healthy infants. Scan J Urol Nephrol, 1996. 156: p. 1808-1812.
8. Jansson, U.-B., et al., Voiding pattern and acquisition of bladder control from birth to age 6 years - a longitudinal study. J Urol, 2005. 174: p. 298 - 293.
9. Kranz, I., et al., On the Epidemiology of Nocturnal Enuresis - A Critical Rewiew of Methods used in descriptive Epidemiological studies on Nocturnal Enuresis. 1994: p. 75-81.
10. McGree, R., et al., A longitudinal study of enuresis from five to nine years. Aust. Paediatr. J., 1984. 20: p. 39-42.
11. Forsythe, W.I. and A. Redmond, Enuresis and spontaneous cure rate - study of 1129 enuretics. Arch Dis Child, 1974. 49: p. 259-263.
12. Fergusson, D.M., L.J. Horwood, and F.T. Shannon, Factors Related to the Age of Attainment of Nocturnal Bladder Control: An 8-Year Longitudinal Study. Pediatrics, 1986. 87(5): p. 884-890.
13. Fergusson, D.M. and L.J. Horwood, Nocturnal enuresis and behavioural problems in adolescence: a 15-year longitudinal study. Pediatrics, 1994. 94(5): p. 662-668.
14. Feehan, M., et al., A 6 year follow-up of childhood enuresis: Prevalence in adolescence and consequences for mental health. J. Paediatr. Child Health, 1990. 26: p. 75-79.
15. Chiozza, M., L, et al., An Italian epidemiological multicentre study of nocturnal enuresis. BJU, 1998. 81.suppl.3: p. 86-89.
16. Yeung, C.K., Nocturnal Enuresis in Hong Kong: Different Chinese Phenotypes. Scan J Urol Nephrol, 1996. suppl.31: p. 17-21.
17. Spee- van der Wekke, J., et al., Childhood nocturnal enuresis in the Netherlands. Urology, 1998. 51: p. 1022.
18. Cher, T.-W., G.-J. Lin, and K.-H. Hsu, Prevalence of nocturnal enuresis and associated familiar factors in primary school children in Taiwan. J Urol, 2002. 168: p. 1142-1146.
19. Serel, T.K., et al., epidemiology of enuresis in Turkish children. Scan J Urol Nephrol, 1997. 31: p. 537.
20. Lee, S.D., et al., An epidemiological study of enuresis in Korean children. BJU Int., 2000. 85: p. 869-873.
21. Kajiwara, M., et al., Nocturnal enuresis and overactive bladder in children: An epidemiological study. Int J Urol, 2006. 13: p. 36-41.
22. Söderstrom, U., et al., Urinary and faecal incontinence: A population-based study. Acta Paediatr, 2004. 93: p. 386-89.
23. Yeung, C.K., et al., differences in characteristics of nocturnal enuresis between children and adolescents: A critical appraisal from a large epidemiological study. BJU Int., 2006. 97(5): p. 1069-73.
24. Kanaheswari, Y., Epidemiology of childhood nocturnal enuresis in Malaysia. J Paediatr Child Health, 2003. 39(2): p. 118-123.
25. Hellström, A.-L., et al., Micturition habits and incontinence in 7-year-ola Swedish school entrants. Eur J Pediatr, 1990. 149(6): p. 434-437.
26. Järvelin, M.R., et al., Enuresis in Seven-Year-Old Children. Acta Paediatr Scan, 1988. 77: p. 148-153.
27. Hellström, A.-L., et al., Micturition habits and incontinence at age 17 - reinvestigation of a cohort studied at age 7. BJU, 1995. 76: p. 231-234.
28. Swithinbank, L.V., J.C. Carr, and P.H. Abrams, Longitudinal Study of Urinary Symptoms in Children. Scan J Urol Nephrol, 1994. Suppl 163: p. 163-167.
29. Swithinbank, L.V., et al., The natural history of urinary symptoms during adolescence. BJU, 1998. 81, Suppl 3: p. 90-93.
30. Hirasing, R.A., et al., Enuresis Nocturna in Adults. Scan J Urol Nephrol, 1997. 31: p. 533-536.
31. Neveus, T., et al., Depth of sleep and sleep habits among enuretic and incontinence children. Acta Paediatr, 1999. 88: p. 748.
32. Bower, W.F., et al., The epidemiology of childhood enuresis in Australia. British Journal of Urology, 1996. 78: p. 602-606.
33. Gümüs, B., et al., Prevalence of nocturnal enuresis and accompanying factors in children aged 7-11 years in Turkey. Acta Paediatr, 1999. 88: p. 1369.
34. Watanabe, H. and A. Kawauchi, Nocturnal Enuresis: Social Aspects and Traetment in Japan. Scan J Urol Nephrol, 1994. suppl. 163: p. 29-38.
35. Liu, X., et al., Attaining Nocturnal Urinary Control, Nocturnal Enuresis, and Behavioral Problems in Chinese Children Aged 6 Through 16 Years. J Am Acard Child Adolesc Phychiatry, 2000. 39(12): p. 1557-1564.
36. Järvelin, M.R., et al., Aetiological and Percipitating Factors for Childhood Enuresis. Acta Paediatr Scan, 1991. 80: p. 361-369.
37. Eiberg, H., Nocturnal enuresis is linked to a specific gene. Scan J Urol Nephrol, 1995. Suppl.173: p. 15.
38. Arnell, H., et al., The genetics of primary nocturnal enuresis: inheritance and suggestion of a second major gene on chromosome 12q. J Med Genet, 1997. 34: p. 360.

39. von Gontard, A., et al., Molecular genetics of nocturnal enuresis: clinical and genetic heterogeneity. Acta Paediatr, 1997. 87: p. 571.

40. Moilanen, I., et al., A follow-up of enuresis from childhood to adolescence. BJU, 1998. 81,suppl.3: p. 94-97.

41. Schulpen, T., The burden of nocturnal enuresis. Acta Paediatr, 1997. 86(9): p. 981-984.

42. van Hoecke, E., et al., Socioeconomic Status as a Common Factor Underlaying the Association Between Enuresis and Psychopathology. J Dev Behav Pediatrics, 2003. 24(2): p. 109-114.

43. Kawauchi, A., et al., Follow-up study of bedwetting from 3 to 5 years of age. Urology, 2001. 58(5): p. 772-776.

44. Lunsing, R.J., et al., Nocturnal enuresis and minor Neurological dysfunction at 12 years: A follow-up study. Dev Medicine and Child Neurology, 1991. 33: p. 439-445.

45. Duel, B.P., et al., A survey of voiding dysfunction in children with attention deficit-hyperactivity disorder. J Urol, 2003. 170: p. 1521-1524.

46. Robson, W.L.M., et al., Enuresis in children with attention deficit hyperactivity disorder. Southern Medical Journal, 1997. 90: p. 503.

47. Wolfish, N.M., R.T. Pivik, and K.A. Busby, Elevated sleep arousal thresholds in enuretic boys: clinical implications. Acta Paediatr, 1997. 86: p. 381-384.

48. Kawauchi, A., et al., Changes in the structure of sleep spindles and delta waves on electroencephalography in patients with nocturnal enuresis. BJU, 1998. 81, suppl. 3: p. 72-75.

49. Hunsballe, J.M., Increased delta component in computerized sleep electroencephalographic analysis suggest abnormally deep sleep in primary monosymptomatic nocturnal enuresis. Scan J Urol Nephrol, 2000. 34: p. 294.

50. Chandra, M., et al., Prevalence of diurnal voiding symptoms and difficult arousal from sleep in children with nocturnal enuresis. J Urol, 2004. 172: p. 311-16.

51. Osungbade, K.O. and F.O. Oshiname, Prevalence and perception of nocturnal enuresis in children of a rural community in southwestern Nigeria. Trop Doc, 2003. 33(4): p. 243-236.

52. Baruzzi, A., et al., Atrial natriuretic peptide and catecholamines in obstructive sleep apnoea. Sleep, 1991. 14: p. 83.

53. Weider, D.J., M.j. Sateia, and R.p. West, Nocturnal enuresis in children with upper airway obstruction. Otolaryngol Head Neck Surg, 1991. 105: p. 427-432.

54. Loening-Baucke, V., Urinary Incontinence and Urinary Tract Infection and Their Resolution With Treatment of Chronic Constipation of Childhood. Pediatrics, 1997. 100(2): p. 228-232.

55. Forbes, F.C., Children with enuresis. Nowdays, a strong suspicion of sexual abuse would prompt full investigation. BMJ, 1998. 316: p. 777.

56. Ferrara, P., et al., Preliminary data on monosymptomatic nocturnal enuresis in children and adolescents with type 1 diabetes. Scan J Urol Nephrol, 2006. 40: p. 238-40.

57. Hoebeke, P., et al., One thousand video-urodynamic studies in children with non-neurogenic bladder sphincter dysfunction. BJU Int., 2001. 87: p. 575-580.

58. Hellerstein, S., Voiding Dysfunction in Pediatric Patients. Clin Pediatr (Phila), 2003. 42(1): p. 43-49.

59. Kajiwara, M., et al., The micturition habits and prevalence of daytime urinary incontinence in Japanese primary school children. J Urol, 2004. 171: p. 403-7.

60. Joinson, C., et al., Psychological problems in children with daytime wetting. Pediatric, 2006. 118(5): p. 1985-93.

61. Bloom, D.A., et al., Toilet habits and continence in children: An opportunity sampling in search of normal parameters. J Urol, 1993. 149: p. 1087-1090.

62. Von Gontard, A., Hollmann,E., Comorbidity of functional urinary incontinence and enkopresis: Somatic and behavioural associations. J Urol, 2004. 171: p. 2644-47.

63. Sureshkumer P., C.P., Roy L.P, Knight J.F.,, et al., Daytime urinary incontinence in primary school children: A population-based survey. The Journal of Pediatrics, 2001. 137(6): p. 814-818.

64. Lettgren, B., et al., Urge incontinence and voiding postponement in children : somatic and phsychosocial factors. Acta Paediatr, 2002. 91: p. 978-984.

65. Zink, S., C.M. Freitag, and A. von Gontard, Behavioural comorbidity differs in subtypes of enuresis and urinary incontinence. J Urol, 2008. 179: p. 295-8.

66. Sandvik, H., Hunskaar, S., Vanvik, A. et al.: Diagnostic classification of female urinary incontinence: an epidemiological survey corrected for validity. J Clin Epidemiol, 48: 339, 1995

67. Thom, D.: Variation in estimates of urinary incontinence prevalence in the community: effects of differences in definition, population characteristics, and study type. [Review] [46 refs]. Journal of the American Geriatrics Society, 46: 473, 1998

68. Sandvik, H., Hunskaar, S., Seim, A. et al.: Validation of a severity index in female urinary incontinence and its implementation in an epidemiological survey. J Epidemiol Community Health, 47: 497, 1993

69. Sandvik, H., Seim, A., Vanvik, A. et al.: A severity index for epidemiological surveys of female urinary incontinence: comparison with 48-hour pad-weighing tests. Neurourol Urodyn, 19: 137, 2000

70. Shumaker, S. A., Wyman, J. F., Uebersax, J. S. et al.: Health-related quality of life measures for women with urinary incontinence: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Continence Program in Women (CPW) Research Group. Qual Life Res, 3: 291, 1994

71. Fultz, N. H., Herzog, A. R.: Measuring urinary incontinence in surveys. Gerontologist, 33: 708, 1993

72. Thom, D. H., Van den Eeden, S. K., Ragins, A. I. et al.: Differences in prevalence of urinary incontinence by race/ethnicity. J Urol, 175: 259, 2006

73. Herzog, A. R., Fultz, N. H.: Prevalence and incidence of urinary incontinence in community-dwelling populations. J Am Geriatr Soc, 38: 273, 1990

74. Fultz, N. H., Herzog, A. R.: Prevalence of urinary incontinence in middle-aged and older women: a survey-based methodological experiment. J Aging Health, 12: 459, 2000

75. Van Oyen, H., Van Oyen, P.: Urinary incontinence in Belgium; prevalence, correlates and psychosocial consequences. Acta Clin Belg, 57: 207, 2002

76. Swithinbank, L. V., Donovan, J. L., Du Heaume, J. C. et al.: Urinary symptoms and incontinence in women: relationships between occurrence, age, and perceived impact. Br J Gen Pract, 49: 897, 1999

77. Yarnell, J. W., Voyle, G. J., Richards, C. J. et al.: The prevalence and severity of urinary incontinence in women. J Epidemiol Community Health, 35: 71, 1981

78. Holst, K., Wilson, P. D.: The prevalence of female urinary incontinence and reasons for not seeking treatment. N Z Med J, 101: 756, 1988

79. Hannestad, Y. S., Rortveit, G., Sandvik, H. et al.: A community-based epidemiological survey of female urinary incontinence: the Norwegian EPINCONT study. Epidemiology of Incontinence in the County of Nord-Tr_ndelag. J Clin Epidemiol, 53: 1150, 2000

80. Lara, C., Nacey, J.: Ethnic differences between Maori, Pacific Island and European New Zealand women in prevalence and attitudes to urinary incontinence. N Z Med J, 107: 374, 1994

81. Rekers, H., Drogendijk, A. C., Valkenburg, H. et al.: Urinary incontinence in women from 35 to 79 years of age: prevalence and consequences. Eur J Obstet Gynecol Reprod Biol, 43: 229, 1992

82. Thomas, T. M., Plymat, K. R., Blannin, J. et al.: Prevalence of urinary incontinence. Br Med J, 281: 1243, 1980

83. Irwin, D. E., Milsom, I., Hunskaar, S. et al.: Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC study. Eur Urol, 50: 1306, 2006

84. Zhu, L., Lang, J., Wang, H. et al.: The prevalence of and potential risk factors for female urinary incontinence in Beijing, China. Menopause, 15: 566, 2008

85. Minassian, V. A., Stewart, W. F., Wood, G. C.: Urinary incontinence in women: variation in prevalence estimates and risk factors. Obstet Gynecol, 111: 324, 2008

86. Tegerstedt, G., Maehle-Schmidt, M., Nyren, O. et al.: Prevalence of symptomatic pelvic organ prolapse in a Swedish population. Int Urogynecol J Pelvic Floor Dysfunct, 16: 497, 2005

87. Melville, J. L., Katon, W., Delaney, K. et al.: Urinary incontinence in US women: a population-based study. Arch Intern Med, 165: 537, 2005

88. Corcos, J., Schick, E.: Prevalence of overactive bladder and incontinence in Canada. Can J Urol, 11: 2278, 2004

89. Danforth, K. N., Townsend, M. K., Lifford, K. et al.: Risk factors for urinary incontinence among middle-aged women. Am J Obstet Gynecol, 194: 339, 2006

90. Homma, Y., Yamaguchi, O., Hayashi, K.: An epidemiological survey of overactive bladder symptoms in Japan. BJU Int, 96: 1314, 2005

91. Waetjen, L. E., Liao, S., Johnson, W. O. et al.: Factors associated with prevalent and incident urinary incontinence in a cohort of midlife women: a longitudinal analysis of data: study of women's health across the nation. Am J Epidemiol, 165: 309, 2007

92. Rohr, G., Stovring, H., Christensen, K. et al.: Characteristics of middle-aged and elderly women with urinary incontinence. Scand J Prim Health Care, 23: 203, 2005

93. Moghaddas, F., Lidfeldt, J., Nerbrand, C. et al.: Prevalence of urinary incontinence in relation to self-reported depression, intake of serotonergic antidepressants, and hormone therapy in middle-aged women: a report from the Women's Health in the Lund Area study. Menopause, 12: 318, 2005

94. Lewis, C. M., Schrader, R., Many, A. et al.: Diabetes and urinary incontinence in 50- to 90-year-old women: a cross-sectional population-based study. Am J Obstet Gynecol, 193: 2154, 2005

95. Mardon, R. E., Halim, S., Pawlson, L. G. et al.: Management of urinary incontinence in Medicare managed care beneficiaries: results from the 2004 Medicare Health Outcomes Survey. Arch Intern Med, 166: 1128, 2006

96. Goode, P. S., Burgio, K. L., Redden, D. T. et al.: Population based study of incidence and predictors of urinary incontinence in black and white older adults. J Urol, 179: 1449, 2008

