Committee 3

Epidemiology and Natural History of Urinary Incontinence (UI)

Chairman

S. HUNSKAAR (NORWAY)

MEMBERS

K. BURGIO (USA),

A.C DIOKNO (USA),

A.R. HERZOG (USA),

K. HJÄLMÅS (SWEDEN),

M.C. LAPITAN (THE PHILIPPINES)

CONTENTS

Epidemiology and Natural History of Urinary Incontinence (UI)

S. HUNSKAAR

K. BURGIO, A.C DIOKNO, A.R. HERZOG, K. HJÄLMÅS, M.C. LAPITAN

I. INTRODUCTION

In this report we focus on the importance of understanding the epidemiology of urinary incontinence (UI) (distribution and determinants) as well as its natural history. We also discuss important topics as differences between epidemiological and clinical approach to a health problem, help seeking behaviour and methodological issues for this research.

The epidemiological population under study for this review will be community dwelling noninstitutionalized persons. The review will include discussion of the prevalence, incidence, natural history, and presence of racial and ethnic differences in the epidemiology of UI. We also reviewed correlates and potential risk factors that have been revealed in epidemiological studies.

The review examines our current state of knowledge of the epidemiology of UI, topics like the epidemiology of *fecal incontinence*, *pelvic organ prolapse* and *pelvic floor weakness* have been omitted. *Overactive bladder* (OAB) is an evolving concept with close relationship to UI. We have studied the scarce epidemiological literature on OAB, and have decided to include the urge incontinence part of OAB only (OAB with incontinence).

We have reviewed a large number of completed studies in the field of UI. We have emphasized high-quality and population based studies. We have also wanted to present studies from a broad variety of countries. 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 equal standard and useful information, but lack of space precluded their inclusion.

Progress has clearly been made during the 3 years since our previous report [1]. Some important areas have been studied with increasing regularity and quality. We have searched the literature for relevant new articles from 1997 to 2000, including clinical trials that might include relevant epidemiological data on UI.

Summary points:

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

II. BASIC EPIDEMIOLOGICAL CONSIDERATIONS

1. GENERAL COMMENTS, DESIGNS AND LEVELS OF EVIDENCE

In this report we emphasize the importance of understanding the epidemiology, and also give a summary of the basic concepts. 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 are always needed when interpreting the results from such studies, as associations found in epidemiological studies may not be the same as causes. In order to strengthen the validity of epidemiological studies longitudinal designs should be preferred, and appropriate control for confounding should be done. Experimental designs will seldom be used.

Recommendations and conclusions should alway be based on a sound evidence. Levels of evidence as used for therapeutic interventions do not fit into epidemiological studies. No uniform guidelines for assessing the results of such studies excists. The level of evidence in observational studies could be judged on the soundness of the exclusion of alternative explanations by statistical and other controls.

Even in many recent studies of UI analyses are very simple. Often only proportions or percentages are used to describe differences in the prevalence of UI in different subgroups. Many analyses do not control for confounders, there is lack of stratification, or multivariate techniques are not used. 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). An RR of 1.0 indicates that the rates in the exposed and nonexposed 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. An 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 between 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 i favour of having incontinence are 2.5 times higher among those with the risk factor than among those without.

Effects may be denoted as strong when RRs or ORs are 2.0 and more, and weak when RRs or ORs are less than 2.0, but there are no generally agreed standards. For a condition with high prevalence, like UI, 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.

2. DEFINITIONS

Studies of disease frequency should rely on a very specific definition of the condition under investigation. The absence of a unifying definition for UI is a fundamental problem which is not resolved. The lack of such a definition leads to problems with assessing sensitivity and specificity of the findings in epidemiological studies. Variations in availability and efficacy of health care around the world may influence the prevalence of UI.

a) Incontinence

UI can be defined in several ways, and one problem when analysing the epidemiological studies is in fact that incontinence *has* been defined in many ways. However, in order to get replicable results and be able to compare different studies, widely accepted definitions are important. The International Continence Society (ICS) defines incontinence as “a condition where involuntary loss of urine is a social or hygienic problem and is objectively demonstrable”. This definition may not be ideal for epidemiological purposes (will be discussed below). More common in epidemiological studies are definitions based on frequency of urine loss, e.g. “any uncontrolled urine loss in the prior year” or “more than two episodes in a month”. Such definitions imply that the studies are in fact studies of period prevalence.

b) Prevalence

Prevalence is defined as the probability of being incontinent 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. Prevalence of all-cause UI is estimated as the ratio of the number of incontinent respondents identified in a cross-sectional survey to the number of all respondents in the survey (i.e., continent and incontinent). Prevalences of specific types and severity levels are estimated in an analogous manner.

c) Incidence

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.

d) Type

Epidemiological surveys often must take a pragmatic approach, and therefore define incontinence type based on the symptoms alone. The classification can be done either by the researchers based on several questions, or by the respondant’s confirmation of a statement or typical description. Clinical assessment allows for more differentiation of subtypes. Possible biases will be discussed below.

e) Severity

Severity of incontinence is another important factor for the prevalence estimate. Severity can be defined by factors like frequency, amount and subjective bother. Examples of how the prevalence differs when based on different definitions for severity will be shown.

Summary points:

- Descriptive epidemiology is the description of disease incidence, prevalence (and mortality) by persons, place and time.
- Analytical epidemiology describes the search 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 multivariate techniques, and strength of associations should be determined by relative risks and odds ratios.

III. EPIDEMIOLOGY OF ENURESIS AND UI IN CHILDREN

1. GENERAL COMMENTS AND DEFINITIONS

The ICS definition of UI as “a condition where involuntary loss of urine is a social or hygienic problem and is objectively demonstrable” is not quite applicable to childhood UI. “Involuntary” is very difficult to assess in children, and since bladder control is gradually developing during childhood, the standards for what is socially acceptable will vary between families and with the age of the child. The objective demonstration of UI in a child rests on assessment by the parents. Different standards and definitions make metaanalysis difficult or impossible. It is thus essential to try to reach an international consensus about the grading of severity of UI in children.

Only isolated bedwetting should be termed *enuresis* according to recommendations issued by the ICCS (International Children’s Continence Society) [2]. The reason is that isolated bedwetting, *nocturnal enuresis,* is a strictly defined type of incontinence characterized by complete bladder emptyings during sleep without any symptoms pointing to bladder dysfunction. In contrast, almost all daywetting children, and children wetting both day and night, have bladder dysfunction of either a functional or organic nature and a great variety in their incontinence patterns. The one exception here is a small group of children with profuse daytime wetting due to incontinent complete bladder emptyings. Psychological factors are underlying this kind of rare childhood UI which is labelled *diurnal enuresis*.

Bedwetting should be characterized as primary or secondary, and as monosymptomatic or polysymptomatic. Monosymptomatic means that the child has no daytime symptoms. Monosymptomatic enuresis will thus be excluded if the child has day wetting, or frequency (>7 voidings per day in a 7-year-old), or infrequent voidings (<3 voidings per day), or imperative urgency. Monosymptomatic enuresis will not be excluded by moderate urgency which is found in 16% of 7-year-olds. Polysymptomatic cases are then defined as the non-monosymptomatic ones (Table 1) [3].

Nocturnal enuresis is also subdivided into *primary enuresis* (the child has not been dry for more than 6 months), and *secondary (onset) enuresis* (the bedwetting has recurred after a dry period lasting more than 6 months). Secondary enuresis may signify behavioural, neurological, infective causes, or chronic retention with overflow, and these require careful consideration.

Table 1 : Subdivision of nocturnal enuresis and diurnal urinary incontinence in children

Enuresis (nocturnal enuresis, bedwetting)

- Primary
  - Monosymptomatic
  - Non-monosymptomatic
- Secondary
  - Monosymptomatic
  - Non-monosymptomatic

Diurnal and nocturnal UI

- Functional (detrusor overactivity)
- Organic
  - Neuropathic
  - Structural
  - Infravesical obstruction
  - Epispadias
  - Other

Diurnal UI (day wettting only)

- Functional (detrusor overactivity)
- Organic
  - Neuropathic
  - Structural
  - Infravesical obstruction
  - Epispadias
  - Other

The healthy infant is socially incontinent but physiologically continent, because micturitions (about once every hour) are discrete and there is no leakage of urine in between micturitions [4]. Opinions regarding at what age the child is expected to become socially dry differ widely between cultures and ethnic groups. Meadow has suggested a reasonable age limit in stating that a wetting child above 5 years of age should be considered incontinent [5], although UI in children due to organic disorders is often evident already in infancy.