97. Anger, J. T., Saigal, C. S., Litwin, M. S.: The prevalence of urinary incontinence among community dwelling adult women: results from the National Health and Nutrition Examination Survey. J Urol, 175: 601, 2006

98. Hsieh, C. H., Su, T. H., Chang, S. T. et al.: Prevalence of and attitude toward urinary incontinence in postmenopausal women. Int J Gynaecol Obstet, 100: 171, 2008

99. Ostbye, T., Seim, A., Krause, K. M. et al.: A 10-year follow-up of urinary and fecal incontinence among the oldest old in the community: the Canadian Study of Health and Aging. Can J Aging, 23: 319, 2004

100. Adelmann, P. K.: Prevalence and detection of urinary incontinence among older Medicaid recipients. J Health Care Poor Underserved, 15: 99, 2004

101. Holroyd-Leduc, J. M., Mehta, K. M., Covinsky, K. E.: Urinary incontinence and its association with death, nursing home admission, and functional decline. J Am Geriatr Soc, 52: 712, 2004

102. Stenzelius, K., Mattiasson, A., Hallberg, I. R. et al.: Symptoms of urinary and faecal incontinence among men and women 75+ in relations to health complaints and quality of life. Neurourol Urodyn, 23: 211, 2004

103. Diokno, A. C., Brock, B. M., Brown, M. B. et al.: Prevalence of urinary incontinence and other urological symptoms in the noninstitutionalized elderly. J Urol, 136: 1022, 1986

104. Molander, U., Milsom, I., Ekelund, P. et al.: An epidemiological study of urinary incontinence and related urogenital symptoms in elderly women. Maturitas, 12: 51, 1990

105. Roberts, R. O., Jacobsen, S. J., Rhodes, T. et al.: Urinary incontinence in a community-based cohort: prevalence and healthcare-seeking. J Am Geriatr Soc, 46: 467, 1998

106. Nuotio, M., Jylhèa, M., Luukkaala, T. et al.: Urinary incontinence in a Finnish population aged 70 and over. Prevalence of types, associated factors and self-reported treatments. Scand J Prim Health Care, 21: 182, 2003

107. H¢rding, U., Pedersen, K. H., Sidenius, K. et al.: Urinary incontinence in 45-year-old women. An epidemiological survey. Scand J Urol Nephrol, 20: 183, 1986

108. Burgio, K. L., Matthews, K. A., Engel, B. T.: Prevalence, incidence and correlates of urinary incontinence in healthy, middle-aged women. J Urol, 146: 1255, 1991

109. Samuelsson, E., Victor, A., Tibblin, G.: A population study of urinary incontinence and nocturia among women aged 20-59 years. Prevalence, well-being and wish for treatment. Acta Obstet Gynecol Scand, 76: 74, 1997

110. Hunskaar, S., Lose, G., Sykes, D. et al.: The prevalence of urinary incontinence in women in four European countries. BJU Int, 93: 324, 2004

111. Sampselle, C. M., Harlow, S. D., Skurnick, J. et al.: Urinary incontinence predictors and life impact in ethnically diverse perimenopausal women. Obstet Gynecol, 100: 1230, 2002

112. Aggazzotti, G., Pesce, F., Grassi, D. et al.: Prevalence of urinary incontinence among institutionalized patients: a cross-sectional epidemiologic study in a midsized city in northern Italy. Urology, 56: 245, 2000

113. Saxer, S., Halfens, R. J., de Bie, R. A. et al.: Prevalence and incidence of urinary incontinence of Swiss nursing home residents at admission and after six, 12 and 24 months. J Clin Nurs, 2008

114. Ouslander, J. G., Kane, R. L., Abrass, I. B.: Urinary incontinence in elderly nursing home patients. Jama, 248: 1194, 1982

115. Hunskaar, S., Ostbye, T., Borrie, M. J.: Prevalence of urinary incontinence in elderly Canadians with special emphasis on the association with dementia, ambulatory function, and institutionalization. Norwegian J Epidemiol, 8: 177, 1998

116. Sgadari, A., Topinkova, E., Bjornson, J. et al.: Urinary incontinence in nursing home residents: a cross-national comparison. Age Ageing, 26 Suppl 2: 49, 1997

117. Ouslander, J. G., Schnelle, J. F.: Incontinence in the nursing home. Ann Intern Med, 122: 438, 1995

118. Anger, J. T., Saigal, C. S., Pace, J. et al.: True prevalence of urinary incontinence among female nursing home residents. Urology, 67: 281, 2006

119. Jumadilova, Z., Zyczynski, T., Paul, B. et al.: Urinary incontinence in the nursing home: resident characteristics

and prevalence of drug treatment. Am J Manag Care, 11: S112, 2005

120. Dimpfl, T., Hesse, U., Schussler, B.: Incidence and cause of postpartum urinary stress incontinence. Eur J Obstet Gynecol Reprod Biol, 43: 29, 1992

121. Marshall, K., Thompson, K. A., Walsh, D. M. et al.: A survey of the incidence of urinary incontinence and constipation during pregnancy and post-partum. Neurourol Urodyn, 17: 429, 1998

122. Morkved, S., Bo, K.: Prevalence of urinary incontinence during pregnancy and postpartum. Int Urogynecol J Pelvic Floor Dysfunct, 10: 394, 1999

123. Hvidman, L., Foldspang, A., Mommsen, S. et al.: Correlates of urinary incontinence in pregnancy. Int Urogynecol J Pelvic Floor Dysfunct, 13: 278, 2002

124. Burgio, K. L., Locher, J. L., Zyczynski, H. et al.: Urinary incontinence during pregnancy in a racially mixed sample: characteristics and predisposing factors. Int Urogynecol J Pelvic Floor Dysfunct, 7: 69, 1996

125. Wesnes, S. L., Rortveit, G., Bo, K. et al.: Urinary incontinence during pregnancy. Obstet Gynecol, 109: 922, 2007

126. Glazener, C. M., Herbison, G. P., MacArthur, C. et al.: New postnatal urinary incontinence: obstetric and other risk factors in primiparae. BJOG, 113: 208, 2006

127. Borello-France, D., Burgio, K. L., Richter, H. E. et al.: Fecal and urinary incontinence in primiparous women. Obstet Gynecol, 108: 863, 2006

128. Altman, D., Ekstrom, A., Gustafsson, C. et al.: Risk of urinary incontinence after childbirth: a 10-year prospective cohort study. Obstet Gynecol, 108: 873, 2006

129. Chaliha, C., Kalia, V., Stanton, S. L. et al.: Antenatal prediction of postpartum urinary and fecal incontinence. Obstet Gynecol, 94: 689, 1999

130. Eason, E., Labrecque, M., Marcoux, S. et al.: Effects of carrying a pregnancy and of method of delivery on urinary incontinence: a prospective cohort study. BMC Pregnancy Childbirth, 4: 4, 2004

131. Eftekhar, T., Hajibaratali, B., Ramezanzadeh, F. et al.: Postpartum evaluation of stress urinary incontinence among primiparas. Int J Gynaecol Obstet, 94: 114, 2006

132. Ekstrom, A., Altman, D., Wiklund, I. et al.: Planned cesarean section versus planned vaginal delivery: comparison of lower urinary tract symptoms. Int Urogynecol J Pelvic Floor Dysfunct, 19: 459, 2008

133. Pregazzi, R., Sartore, A., Troiano, L. et al.: Postpartum urinary symptoms: prevalence and risk factors. Eur J Obstet Gynecol Reprod Biol, 103: 179, 2002

134. Farrell, S. A., Allen, V. M., Baskett, T. F.: Parturition and urinary incontinence in primiparas. Obstet Gynecol, 97: 350, 2001

135. Groutz, A., Rimon, E., Peled, S. et al.: Cesarean section: does it really prevent the development of postpartum stress urinary incontinence? A prospective study of 363 women one year after their first delivery. Neurourol Urodyn, 23: 2, 2004

136. Pregazzi, R., Sartore, A., Bortoli, P. et al.: Immediate postpartum perineal examination as a predictor of puerperal pelvic floor dysfunction. Obstet Gynecol, 99: 581, 2002

137. Wijma, J., Potters, A. E., de Wolf, B. T. et al.: Anatomical and functional changes in the lower urinary tract following spontaneous vaginal delivery. BJOG, 110: 658, 2003

138. Arya, L. A., Jackson, N. D., Myers, D. L. et al.: Risk of new-onset urinary incontinence after forceps and vacuum delivery in primiparous women. Am J Obstet Gynecol, 185: 1318, 2001

139. Schytt, E., Lindmark, G., Waldenstrom, U.: Symptoms of stress incontinence 1 year after childbirth: prevalence and predictors in a national Swedish sample. Acta Obstet Gynecol Scand, 83: 928, 2004

140. Fultz, N., Herzog, A. R., Raghunathan, T. E. et al.: Prevalence and Severity of Urinary Incontinence in Older African And Caucasian Women. Journal of Gerontology: Medical Sciences, 54A: M299, 1999

141. Nygaard, I., Turvey, C., Burns, T. L. et al.: Urinary incontinence and depression in middle-aged United States women. Obstet Gynecol, 101: 149, 2003

142. Grodstein, F., Fretts, R., Lifford, K. et al.: Association of age, race, and obstetric history with urinary symptoms among women in the Nurses' Health Study. Am J Obstet Gynecol, 189: 428, 2003

143. Jackson, R. A., Vittinghoff, E., Kanaya, A. M. et al.: Urinary incontinence in elderly women: findings from the Health, Aging, and Body Composition Study. Obstet Gynecol, 104: 301, 2004

144. Dooley, Y., Kenton, K., Cao, G. et al.: Urinary incontinence prevalence: results from the National Health and Nutrition Examination Survey. J Urol, 179: 656, 2008

145. Fenner, D. E., Trowbridge, E. R., Patel, D. L. et al.: Establishing the prevalence of incontinence study: racial differences in women's patterns of urinary incontinence. J Urol, 179: 1455, 2008

146. Markland, A. D., Gerety, M. B., Goode, P. S. et al.: Urinary incontinence in community-dwelling older Mexican American and European American women. Arch Gerontol Geriatr, 2008

147. Markland, A. D., Goode, P. S., Burgio, K. L. et al.: Correlates of urinary, fecal, and dual incontinence in older African-American and white men and women. J Am Geriatr Soc, 56: 285, 2008

148. Daneshgari, F., Imrey, P. B., Risendal, B. et al.: Differences in urinary incontinence between Hispanic and non-Hispanic white women: a population-based study. BJU Int, 101: 575, 2008

149. Nygaard, I., Turvey, C., Burns, T. L. et al.: Urinary incontinence and depression in middle-aged United States women. Obstet Gynecol, 101: 149, 2003

150. Howard, D., Delancey, J. O., Tunn, R. et al.: Racial differences in the structure and function of the stress urinary continence mechanism. Obstet Gynecol, 95: 713, 2000

151. Knobel, J.: Stress incontinence in the Black female. S Afr Med J, 49: 430, 1975

152. Wehrberger, C., Temml, C., Ponholzer, A. et al.: Incidence and remission of female urinary incontinence over 6.5 years: analysis of a health screening project. Eur Urol, 50: 327, 2006

153. Samuelsson, E. C., Victor, F. T., Svardsudd, K. F.: Five-year incidence and remission rates of female urinary incontinence in a Swedish population less than 65 years old. Am J Obstet Gynecol, 183: 568, 2000

154. Hagglund, D., Walker-Engstrom, M. L., Larsson, G. et al.: Changes in urinary incontinence and quality of life after four years. A population-based study of women aged 22-50 years. Scand J Prim Health Care, 22: 112, 2004

155. Townsend, M. K., Danforth, K. N., Lifford, K. L. et al.: Incidence and remission of urinary incontinence in middle-aged women. Am J Obstet Gynecol, 197: 167 e1, 2007

156. Dallosso, H., Matthews, R., McGrother, C. et al.: Diet as a risk factor for the development of stress urinary incontinence: a longitudinal study in women. Eur J Clin Nutr, 58: 920, 2004

157. McGrother, C. W., Donaldson, M. M., Shaw, C. et al.: Storage symptoms of the bladder: prevalence, incidence and need for services in the UK. BJU Int, 93: 763, 2004

158. Liu, C., Andrews, G. R.: Prevalence and incidence of urinary incontinence in the elderly: a longitudinal study in South Australia. Chin Med J (Engl), 115: 119, 2002

159. Maggi, S., Minicuci, N., Langlois, J. et al.: Prevalence rate of urinary incontinence in community-dwelling elderly individuals: the Veneto study. J Gerontol A Biol Sci Med Sci, 56: M14, 2001

160. Chiarelli, P., Brown, W., McElduff, P.: Leaking Urine: Prevalence and Associated Factors in Australian Women. Neurourol Urodyn, 18: 567, 1999

161. Nygaard, I. E., Lemke, J. H.: Urinary incontinence in rural older women: prevalence, incidence and remission. J Am Geriatr Soc, 44: 1049, 1996

162. Brown, J. S., Seeley, D. G., Fong, J. et al.: Urinary incontinence in older women: who is at risk? Study of Osteoporotic Fractures Research Group. Obstet Gynecol, 87: 715, 1996

163. Milsom, I., Ekelund, P., Molander, U. et al.: The influence of age, parity, oral contraception, hysterectomy and menopause on the prevalence of urinary incontinence in women. J Urol, 149: 1459, 1993

164. Dolan, L. M., Casson, K., McDonald, P. et al.: Urinary incontinence in Northern Ireland: a prevalence study. BJU Int, 83: 760, 1999

165. Roe, B., Doll, H.: Lifestyle Factors and Continence Status: Comparison of Self-Report Data from a Postal Survey in England. J WOCN, 26: 312, 1999

166. Schmidbauer, J., Temml, C., Schatzl, G. et al.: Risk factors for urinary incontinence in both sexes. Analysis of a health screening project. Eur Urol, 39: 565, 2001

167. Peyrat, L., Haillot, O., Bruyere, F. et al.: Prevalence and risk factors of urinary incontinence in young and middle-aged women. BJU Int, 89: 61, 2002

168. Goldberg, R. P., Kwon, C., Gandhi, S. et al.: Urinary incontinence among mothers of multiples: the protective effect of cesarean delivery. Am J Obstet Gynecol, 188: 1447, 2003

169. Diokno, A. C., Brown, M. B., Brock, B. M. et al.: Clinical and cystometric characteristics of continent and incontinent noninstitutionalized elderly. J Urol, 140: 567, 1988

170. Staskin, D. R.: Age-related physiologic and pathologic changes affecting lower urinary tract function. Clin Geriatr Med, 2: 701, 1986

171. Burgio, K. L., Zyczynski, H., Locher, J. L. et al.: Urinary incontinence in the 12-month postpartum period. Obstet Gynecol, 102: 1291, 2003

172. Hvidman, L., Foldspang, A., Mommsen, S. et al.: Postpartum urinary incontinence. Acta Obstet Gynecol Scand, 82: 556, 2003

173. Viktrup, L., Lose, G.: The risk of stress incontinence 5 years after first delivery. Am J Obstet Gynecol, 185: 82, 2001

174. Viktrup, L., Rortveit, G., Lose, G.: Risk of stress urinary incontinence twelve years after the first pregnancy and delivery. Obstet Gynecol, 108: 248, 2006

175. Sommer, P., Bauer, T., Nielsen, K. K. et al.: Voiding patterns and prevalence of incontinence in women. A questionnaire survey. Br J Urol, 66: 12, 1990

176. Elving, L. B., Foldspang, A., Lam, G. W. et al.: Descriptive epidemiology of urinary incontinence in 3,100 women age 30-59. Scand J Urol Nephrol, 125 (suppl): 37, 1989

177. Harrison, G. L., Memel, D. S.: Urinary incontinence in women: its prevalence and its management in a health promotion clinic. Br J Gen Pract, 44: 149, 1994

178. Simeonova, Z., Bengtsson, C.: Prevalence of urinary incontinence among women at a Swedish primary health care centre. Scand J Prim Health Care, 8: 203, 1990

179. Jolleys, J. V.: Reported prevalence of urinary incontinence in women in a general practice. Br Med J, 296: 1300, 1988

180. Yarnell, J. W., Voyle, G. J., Sweetnam, P. M. et al.: Factors associated with urinary incontinence in women. J Epidemiol Community Health, 36: 58, 1982

181. Crist, T., Shingleton, H. M., Koch, G. G.: Stress incontinence and the nulliparous patient. Obstet Gynecol Clin North Am, 40: 13, 1972

182. Foldspang, A., Mommsen, S., Lam, G. W. et al.: Parity as a correlate of adult female urinary incontinence prevalence. J Epidemiol Community Health, 46: 595, 1992

183. Persson, J., Wolner-Hanssen, P., Rydhstroem, H.: Obstetric risk factors for stress urinary incontinence: a population-based study. Obstetrics & Gynecology, 96: 440, 2000

184. Sherburn, M., Guthrie, J. R., Dudley, E. C. et al.: Is incontinence associated with menopause? Obstet Gynecol, 98: 628, 2001

185. Rortveit, G., Hannestad, Y. S., Daltveit, A. K. et al.: Age- and type-dependent effects of parity on urinary incontinence: the Norwegian EPINCONT study. Obstet Gynecol, 98: 1004, 2001

186. Faâundes, A., Guarisi, T., Pinto-Neto, A. M.: The risk of urinary incontinence of parous women who delivered only by cesarean section. Int J Gynaecol Obstet, 72: 41, 2001

187. Alling M_ller, L., Lose, G., J_rgensen, T.: Risk factors for lower urinary tract symptoms in women 40 to 60 years of age. Obstet Gynecol, 96: 446, 2000

188. Kuh, D., Cardozo, L., Hardy, R.: Urinary incontinence in middle aged women: childhood enuresis and other lifetime risk factors in a British prospective cohort. Journal of Epidemiology & Community Health, 53: 453, 1999

189. Samuelsson, E., Victor, A., Svardsudd, K.: Determinants of urinary incontinence in a population of young and middle-aged women. Acta Obstetricia et Gynecologica Scandinavica, 79: 208, 2000

190. Pregazzi, R., Sartore, A., Troiano, L. et al.: Postpartum urinary symptoms: prevalence and risk factors. Eur J Obstet Gynecol Reprod Biol, 103: 179, 2002

191. Handa, V. L., Harris, T. A., Ostergard, D. R.: Protecting the pelvic floor: obstetric management to prevent incontinence and pelvic organ prolapse. Obstet Gynecol, 88: 470, 1996

192. Viktrup, L., Lose, G., Rolff, M. et al.: The symptom of stress incontinence caused by pregnancy or delivery in primiparas. Obstet Gynecol, 79: 945, 1992

193. H_jberg, K. E., Salvig, J. D., Winsl_w, N. A. et al.: Urinary incontinence: prevalence and risk factors at 16 weeks of gestation. Br J Obstet Gynaecol, 106: 842, 1999

194. Wilson, P. D., Herbison, R. M., Herbison, G. P.: Obstetric practice and the prevalence of urinary incontinence three months after delivery. Br J Obstet Gynaecol, 103: 154, 1996

195. Mason, L., Glenn, S., Walton, I. et al.: The prevalence of stress incontinence during pregnancy and following delivery. Midwifery, 15: 120, 1999

196. MacLennan, A. H., Taylor, A. W., Wilson, D. H. et al.: The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. Bjog, 107: 1460, 2000

197. Hannah, M. E., Hannah, W. J., Hodnett, E. D. et al.: Outcomes at 3 months after planned cesarean vs planned vaginal delivery for breech presentation at term: the international randomized Term Breech Trial. Jama, 287: 1822, 2002

198. Rortveit, G., Daltveit, A. K., Hannestad, Y. S. et al.: Urinary incontinence after vaginal delivery or cesarean section. N Engl J Med, 348: 900, 2003

199. Chaliha, C., Digesu, A., Hutchings, A. et al.: Caesarean section is protective against stress urinary incontinence: an analysis of women with multiple deliveries. Bjog, 111: 754, 2004

200. Altman, D., Ekstrom, A., Forsgren, C. et al.: Symptoms of anal and urinary incontinence following cesarean section or spontaneous vaginal delivery. Am J Obstet Gynecol, 197: 512 e1, 2007

201. Press, J. Z., Klein, M. C., Kaczorowski, J. et al.: Does cesarean section reduce postpartum urinary incontinence? A systematic review. Birth, 34: 228, 2007

202. Rortveit, G., Hunskaar, S.: Urinary incontinence and age at the first and last delivery: the Norwegian HUNT/EPINCONT study. Am J Obstet Gynecol, 195: 433, 2006

203. Kuh, D., Cardozo, L., Hardy, R.: Urinary incontinence in middle aged women: childhood enuresis and other lifetime risk factors in a British prospective cohort. J Epidemiol Community Health, 53: 453, 1999

204. Miller, Y. D., Brown, W. J., Russell, A. et al.: Urinary incontinence across the lifespan. Neurourol Urodyn, 22: 550, 2003

205. Thom, D. H., van den Eeden, S. K., Brown, J. S.: Evaluation of parturition and other reproductive variables as risk factors for urinary incontinence in later life. Obstet Gynecol, 90: 983, 1997

206. Foldspang, A., Mommsen, S., Djurhuus, J. C.: Prevalent urinary incontinence as a correlate of pregnancy, vaginal childbirth, and obstetric techniques. Am J Public Health, 89: 209, 1999

207. Chaliha, C., Kalia, V., Stanton, S. L. et al.: Antenatal prediction of postpartum urinary and fecal incontinence. Obstet Gynecol, 94: 689, 1999

208. Rortveit, G., Daltveit, A. K., Hannestad, Y. S. et al.: Vaginal delivery parameters and urinary incontinence: the Norwegian EPINCONT study. Am J Obstet Gynecol, 189: 1268, 2003

209. Danso, K. A., Martey, J. O., Wall, L. L. et al.: The epidemiology of genitourinary fistulae in Kumasi, Ghana 1977-1992. International Urogynecology Journal, 7: 117, 1996

210. Han, M. O., Lee, N. Y., Park, H. S.: Abdominal obesity is associated with stress urinary incontinence in Korean women. Int Urogynecol J Pelvic Floor Dysfunct, 17: 35, 2006

211. Bortolotti, A., Bernardini, B., Colli, E. et al.: Prevalence and risk factors for urinary incontinence in Italy. Eur Urol, 37: 30, 2000

212. Holtedahl, K., Hunskaar, S.: Prevalence, 1-year incidence and factors associated with urinary incontinence: a population based study of women 50-74 years of age in primary care. Maturitas, 28: 205, 1998

213. Brown, J. S., Grady, D., Ouslander, J. G. et al.: Prevalence of urinary incontinence and associated risk factors in postmenopausal women. Heart & Estrogen/Progestin Replacement Study (HERS) Research Group. Obstet Gynecol, 94: 66, 1999

214. Mommsen, S., Foldspang, A.: Body mass index and adult female urinary incontinence. World J Urol, 12: 319, 1994

215. Hvidman, L., Foldspang, A., Mommsen, S. et al.: Menstrual cycle, female hormone use and urinary incontinence in premenopausal women. Int Urogynecol J Pelvic Floor Dysfunct, 14: 56, 2003

216. Buchsbaum, G. M., Chin, M., Glantz, C. et al.: Prevalence of urinary incontinence and associated risk factors in a cohort of nuns. Obstet Gynecol, 100: 226, 2002

217. Hvidman, L., Foldspang, A., Mommsen, S. et al.: Correlates of urinary incontinence in pregnancy. Int Urogynecol J Pelvic Floor Dysfunct, 13: 278, 2002

218. Hannestad, Y. S., Rortveit, G., Daltveit, A. K. et al.: Are smoking and other lifestyle factors associated with female urinary incontinence? The Norwegian EPINCONT Study. Bjog, 110: 247, 2003

219. Huang, A. J., Thom, D. H., Kanaya, A. M. et al.: Urinary incontinence and pelvic floor dysfunction in Asian-American women. Am J Obstet Gynecol, 195: 1331, 2006

220. Brown, J. S., Wing, R., Barrett-Connor, E. et al.: Lifestyle intervention is associated with lower prevalence of urinary incontinence: the Diabetes Prevention Program. Diabetes Care, 29: 385, 2006

221. Jackson, S. L., Scholes, D., Boyko, E. J. et al.: Predictors of urinary incontinence in a prospective cohort of postmenopausal women. Obstet Gynecol, 108: 855, 2006

222. Azuma, R., Murakami, K., Iwamoto, M. et al.: Prevalence and risk factors of urinary incontinence and its influence on the quality of life of Japanese women. Nurs Health Sci, 10: 151, 2008

223. Townsend, M. K., Danforth, K. N., Rosner, B. et al.: Body mass index, weight gain, and incident urinary incontinence in middle-aged women. Obstet Gynecol, 110: 346, 2007

224. Bump, R. C., Sugerman, H. J., Fantl, J. A. et al.: Obesity and lower urinary tract function in women: surgically induced weight loss. Am J Obstet Gynecol, 167: 392, 1992

225. Deitel, M., Stone, E., Kassam, H. A. et al.: Gynecologic-obstetric changes after loss of massive excess weight following bariatric surgery. J Am Coll Nutr, 7: 147, 1988

226. Burgio, K. L., Richter, H. E., Clements, R. H. et al.: Changes in urinary and fecal incontinence symptoms with weight loss surgery in morbidly obese women. Obstet Gynecol, 110: 1034, 2007

227. Subak, L. L., Whitcomb, E., Shen, H. et al.: Weight loss: a novel and effective treatment for urinary incontinence. J Urol, 174: 190, 2005

228. Brown, J. S., Vittinghoff, E., Lin, F. et al.: Prevalence and risk factors for urinary incontinence in women with type 2 diabetes and impaired fasting glucose: findings from the National Health and Nutrition Examination Survey (NHANES) 2001-2002. Diabetes Care, 29: 1307, 2006

229. Brown, J. S., Seeley, D. G., Fong, J. et al.: Urinary incontinence in older women: who is at risk? Study of Osteoporotic Fractures Research Group. Obstet Gynecol, 87: 715, 1996

230. Diokno, A. C.: Diagnostic categories of incontinence and the role of urodynamic testing. J Am Geriatr Soc, 38: 300, 1990

231. Grady, D., Brown, J. S., Vittinghoff, E. et al.: Postmenopausal hormones and incontinence: the Heart and Estrogen/Progestin Replacement Study. Obstet Gynecol, 97: 116, 2001

232. Steinauer, J. E., Waetjen, L. E., Vittinghoff, E. et al.: Postmenopausal hormone therapy: does it cause incontinence? Obstet Gynecol, 106: 940, 2005

233. Hendrix, S. L., Cochrane, B. B., Nygaard, I. E. et al.: Effects of estrogen with and without progestin on urinary incontinence. JAMA, 293: 935, 2005

234. Hendrix, S. L., McNeeley, S. G.: Effect of selective estrogen receptor modulators on reproductive tissues other than endometrium. Ann N Y Acad Sci, 949: 243, 2001

235. Goldstein, S. R., Nanavati, N.: Adverse events that are associated with the selective estrogen receptor modulator levormeloxifene in an aborted phase III osteoporosis treatment study. Am J Obstet Gynecol, 187: 521, 2002

236. Waetjen, L. E., Brown, J. S., Modelska, K. et al.: Effect of raloxifene on urinary incontinence: a randomized controlled trial. Obstet Gynecol, 103: 261, 2004

237. Lifford, K. L., Curhan, G. C., Hu, F. B. et al.: Type 2 diabetes

mellitus and risk of developing urinary incontinence. J Am Geriatr Soc, 53: 1851, 2005

238. Ebbesen, M. H., Hannestad, Y. S., Midthjell, K. et al.: Diabetes and urinary incontinence - prevalence data from Norway. Acta Obstet Gynecol Scand: 1, 2007

239. Waetjen, L. E., Feng, W. Y., Ye, J. et al.: Factors associated with worsening and improving urinary incontinence across the menopausal transition. Obstet Gynecol, 111: 667, 2008

240. Taylor, T., Smith, A. N., Fulton, M.: Effects of hysterectomy on bowel and bladder function. Int J Colorectal Dis, 5: 228, 1990

241. Parys, B. T., Haylen, B. T., Hutton, J. L. et al.: The effects of simple hysterectomy on vesicourethral function. Br J Urol, 64: 594, 1989

242. Parys, B. T., Woolfenden, K. A., Parsons, K. F.: Bladder dysfunction after simple hysterectomy: urodynamic and neurological evaluation. Eur Urol, 17: 129, 1990

243. Coughlan, B. M., Smith, J. M., Moriarity, C. T.: Does simple hysterectomy affect lower urinary tract function--a urodynamic investigation. Ir J Med Sci, 158: 215, 1989

244. Kujansuu, E., Teisala, K., Punnonen, R.: Urethral closure function after total and subtotal hysterectomy measured by urethrocystometry. Gynecol Obstet Invest, 27: 105, 1989

245. Farquharson, D. I., Shingleton, H. M., Orr, J. W., Jr. et al.: The short-term effect of radical hysterectomy on urethral and bladder function. Br J Obstet Gynaecol, 94: 351, 1987

246. Vervest, H. A., van Venrooij, G. E., Barents, J. W. et al.: Non-radical hysterectomy and the function of the lower urinary tract. I: Urodynamic quantification of changes in storage function. Acta Obstet Gynecol Scand, 68: 221, 1989

247. Hansen, B. M., Bonnesen, T., Hvidberg, J. E. et al.: Changes in symptoms and colpo-cystourethrography in 35 patients before and after total abdominal hysterectomy: a prospective study. Urol Int, 40: 224, 1985

248. Virtanen, H., Makinen, J., Tenho, T. et al.: Effects of abdominal hysterectomy on urinary and sexual symptoms. Br J Urol, 72: 868, 1993

249. Carlson, K. J., Miller, B. A., Fowler, F. J., Jr.: The Maine Women's Health Study: I. Outcomes of hysterectomy. Obstet Gynecol, 83: 556, 1994

250. Griffith-Jones, M. D., Jarvis, G. J., McNamara, H. M.: Adverse urinary symptoms after total abdominal hysterectomy--fact or fiction? Br J Urol, 67: 295, 1991

251. Bhattacharya, S., Mollison, J., Pinion, S. et al.: A comparison of bladder and ovarian function two years following hysterectomy or endometrial ablation. Br J Obstet Gynaecol, 103: 898, 1996

252. Iosif, C. S., Bekassy, Z., Rydhstrom, H.: Prevalence of urinary incontinence in middle-aged women. Int J Gynaecol Obstet, 26: 255, 1988

253. Neumann, G. A., Lauszus, F. F., Ljungstrom, B. et al.: Incidence and remission of urinary incontinence after hysterectomy--a 3-year follow-up study. Int Urogynecol J Pelvic Floor Dysfunct, 18: 379, 2007

254. Gimbel, H., Zobbe, V., Andersen, B. M. et al.: Randomised controlled trial of total compared with subtotal hysterectomy with one-year follow up results. Bjog, 110: 1088, 2003

255. Altman, D., Granath, F., Cnattingius, S. et al.: Hysterectomy and risk of stress-urinary-incontinence surgery: nationwide cohort study. Lancet, 370: 1494, 2007

256. Lifford, K. L., Townsend, M. K., Curhan, G. C. et al.: The Epidemiology of Urinary Incontinence in Older Women: Incidence, Progression, and Remission. J Am Geriatr Soc, 2008

257. Tseng, I. J., Chen, Y. T., Chen, M. T. et al.: Prevalence of urinary incontinence and intention to seek treatment in the elderly. J Formos Med Assoc, 99: 753, 2000

258. Chen, G. D., Lin, T. L., Hu, S. W. et al.: Prevalence and correlation of urinary incontinence and overactive bladder in Taiwanese women. Neurourol Urodyn, 22: 109, 2003

259. Nuotio, M., Jylha, M., Luukkaala, T. et al.: Urinary incontinence in a Finnish population aged 70 and over. Prevalence of types, associated factors and self-reported treatments. Scand J Prim Health Care, 21: 182, 2003

260. Parazzini, F., Chiaffarino, F., Lavezzari, M. et al.: Risk factors for stress, urge or mixed urinary incontinence in Italy. Bjog, 110: 927, 2003

261. Buchsbaum, G. M., Chin, M., Glantz, C. et al.: Prevalence of urinary incontinence and associated risk factors in a cohort of nuns. Obstet Gynecol, 100: 226, 2002