2. PREVALENCE OF NOCTURNAL ENURESIS

Most epidemiological studies link primary and secondary enuresis together and include also mon-symptomatic and poly-symptomatic cases. Also, enuresis is defined in different ways, and in many papers there is no frequency defined at all. The best studies are longitudinal cohort studies, but many are cross-sectional. In some cultures the parents are more complacent about the bedwetting than in others and do not regard it as a problem requiring attention. These problems in understanding epidemiology were summarized by Krantz et al. [6], who also reviewed the epidemiological studies that had been published until 1993.

Nocturnal enuresis, NE, is caused by relative nocturnal polyuria and/or nocturnal bladder overactivity combined with lack of arousal at the time when the bladder needs to be emptied. These factors have different weight in different enuretic children. The pathophysiology of NE is thus a mixed mechanism which explains difficulties encountered when trying to define enuresis in a consistent way. Stringent epidemiological studies would need to evaluate nocturnal urine production, nocturnal bladder activity, and sleep and arousal in each of the probands. Needless to say, there is no large population based study using such diagnostic evaluation.

a) Longitudinal cohort studies

A child development study has followed 1139 children born in one year in Dunedin (New Zealand) and obtained an impressive 92% follow-up 9 years later [7, 8]. It was found that primary enuresis usually remits with age (Figure 1). Another birth cohort born in 1977 in Christchurch (New Zealand) was followed up annually. By the age of 8 they found that 7% had nocturnal enuresis although this included some who had relapsed after initially becoming dry [9].

b) Survey Studies

Prevalence is defined at a specific point in time, but many studies have reported prevalence of enuresis without separately recounting data at specific ages, say seven-year-olds. Prevalence of NE at age seven is significant since many children then start school meaning more exposure to the environment and thus a greater awareness of the problem. At this age, the prevalence of nocturnal enuresis seems to be between 7 and 9 per cent (Table 2).

In more detail, the study by Hellström [10] found nocturnal enuresis in 7% of girls (of whom 3% monosymptomatic), and 12% of boys (of whom 7% monosymptomatic). Recent studies on the prevalence of nocturnal enuresis in different parts of the world are summarized in Table 3. In the early ages the prevalence in boys is reported to be higher than in girls by a 2:1 ratio in Western countries. In studies from other countries the figures are more similar in boys and girls, but there is always a predominance of boys. It seems that the sex difference diminishes by age and becomes less visible and less proven among elderly children.

In the French study [15] (Table 3), the severity and consequences of enuresis were studied in a sub-sample of 228 children (out of the 349 who had reported enuresis). In the sub-sample, 66% had more than one wet night per month, 37% more than one wet night per week, and 22% wet the bed every night. Regarding consequences, 42% of the 228 were “bothered a lot” while 15% were “not bothered at all” by their enuresis. In contrast, 92% of the 228 mothers declared that the enuresis had no effect on family life or the child’s behaviour at school. Fourteen per cent of mothers punished their child and only 13% intended to seek treatment for their child. The study from The Netherlands [18] (Table 3) reported that the prevalence of enuresis was higher in the Turkish/Moroccan ethnic group (14%) than in Dutch children (6%), and higher in children in special education (especially mentally retarded, 14%) than in children in mainstream education (6%).

Even if there are some ethnic and cultural differences of enuresis prevalence, with higher rates generally reported from Eastern countries, there is nonetheless a remarkable similarity of prevalence rates of nocturnal enuresis in populations from all parts of the world.

3. POTENTIAL RISK FACTORS OF NOCTURNAL ENURESIS

Several risk factors have been established or suggested by epidemiological studies, the most important will be discussed here (Table 4).

a) Family history

Nocturnal enuresis is a hereditary disorder. The mode of inheritance is autosomal dominant so if both parents were enuretic as children, the risk for their offspring is 77%, while if only one parent had NE, the risk is about 45%. In the Italian study by Chiozza [17], the odds ratio was highly significant for a family history of NE in

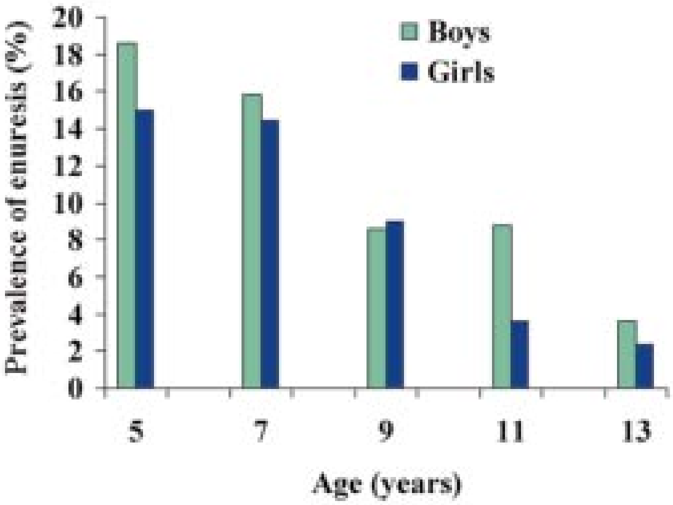

Figure 1 : Prevalence of enuresis, primary and secondary combined, for boys and girls separately. Data from [27] with permission. Comment: The prevalence in this study is higher than in most studies and the sex difference is less

Table 2 : Prevalence (% and 95% CI) of nocturnal enuresis at age seven. Results from two cross-sectional studies from Nordic countries, Hellström [10] and Järvelin [11] and one longitudinal study by Fergusson [9] from New Zealand. (Cited from Krantz et al [6]).

Author	Study period	Prevalence (% and 95% CI)
Hellström	1982	9.5 (8.5; 10.5)
Järvelin	1984	6.4 (5.3; 7.3)
Fergusson	1977-85	10.3 (8.3; 12.3)

Table 3 : Prevalence of nocturnal enuresis in recent studies. NE: Nocturnal enuresis, bedwetting. PMNE: Primary monosymptomatic nocturnal enuresis

Author	Year	Ref	Definition	Sample	Age	Prevalence (%)
Swithinbank	1994	12	>once/3 months	1 176	11-12	6 (boys), 4 (girls)
Bower	1996	13	once/month	2 292	5-12	19
Watanabe	1994	14	PMNE		7	15 (boys), 8 (girls)
Lottmann	1999	15	all NE	3 803	5-10 5-7	9 11
Nevéus	1999	16	once/month	1 413	6-10	8
Chiozza	1998	17	all NE	6 892	6-14	4
				674	7	8 (boys), 6 (girls)
Van der Wekke	1998	18	all NE	5 630	5-15 7-9 13-15	6 8 1
Serel	1997	19	all NE	5 523	7-12 7	14 (boys), 8 (girls) 16 (boys), 15 (girls)
Gümüs	1999	20	all NE	1 703	7-11	17 (boys), 11 (girls)
Kalo	1996	21	all NE	740	6-16 7	16 (boys), 14 (girls) 10 (boys), 14 (girls)
Lee	2000	22	all NE	7 012	7-12 7	9 14
Yeung	1997	23	all NE		3 521	4-12 4 7 5 (boys), 0.5 (girls)

Table 4 : Risk factors reviewed in this text

Nocturnal enuresis
- Family history
- Behavioural disturbances
- Nocturnal polyuria
- Sleep and arousal
- Nocturnal bladder dysfunction
- Other factors

Day and day/night wetting
- Family history
- Psychological disorders and sexual abuse
- Disorders of bladder-sphincter nerve control
- Urinary tract infections
- Infravesical obstruction
- Epispadias

siblings (3.1, 95% CI 1.8-5.6). By molecular genetic methods foci have been found on chromosomes 13, 12, 8, and 22 [24], [25]. A picture of pronounced heterogeneity of both genotype and phenotype is emanating [26]. The etiology of NE is thus characterized by a complex interaction of genetic and environmental factors.