262. Van Oyen, H., Van Oyen, P.: Urinary incontinence in Belgium; prevalence, correlates and psychosocial consequences. Acta Clin Belg, 57: 207, 2002

263. Ozerdogan, N., Beji, N. K., Yalcin, O.: Urinary incontinence: its prevalence, risk factors and effects on the quality of life of women living in a region of Turkey. Gynecol Obstet Invest, 58: 145, 2004

264. Oskay, U. Y., Beji, N. K., Yalcin, O.: A study on urogenital complaints of postmenopausal women aged 50 and over. Acta Obstet Gynecol Scand, 84: 72, 2005

265. Kocak, I., Okyay, P., Dundar, M. et al.: Female urinary incontinence in the west of Turkey: prevalence, risk factors and impact on quality of life. Eur Urol, 48: 634, 2005

266. Moore, E. E., Jackson, S. L., Boyko, E. J. et al.: Urinary incontinence and urinary tract infection: temporal relationships in postmenopausal women. Obstet Gynecol, 111: 317, 2008

267. Brandeis, G. H., Baumann, M. M., Hossain, M. et al.: The prevalence of potentially remediable urinary incontinence in frail older people: a study using the Minimum Data Set. J Am Geriatr Soc, 45: 179, 1997

268. Herzog, A. R., Fultz, N. H., Brock, B. M. et al.: Urinary incontinence and psychological distress among older adults. Psychol Aging, 3: 115, 1988

269. Tinetti, M. E., Inouye, S. K., Gill, T. M. et al.: Shared risk factors for falls, incontinence, and functional dependence. Unifying the approach to geriatric syndromes :see comments:. Jama, 273: 1348, 1995

270. Nelson, R., Furner, S., Jesudason, V.: Urinary incontinence in Wisconsin skilled nursing facilities: prevalence and associations in common with fecal incontinence. J Aging Health, 13: 539, 2001

271. Landi, F., Cesari, M., Russo, A. et al.: Potentially reversible risk factors and urinary incontinence in frail older people living in community. Age Ageing, 32: 194, 2003

272. Palmer, M. H., Baumgarten, M., Langenberg, P. et al.: Risk factors for hospital-acquired incontinence in elderly female hip fracture patients. J Gerontol A Biol Sci Med Sci, 57: M672, 2002

273. McGrother, C. W., Donaldson, M. M., Hayward, T. et al.: Urinary storage symptoms and comorbidities: a prospective population cohort study in middle-aged and older women. Age Ageing, 35: 16, 2006

274. McGrother, C. W., Donaldson, M. M., Thompson, J. R.: Re: "Prevalence of and risk factors for urine leakage in a racially and ethnically diverse population of adults: the Boston Area Community Health (BACH) Survey". Am J Epidemiol, 168: 234; author reply 234, 2008

275. Huang, A. J., Brown, J. S., Thom, D. H. et al.: Urinary incontinence in older community-dwelling women: the role of cognitive and physical function decline. Obstet Gynecol, 109: 909, 2007

276. Borrie, M. J., Davidson, H. A.: Incontinence in institutions:

costs and contributing factors. Can Med Assoc J, 147: 322, 1992

277. Ouslander, J. G., Morishita, L., Blaustein, J. et al.: Clinical, functional, and psychosocial characteristics of an incontinent nursing home population. J Gerontol, 42: 631, 1987

278. Ouslander, J. G., Uman, G. C., Urman, H. N. et al.: Incontinence among nursing home patients: clinical and functional correlates. J Am Geriatr Soc, 35: 324, 1987

279. Rait, G., Fletcher, A., Smeeth, L. et al.: Prevalence of cognitive impairment: results from the MRC trial of assessment and management of older people in the community. Age Ageing, 34: 242, 2005

280. Wetle, T., Scherr, P., Branch, L. G. et al.: Difficulty with holding urine among older persons in a geographically defined community: prevalence and correlates. J Am Geriatr Soc, 43: 349, 1995

281. Espey, M. J., Du, H. J., Downie, J. W.: Serotonergic modulation of spinal ascending activity and sacral reflex activity evoked by pelvic nerve stimulation in cats. Brain Res, 798: 101, 1998

282. Iosif, C. S., Bekassy, Z.: Prevalence of genito-urinary symptoms in the late menopause. Acta Obstet Gynecol Scand, 63: 257, 1984

283. Versi, E.: Incontinence in the climacteric. Clin Obstet Gynecol, 33: 392, 1990

284. Nygaard, I., Girts, T., Fultz, N. H. et al.: Is urinary incontinence a barrier to exercise in women? Obstet Gynecol, 106: 307, 2005

285. Danforth, K. N., Shah, A. D., Townsend, M. K. et al.: Physical activity and urinary incontinence among healthy, older women. Obstet Gynecol, 109: 721, 2007

286. Bump, R. C., McClish, D. K.: Cigarette smoking and urinary incontinence in women. Am J Obstet Gynecol, 167: 1213, 1992

287. Swanson, J. G., Kaczorowski, J., Skelly, J. et al.: Urinary incontinence: common problem among women over 45. Can Fam Physician, 51: 84, 2005

288. Fultz, N. H., Herzog, A. R., Raghunathan, T. E. et al.: Prevalence and severity of urinary incontinence in older African American and Caucasian women. J Gerontol A Biol Sci Med Sci, 54: M299, 1999

289. Arya, L. A., Myers, D. L., Jackson, N. D.: Dietary caffeine intake and the risk for detrusor instability: a case-control study. Obstet Gynecol, 96: 85, 2000

290. Teleman, P. M., Lidfeldt, J., Nerbrand, C. et al.: Overactive bladder: prevalence, risk factors and relation to stress incontinence in middle-aged women. BJOG, 111: 600, 2004

291. Dallosso, H. M., McGrother, C. W., Matthews, R. J. et al.: The association of diet and other lifestyle factors with overactive bladder and stress incontinence: a longitudinal study in women. BJU Int, 92: 69, 2003

292. Rohr, G., Kragstrup, J., Gaist, D. et al.: Genetic and environmental influences on urinary incontinence: a Danish population-based twin study of middle-aged and elderly women. Acta Obstet Gynecol Scand, 83: 978, 2004

293. Dietz, H. P., Hansell, N. K., Grace, M. E. et al.: Bladder neck mobility is a heritable trait. BJOG, 112: 334, 2005

294. Mushkat, Y., Bukovsky, I., Langer, R.: Female urinary stress incontinence--does it have familial prevalence? Am J Obstet Gynecol, 174: 617, 1996

295. Elia, G., Bergman, J., Dye, T. D.: Familial incidence of urinary incontinence. Am J Obstet Gynecol, 187: 53, 2002

296. Ertunc, D., Tok, E. C., Pata, O. et al.: Is stress urinary incontinence a familial condition? Acta Obstet Gynecol Scand, 83: 912, 2004

297. Hannestad, Y. S., Lie, R. T., Rortveit, G. et al.: Female urinary incontinence - Running in the family? Neurourol Urodyn, 22: 448, 2003

298. Tennstedt, S. L., Link, C. L., Steers, W. D. et al.: Prevalence of and risk factors for urine leakage in a racially and ethnically diverse population of adults: the Boston Area Community Health (BACH) Survey. Am J Epidemiol, 167: 390, 2008

299. Finkelstein, M. M., Skelly, J., Kaczorowski, J. et al.: Incontinence Quality of Life Instrument in a survey of primary care physicians. J Fam Pract, 51: 952, 2002

300. Diokno, A. C., Brock, B. M., Herzog, A. R. et al.: Medical correlates of urinary incontinence in the elderly. Urology, 36: 129, 1990

301. Thom, D. H., Haan, M. N., Van Den Eeden, S. K.: Medically recognized urinary incontinence and risks of hospitalization, nursing home admission and mortality. Age Ageing, 26: 367, 1997

302. Landi, F., Cesari, M., Russo, A. et al.: Benzodiazepines and the risk of urinary incontinence in frail older persons living in the community. Clin Pharmacol Ther, 72: 729, 2002

303. Montella, J., Wordell, C. J.: The effects of drugs on the lower urinary tract. In: Urogynecology and urodynamics. Edited by D. Ostergard and A. Bent. Baltimore: Williams and Wilkins, 1996

304. Movig, K. L., Leufkens, H. G., Belitser, S. V. et al.: Selective serotonin reuptake inhibitor-induced urinary incontinence. Pharmacoepidemiol Drug Saf, 11: 271, 2002

305. Foldspang, A., Mommsen, S.: Adult female urinary incontinence and childhood bedwetting :see comments:. J Urol, 152: 85, 1994

306. Fitzgerald, M. P., Thom, D. H., Wassel-Fyr, C. et al.: Childhood urinary symptoms predict adult overactive bladder symptoms. J Urol, 175: 989, 2006

307. Thom, D. H., Haan, M. N., Van den Eeden, S. K.: Medically recognized urinary incontinence and risks of hospitalization, nursing home admission and mortality. Age & Ageing, 26: 367, 1997

308. van der Vaart, C. H., de Leeuw, J. R., Roovers, J. P. et al.: The effect of urinary incontinence and overactive bladder symptoms on quality of life in young women. BJU Int, 90: 544, 2002

309. Brown, J. S., Posner, S. F., Stewart, A. L.: Urge incontinence: new health-related quality of life measures. J Am Geriatr Soc, 47: 980, 1999

310. DuBeau, C. E., Kiely, D. K., Resnick, N. M.: Quality of life impact of urge incontinence in older persons: a new measure and conceptual structure. J Am Geriatr Soc, 47: 989, 1999

311. Uebersax, J. S., Wyman, J. F., Shumaker, S. A. et al.: Short forms to assess life quality and symptom distress for urinary incontinence in women: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Continence Program for Women Research Group. Neurourol Urodyn, 14: 131, 1995

312. Kelleher, C. J., Cardozo, L. D., Khullar, V. et al.: A new questionnaire to assess the quality of life of urinary incontinent women. Br J Obstet Gynaecol, 104: 1374, 1997

313. Wagner, T. H., Patrick, D. L., Bavendam, T. G. et al.: Quality of life of persons with urinary incontinence: development of a new measure. Urology, 47: 67, 1996

314. Jackson, S., Donovan, J., Brookes, S. et al.: The Bristol Female Lower Urinary Tract Symptoms questionnaire: development and psychometric testing. Br J Urol, 77: 805, 1996

315. Huang, A. J., Brown, J. S., Kanaya, A. M. et al.: Quality-of-life impact and treatment of urinary incontinence in ethnically diverse older women. Arch Intern Med, 166: 2000, 2006

316. Brown, J. S., Vittinghoff, E., Wyman, J. F. et al.: Urinary incontinence: does it increase risk for falls and fractures? Study of Osteoporotic Fractures Research Group. J Am Geriatr Soc, 48: 721, 2000

317. Kron, M., Loy, S., Sturm, E. et al.: Risk indicators for falls in institutionalized frail elderly. Am J Epidemiol, 158: 645, 2003

318. Wilson, L., Brown, J. S., Shin, G. P. et al.: Annual direct cost of urinary incontinence. Obstet Gynecol, 98: 398, 2001

319. Subak, L. L., Brown, J. S., Kraus, S. R. et al.: The "costs" of urinary incontinence for women. Obstet Gynecol, 107: 908, 2006

320. Thom, D. H., Nygaard, I. E., Calhoun, E. A.: Urologic diseases in America project: urinary incontinence in women-national trends in hospitalizations, office visits, treatment and economic impact. J Urol, 173: 1295, 2005

321. Diokno, A. C., Burgio, K., Fultz, N. H. et al.: Medical and self-care practices reported by women with urinary incontinence. Am J Manag Care, 10: 69, 2004

322. Hannestad, Y. S., Rortveit, G., Hunskaar, S.: Help-seeking and associated factors in female urinary incontinence. The Norwegian EPINCONT Study. Epidemiology of Incontinence in the County of Nord-Trondelag. Scand J Prim Health Care, 20: 102, 2002

323. O'Donnell, M., Lose, G., Sykes, D. et al.: Help-seeking behaviour and associated factors among women with urinary incontinence in France, Germany, Spain and the United Kingdom. Eur Urol, 47: 385, 2005

324. Harris, S. S., Link, C. L., Tennstedt, S. L. et al.: Care seeking and treatment for urinary incontinence in a diverse population. J Urol, 177: 680, 2007

325. Gasquet, I., Tcherny-Lessenot, S., Gaudebout, P. et al.: Influence of the severity of stress urinary incontinence on quality of life, health care seeking, and treatment: A national cross-sectional survey. Eur Urol, 50: 818, 2006

326. Hagglund, D., Walker-Engstrom, M. L., Larsson, G. et al.: Reasons why women with long-term urinary incontinence do not seek professional help: a cross-sectional population-based cohort study. Int Urogynecol J Pelvic Floor Dysfunct, 14: 296, 2003

327. Kinchen, K. S., Burgio, K., Diokno, A. C. et al.: Factors associated with women's decisions to seek treatment for urinary incontinence. J Womens Health (Larchmt), 12: 687, 2003

328. Goldstein, M., Hawthorne, M. E., Engeberg, S. et al.: Urinary incontinence. Why people do not seek help. J Gerontol Nurs, 18: 15, 1992

329. Diokno, A. C.: Medical management of urinary incontinence. Gastroenterology, 126: S77, 2004

330. Reymert, J., Hunskaar, S.: Why do only a minority of perimenopausal women with urinary incontinence consult a doctor? Scand J Prim Health Care, 12: 180, 1994

331. Dugan, E., Roberts, C. P., Cohen, S. J. et al.: Why older community-dwelling adults do not discuss urinary incontinence with their primary care physicians. J Am Geriatr Soc, 49: 462, 2001

332. Norton, P. A., MacDonald, L. D., Sedgwick, P. M. et al.: Distress and delay associated with urinary incontinence, frequency, and urgency in women. Br Med J, 297: 1187, 1988

333. Rizk, D. E., Shaheen, H., Thomas, L. et al.: The prevalence and determinants of health care-seeking behavior for urinary incontinence in United Arab Emirates women. Int Urogynecol J Pelvic Floor Dysfunct, 10: 160, 1999

334. Roe, B., Doll, H., Wilson, K.: Help seeking behaviour and health and social services utilisation by people suffering from urinary incontinence. International Journal of Nursing Studies, 36: 245, 1999

335. MacKay, K., Hemmett, L.: Needs assessment of women with urinary incontinence in a district health authority. Br J Gen Pract, 51: 801, 2001

336. Mitteness, L. S.: So what do you expect when you're 85?: Urinary incontinence in late life. Res Soc Health Care, 6: 177, 1987

337. Shaw, C.: A review of the psychosocial predictors of help-seeking behaviour and impact on quality of life in people with urinary incontinence. J Clin Nurs, 10: 15, 2001

338. Sandvik, H., Hunskaar, S., Eriksen, B. C.: Management of urinary incontinence in women in general practice: actions taken at the first consultation. Scand J Prim Health Care, 8: 3, 1990

339. Herzog, A. R., Fultz, N. H., Normolle, D. P. et al.: Methods used to manage urinary incontinence by older adults in the community. J Am Geriatr Soc, 37: 339, 1989

340. Seim, A., Sandvik, H., Hermstad, R. et al.: Female urinary incontinence--consultation behaviour and patient experiences: an epidemiological survey in a Norwegian community. Fam Pract, 12: 18, 1995

341. Diokno, A. C., Burgio, K., Fultz, H. et al.: Prevalence and outcomes of continence surgery in community dwelling women. J Urol, 170: 507, 2003

342. Schulman, C., Claes, H., Matthijs, J.: Urinary incontinence in Belgium: a population-based epidemiological survey. European Urology, 32: 315, 1997

343. Seim, A., Eriksen, B. C., Hunskaar, S.: A study of female urinary incontinence in general practice. Demography, medical history, and clinical findings. Scand J Urol Nephrol, 30: 465, 1996

344. Sandvik, H., Hunskaar, S.: Incontinence pads - prevalence of use and individual consumption. Scand J Soc Med, 21: 120, 1993

345. Sandvik, H., Kveine, E., Hunskaar, S.: Female urinary incontinence - psychosocial impact, self care, and consultations. Scand J Caring Sci, 7: 53, 1993

346. Andersen, J. T., Sander, P.: Minimal care--a new concept for the management of urinary incontinence in an open access, interdisciplinary incontinence clinic. The way ahead? Scand J Urol Nephrol Suppl, 179: 55, 1996

347. Thom D. : Variations in estimated of urinary incontinence prevalence in the community: Effects of Differences in Definitions, Population Characteristics, and Study Type. J. Amer Geriatrics Society, 46: 473, 1998.

348. Koyama W, Koyanagi A, Mihara S, Kawazu S, Uemura T, Nakano H et al. : Prevalence and conditions or urinary incontinenc among the elderly. Methods Inf Med, 37: 151, 1998.

349. Boyle P, Boyle P, Robertson C, Mazzetta C, Keech M, Hobbs FD, Fourcade R, Kiemeney L, Lee C; The UrEpik Study Group. : The prevalence of male urinary incontinence in four centres: the UREPIK study. BJU Int., 92 : 943, 2003.

350. Engstrom G, Walker-Engstrom ML, Loof L, Leppert J. : Prevalence of three lower urinary tract symptoms in men - a population-based study. Fam Pract, 20 : 7, 2003 .

351. Van Oyen H, Van Oyen P.: Urinary incontinence in Belgium; prevalence, correlates and psychosocial consequences. Acta Clin Belg., 57: 207, 2002.

352. Schmidbauer J, Temml C, Schatzl G, Haidinger G, Madersbacher S.: Risk factors for urinary incontinence in both sexes. Analysis of a health screening project. Eur Urol., 39: 565, 2001.

353. Maral I, Ozkardes H, Peskircioglu L, and Bumin MA. : Prevalence of stress urinary incontinence in both sexes at or after age 15 years: a cross-sectional study. J Urol., 165: 408, 2001.

354. Bortolotti A., Bernardini B., Colli, E., Dibendetto, P., Nacci, G.G., Landoni, M. et al : Prevalence and risk factors for urinary incontinence in Italy. Eur. Urol., 37: 30, 2000.

355. Smoger SH, Felice TL, and Kloecker GH. : Urinary incontinence among male veterans receiving care in primary care clinics. Ann Intern Med., 132: 547, 2000.

356. Ueda T, Tamaki M, Kageyama S, Yoshimura N, and Yoshida O. : Urinary incontinence among community-dwelling people aged 40 years or older in Japan: prevalence, risk factors, knowledge and self-perception. Int J Urol., 7: 95, 2000.

357. Roberts RO, Jacobsen SJ, Reilly WT, Pemberton JH, Lieber MM, and Talley NJ.: Prevalence of combined fecal and urinary incontinence: a community-based study. J Am Geriatr Soc., 47: 837, 1999.

358. Roberts RO, Jacobsen SJ, Rhodes T, Reilly WT, Girman CJ, Talley NJ, and Lieber MM. : Urinary incontinence in a community-based cohort: prevalence and ealthcare-seeking. J Am Geriatr Soc., 46: 467,1998.

359. Schulman C, Claesm H, and Mattijs J: Urinary incontiennce in Belgium: a population-based epidemiological survey. Eur. Urol., 32: 315, 1997.

360. Malmsten UG, Milsom I, Molander U, and Norlen LJ.: Urinary incontinence and lower urinary tract symptoms: an epidemiological study of men aged 45-99 years. J Urol., 158: 1733, 1997.

361. Brocklehurst JC. Urinary incontinence in the community – analysis of a MORI poll. BMJ 306: 832-4, 1993.

362. Irwin DE, Milsom I, Hunskaar S, Reilly K, Kopp Z, Herschorn S, et al., Population-based survey of urinary incontinence, overactive bladder and other lower urinary tract symptoms in five countries : Results of the EPIC study. Eur Urol 50m 1306-15, 2006.

363. Legace EA, Hansen W, and Hickner JM. Prevalence and severity of urinary incontinence in ambulatory adults : an UPRNet study. J fam Pract 36(6): 610-5, 1993.

364. McGrother CW, Donaldson MMK, Shaw C, Matthews RJ, Hayward TA, Dallosso HM, Jagger c, et al. Storage symptoms of the bladder : prevalence, incidence and need for services in the UK. BJU Int 93: 763-69, 2004.

365. OBrien J, Austin M, Sethi P, and O'Boyle P : Urinary incontinence : prevalence need for treatment, and effectiveness of intervention by nurse. BMJ, 303: 1308-12, 1991.

366. Parrazzini F, Lavezzari M, and Artibani W. Prevalence of overactive bladder and urinary incontinence. J Fam Pract 5:1072-4, 2002.

367. Roe B. Prevalence of urinary incontinence and its relationship with health status. J Clin Nursing 9: 178-88, 2000.

368. Diokno, A.C., Brock, B.M., Brown, M.B., and Herzog, R. : Prevalence of urinary incontinence and other urological symptoms in the non-institutionalized elderly. J. Urol., 136: 1022, 1986.

369. Dios-Diz JM, Rodriguez-Lama M, Martinez-Calvo JR, Rodriguez-Perez C, Melero-Brezo M, Garcia-Cepeda JR. : [Prevalence of urinary incontinence in the population aged more than 64 years in Galicia, Spain]. Gac Sanit, 17 : 409, 2003.

370. Stoddart H, Donovan J, Whitley E, Sharp D, and Harvey I. : Urinary incontinence in older people in the community: a neglected problem? Br J Gen Pract., 51: 548, 2001.

371. Aggazzotti G, Pesce F, Grassi D, Fantuzzi G, Righi E, De Vita D, Santacroce S., and Artibani W.: Prevalence of urinary incontinence among institutionalized patients: a cross-sectional epidemiologic study in a midsized city in northern Italy. Urology, 56: 245, 2000.

372. Gavira Iglesias FJ, Caridad y Ocerin JM, Perez del Molino Martin J, Valderrama Gama E, Lopez Perez M, Romero Lopez M, et al. : Prevalence and psychosocial impact of urinary incontinence in older people of a Spanish rural population. J Gerontol A Biol Sci Med Sci., 55 : M207, 2000.

373. Damian J., Martin-Moreno, J.M., Lobo F., Bonache J., Cervino J., Redondo-Marquez, L., et al.: Prevalence of urinary incontinence among Spanish older people living at home. Eur. Urol., 34: 333, 1998.

374. Umlauf MG, AND Sherman SM.: Symptoms of urinary incontinence among older community-dwelling men.J Wound Ostomy Continence Nurs., 23: 314, 1996.

375. Nuotio M, Jylha M, Luukkaala T, and Tammela TL. : Urinary incontinence in a Finnish population aged 70 and over. Prevalence of types, associated factors and self-reported treatments. Scand J Prim Health Care., 21: 182, 2003.

376. Herzog, AR and Fultz NH.: Epidemiology of urinary incontinence: prevalence, incidence and correlates in community populations. Urology Suppl., 36: 2, 1990.

377. Janssen I. Morbidity and mortality risk associated with an overweight BMI in older women and women. Obesity 17: 1827-80, 2007.

378. Landi F, Cesari M, Russo A, Onder G, Lattansio F, Bernabe R, Silvernet-HC Study group. Potentially reversible risk factors and urinary incontinence in frail older people living in community. Age and Ageing 32: 194-9, 2003.

379. Thom C, Haan MN, and Van Den Eeden SK. Medically recognized urinary incontinence and risks of hospitalization, nursing home admission and mortality. Age and Ageing 26(5) : 367-9,1997.

380. Yarnell JW, and St Lege AS.: The prevalence, severity and factors associated with urinary incontinence in a random sample of the elderly. Age Ageing, 8: 81, 1979.

381. Thomas TM, Plymat KR, Blannin J and Meade TW: Prevalence of urinary incontinence. BMJ., 281:1243, 1980.

382. Temml, C., Haidinger, G., Schmidbauer, J., Schatzl, G., and Madershbacher, S.: Urinary incontinence in both sexes: Prevalence rates and impact on quality of life and sexual life. Nuerourol. Urodyn., 19: 259, 2000.

383. Marriapan P and Chong WL. Prevalence and correlations of lower urinary tract symptoms, erectile dysfunction and incontinence in men from a multiethnic Asian population : results of a regional population-based survey and comparison with industrialized nations. BJU Int 98: 1264-8, 2006.

384. Chaojie L, and Andrews GR. Prevalence and incidence of urinary incontinence in the elderly : a longitudinal study in South Australia. Chinese Med J 115(1) : 119-22, 2002.

385. Irwin DE, Milsom I, Hunskaar S, Reilly K, Kopp Z, Herschorn S, Coyne K, Kelleher C, Hampel C, Artibani W, Abrams P. Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC study. Eur Urol 50 (6), 1306-14, Dec 2006.

386. Herschorn A, Gajewski J, Schulz J, and Corcos J : A population-based study of urinary symptoms and incontninence : the Canadian urinary bladder survey. BJU Int 101: 52-8, 2007.

387. Sladden MJ, Hughes AM, Hirst GH, and Ward JE.: A community study of lower urinary tract symptoms in older men in Sydney, Australia. Aust N.Z. J. Surg, 70: 322, 2000.

388. Diokno AC, Estanol MVC, Ibrahim IA, and Balasubamanian M. Prevalence of urinary incontiennce in community dwelling men : a cross sectional nationwide epidemiology survey. Int Urol Nephrol 39: 129-36, 2007.

389. Muscatello DJ, Rissel C, and Szonyi G. Urinary symptoms and incontinence in an urban community prevalence and

associated factors in older men and women. Int Med J 31:151-160, 2001.

390. Nelson RL, and Furner SE. Risk factors for the development of fecal and urinary incontinence in Winconsin nursing home residents. Maturitas 52: 26-31, 2005.

391. Kikuchi A, Niu K, Ikeda Y, Hozawa A, Nakagawa H, Guo H, Ohmori-Marsuda K, et al. Association between physical activity and urinary incontinence in a community-based elderly population aged 70 years and over. Eur Urol 52: 868-75, 2007.

392. Resnick NM and Yalla SV.: Detrusor hyperactivity with impaired contractile function : an unrecognized but common cause of incontinence in elderly patients. JAMA., 257: 3076, 1987.

393. Patel M, Coshall C, Rudd AG, and Wolfe CD.: Natural history and effects on 2-year outcomes of urinary incontinence after stroke. Stroke, 32 (1): 122-7, 2001.

394. Hunskaar S.: One hundred and fifty men with urinary incontinence. I. Demography and medical history. Scan. J. Prim. Health Care, 10: 21, 1992.

395. Diokno A, Brock B, Hersog A, and Bromberg J: Medical correlates of urinary incontinence in the elderly. Urology, 36: 129, 1990.

396. van Melick HH, van Venrooij GE, Eckhardt MD, and Boon TA.: A randomized controlled trial comparing transurethral resection of the prostate, contact laser prostatectomy and electrovaporization in men with benign prostatic hyperplasia: analysis of subjective changes, morbidity and mortality. J Urol., 169: 1411, 2003.

397. Demirkesen O, Onal B, Tunc B, Alici B, Onder AU, Ozalp AU, Cetinel B.. Assessment of the continence status and patients' satisfaction after retropubic radical prostatectomy: a questionnaire based study. Int Urol Nephrol. 39(2):531-6, 2007.

398. Kundu SD, Roehl KA, Eggender SE, Antenor JV, Han M and Catalona WJ. Potency, continence and complications in 3477 consecutive radical retropubic prostatectomies. J Urol 172: 2227-2231, dec 2004.

399. Salomon L, Saint F, Anastasiadis AG, Sebe P, Chopin D, Abbou CC.: Combined reporting of cancer control and functional results of radical prostatectomy. Eur Urol., 44: 656, 2003

400. Moinzadeh A, Shunaigat AN, and Libertino JA.: Urinary incontinence after radical retropubic prostatectomy: the outcome of a surgical technique. BJU Int., 92: 355, 2003.

401. Maffezzini M, Seveso M, Taverna G, Giusti G, Benetti A, and Graziotti P.: Evaluation of complications and results in a contemporary series of 300 consecutive radical retropubic prostatectomies with the anatomic approach at a single institution. Urology, 61: 982, 2003.

402. Deliveliotis C, Protogerou V, Alargof E, and Varkarakis J.: Radical prostatectomy: bladder neck preservation and puboprostatic ligament sparing--effects on continence and positive margins. Urology, 60: 855, 2002.

403. Benoit RM, Naslund MJ, and Cohen JK.: Complications after radical retropubic prostatectomy in the medicare population. Urology, 56: 116, 2000.

404. Walsh PC, Marschke P, Ricker D and Burnett AL.: Patient-reported urinary continence and sexual function after anatomic radical prostatectomy. Urology, 55: 58, 2000.

405. Poon M, Ruckle H, Bamshad DBR, Rsai C, Webster R, and Lui P.: Radical retropubic prostatectomy : bladder neck preservation versus reconstruction. J. Urol., 163: 194, 2000.

406. Catalona WJ, Carvalhal GF, Mager DE, and Smith DS.: Potency, continence and complication rates in 19870 consecutive radical retropubic prostatectomies. J. Urol., 162: 433, 1999.

407. Horie S, Tobisu KI, Fujimoto H, Doi N. and Kakizoe T.: Urinary incontinence after non-nervesparing radical prostatectomy with neoadjuvant androgen deprivation. Urology, 53: 561, 1999.

408. Goluboff ET, Saidi JA, Mazer S, Bagiella E, Heitjan DF, Benson MC. Et. Al.: Urinary continence after radical prostatectomy: The Columbia experience. J. Urol., 159: 1276, 1998.

409. Weldon VE, Tavel FR, and Neuwirth H. : Continence, potency and morbidity after radical perineal prostatectomy. J Urol., 158: 1470, 1997.

410. Lowe BA. : Comparison of bladder neck preservation to bladder neck resection in maintaining postprostatectomy urinary incontinence. Urology, 48: 889, 1996.

411. Hu JC, Gold KF, Pashos CL, Mehta SS, and Litwin MS.: Temporal trends in radical prostatectomy complications from 1991 to 1998. J Urol, 169: 1443, 2003.

412. Augustin H, Pummer K, Daghofer F, Habermann H, Primus G, and Hubmer G. Patient self-reporting questionnaire on urological morbidity and bother after radical retropubic prostatectomy. Eur Urol., 42: 112, 2002.

413. Sebesta M, Cespedes RD, Luhman E, Optenberg S, and Thompson IM.: Questionnaire-based outcomes of urinary incontinence and satisfaction rates after radical prostatectomy in a national study population. Urology, 60: 1055, 2002.

414. Potosky, A.L., Legler, J., Albertsen, P.C., Stanford, J.L., Gilliland, F.D., Hamilton, A.S., Eley, J.W., Stephenson, R.A., Harlan, L.C.: Health outcomes after prostatectomy or radiotherapy for prostate cancer : results from the Prostate Cancer Outcomes Study. J Natl Cancer Inst, 92: 1582, 2000.

415. Arai Y, Okubo K, Aoki Y, Maekawa S, Okada T, Maeda H, Ogawa O, and Kato T. : Patient-reported quality of life after radical prostatectomy for prostate cancer. Int J Urol., 6: 78, 1999.

416. Bishoff JT, Motley G., Optenberg SA, Stein CR, Moon KA, Browning SM. Et al.: Incidence of fecal and urinary incontinence following radical perineal and retropubic prostatectomy in a national population. J. Urol., 160: 454, 1998.

417. Egawa S, Minei S, Iwamura M, Uchida T, and Koshiba K. : Urinary continence following radical prostatectomy. Jpn J Clin Oncol., 27: 71, 1997.

418. Gray M, Petroni GR, and Theodorescu D.: Urinary function after radical prostatectomy: a comparison of the retropubic and perineal approaches. Urology, 53: 881, 1999.

419. Olsson LE, Salomon L, Nadu A, Hoznek A, Cicco A, Saint F, Chopin D, and Abbou CC. :Prospective patient-reported continence after laparoscopic radical prostatectomy. Urology, 58: 570, 2001.

420. LaFontaine P, Chan D, Partin AW, Gurganus R, Hortopan SC, and Marshall FF. : Minilaparotomy radical retropubic prostatectomy: updated technique and results. Semin Urol Oncol., 18: 19, 2000.

421. Galli S, Simonato A, Bozzola A, Gregori A, Lissiani A, Scaburri A, Gabardi F. Oncologic outcome and continence revcovery after laparoscopic radical prostatectomy: 3 years' follow-up in a "second generation center'. Eur Urol 49 : 859-865, 2006.