b) Behavioural disturbances

Behavioural disturbances were for a long time thought to be associated with NE. Children with *secondary* enuresis may be more inclined to have associated mental health disorders later in adolescence. It is this group who might benefit from thorough assessment of any underlying emotional disturbance or psychopathology. A lack of association between *primary* NE and psychopathology in later childhood and adolescence was reported in 1990 by Feehan et al. [27] which seemed to confirm similar findings in an eight year follow-up study published in 1986 by Fergusson et al. [9]. But while it seems clear that psychopathology is not, with few exceptions, the *cause of* primary NE, evidence is accumulating that psychological consequences *caused by* enuresis should not be underestimated [28 - 30].

c) Nocturnal polyuria

In 1985, a virtual absence of day/night variation of vasopressin accompanied by nocturnal polyuria was found in children with NE [31]. For the first time, a coherent physiological explanation for NE, or at least a large part of the NE population, had been presented. Currently, there is a consensus that nocturnal polyuria is an important pathogenetic factor in two thirds of NE patients (those are the patients responding to desmopressin, DDAVP) while most of the remaining third have inadequate nocturnal bladder storage.

d) Sleep and arousal

Known physiological factors cannot explain why the enuretic child does not wake up to the sensation of a full or contracting bladder. Parents have expressed that enuretic children are very difficult to arouse or rather, as the parents put it, “sleeps very deeply”. Some recent studies seem to support the parents’ view. By using auditory signals [32], computerized EEG analysis [33], or enquiries [16], a defect in arousal has been largely validated. In the study by Nevéus, odds ratios were significantly high for high arousal threshold (2.7), pavor nocturnus (2.4), and confusion when awoken from sleep (3.4). Computerized EEG energy analysis has indicated both greater depth of sleep and impaired arousal in enuretics [34].

e) Nocturnal bladder dysfunction

Although bladder function in NE has been considered normal in many studies in the past, evidence about the bladder’s pathophysiological role for NE has accumulated during recent years. As many as one third of all enuretic children may have a nocturnal detrusor overactivity. Especially non-polyuric bedwetters, those who do not respond to desmopressin, should be suspected to have a malfunctioning bladder with reduced capacity.

In this context it is interesting to note that there seems to be an association between childhood NE and adult detrusor overactivity. People with a past history of NE have a greater risk of bladder dysfunction developing in, *or remaining into,* adult life. In a random cross-section of 2613 women 30-59 years of age, 17% reported incontinence. Childhood bedwetting was associated with a prevalence of urge incontinence with odds ratios between 1.8 and 2.2 depending on the age at which dryness was achieved [35]. In another retrospective study of 1000 urodynamic records on 500 men and 500 women, 10% of the men had idiopathic detrusor instability of whom 63% had a past history of NE, while 29% of the women had detrusor instability of whom 38% had a past history of NE beyond the age of 6 [36] The difference between men and women may reflect the gender difference in childhood bedwetting.

f) Other risk factors for nocturnal enuresis

Sleep apnoea has been associated with enuresis in some patients [37]. *Upper airway obstruction* also seems to be a cause of NE, since it is not uncommon for enuretic children to have their NE resolved after removal of large adenoids or tonsils. One study reports significant decrease or complete cure of NE in 87 (76%) of 115 enuretic children (of whom 103 with primary NE) after surgical removal of upper airway obstruction [38]. *Constipation* may cause secondary NE or make primary NE persist [39]. The important implication is that constipation has to be identified and treated in every child with NE. The polyuria in *diabetes mellitus and insipidus* increases the risk for NE which is most often of the secondary type. Children with *minor neurological dysfunction* are more vulnerable to NE, particularly if belonging to a lower social class [40]. Children *with attention deficit hyperactivity disorders (ADHD)* are 2.7 times more likely to have enuresis than the general child population [41]. Regrettably, *sexual abuse* must nowadays also count among factors that may lead to NE (most often secondary and non-monosymptomatic). A strong suspicion would prompt full investigation [42].

In addition, children with *urinary tract infection*, *infravesical obstruction, neuropathic bladder, psychiatric disorders*, and other conditions may be wetting their beds. Their nocturnal incontinence is, however, with very few exceptions combined with daytime symptoms, in particular daywetting. Primary monosymptomatic NE with bedwetting as the *one and only* symptom, is a well circumscribed condition that should be identified when present, thus avoiding clinical confusion generated by the huge array of childhood disorders that may have bedwetting as *one* of several symptoms.

4. LONG TERM OUTCOME OF NOCTURNAL ENURESIS

a) Remission and natural history

Longitudinal studies on the natural history of enuresis are no longer possible to perform since a large part of enuretic children receive treatment. Previous studies have confirmed the common knowledge that most children who are enuretic eventually become dry at night. The spontaneous cure rate was 14% annually between the ages of 5 and 9, 16% between 10 and 14, and 16% in the 15 to 19 year old group in the study by Forsythe and Redmond [43]. In their group there remained 33 patients who were still bedwetting at the age of more than 20 years (3%). Similar rates of prevalence relating to age were noted by Feehan et al. [27].

b) Prevalence of nocturnal enuresis in adults

The first really reliable epidemiological study on enuresis in adults in a randomly selected sample was performed in The Netherlands in 1996 [44]. To the question “Did you wet your bed at least once during the past 4 weeks?” 57 of 11 406 respondents answered “yes”, thus giving an overall enuresis prevalence of 0.5%. There was no significant difference between age groups. Primary NE was reported by 50% of the men and 19% of the women, the rest had secondary enuresis.

c) Long term follow-up studies

Ten years after evaluating 3556 seven-year-olds [10], a randomly selected subsample was reinvestigated [45]. In addition, 452 teenagers out of the 461 in the original cohort who had reported symptoms (imperative urge, day wetting, emptying difficulties, or bedwetting) were contacted. Bedwetting which had been reported by 12% of boys and 7% of girls aged 7 had decreased to only one girl (0.3%) still wetting at least once a month in the randomized group, and 1 boy (0.5%) and 2 girls (1%) still wetting in the symptomatic group. Almost all of the enuretic children had received treatment starting around 7-8 years of age. Swithinbank et al found a similar decline of prevalence of NE when they investigated the original cohort of 11-12-year-old schoolchildren again at 15-16 years [12]. Nocturnal enuresis was reported by 5% of children at 11-12 years but only 1% at 15-16 years. A Finnish study [30] looked at 501 14-year-olds who had reported NE at age 8 in an original study of 5603 children. At age 14, only 16 adolescents remained enuretic (0.3% of the original total sample).

The enuresis prevalence of 0.5% in otherwise healthy adults in the Dutch study [44] refers to a largely untreated population. Fifty percent of the men had primary enuresis so they had never been consistently dry at night. Assuming a prevalence of enuresis of 8% in 7-year-old boys, this could be translated to mean that the risk for an enuretic boy to remain enuretic for the rest of his life is 3% if he does not receive active treatment during childhood. Three per cent equals the prevalence found in patients after the age of 20 years in the study by Forsythe and Redmond [43] and in the Finnish 14-year-olds [30]. It is still not clear whether active treatment of NE in childhood is able to reduce the number of adult enuretics.

5. PREVALENCE OF DAY WETTING, AND DAY/NIGHT WETTING

Diurnal UI as well as combined diurnal and nocturnal UI will here be reported as two manifestations of the same basic disorder; *overactive bladder* (urgency, frequency and urge incontinence). A few children have a clinical picture resembling the detrusor-sphincter dyssynergia of the neuropathic bladder, but without a neurological malformation or disease, this condition is termed *non-neuropathic bladder-sphincter dysfunction* (NNBSD) or in short *dysfunctional voiding* [46].

a) Prevalence in survey studies

Compared to nocturnal enuresis, the prevalence of diurnal or combined diurnal and nocturnal incontinence in childhood has not been extensively studied. No true longitudinal cohort studies have been published.

Prevalence of diurnal or combined diurnal and nocturnal incontinence has been reported in a few cross-sectional studies (Table 5). Diurnal or combined diurnal and nocturnal UI at least once a week seems to occur in about 2-4% of 7-year old children and is more common in girls than in boys.