422. McKammon KA, Kolm P, Main B, and Schellhammer PF: Comparative quality of life analysis after radical retropubic prostatectomy: objective and subjective analysis. Urology, 49: 225, 1997.

423. Donnellan SM, Duncan HJ., MacGregor RJ., and Russel, JM. : Prospective assessment of incontinence after radical retropubic prostatectomy: objective and subjective analysis. Urology 49: 225, 1997.

424. Ojdeby G, Claezon A, Brekkan E, Haggman M, and Norlen BJ: Urinary incontinence and sexual impotence after radical prostatectomy. Scand. J. Urol. Nephrol., 30: 473, 1996.

425. Saranchuk JW, Kattan MW, Elkin E, Taurier K, Scardino PT, and Eastham, JA. Achieving optimal outcomes after radical prostatectomy. J Clin Oncol 23 (18) : 4146-51, June 2005.

426. Harris MJ. The anatomic radical perineal prostatectomy : An outcomes-based evolution. Eur Urol 52 : 81-88, 2007.

427. Jacobsen NB, Moore KN, Estey E, and Voaklander D. Open versus laparoscopic radical prostatectomy : A prostective comparison of postoperative urinary incontinence rates. J Urol, 177 : 615-619, Feb 2007.

428. Link RE, Su LM, Sullivan W, Bhayani S, and Pavlovich CP. Health related quality of life before and after laparoscopic radical prostatectomy. J Urol 173 : 173-9, January 2005.

429. Jonler M, Madsen FA, Rhodes PR, Sall M, Messing EM, Brusketwitz RB. A prospective study of quantification of urinary incontinence and quality of life in patients undergoing radical retropubic prostatectomy. Urology 48 : 433-40, 1996.

430. Penson DF, McLerran D, Feng Z, Li L, Albertsen PC, Gilland FD et al. 5-year urinary and sexual outcomes after radical prostatectomy : Results from the prostate cancer outcomes study. J Urol 173:1701-5, May 2005.

431. Wei JT, Dunn RL, Marcovich R, Montie JE, and Sanda MG.: Prospective assessment of patient reported urinary continence after radical prostatectomy. J Urol., 164: 744, 2000.

432. Eastham JA, Kattan MW, Rogers E, Goan JR, Ohori M, Boone TB, and Scardino PT.: Risk factors for urinary incontinence after radical prostatectomy. J Urol., 156: 1707, 1996.

433. Van Kampen M, De Weerdt W, Van Poppel H, Castell Campesino A., Stragier J. and Baert, L.: Prediction of urinary continence following radical prostatectomy. Urol. Int., 60: 80, 1998.

434. Srougi M, Nesrallah LJ, Kauffmann JR, Nesrallah A, and Leite KR.: Urinary continence and pathological outcome after bladder neck preservation during radical retropubic prostatectomy: a randomized prospective trial. J Urol., 165: 815, 2001.

435. Noh C, Kshirsagar A, and Mohler JL.: Outcomes after radical retropubic prostatectomy. Urology, 61: 412, 2003.

436. Kao TC., Garner D., Foley J., Seay T., Friedrichs P., Thrasher JB., et al : Multicenter patient self-reporting questionnaire on impotence, incontinence and stricture after radical prostatectomy. J. Urol., 163: 858, 2000.

437. Moul JW, Mooneyhan RM, Kao TC, McLeod DG, and Cruess DF.: Preoperative and operative factors to predict incontinence, impotence and stricture after radical prostatectomy. Prostate Cancer Prostatic Dis., 1: 242, 1998.

438. Lee SE, Byun SS, Lee HJ, Song SH, Chang IH, Kim YJ, Gill MC, and Hong SK. Impact of variations in prostatic apex shape on early recovery on urinary continence after radical retropubic prostatectomy. Urology 68 : 137-41, 2006.

439. Johnson TK, Gilliland FD, Hoffman RM, Deapen D, Penson DF, Stanforn J>, e tal. Racial/ethnic differences in functional outcomes in the 5 years after diagnosis of localized prostate concer. J Clin Onco 22(20) : 4193-4201, Oct 2004.

440. Formenti SC, Lieskovsky G, Skinner D, Tsao-Wei DD, Groshen S, and Petrovich Z.: Update on impact of moderate dose of adjuvant radiation on urinary continence and sexual potency in prostate cancer patients treated with nerve-sparing prostatectomy. Urology, 56: 453, 2000.

441. Hofmann T, Gaensheimer S, Buchner A, Rohloff R, and Schilling A. An unrandomized prospective comparison of urinary continence, bowel symptoms and the need for further procedures in patients with and with no adjuvant radiation after radical prostatectomy. BJU Int 92 : 360-4, 2003.

442. Castro D, Espuna M, Prieto M, Badia X. Prevalence of overactive bladder in Spain: a population-based study [Spanish]. Arch Esp Urol 2005;58:131-138. Chen GD, Lin TL, Hu SW, Chen YC, Lin LY. Prevalences and correlation of urinary incontinence and overactive bladder in Taiwanese women. Neurourol Urodyn 2003;22:109-117.

443. Choo MS, KU JH, Lee JB, Lee DH, Kim JC, Kim HJ, Lee JJ, Park WH. Cross-cultureal differences for adapting overactive bladder symptoms: results of an epidemiologic survey in Korea. World J Urol 2007;25:505-511.

444. Corcos J, Schick E. Prevalence of overactive bladder and incontinence in Canada. Can J Urol 2004;11:2278-2284.

445. Herschorn S, Gajewski J, Schulz J, Corcos J. A population-based study of urinary symptoms and incontinence: the Canadian Urinary Bladder Survey. BJU Int 2007;101:52-58.

446. Homma Y, Yamaguchi O, Hayashi K. An epidemtiological survey of overactive bladder symptoms in Japan. BJU Int 2005;96:1314-1318.

447. Irwin DE, Milsom I, Hunskaar S, Reilly K, Kopp Z, Herschorn S, Coyne K, Kelleher C, Hampel C, Artibani W, Abrams P. Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC study. Eur Urol 2006;50:1306-1315.

448. Kajiwara M, Inoue K, Kato M, Ushui A, Kurihara M, Usui T. Nocturnal enuresis and overactive bladder in children: an epidemiological study. Int J Urol 2006;13:36-41.

449. Kajiwara M, Inoue K, Mutaguchi K, Usui T. The prevalence of overactive bladder and noturnal enuresis in Japanese early adolescents: a questionnaire survey. Hinyokika Kiyo 2006;52:107-111.

450. Lawrence JM, Lukacz ES, Amy Liu IL, Nager CW, Luber KM. Pelvic floor disorders, diabetes, and obesity in women: findings from the Kaiser Permanente Continence Associated Risk Epidemiology Study. Diabetes Care 2007;30:2536-2541.

451. McGrother CW, Donaldson MMK, Hatward T, Matthews R, Dallosso HM, Hyde C. Urinary storage symptoms and comorbidities: a prospective population cohort study in middle-aged and older women. Age Ageing 2006;35:16-24.

452. Milsom I. Abrams P, Cardoza L, Roberts RG, Thuroff J, Wein AJ. How widespread are the symptoms of an overactive bladder and how are they managed? A population-based prevalence study. BJU Int 2001;87:760-766.

453. Stewart WF, Van Rooyen JB, Cundiff GW, Abrams P, Herzog AR, Corey R, Hunt TL, Wein AJ. Prevalence and burden of overactive bladder in the United States. Worl J Urol 2003;20:327-336.

454. Teloken C, Caraver F, Weber FA, Teloken PE, Moraes JF, Sogari PR, Graziottin TM. Overactive bladder: prevalence and implications in Brazil. Eur Urol 2006;49:1087-1092.

455. Temml C, Heidler S, Ponholzer A, Madersbacher S. Prevalence of the overactive bladder symdrome by applying the Inernational Continence Society definition. Eur Urol 2005;48:622-627.

456. Zhang W, Song Y, He X, Huang H, Xu B, Song J. Prevalence and risk factors of overactive bladder syndrome in Fuzhou Chinese women. Neurourol Urodyn 2006;25:717-721.

457. Titi M, Jenkins JT, Urie A, Molloy RG. Prospective study of the diagnostic evaluation of faecal incontinence and leakage in male patients. Colorectal Dis. 2007;9:647-52.

458. Leigh RJ, Turnberg LA. Faecal incontinence: the unvoiced symptom. Lancet. 1982;1:1349-1351.

459. Thomas TM, Egan M, Walgrove A, Meade TW. The prevalence of faecal and double incontinence. Comm Med. 1984;6:216-220.

460. Nelson RL, Norton N, Cautley E, Furner S. Community based prevalence of AI. JAMA. 1995;274:559-562.

461. Macmillan AK, Merrie AEH, Marshall RJ, Parry BR. The prevalence of faecal incontinence in community dwelling adults: a systematic review. Dis. Colon & Rectum.2004.47.1341-49.

462. Pretlove SJ, Radley S, Toozs-Hobson PM, Thompson PJ, Coomarasamy A, Khan KS. Prevalence of anal incontinence according to age and gender: a systematic review and meta-regression analysis.

463. Bai Y, Chen H, Hao J, Huang Y, Wang W. Long term outcome and quality of life after the Swenson procedure for Hirschprung's disease. J Pediatric Surg. 2002;37:639-42.

464. Javid PJ, Barnhart DC, Hirschi RB, Coran AG, Harmon CM. Immediate and long term results of surgical management of low imperforate anus in girls. J Pediatric Surg. 1998;33:198-203

465. Rintala RJ, Lindahl H. Is normal bowel function possible after repair of intermediate and high anorectal malformations? J Pediatric Surg. 1995;30:491-4.

466. Hartman EE, Oort FJ, Aronson DC, Hanneman JG, van der Zee DC, et al. Critical factors affecting the quality of life of adult patients with anorectal malformations or Hirschprung's Disease. Am J Gastroent.2004;xx:907-13.

467. Forrester MB, Merz RD. Descriptive epidemiology of anal atresia in Hawaii, 1986-1999. Teratology.2002;66supp.:S12-6.

468. Largo RH, Gianciaruso M, Prader A. Development of intestinal and bladder control from birth until the 18th year of age. Longitudinal Study. Schweiz Med Wochenschr. 1978;108:155-60.

469. Nelson RL. Outcome of surgical procedures for anal fissure. Cochrane Library.2008, #2.

470. Nelson RL. Non-surgical therapy for anal fissure. Cochrane Library.2008, #2.

471. Nelson RL, Furner SE, Westercamp M. A systematic review of the efficacy of cesarean section in prevention of anal incontinence. In press. Cochrane Library.

472. Small KA, Wynne JM. Evaluating the pelvic floor in obstetric patients. Aust New Zeal Obstet Gynecol. 1990;30:41-45.

473. Madoff RD, Williams JG, Caushaj PF. Current concepts: Fecal incontinence. N Engl J Med. 1992;326:1002-1007.

474. Abou-Zahr C. Obstructed labour. In: Murray CJL, Lopez AD, eds. Health dimensions of sex and reproduction: the global burden of sexually transmitted diseases, HIV, maternal conditions, perinatal disorders and congenital anomalies. Cambridge, Mass. Harvard University Press, 1996.

475. Drossman DA. What can be done to control incontinence associated with the irritable bowel syndrome? Am J Gastroenterol. 1989;84:355-357.

476. Schiller LR, Santa Ana CA, Schmulen AC, Hendler RS, Harford WV, Fordtran JS. Pathogenesis of fecal incontinence in dia-betes mellitus. N Engl J Med. 1982;307:1666-1671.

477. Campbell AJ, Reinken J, McCosh L. Incontinence in the elderly: prevalence and prognosis. Age Ageing. 1985;14:65-70.

478. Denis P, Bercoff E, Bizien MF, Brocker P, Chassagne P, Lamouliatte H et al. Etude de la prevalence de l'incontinence anale chez l'adulte. Gastroent Clin Biol. 1992;16:344-350.

479. Drossman DA, Li Z, Andruzzi E, Temple RD, Talley NJ, Thomp-son WG et al. U.S. householder survey of functional gastrointestinal disorders: Prevalence, sociodemography, and health impact. Dig Dis Sci. 1993;38:1569-1580.

480. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity, and mode of delivery. BJOG. 2000;107:1460-1470.

481. Giebel GD, Lefering R, Troidl H, Blochl H. Prevalence of fecal incontinence: what can be expected? Int J Colorectal Dis. 1998;13:73-77.

482. Lam L, Kennedy M, Chen F. Prevalence of faecal incontinence: obstetric and constipation risk factors: a population based study. Colorectal Dis. 1999;1:197-203.

483. Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors. Med J Aust. 2002;176:54-57.

484. Lynch AJ, Dobbs BR, Keating J, Frizelle FA. The prevalence of faecal incontinence and constipation in a general New Zealand population: a postal survey. NZ Med J. 2001;114:474-477.

485. Perry S, Shaw C, McGrother C, Mathews RJ, Assassa RP, Dallosso H et al. Prevalence of faecal incontinence in adults aged 40 years or more living in the community. Gut. 2002;50: 480-484.

486. Chaliha C, Kalia V, Stanton SL, Monga A, Sultan AH. Antenatal prediction of postpartum urinary and fecal incontinence. Obstet Gynecol. 1999;94:689-694.

487. Eason E, Labrecque M, Marcoux S, Mondor M. AI after childbirth. CMAJ. 2002;166:326-330.

488. Sangalli MR, Floris L, Faltin D, Weil A. AI in women with third or fourth degree perineal tears and subsequent vaginal deliveries. Aust NZ J Obstet Gynaecol. 2000;40:244-248.

489. Okonkwo JE, Obionu CN, Okonkwo CV, Obiechina NJ. AI among Igbo women. Int J Clin Pract. 2002;56:178-180.

490. Rizk DE, Hassan MY, Shaheen H, Chervian JV, Micallef R, Dunn E. The prevalence and determinants of health care seeking behavior for fecal incontinence in multiparous United Arab Emirates females. Dis Colon Rectum. 2001;44:1850-1856.

491. Alnaif B, Drutz HP. The prevalence of urinary and fecal incontinence in Canadian secondary school teenage girls: questionnaire study and review of the literature. Int J Urogynecol/J Pelvic Floor Dysfunct. 2001;12:134-137.

492. Faltin DL, Sangalli MR, Curtin F, Morabia A, Weil A. Prevalence of AI and other anorectal symptoms in women. Int J Urogynecol/J Pelvic Floor Dysfunct. 2001;121: 117-120.

493. Ishigooka M, Hashimoto T, Izumiya K, Sasagawa I, Nakada T. Incidence of AI after long term follow up of patients treated by ureterosigmoidostomy. Int Urol Nephrol. 1993;25:455-460.

494. Adolfsson J, Helgason AR, Dickman P, Steineck G. Urinary and bowel symptoms in men with and without prostate cancer: results from an observational study in the Stockholm area. Eur Urol. 1998;33:11-16.

495. Bytzer P, Talley NJ, Leemon M, Young LJ, Jones MP, Horowitz M. Prevalence of gastrointestinal symptoms associated with diabetes mellitus: a population bases survey of 15000 adults. Arch Intern Med. 2001;161:1989-1996.

496. Kok ALM, Voorhorst FJ, Burger CW, van Houten P, Kenemans P, Jansens J. Urinary and faecal incontinence in community-residing elderly women. Age Ageing. 1992;21:211-215.

497. Talley NJ, O'Keefe EA, Zinsmeister AR, Melton LJ. Prevalence of gastrointestinal symptoms in the elderly: a population based study. Gastroenterol. 1992;102:895-901.

498. Nakanishi N, Tatara K, Nakajima K, Takabayashi H, Takahashi S, Naramura H et al. Urinary and fecal incontinence in a community-residing elderly population: prevalence, correlates and prognosis. Nippon Koshu Eisei Zasshi. 1997;44:192-200.

499. Roberts RO, Jacobsen SJ, Reilly WT, Pemberton JH, Lieber MM, Talley NJ. Prevalence of combined fecal and urinary incontinence: a community-based study. J Am Geriatr Soc. 1999;47:837-841.

500. Edwards NI, Jones D. The prevalence of faecal incontinence in older people living at home. Age Ageing. 2001;30:503-507.

501. Verhagen TE, Lagro-Janssen AL. Fecal incontinence in community dwelling elderly: findings from a study of prevalence, consultation of physicians, psychosocial aspects and treatment. Ned Tijdschr Geneeskd. 2001;145:741-745.

502. Tpokinova E, Neuwirth J, Stankova M, Mellanova A, Haas T. Urinary and fecal incontinence in geriatric facilities in the Czech Republic. Cas Lek Cesk. 1997;136:573-577.

503. Nelson RL, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes. Dis Colon Rectum. 1998;41:1226-1229.

504. Borrie MJ, Davidson HA. Incontinence in institutions: costs and contributing factors. CMAJ. 1992;147:322-328.

505. Damon H, Guye O, Seigneurin A,Long F, Faucheron AL, Grandjean JP, Mellier G, Vallancogne G, FayardO, Henry L, Guyot P, Barth X, Mion F. Prevalence of anal incontinence in adults impact on quality of life. Gastroenterologie. 2006;30:37-43.

506. Bharucha AE, Zinsmeister AR, Locke GR, Seide BM, McKeon K, Schleck CD, Melton III J. Rick factors for fecal incontinence: a population-based study in women. Am J Gastro.206;101:1305-12.

507. Fritel X, Ringa V, Varnoux N, Zins M, Breart G. Mode of delivery and fecal incontinence at midlife; a study of 2640 women in the Gazel cohort. Obstet & Gynecol.2007;110:31-8.

508. Lawrence JM, Lukasz ES, Nager CW,Hsu JWY, Luber KM. Prevalence and co-occurence of pelvic floor disorders in community-dwelling women. Obstet.& Gynecol. 2008; 111:678-85.

509. Borello-France D, Burgio KL, Richter HE, Zyczynski H, Fitzgerald MP, Whitehead W, Fine P, Nygaard I, Handa VL, Visco AG, Weber AM, Brown MB. Fecal andurinary incontience in primiparous women. Obstet & Gynecol. 2006;108:863-72.

510. Erekson EA, Sung VW, Myers DL. Effect of body mass index on the risk of anal incontinence and defecatory dysfunction in women. Am J Obstet& Gyencol. 2008;198:596-99.

511. Altman D, Falconer C, Rossner S,Melin I. The risk of anal incontinence in obese women. Int. Urogynecol. J. 2007;18:1283-9.

512. Burgio KL, Richter HE, Clements RH, Redden DT, Goode PS. Changes in urinary and fecal incontinence symptoms with weight loss surgery in morbidly obese women. Obstet Gynecol. 2007;110:1034-40.

513. Oberwalder M, Connor J, Wexner SD. Meta-analysis to determine the incidence of obstetric anal sphincter damage. Brit. J Surg. 2003;90:1333-7

514. Nelson RL. Epidemiology of fecal Incontinence. Gastroenterology.2004;126 (Suppl. 1): s3-7.

515. Goffeng AR, Andersch B, Andersson M, Berndtsson I, Hulten L, Oreslan T. Objective methods cannot predict anal incontinence after primary repair of extensive anal tears.Acta Obstet Gynecol Scand. 1998;77:439-43

516. Guttierz AB, Madoff RD, Lowry AC, Parker SC,Buie WD, Baxter NN. Long term results of anterior sphincteroplasty. Dis Colon & Rectum. 2004;47:727-32

517. Halverson AN, Hull TL. Long-term outcome of overlapping anal sphincter repair. Dis Colon & Rectum. 2002;45:345-8

518. Karoui S, Leroi AM, Koning E, Menar JF, Michot F, Dens P. Results of sphincteroplast in 86 patients with anal incontinence. Dis Colon & Rectum. 2000;43:813-20

519. Malouf AJ, Norton CS, Engel AF, Nicholls RJ, Kamm MA. Long-term resuls of overlapping anterior anal sphincter repair for obstetrical trauma. Lancet. 2000;355:260-5

520. Pinta T, Kyaanpaa ML, Salmi T, Jaarvinen HJ, Luukkonen P. Delayed sphincter repair for obstetric ruptures: analysis of failure. Colorectal Dis. 2003;5:73-8

521. Rothbart J, Bemelman WA, Meijerink WJ. Long term results of anterior anal sphincter repair for recal incontinence due to obstetric injury. Dig. Surg. 2000;17:390-4

522. Vaizey CJ, Norton C, Thornton MJ, Nicholls RJ, Kamm MA. Long term results of repeat anterior anal sphincter repair. Dis Colon & Rectum.2004;47:858-63

523. MacArthur C, Glazener CMA, Lancashire R, Herbison P, et al. Faecal incontinence and mode of first and subsequent deliveries: a six year longitudinal study. Br J Obstet Gynecol.2005;112:1075-82.

524. Zetterstrom J, Lopez A, Anzen B, Dolk A, Norman M, Mellgren A. Analincontinence after vaginal delivery: a prospective study in pimiparous women. Obstet Gynecol.1999;106:324-30

525. Devine JB, Ostergard DR, Noblett KL. Long tern complications of the second stage oof labor. Contemp. Obstet Gynecol.1999:119-26.

526. Altman D, Zetterestrom J, Lopez A, Pollack J, Nordenstam J, Mellgren A. Effect of hysterectomy on bowel function. Dis. Colon & Rectum.2004;47:502-9.

527. MacKenzie WR, Hoxie NJ, Proctor ME, Gradus MS, Blair KA, Petersen DE et al. A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. N Eng J Med. 1994;331:161-167.

528. Sullivan SN, Wong C. Runner's diarrhea. Different patterns and associated factors. J Clin Gastroenterol. 1992;14:101-104.

529. Pernikoff BJ, Eisenstat TE, Rubin RJ, Oliver GC, Salvati EP. Reappraisal of partial lateral internal sphincterotomy. Dis Colon Rectum. 1994;37:1291-1295.

530. del Pino A, Nelson RL, Pearl RK, Abcarian H. Island flap anoplasty for treatment of transsphincteric fistula-in-ano. Dis Colon Rectum. 1996;39:224-226.

531. Gorfine SR. Treatment of benign anal disease with topical nitroglycerin. Dis Colon & Rectum. 1995;38:453-457.

532. Johannsson HO, Graf W, Pahlman L. Long term results of hemorrhoidectomy. Eur J Surg.2002;168:485-9.

533. Wald A. Systemic diseases causing disorders of defecation and continence. Sem Gastrointest. Dis. 1995;6:194-202.

534. Bliss DZ, Johnson S, Savik K, Clabots CR, Gerding DN. Fecal incontinence in hospitalized patients who are acutely ill. Nursing Res. 2000;49:101-108.

535. Nakayama H, Jorgensen HS, Pedersen PM, Raaschou HO, Olsen TS. Prevelance and risk factors of incontinence after stroke: The Copenhagen Stroke Study. Stroke. 1997;28:58-62.

536. Porell F, Caro FG, Silva A, Monane M. A longitudinal analysis of nursing home outcomes. Health Svc.s Rsch. 1998;33:835-65.

537. Chassange P, Landrin I, Neveu C, Czernichow M, Doucet J, Denis P, Bercoff E. Fecal incontinence in the institutionalized elderly: incidence, risk factors and prognosis. Am J Med.1999;106:185-190.

538. Nelson RL, Furner SE. The development of incontinence in Wisconsin nursing home residents. Submitted.

539. Fornell EU, Wingren G, Kjoelhede P. Factors associated

with pelvic floor dysfunction with emphasis on urinary and fecal incontinence and genital prolapse: an epidemiological study. Act. Obstet. Gynecol. Scand. 2004;83:383-389.

540. Culligan PJ, Myers JA, Goldberg RP, Blackwell L,Gohmann SF, Abell TD. Elective cesarean section to prevent anal incontinence and brachial plexus injuries associated with macrosomia – a decision analysis. Int Urogyencol J. 2005;16:19-28

541. Faltin DL, Boulvain M, Floris LA, Irion O. Diagnosis of anal sphincter tears to prevent fecal incontinence; a randomized controlled trial. Obstet & Gynecol. 2005;106:6-13.

542. Shamliyan T, Wyman J, Bliss DZ, Kane RL, Wilt TJ. Prevention of urinary and fecal incontinence in adults. Evid Rep Technol Assess (Full Rep). 2007 Dec;(161):1-379.

543. Bump, R. C., Mattiasson, A., Bo, K. et al.: The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol, 175: 10, 1996

544. Hall, A. F., Theofrastous, J. P., Cundiff, G. W. et al.: Interobserver and intraobserver reliability of the proposed International Continence Society, Society of Gynecologic Surgeons, and American Urogynecologic Society pelvic organ prolapse classification system. Am J Obstet Gynecol, 175: 1467, 1996

545. Weber, A. M., Abrams, P., Brubaker, L. et al.: The standardization of terminology for researchers in female pelvic floor disorders. Int Urogynecol J Pelvic Floor Dysfunct, 12: 178, 2001

546. Ellerkmann, R. M., Cundiff, G. W., Melick, C. F. et al.: Correlation of symptoms with location and severity of pelvic organ prolapse. Am J Obstet Gynecol, 185: 1332, 2001

547. Lukacz, E. S., Lawrence, J. M., Buckwalter, J. G. et al.: Epidemiology of prolapse and incontinence questionnaire: validation of a new epidemiologic survey. Int Urogynecol J Pelvic Floor Dysfunct, 16: 272, 2005

548. Ghetti, C., Gregory, W. T., Edwards, S. R. et al.: Pelvic organ descent and symptoms of pelvic floor disorders. Am J Obstet Gynecol, 193: 53, 2005

549. Swift, S. E.: The distribution of pelvic organ support in a population of female subjects seen for routine gynecologic health care. Am J Obstet Gynecol, 183: 277, 2000

550. Hendrix, S. L., Clark, A., Nygaard, I. et al.: Pelvic organ prolapse in the Women's Health Initiative: gravity and gravidity. Am J Obstet Gynecol, 186: 1160, 2002

551. Handa, V. L., Garrett, E., Hendrix, S. et al.: Progression and remission of pelvic organ prolapse: a longitudinal study of menopausal women. Am J Obstet Gynecol, 190: 27, 2004

552. L awrence, J. M., Lukacz, E. S., Nager, C. W. et al.: Prevalence and co-occurrence of pelvic floor disorders in community-dwelling women. Obstet Gynecol, 111: 678, 2008

553. Bradley, C. S., Zimmerman, M. B., Qi, Y. et al.: Natural history of pelvic organ prolapse in postmenopausal women. Obstet Gynecol, 109: 848, 2007

554. Nygaard, I., Bradley, C., Brandt, D.: Pelvic organ prolapse in older women: prevalence and risk factors. Obstet Gynecol, 104: 489, 2004

555. Tegerstedt, G., Hammarstrom, M.: Operation for pelvic organ prolapse: a follow-up study. Acta Obstet Gynecol Scand, 83: 758, 2004

556. Rortveit, G., Brown, J. S., Thom, D. H. et al.: Symptomatic pelvic organ prolapse: prevalence and risk factors in a population-based, racially diverse cohort. Obstet Gynecol, 109: 1396, 2007

557. Eva, U. F., Gun, W., Preben, K.: Prevalence of urinary and fecal incontinence and symptoms of genital prolapse in women. Acta Obstet Gynecol Scand, 82: 280, 2003

558. Samuelsson, E. C., Victor, F. T., Tibblin, G. et al.: Signs of genital prolapse in a Swedish population of women 20 to 59 years of age and possible related factors. Am J Obstet Gynecol, 180: 299, 1999

559. Kumari, S., Walia, I., Singh, A.: Self-reported uterine prolapse in a resettlement colony of north India. J Midwifery Womens Health, 45: 343, 2000

560. MacLennan, A. H., Taylor, A. W., Wilson, D. H. et al.: The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. BJOG, 107: 1460, 2000

561. Mant, J., Painter, R., Vessey, M.: Epidemiology of genital prolapse: observations from the Oxford Family Planning Association Study. Br J Obstet Gynaecol, 104: 579, 1997

562. Boyles, S. H., Weber, A. M., Meyn, L.: Procedures for pelvic organ prolapse in the United States, 1979-1997. Am J Obstet Gynecol, 188: 108, 2003

563. Brown, J. S., Waetjen, L. E., Subak, L. L. et al.: Pelvic organ prolapse surgery in the United States, 1997. Am J Obstet Gynecol, 186: 712, 2002

564. Olsen, A. L., Smith, V. J., Bergstrom, J. O. et al.: Epidemiology of surgically managed pelvic organ prolapse and urinary incontinence. Obstet Gynecol, 89: 501, 1997

565. Shah, A. D., Kohli, N., Rajan, S. S. et al.: The age distribution, rates, and types of surgery for pelvic organ prolapse in the USA. Int Urogynecol J Pelvic Floor Dysfunct, 19: 421, 2008

566. Jelovsek JE, Barber MD, Paraiso MF, Walters MD. Functional bowel and anorectal disorders in patients with pelvic organ prolapse and incontinence. Am J Obstet Gynecol 2005;193:2105-11.

567. Kahn MA, Breitkopf CR, Valley MT, Woodman PJ, O'Boyle AL, Bland DI, Schaffer JI, Grady JJ, Swift SE. Pelvic Organ Support Study (POSST) and bowel symptoms: straining at stool is associated with perineal and anterior vaginal descent in a general gynecologic population. Am J Obstet Gynecol 2005;192:1516-22.

568. Morgan DM, DeLancey JO, Guire KE, Fenner DE. Symptoms of anal incontinence and difficult defecation among women with prolapse and a matched control cohort. Am J Obstet Gynecol 2007;197:509 e1-6.

569. Snooks SJ, Barnes PR, Swash M, Henry MM. Damage to the innervation of the pelvic floor musculature in chronic constipation. Gastroenterology. 1985;89:977-81.

570. Lembo A, Camilleri M. Chronic constipation. N Engl J Med 2003;349:1360-8.

571. Spence-Jones C, Kamm MA, Henry MM, Hudson CN. Bowel dysfunction: a pathogenic factor in uterovaginal prolapse and urinary stress incontinence. Br J Obstet Gynaecol 1994;101:147-52.

572. Arya LA, Novi JM, Shaunik A, Morgan MA, Bradley CS. Pelvic organ prolapse, constipation, and dietary fiber intake in women: a case-control study. Am J Obstet Gynecol 2005;192:1687-91.

573. Rortveit G, Brown JS, Thom DH, Van Den Eeden SK, Creasman JM, Subak LL. Symptomatic pelvic organ prolapse: prevalence and risk factors in a population-based, racially diverse cohort. Obstet Gynecol 2007;109:1396-403.

574. Varma MG, Hart SL, Brown JS, Creasman JM, Van Den Eeden SK, Thom DH. Obstructive Defecation in Middle-aged Women. Dig Dis Sci 2008.

575. Hendrix SL, Clark A, Nygaard I, Aragaki A, Barnabei V, McTiernan A. Pelvic organ prolapse in the Women's Health Initiative: gravity and gravidity. Am J Obstet Gynecol 2002;186:1160-6.

576. Tegerstedt G, Maehle-Schmidt M, Nyren O, Hammarstrom M. Prevalence of symptomatic pelvic organ prolapse in a

Swedish population. Int Urogynecol J Pelvic Floor Dysfunct 2005;16:497-503.

577. Bradley CS, Kennedy CM, Nygaard IE. Pelvic floor symptoms and lifestyle factors in older women. J Womens Health (Larchmt) 2005;14:128-36.

578. Yoshioka K, Matsui Y, Yamada O, Sakaguchi M, Takada H, Hioki K, Yamamoto M, Kitada M, Sawaragi I. Physiologic and anatomic assessment of patients with rectocele. Diseases of the Colon & Rectum. 1991;34:704-8.

579. Kelvin FM, Maglinte DD, Hornback JA, Benson JT. Pelvic prolapse: assessment with evacuation proctography (defecography)[comment]. Radiology. 1992;184:547-51.

580. Altman D, Lopez A, Kierkegaard J, Zetterstrom J, Falconer C, Pollack J, Mellgren A. Assessment of posterior vaginal wall prolapse: comparison of physical findings to cystodefecoperitoneography. Int Urogynecol J Pelvic Floor Dysfunct 2005;16:96-103; discussion 103.

581. Weber AM, Walters MD, Ballard LA, Booher DL, Piedmonte MR. Posterior vaginal prolapse and bowel function. American Journal of Obstetrics & Gynecology. 1998;179:1446-9; discussion 1449-50.