6. POTENTIAL RISK FACTORS OF DAY AND DAY/NIGHT WETTING

a) Family history

Day wetting seems to be more common in children of mothers who had diurnal UI themselves. This clinical impression has not been the object of epidemiological research. But Chiozza et al. [17] found that the highest risk for familiality of enuresis was shown by *non-monosymptomatic* NE (with diurnal frequency and urgency; OR 12.3), suggesting a hereditary background not only for bedwetting itself but also for associated bladder overactivity.

b) Psychological disorders and sexual abuse

Children in stress or with mental disorders have a higher incidence of diurnal UI as well as NE, mostly of the secondary type [48]. A special case is the rare disorder of *diurnal enuresis* which is the only kind of diurnal UI which takes place as a complete, incontinent micturition. Sexual abuse may also lead to secondary day wetting.

c) Disorders of bladder-sphincter nerve control

With few exceptions, children with *myelomeningocele* have neuropathic bladder-sphincter dysfunction with UI due to detrusor hyperreflexia and/or sphincter denervation. Skin-covered *occult spinal dysraphism* may be mistaken for non-neuropathic dysfunctional bladder. At least half of the children with *spastic cerebral palsy* have clinically silent bladder dysfunction. *Nerves* in the pelvic area may be injured by trauma such as pelvic fractures, but more common is iatrogenic trauma during surgery for imperforate anus or pelvic tumors.

d) Urinary tract infections (UTI)

Regular and complete voidings are the most efficient way to prevent urinary infection. A bacterial cystitis induces temporary detrusor overactivity. Thus, bladder dysfunction may cause UTI, and UTI may cause bladder dysfunction. The UTI may be asymptomatic. Girls with asymptomatic bacteriuria (ABU) have symptoms of an overactive bladder, such as urgency and diurnal UI, in a high percentage [49]

e) Infravesical obstruction

Boys born with posterior urethral valves are incontinent in about 20% on long-term follow up after surgical treatment [50]

f) Epispadias

Epispadias may include a malformation of the sphincter resulting in constantly dribbling UI.

7. LONG TERM OUTCOME OF DAY AND DAY/NIGHT WETTING

a) Epidemiology of dryness in the day

Few studies recount the prevalence of daytime dryness. In Minnesota, 60% of children were dry in the day at two and a half years of age. Between 4 and 5 years of age, 92-95% of children in Sweden, The Netherlands and USA were dry in the day. Corresponding figures for the age group between 5 and 6 years were 95-96%, and

Table 5 : Prevalence of diurnal or combined diurnal and nocturnal UI. NNBSD: Dysfunctional voiding (see text)

Author, year	Ref	Definition	Age	Prevalence (%)
Hellstrøm 1982	10	Once a week Day wetting only NNBSD	7	2.1 (boys), 3.1 (girls) 0.5 (boys), 1.1 (girls) 1.0 (boys), 2.0 (girls)
Järvelin 1984	11	Isolated diurnal UI Diurnal/nocturnal	7	1.8 1.6
Bloom 1993	47	Once in 2 weeks	6-8	9.0
Neveus 1999	16	Diurnal UI only	6-11	4.4
Swithinbank 1994	8	Diurnal UI	11-12	0.2 (boys), 0.9 (girls)
Lee 2000	22	Diurnal UI	7-12	2.2
			7	3.9
		Diurnal/nocturnal		1.4
			7	2.8

94-97 for the age group between 6 and 7 years [51]. It thus seems that children who are and remain dry in the day attain their diurnal continence already between 4-5 years of age.

b) Long term follow-up studies

Hellström et al found day wetting at least once a week at age 17 in 0.2% of boys and 0.7% of girls and as compared to 2.1% and 3.1%, respectively, at age 7 years [45]. Swithinbank et al had found a prevalence of day wetting (including also "occasional" wetting) in 12.5% in children at age 10-11 years declining to 3.0% at age 15-16 years [12]. Based on these, it seems that the prevalence of all kinds of diurnal UI will diminish with 1-2% per year of life from age 10-11 to age 15-16 years, while diurnal UI at least once a week seems to diminish with 0.2% per year of life from age 7 to age 17 years. Because of treatment the studies may not recount the true natural history.

Summary points

Nocturnal enuresis

- Primary enuresis refers to bedwetting occurring without a dry continence break of more than 6 months. Secondary enuresis refers to recurrence after a longer interval of dryness. Most epidemiological studies in the past have unfortunately combined primary and secondary enuresis together. Equally, the majority of previous studies have not kept monosymptomatic (no daytime symptoms) apart from non-monosymptomatic NE.

- The prevalence of nocturnal enuresis at age 7 in Western countries seems to be around 8%, and in an largely untreated adult population 0.5%.

- Primary nocturnal enuresis usually remits with age. The spontaneous cure rate seems to be around 15% annually between the ages of 5 and 19.

- The risk for an enuretic 7-year-old boy to remain enuretic throughout life may be calculated to 3%.

- Potential risk factors for enuresis in children reviewed in this test include family history, behavioural disturbances, nocturnal polyuria, sleep and arousal, nocturnal bladder dysfunction, sleep apnoea and upper airway obstruction, constipation, diabetes, minor neurological dysfunction and ADHD, and sexual abuse.

- It seems clear that psychopathology is not the *cause of* primary NE, while evidence is accumulating that NE has psychological consequences.

- Nocturnal polyuria seems to be the major pathogenetic factor in 2/3 of bedwetting children (those responding to antidiuretic treatment with desmopressin), while NE in the remaining third seems to be caused by bladder dysfunction that may occur only during sleep.

- There is some evidence that people with a past history of enuresis have a greater risk of bladder dysfunction developing in, or remaining into, adult life.

Day and day/night wetting

- Children who are and remain dry in the day seem to attain their diurnal continence already between 4-5 years of age.

- Diurnal UI, or combined diurnal and nocturnal UI, in children is caused by overactive bladder in the great majority of cases.

- Potential risk factors for diurnal UI in children include family history, psychological disorders, disorders of bladder-sphincter nerve control, urinary tract infections, infravesical obstruction, epispadias, and sexual abuse.

- The prevalence of diurnal UI at least once a week at age 7 seems to be around 2-4%, declining with about 0.2% per additional year of life.

- More research is needed in order to elucidate the epidemiology and genetics of childhood diurnal UI and its association with bladder dysfunction.

IV. EPIDEMIOLOGY OF UI IN WOMEN

1. PREVALENCE

A large number of epidemiological studies of UI in women have been published. Some cover a wide age span while others are surveys of a single age cohort or specific group like pregnant women. Differences in sample, definition and measurement, and survey methodology continue to make reviews challenging. Table 6 lists some of the prevalence figures reported for women sampled from community-residing populations, showing widely varying estimates. Several reviews of epidemiologic studies of UI are available, including some quite recent ones [52 - 58].

More epidemiologic research is available on older women of all ages because UI is considered to be a health condition of older age. A 1988 review of several European and American epidemiologic studies of older women living in the community identified a 10-40 %

Table 6 : Examples of prevalance of any UI in studies of women living in the community

First author	Ref	Year	N	Age	Prevalence (%)
Yarnell	59	1981	1 000	17+	45
Holst	60	1988	851	18+	31
Sommer	61	1990	414	20-79	40
Brocklehurst	62	1993	2 124	30+	9
Sandvik	63	1993	1 820	20+	29
Swithinbank	64	1999	2 075	18+	69
Hannestad	65	2000	27 936	20+	25
Temml	66	2000	1 262	20+	26
Bartolotti	67	2000	5 488	40+	11
Moller	68	2000	2 860	40-60	72

range of prevalence estimates of the experience of any UI and suggested a UI prevalence of 40% [55]. More recent reviews [56 - 58] that summarize the literature through about 1997 confirm this or even a wider range of prevalences and continue to suggest that the wide range can be attributed to the definition of UI and the sample [58] and potentially to the format of the questions about UI [55, 57]. Since these recent reviews several additional studies have been published on the prevalence of UI among older women [65 - 67, 69 - 73]. But again, a range of prevalence between 10 and 40% or even wider remains a reasonable estimate. Table 7 lists specific prevalence estimates of any urinary incontinence among older women living in the community.

The prevalence of any UI by age is addressed by several of these studies. In our previous report [1] we described the general finding of higher prevalence estimates in older ages. We also noted the intriguing suggestion of an early prevalence peak in midlife and then a steady increase among the aged. A recent study by Hannestad and her colleagues [65] of women of all ages examplifies this latter pattern. The study finds a gradual increase of prevalences across adulthood until age 50 when prevalence reaches 30%, a stabilization or even slight decline until age 70 when prevalences start rising again (Fig 2).

Prevalence has always been higher in institutions because residents in institutions tend to be older and more impaired than commuity residing women. A recent review [56] suggests a range from 6 to 72% of institutional prevalences. Several recent studies from around the world [80 - 87] — some of which use samples of institutions rather than individual institutions — suggest prevalences of 50% or higher.

The range of prevalences in institutions is likely a consequence of the definition and admission criteria of residential care which vary throughout the world. In Australia, for instance, they covers the spectrum from requiring very low levels of supportive care to the highest levels of assistance as found in most high level nursing homes in other countries of the world. And even within a given country definition and criteria vary from city to city and facility to facility. For this reason it is important to sample residential care facilities in the same fashion as individuals in order to obtain representative results about prevalences in such facilities.

The process underlying the relatively high prevalences in residential care facilities remains somewhat less clear. A Canadian study [77] documented a statistically nonsignificant odds ratio of 1.4 for UI among women in institutions compared to community living women after statistically controlling for age, dementia and ambulatory function. Thom and co-workers showed that existing urinary incontinence increases the risk of subsequent hospitalisation and substantially increases the risk of admission into nursing homes, independent of age, gender or the presence of any co-morbid condition [88] These investigators demonstrated that the relative risk of admission to a nursing home was two times greater for incontinent women and 3.2 times greater for incontinent men after adjustment for age, cohort factors and comorbid conditions. These findings as well as others suggest that incontinence may be a contributing factor to institutionalization. However, at this point an alternative explanation of residential care contributing in some way to the development of incontinence cannot be excluded with confidence.

2. TYPE

In surveys based on questionnaires or interviews only *symptoms* can be registered. Typically stress incontinence is identified when the respondent reports it to

Table 7 : Examples of prevalence of any UI in studies of older women living in the community

First author	Ref	Year	N	Age	Prevalence (%)
Yarnell	59	1981	1 060	65+	50
Diokno	74	1986	1 150	60+	38
Molander	75	1990	4 206	70-90	17
Sandvik	63	1993	1 820	60+	32
Brocklehurst	62	1993	840	60+	12
Holtedahl	76	1998	507	60-74	47
Hunskaar	77	1998	1 876	65+	17
Roberts	78	1998	762	50+	58
Chiarelli	79	1999	12 417	70-75	35
Iglesias	70	2000	486	65+	42
Maggi	71	2001	1 531	65+	22

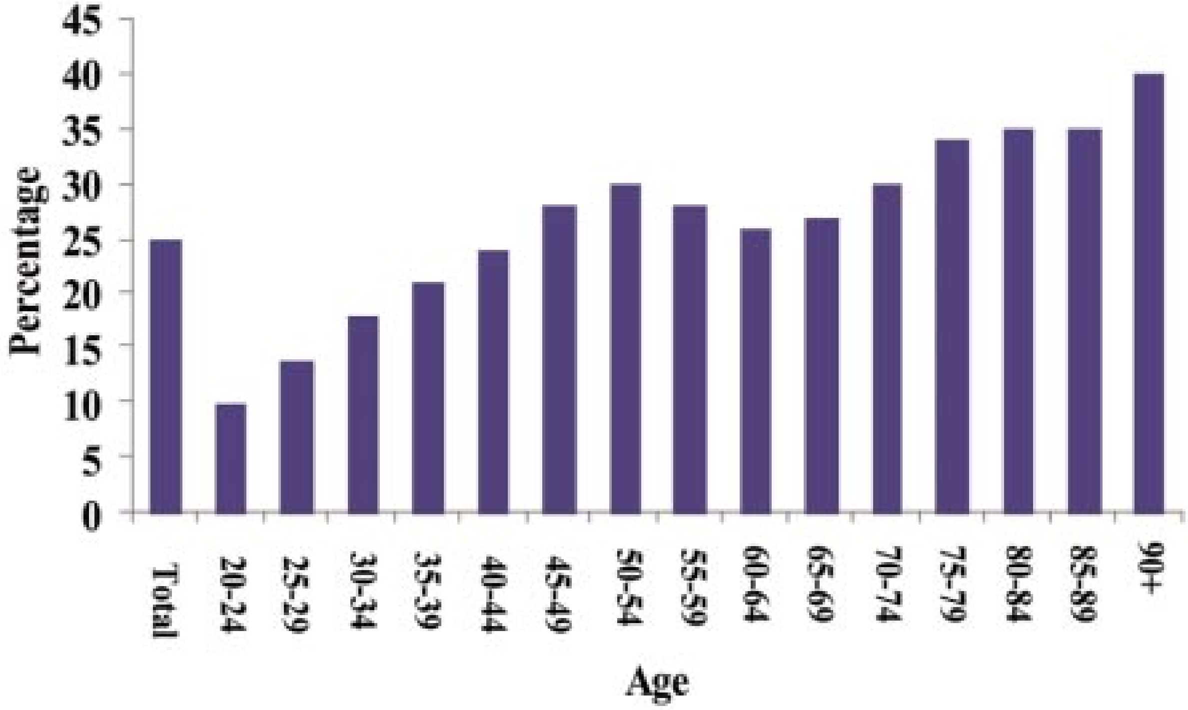

Figure 2 : Prevalence of UI (any leakage) in women 20 years+. Data from the EPINCONT study [65]

occur with physical activity, urge incontinence when it occurs in the context of a sudden urge to urinate. Diagnoses such as motor urge incontinence or genuine stress incontinence require the use of urodynamic equipment and thus cannot be made based on data from questionnaires or interviews alone.

Several authors have reported the relative proportions of stress, urge, and mixed symptoms. A few authors reported stress and urge separately without a mixed category. In Table 8 the distribution of UI types are shown, as they are found in some surveys. Overall, approximately half of all incontinent women are classified as stress incontinent, the highest proportion among urge, mixed, and stress types. A smaller proportion is classified as mixed incontinent, the smallest one as urge incontinent. The recent NOBLE telephone survey found an overall prevalence of overactive bladder with urge incontinence of 9.6% in women 18+, increasing with age from less than 5% in age group 18-44 to 19% in age group 65+ [97].

Proportions of types of UI differ by age. A survey of older women suggests that mixed incontinence predominates [74]. A survey of young and middle-aged women suggests that pure stress incontinence predominates in that age group [96]. A recent study which includes the entire age range by Hannested et al. [65] demonstrates a fairly regular increase in prevalence of mixed incontinence across the age range, and a regular decrease in prevalence of stress incontinence (Fig 3). The question has been raised about the use of self reports in epidemiological studies, and low accuracy has been found [98]. Sandvik and coauthors [95] validated diagnostic questions used in a survey against a final diagnosis made by a gynecologist after urodynamic evaluation. After using the validity (sensitivity and specificity) as basis for correcting the type distribution, the percentage of stress incontinence increased from 51 to 77 %, mixed incontinence was reduced from 39 to 11% and urge incontinence increased from 10 to 12 %. The authors warn that mixed incontinence may be overreported in epidemiological surveys, and that correction for validity indicates that a larger majority of women than hitherto reported may have pure stress incontinence. However, there are also some limitations in the use of urodynamics in documentating the presence of involuntary detrusor contractions, with both false positives and false negatives occurring. Therefore the use of urodynamic testing as a gold standard may not be entirely appropriate in establishing the true prevalence of the different types, especially urge incontinence.

Unfortunately, not all studies have carefully assessed the different types (and even fewer have examined their correlates). Therefore, proportions of stress, urge and mixed types among women are difficult to estimate and estimates vary considerably. But there are intriguing differences between the different types suggesting that the types may reflect quite different pathologies and that differentiating the types in future research might prove very fruitful.