582. Ellerkmann RM, Cundiff GW, Melick CF, Nihira MA, Leffler K, Bent AE. Correlation of symptoms with location and severity of pelvic organ prolapse. American Journal of Obstetrics & Gynecology. 2001;185:1332-7.

583. Bradley CS, Brown MB, Cundiff GW, Goode PS, Kenton KS, Nygaard IE, Whitehead WE, Wren PA, Weber AM. Bowel symptoms in women planning surgery for pelvic organ prolapse. Am J Obstet Gynecol 2006.

584. Klingele CJ, Bharucha AE, Fletcher JG, Gebhart JB, Riederer SG, Zinsmeister AR. Pelvic organ prolapse in defecatory disorders. Obstet Gynecol 2005;106:315-20.

585. Altman D, Falconer C, Cnattingius S, Granath F. Pelvic organ prolapse surgery following hysterectomy on benign indications. Am J Obstet Gynecol 2008.

586. Mant J, Painter R, Vessey M. Epidemiology of genital prolapse: observations from the Oxford Family Planning Association Study. British Journal of Obstetrics & Gynaecology. 1997;104:579-85.

587. Swift SE, Pound T, Dias JK. Case-control study of etiologic factors in the development of severe pelvic organ prolapse. Int Urogynecol J Pelvic Floor Dysfunct 2001;12:187-92.

588. Swift SE. The distribution of pelvic organ support in a population of female subjects seen for routine gynecologic health care. Am J Obstet Gynecol 2000;183:277-85.

589. Dallenbach P, Kaelin-Gambirasio I, Dubuisson JB, Boulvain M. Risk factors for pelvic organ prolapse repair after hysterectomy. Obstet Gynecol 2007;110:625-32.

590. Forsgren C, Zetterstrom J, Lopez A, Nordenstam J, Anzen B, Altman D. Effects of hysterectomy on bowel function: a three-year, prospective cohort study. Dis Colon Rectum 2007;50:1139-45.

591. DeLancey JO. Anatomic aspects of vaginal eversion after hysterectomy. Am J Obstet Gynecol 1992;166:1717-28.

592. Marchionni M, Bracco GL, Checcucci V, Carabaneanu A, Coccia EM, Mecacci F, Scarselli G. True incidence of vaginal vault prolapse. Thirteen years of experience. J Reprod Med 1999;44:679-84.

593. Altman D, Zetterstrom J, Schultz I, Nordenstam J, Hjern F, Lopez A, Mellgren A. Pelvic organ prolapse and urinary incontinence in women with surgically managed rectal prolapse: a population-based case-control study. Dis Colon Rectum 2006;49:28-35.

594. Moalli PA, Jones Ivy S, Meyn LA, Zyczynski HM. Risk factors associated with pelvic floor disorders in women undergoing surgical repair. Obstet Gynecol 2003;101:869-74.

595. Wiskind AK, Creighton SM, Stanton SL. The incidence of genital prolapse after the Burch colposuspension. Am J Obstet Gynecol 1992;167:399-404; discussion 404-5.

596. Kjolhede P. Genital prolapse in women treated successfully and unsuccessfully by the Burch colposuspension. Acta Obstet Gynecol Scand 1998;77:444-50.

597. Denman M, Gregory W, Boyles S, Smith V, Edwards S, Clark A. Reoperation 10 years after surgically managed pelvic organ prolapse and urinary incontinence. Am J Obstet Gynecol 2008;198:555.e1-5.

598. Mushkat Y, Bukovsky I, Langer R. Female urinary stress incontinence--does it have familial prevalence? Am J Obstet Gynecol 1996;174:617-9.

599. Elia G, Bergman J, Dye TD. Familial incidence of urinary incontinence. Am J Obstet Gynecol 2002;187:53-5.

600. Hannestad YS, Lie RT, Rortveit G, Hunskaar S. Familial risk of urinary incontinence in women: population based cross sectional study. BMJ 2004;329:889-91.

601. Ertunc D, Tok EC, Pata O, Dilek U, Ozdemir G, Dilek S. Is stress urinary incontinence a familial condition? Acta Obstet Gynecol Scand 2004;83:912-6.

602. Jack GS, Nikolova G, Vilain E, Raz S, Rodriguez LV. Familial transmission of genitovaginal prolapse. Int Urogynecol J Pelvic Floor Dysfunct 2006;17:498-501.

603. McLennan MT, Harris JK, Kariuki B, Meyer S. Family history as a risk factor for pelvic organ prolapse. Int Urogynecol J Pelvic Floor Dysfunct 2008.

604. Twiss C, Triaca V, Rodriguez LV. Familial transmission of urogenital prolapse and incontinence. Curr Opin Obstet Gynecol 2007;19:464-8.

605. Chiaffarino F, Chatenoud L, Dindelli M, Meschia M, Buonaguidi A, Amicarelli F, Surace M, Bertola E, Di Cintio E, Parazzini F. Reproductive factors, family history, occupation and risk of urogenital prolapse. European Journal of Obstetrics, Gynecology, & Reproductive Biology 1999;82:63-7.

606. Rinne KM, Kirkinen PP. What predisposes young women to genital prolapse? European Journal of Obstetrics, Gynecology, & Reproductive Biology. 1999;84:23-5.

607. Bump RC. Racial comparisons and contrasts in urinary incontinence and pelvic organ prolapse. Obstet Gynecol 1993;81:421-5.

608. Duong TH, Korn AP. A comparison of urinary incontinence among African American, Asian, Hispanic, and white women. Am J Obstet Gynecol 2001;184:1083-6.

609. Sze EH, Jones WP, Ferguson JL, Barker CD, Dolezal JM. Prevalence of urinary incontinence symptoms among black, white, and Hispanic women. Obstet Gynecol 2002;99:572-5.

610. Fenner DE, Trowbridge ER, Patel DL, Fultz NH, Miller JM, Howard D, DeLancey JO. Establishing the prevalence of incontinence study: racial differences in women's patterns of urinary incontinence. J Urol 2008;179:1455-60.

611. Graham CA, Mallett VT. Race as a predictor of urinary incontinence and pelvic organ prolapse. Am J Obstet Gynecol 2001;185:116-20.

612. Sewell CA, Chang E, Sultana CJ. Prevalence of genital prolapse in 3 ethnic groups. J Reprod Med 2007;52:769-73.

613. Scherf C, Morison L, Fiander A, Ekpo G, Walraven G. Epidemiology of pelvic organ prolapse in rural Gambia, West Africa. BJOG: an International Journal of Obstetrics & Gynaecology 2002;109:431-6.

614. Hansell NK, Dietz HP, Treloar SA, Clarke B, Martin NG. Genetic covariation of pelvic organ and elbow mobility in twins and their sisters. Twin Res 2004;7:254-60.

615. Rohr G, Kragstrup J, Gaist D, Christensen K. Genetic and environmental influences on urinary incontinence: a Danish population-based twin study of middle-aged and elderly women. Acta Obstet Gynecol Scand 2004;83:978-82.

616. Dietz HP, Hansell NK, Grace ME, Eldridge AM, Clarke B, Martin NG. Bladder neck mobility is a heritable trait. Bjog 2005;112:334-9.

617. Lykken DT, McGue M, Tellegen A. Recruitment bias in twin research: the rule of two-thirds reconsidered. Behav Genet 1987;17:343-62.

618. Altman D, Forsman M, Falconer C, Lichtenstein P. Genetic Influence on Stress Urinary Incontinence and Pelvic Organ Prolapse. Eur Urol 2007.

619. Slemenda CW, Christian JC, Williams CJ, Norton JA, Johnston CC, Jr. Genetic determinants of bone mass in adult women: a reevaluation of the twin model and the potential importance of gene interaction on heritability estimates. J Bone Miner Res 1991;6:561-7.

620. Spector TD, Cicuttini F, Baker J, Loughlin J, Hart D. Genetic influences on osteoarthritis in women: a twin study. Bmj 1996;312:940-3.

621. Clayton D, McKeigue PM. Epidemiological methods for studying genes and environmental factors in complex diseases. Lancet 2001;358:1356-60.

622. Skorupski P, Krol J, Starega J, Adamiak A, Jankiewicz K, Rechberger T. An alpha-1 chain of type I collagen Sp1-binding site polymorphism in women suffering from stress urinary incontinence. Am J Obstet Gynecol 2006;194:346-50.

623. Andersson KE. A role for nitric oxide synthase in urinary "urge syndrome". Nat Med 1997;3:502-4.

624. Liu X, Zhao Y, Pawlyk B, Damaser M, Li T. Failure of elastic fiber homeostasis leads to pelvic floor disorders. Am J Pathol 2006;168:519-28.

625. Poncet S, Meyer S, Richard C, Aubert JD, Juillerat-Jeanneret L. The expression and function of the endothelin system in contractile properties of vaginal myofibroblasts of women with uterovaginal prolapse. Am J Obstet Gynecol 2005;192:426-32.

626. Choy KW, Yip SK. Necessary to evaluate the possible involvement of endothelin-1 gene polymorphisms in urinary dysfunction? Am J Obstet Gynecol 2006;194:300-1; author reply 301-2.

627. Nikolova G, Lee H, Berkovitz S, Nelson S, Sinsheimer J, Vilain E, Rodriguez LV. Sequence variant in the laminin gamma1 (LAMC1) gene associated with familial pelvic organ prolapse. Hum Genet 2007;120:847-56.

628. O'Boyle AL, Woodman PJ, O'Boyle JD, Davis GD, Swift SE. Pelvic organ support in nulliparous pregnant and nonpregnant women: a case control study. Am J Obstet Gynecol 2002;187:99-102.

629. Sze EH, Sherard GB, 3rd, Dolezal JM. Pregnancy, labor, delivery, and pelvic organ prolapse. Obstet Gynecol 2002;100:981-6.

630. Carley ME, Turner RJ, Scott DE, Alexander JM. Obstetric history in women with surgically corrected adult urinary incontinence or pelvic organ prolapse. Journal of the American Association of Gynecologic Laparoscopists 1999;6:85-9.

631. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. Obstet Gynecol 2006;107:1253-60.

632. Uma R, Libby G, Murphy DJ. Obstetric management of a woman's first delivery and the implications for pelvic floor surgery in later life. Bjog 2005;112:1043-6.

633. Tegerstedt G, Miedel A, Maehle-Schmidt M, Nyren O, Hammarstrom M. Obstetric risk factors for symptomatic prolapse: a population-based approach. Am J Obstet Gynecol 2006;194:75-81.

634. MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. BJOG: an International Journal of Obstetrics & Gynaecology 2000;107:1460-70.

635. Risk factors for genital prolapse in non-hysterectomized women around menopause. Results from a large cross-sectional study in menopausal clinics in Italy. Progetto Menopausa Italia Study Group. Eur J Obstet Gynecol Reprod Biol 2000;93:135-40.

636. Carley ME, Schaffer J. Urinary incontinence and pelvic organ prolapse in women with Marfan or Ehlers Danlos syndrome. American Journal of Obstetrics & Gynecology. 2000;182:1021-3.

637. McIntosh LJ, Mallett VT, Frahm JD, Richardson DA, Evans MI. Gynecologic disorders in women with Ehlers-Danlos syndrome. J Soc Gynecol Investig 1995;2:559-64.

638. Lind LR, Lucente V, Kohn N. Thoracic kyphosis and the prevalence of advanced uterine prolapse. Obstet Gynecol 1996;87:605-9.

639. Nguyen JK, Lind LR, Choe JY, McKindsey F, Sinow R, Bhatia NN. Lumbosacral spine and pelvic inlet changes associated with pelvic organ prolapse. Obstet Gynecol 2000;95:332-6.

640. Marshman D, Percy J, Fielding I, Delbridge L. Rectal prolapse: relationship with joint mobility. Australian & New Zealand Journal of Surgery 1987;57:827-9.

641. Norton PA, Baker JE, Sharp HC, Warenski JC. Genitourinary prolapse and joint hypermobility in women. Obstetrics & Gynecology. 1995;85:225-8.

642. Samuelsson EC, Arne Victor FT, Tibblin G, Svardsudd KF. Signs of genital prolapse in a Swedish population of women 20 to 59 years of age and possible related factors. American Journal of Obstetrics & Gynecology 1999;180:299-305.

643. Forsman M, Iliadou A, Magnusson P, Falconer C, Altman D. Diabetes and obesity related risks for pelvic reconstructive surgery in a cohort of Swedish twins. Manuscript submitted. 2008.

644. Strinic T, Eterovic D, Dujic Z, Markovic V, Tocilj J. Spirometric disorders in women with genital descensus. Acta Obstet Gynecol Scand 1997;76:879-83.

645. Nygaard I, Bradley C, Brandt D. Pelvic organ prolapse in older women: prevalence and risk factors. Obstet Gynecol 2004;104:489-97.

646. Woodman PJ, Swift SE, O'Boyle AL, Valley MT, Bland DR, Kahn MA, Schaffer JI. Prevalence of severe pelvic organ prolapse in relation to job description and socioeconomic status: a multicenter cross-sectional study. Int Urogynecol J Pelvic Floor Dysfunct 2006;17:340-5.

647. Strinic T, Bukovic D, Roje D, Milic N, Pavic M, Turcic P. Epidemiology of pelvic floor disorders between urban and rural female inhabitants. Coll Antropol 2007;31:483-7.

648. Jorgensen S, Hein HO, Gyntelberg F. Heavy lifting at work and risk of genital prolapse and herniated lumbar disc in assistant nurses. Occup Med (Lond) 1994;44:47-9.

649. Larsen WI, Yavorek T. Pelvic prolapse and urinary incontinence in nulliparous college women in relation to paratrooper training. Int Urogynecol J Pelvic Floor Dysfunct 2007;18:769-71.

650. Kopp Z, Irwin DE, Agatep B, Milsom I, Abrams P. Worldwide Estimates of Current and Future Individuals (≥20 years) With Lower Urinary Tract Symptoms Including Urinary Incontinence and Overactive Bladder. Poster presentation, 4th International Consultation on Incontinence, Paris 2008.

651. Irwin DE, Milsom I, Hunskaar S, Reilly K, Kopp Z, Herschorn S, Coyne K, Kelleher C, Hampel C, Artibani W, Abrams P. Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC study. Eur Urol 2006;50:1306-1315.

652. US Census Bureau, International Data Base. Data updated 3-27-2008. Retrieved April 14, 2008 2008

653. Homma Y, Yamaguchi O, Hayashi K. An epidemtiological survey of overactive bladder symptoms in Japan. BJU Int 2005;96:1314-1318.

654. Herschorn S, Gajewski J, Schulz J, Corcos J. A population-based study of urinary symptoms and incontinence: the Canadian Urinary Bladder Survey. BJU Int 2007;101:52-58.

655. Irwin DE, Milsom I, Kopp Z, Abrams P; EPIC Study Group. Symptom bother and health care-seeking behavior among individuals with overactive bladder. Eur Urol. 2008;53(5):1029-37.

656. Coyne KS, Sexton CC, Irwin DE, Kopp ZS, Kelleher CJ, Milsom I. The impact of overactive bladder, incontinence and other lower urinary tract symptoms on quality of life, work productivity, sexuality and emotional well-being in men and women: results from the EPIC study. BJU Int. 2008 Jun;101(11):1388-95.

657. Roe B, Doll H. Prevalence of urinary incontinence and its relationship with health status. J Clin Nurs 2000: 9:178.

658. Dallosso HM, Mathews RJ, McGrother CW, Clarke M, Perry S I, Shaw C et al. An investigation into nonresponse bias in a postal survey on urinary symptoms. BJU Int 2003: 91:631.

659. Seim A, Sivertsen B, Eriksen BC, Hunskaar S. Treatment of urinary incontinence in women in general practice: observational study. BMJ 1996: 312 :1459.

660. Nelson R, Norton N,Cautley E, Furner S, Community-based prevalence of anal incontinence. JAMA 1995:274:559.

661. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, and STROBE Initiative. Strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. BMJ 2007; 335;806-808.

662. Coyne KS, Sexton CC, Milsom I, Irwin DE, Kopp ZS, Chapple CR, Kaplan SA, Tubaro A, Thompson C, Aiyer LP, Wein AJ. The prevalence of lower urinary tract symptoms in the United States, the United Kingdom and Sweden: Results from EpiLUTS. BJU Int (In Press 2008).