3. SEVERITY

Most studies have operationalized the measurement of severe incontinence by measuring frequency of urine loss. Those reporting weekly or even more frequent loss are typically assigned to the severe category. Fewer studies have also used a quantity measure by asking about amount of loss, dampening of clothes, extra laundry, restrictions in activity, or the use of protective pads. The first approach may be considered a simple attempt to operationalize the frequency of urine loss, the second approach reflects quantity but also perceptional differences, personal hygiene and coping ability. Some studies have explicitly combined a frequency and a quantity measure [63, 74] Sandvik's Severity index [63, 99] is calculated by multiplying the reported frequency (four levels) by the amount of leakage (dichotomized to two levels). The resulting index value is further categorized into slight, moderate and severe. Typically, slight incontinence denotes leakage of drops a few times a month, moderate incontinence daily leakage of drops, and severe incontinence larger amounts at least once a week. The severity index has been validated against a 48 hour «pad weighing» test. According to this test, slight incontinence means a leakage of 6 g/24 hours (95% CI 2-9), moderate incontinence means a leakage of 17 g/24 hours (95% CI 13-22) and severe incontinence means a leakage of 56 g/24 hours (95% CI 44-67). The severity index is thus a semi-objective and quantitative measure, and does not include the woman`s subjective perception of her leakage as being a problem or not. A recent validation from Scotland concludes that the Severity index is a short, simple, valid, reliable, and sensitive measure of urinary incontinence in women, and that it can be recommended for routine use [100].

It can be shown that the prevalence is dependent on "thresholds" for diagnosis or severity For example, Sandvik [63] found that nearly half of cases were classified as having slight incontinence and only 27 % as severe. The author also investigated the "botherness" factor, and found that different levels of botherness significantly affected the prevalence estimates. If only those with moderate or severe incontinence are considered, and including only those who are bothered by

Table 8 : Relative proportions of different types of UI among women living in the community

First author	Ref	Year	Age	N	Stress (%)	Urge (%)	Mixed (%)
Yarnell	59	1981	18+	1 000	49	21	30
Iosif	89	1984	61	902	40	27	33
Diokno	74	1986	60+	1 955	29	10	61
Hording	90	1986	45	515	75	11	14
Holst	60	1988	18+	851	52	25	23
Elving	91	1989	30-59	2 631	48	7	45
Sommer	61	1990	20-79	414	38	33	29
Molander	75	1990	70-90	4 206	24	49	27
Burgio	93	1991	42-50	541	50	12	38
Harrison	92	1994	20+	314	48	9	44
Lara	94	1994	18+	556	48	27	21
Sandvik	95	1995	20+	1 820	51	10	39
Samuelsson	96	1997	20-60	487	68	9	23
Bartolotti	67	2000	40+	5 488	61	26	13
Hannestad	65	2000	20+	27 936	52	37	11
		Median (range)			**49 (24-75)**	**21 (7-49)**	**29 (11-61)**

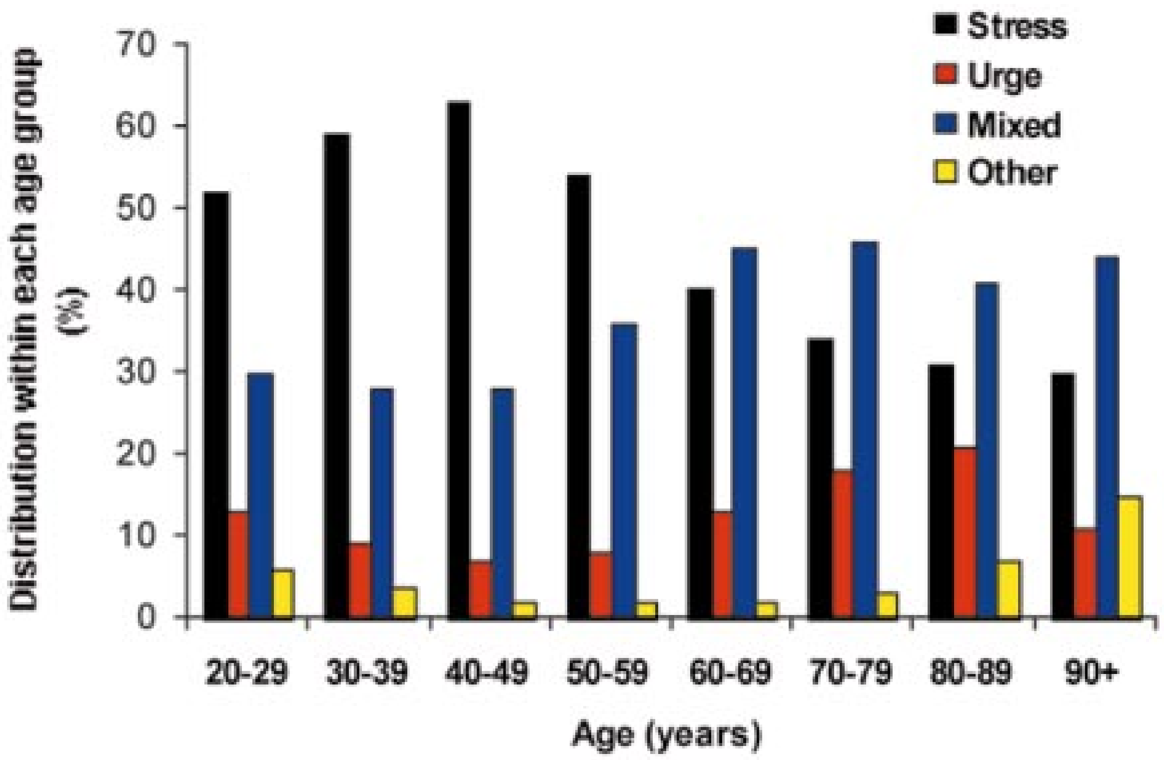

Figure 3 : Distribution of different types of UI in age groups. Data from the EPINCONT study [65]

their leakage, the findings from that study indicate that approximately one fifth of incontinent women suffer from severe incontinence. These results have recently been confirmed in the EPINCONT study [65] (Figure 4 and 5)

The severity of incontinence varies between the different types. The fraction of severe incontinence is much lower in the stress group compared to the urge and mixed groups. In the EPINCONT study [65], slight incontinence was found in 53% in the stress group, 39% in the urge group and 31% in the mixed group (Figure 6). Within each type of incontinence, severity increased with increasing age.

Even though the definition of severe or "significant" UI varies between authors, its prevalence is considerably less variable across different studies than prevalence of any UI. Prevalence estimates range between 3% and 17% (Table 9). The lesser variance among these estimates suggests that severe incontinence is less easy to deny and better understood by participants than "any incontinence" and thus may represent a more reliable figure.

Several studies found that the prevalence of significant incontinence tended to increase with advancing age [59, 61, 65, 70] In the EPINCONT study the percentages were 2.6, 6.5, 8.6, and 13.0 for age groups 20-39, 40-54, 55-69, and 70+, respectively [65].

In summary, it is quite clear that the prevalence estimates depend on which definition of severity is used. Nevertheless, prevalence estimates of severe or significant UI display a less wide range than any UI, ranging from 3 to 17%, with a cluster between 6-10%. Moreover, it is not well established what level of severity should be regarded as clinically significant and might be indicative for treatment.

4. INCIDENCE, REMISSION AND NATURAL HISTORY

The epidemiological data are sparse about the development or the natural history of UI and its types and severity levels. Data are needed regarding the transition from continence to various levels of severity and type of incontinence.

A few studies have reported on the incidence of UI. A study of community dwelling women aged 60 years or older found that 20% of the originally continent women had developed any UI during the 1-year study period [102]. A cohort of healthy middle aged women was examined over three years [93]. Of the previously continent women 8% reported at least monthly leakage, higher rates have been found in the elderly [103]. Two recent studies of young and middle-aged women reported 1-year incidence rates of 6% and 3% [68], [104].

Likewise, rates of remission (the probability of becoming continent among previously incontinent women) vary considerable across the few studies that have investigated them, ranging over one year between 10% for older women [102], 6% [96] and 38% [68] among middle-aged and younger women. What is very clear from these findings is that substantial incidence rates are paralleled by equally substantial remission rates. It is not clear whether the level of remission reflects active treatment or intervention or whether it is part of the natural course of incontinence. Although questions

Table 9 : The prevalence of "any" and "severe" UI among older women living in the community.

First author	Ref	Year	N	Age	Any UI (%)	Severe UI (%)
Vetter	101	1981	1 342	70+	14	5
Diokno	74	1986	1 955	60+	38	3
Burgio	93	1991	541	42-50	58	17
Brocklehurst	62	1993	840	60+	12	8
Sandvik	63	1993	1 820	18+	29	6
Hunskaar	77	1998	1 876	65+	17	7
Bartolotti	67	2000	5 488	40+	11	6
Hannestad	65	2000	27 936	20+	25	7
Samuelsson	96	2000	487	20-60	28	8
Moller	68	2000	2 860	40-60	72	16
Maggi	71	2001	1 531	65+	22	11
		Median (range)			**29 (11-72)**	**7 (3-17)**

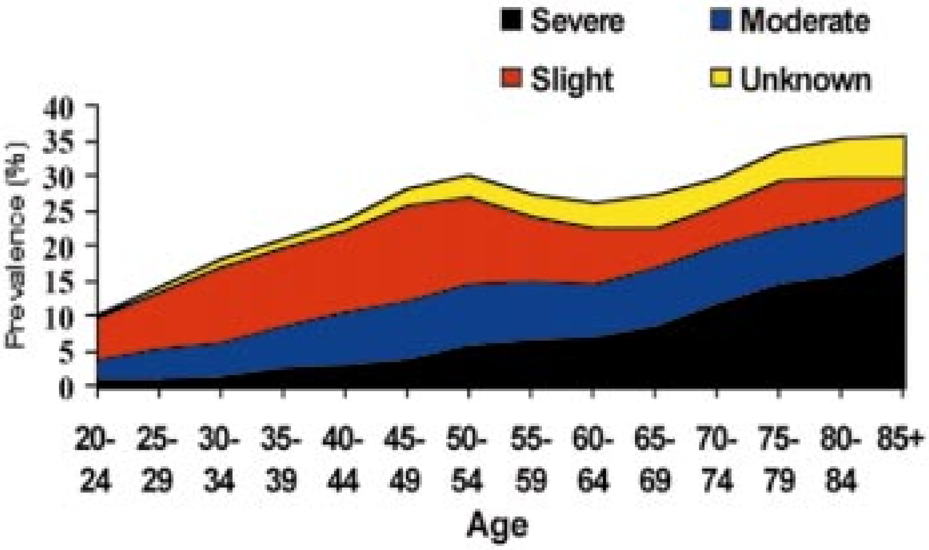

Figure 4 : Prevalence of UI by age group and severity. Data from the EPINCONT study 65

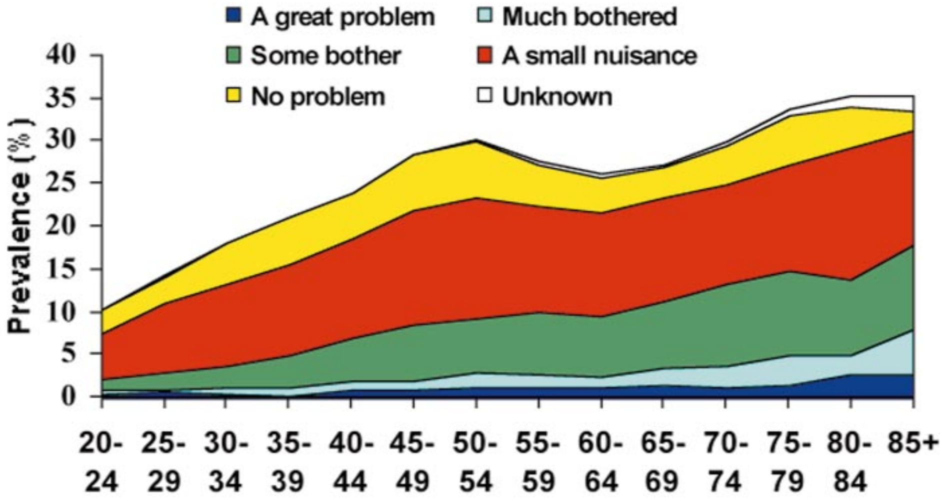

Figure 5 : Prevalence of UI by age group and impact. Data from the EPINCONT study 65

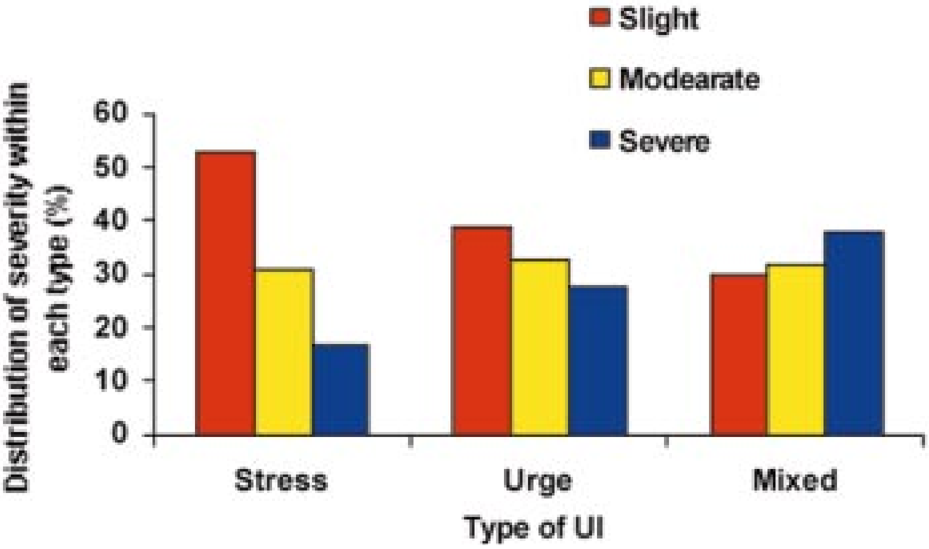

Figure 6 : Severity of the different types of UI. Data from the EPINCONT study 65

about causal factors had been investigated in the studies, few relevant findings emerged.

In summary, longitudinal studies of incidence, remission and natural history are scarce and should be encouraged because these types of studies provide information about the course of UI and are best suited to investigate its causes and consequences. Incidence rates are not insubstantial, but it has proven difficult to establish predictors for incident UI. It is also well documented that remission can take place, but again predictors are not well understood. Remission may be related to natural recovery or to medical care, but unreliable measurements cannot be excluded either.

5. RACIAL AND ETHNIC DIFFERENCES

Most epidemiological studies of UI have been conducted on white populations. There are several studies of non-caucasian women showing a wide variation in prevalence [72, 73, 94, 105 - 112] The studies have used different methods and definitions, and the quality is heterogenious. Therefore the results are difficult to compare, and most of the studies do not lend themselves easily to crosscultural or crossnational comparisons.

Regarding black women, some data exist, and they provide some evidence that white women may be more susceptible to UI than black women. For example, some early studies examined UI predominately in the black African population. Reports by Heyn's state that black South Africans rarely developed stress incontinence and developed the related disorder of the pelvic floor prolapse at a rate 80 times lower than whites [113, 114]. Later studies of South African women attempted to explain the rarity of stress incontinence among blacks as a function of differing urethral pressures and length as well as pubococcygeal muscle strength [115, 116]. No difference in the prevalence of minor nulliparous stress incontinence among white (46 %), Indian (42 %) and black South African nurses (40 %) was demonstrated. While pelvic floor prolapse has been reported to be exceedingly rare among black South Africans it is the most common indication for major surgery in the Pokot people along the Kenyan Ugandan border [117]. In the US, Howard and her colleagues [118] report clinical data suggesting that African American women have higher uretral closure pressure, larger urethral volume, and greater vesical mobility.

Given the variability in methodology across studies, ideally comparative data are collected on different racial groups using an equivalent methodology. In the United States, the Establish Populations for Epidemiologic Studies of the Elderly reported an equal prevalence of UI among Caucasians and African American women [119]. In contrast Burgio et al. reported significant differences in regular and infrequent incontinence in African American women (18% and 15% respectively) compared to white women (32% and 28%) [93]. However only 51 of 541 women in this study were African American. In a clinical study of patients referred for evaluation of UI or prolapse, Bump and colleagues found that a larger proportion of white than African American women reported symptoms of stress incontinence (31% vs. 7%), and a larger proportion were diagnosed urodynamically as having genuine stress incontinence (61% vs. 27%) [120]. In his report, white women had a prevalence of pure genuine stress incontinence 2.3 times higher than African American women. This difference in physiologic subtypes was supported by a recent presentation of similar studies, confirming a significant difference in the predominance of stress incontinence in the white women [121]. This difference may, however, be explained by racial differences in help seeking behaviour, since it involved clinical groups. Racial differences have also been reported among pregnant women. However, the differences were evident only for stress incontinence and not for urge incontinence or other types of urine leakage [122].

Recently several large population-based studies have reported on differences between African and White American women. Fultz and her colleagues [57] found lower prevalences of urinary incontinence among African than among White Americans in well-controlled analyses; the difference was only reliable for moderate and severe forms of incontinence though. Brown and her colleagues [123] [124] showed lower prevalence of stress and mixed UI among African than White Americans, even after controlling on age and a number of health problems. Thom and his colleagues [125] reported prevalences of general UI to be higher among White Americans and were able to explain the difference by health problems and parturition factors.

In summary, there remains a paucity of information regarding UI in non-caucasian women world wide. The existing comparative data on differences between Caucasian and African American women in the US, although not entirely consistent, suggests higher prevalence of UI among Caucasian women that may be due primarily to racial differences in stress UI and attributable to pelvic and urodynamic features possibly related to childbirth. More needs to be learned about risk factors, whether they operate in a similar fashion within the different races, which ones might be able to explain racial differences, and whether and how the impact of incontinence differs by racial and cultural context.

6. POTENTIAL RISK FACTORS

Epidemiological studies conducted in various populations reveal a number of variables related to UI including several possible risk factors or contributing variables (Table 10). Most of the data regarding risk factors for the development of UI have been derived from cross sectional studies of volunteer and clinical subjects. Risk factors like smoking, menopause, restricted mobility, chronic cough, chronic straining for constipation, and urogenital surgery have not been as rigorously studied as age, parity, and obesity. This provides us with information of limited generalizability and restricts the level of inference regarding causality.

Well controlled analyses of potential risk factors and predictors are limited. Little is known about their relative and absolute value. Risk factors or causes of UI need to be investigated in a prospective or longitudinal design in order to establish the temporal ordering between risk factors and onset of UI. Unfortunately, very few longitudinal studies of UI have been conducted. Therefore this review of health-related factors is based primarily on cross-sectional studies and can only identify correlates.

Table 10 : Established and suggested risk factors for UI in women reviewed in this text

- Age
- Pregnancy
- Childbirth
- Menopause
- Hysterectomy
- Obesity
- Lower urinary tract symptoms
- Functional impairment
- Cognitive impairment
- Occupational risks
- Family history and genetics
- Other factors

a) Age

Because UI is so common among older women, it is often regarded as a normal and inevitable part of the aging process. Most studies indicate that UI is indeed correlated with age [59, 65, 71, 79, 80, 95, 103, 123, 126 - 130]. In one well-known study, a random sample of 842 women 17-64 years of age were interviewed [59]. The prevalence rates increased steadily with age. In another large survey, prevalence of UI in women 46-86 years old increased progressively over seven birth cohorts (1900 - 1940) from 12 % to 25 % [127] More recent studies have yielded similar results in large samples of women. One study of 27,936 women 20+ years of age replicated the significant association between age and incontinence [65] (see Figure 2). Another study found a significant effect for age in a study of 40,155 women in three age groups: young (18 – 23), middle-aged (45 – 50), and older (70 – 75). Though age was significant, there were no differences between the middle-aged and older women [79]. Some studies have also found that age was a significant risk factor for urge incontinence, but not for stress incontinence [103, 123]. Studies that have reported a nonsignificant relationship between incontinence and age generally have had smaller sample size and more narrow age range perhaps restricting the ability to detect this effect [58, 76, 78]

Incontinence is not to be considered normal with aging; however, there are changes in the bladder and the pelvic structures that occur with age and which can contribute to UI [131, 132] Further, UI is often attributable to medical problems or diseases that can disrupt the mechanisms of continence (e.g., diabetes mellitus), many of which are more common among older adults.

b) Pregnancy

UI in women is often assumed to be attributable to the effects of pregnancy and childbirth. The literature shows that UI is a more common occurrence among pregnant women compared with other groups of women. Prevalence rates of 31 % and 60 % have been reported [122, 133]. UI during pregnancy is a self-limiting condition for most women. Viktrup et al found a prevalence rate of 28% who developed stress incontinence during pregnancy, and 16% became free of symptoms in the puerperium [134]. However, there is some speculation that women who are incontinent during pregnancy may be predisposed to experiencing UI at later times in their lives, such as during a subsequent pregnancy, or as they age.

It is still questionable whether pregnancy itself is a risk factor for UI in later life or if it is the vaginal delivery that is the main risk factor. Iosif et al found a prevalence of 9% of persisting stress UI after cesarean deliveries [135]. Viktrup et al compared continent women having delivered vaginally with women who underwent a cesarean section and found a difference in favour of cesarean. However, three months after delivery, the difference became statistically insignificant [134].

c) Childbirth

The role of childbearing in predisposing women to UI is supported by several studies that have demonstrated a link between UI and parity [61, 91, 92, 122, 128, 136 - 139] There are several explanations that may be offered [140]. First, childbirth may result in pelvic floor laxity as a consequence of weakening and stretching of the muscles and connective tissue during delivery. Second, damage may occur as a result of spontaneous lacerations and episiotomies during delivery. The result of these events can be impaired support of the pelvic organs and alteration in their positions. A third possibility is that the stretching of the pelvic tissues during vaginal delivery may damage the pudendal and pelvic nerves, as well as the muscles and connective tissue of the pelvic floor, and can interfere with the ability of the striated urethral sphincter to contract promptly and efficiently in response to increases in intra-abdominal pressure or detrusor contractions.

The evidence for the relationship between childbearing and UI is presented in several studies. For example, Thomas and colleagues reported that UI was most likely to occur in parous rather than nulliparous women at all ages (15-64 years) [141]. Furthermore, UI was most common in women who had four or more children. Others have found a threshold at one delivery [142, 144]. Holst and Wilson found that UI was less common in nulliparous women but found no association with rates of UI and increasing parity [60]. However, Samuelsson et al and Jolleys suggested that the relationship between increasing rates of UI and increasing parity was linear [136, 145]. In addition, pregnant women with UI have been found in one study to have a history of more pregnancies and more births than pregnant women who were continent [122]. Vaginal delivery in particular is believed to cause pelvic neuropathy that could instigate UI [143, 146, 147].

Only 3% of women were found to have troublesome stress incontinence one year after their first vaginal birth [134]. This association between childbirth and UI diminishes with age. Foldspang found that although parity was a risk for stress incontinence, the effect decreased with age [139]. Two studies of middle-aged women have found no association between parity and UI. Hording and colleagues found that frequency of UI in 45-year-old women was not associated with frequency of births [90]. Similarly, Burgio and colleagues found that healthy perimenopausal women with UI were not any more likely to have delivered more children than continent women [93].

Until recently age as a confounder has been little focused upon when studying parity and UI. A huge 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 old women (70-75 years) the association disappeared [79]. Similar results were found in a Norwegian study [144]. Nevertheless, the literature shows that age is a stronger risk factor than parity

Also there has been done little research whether time since delivery or age at delivery will influence the effect of parity. Foldspang et al found increased risk of UI with increasing age at the last childbirth for women aged 30-44 years [139]. One study found that women over 30 years old at their first delivery were at higher risk [143], while another found that increasing age at the first delivery had an influence on UI [148].

The effect of parity on the severity of UI has been little investigated. Kuh et al found a statistically significant trend for increasing severity with increasing parity [143], while Rortveit et al found no effect on severity [144]. Generally, stress UI is believed to be the incontinence type associated with parity. Kuh et al have showed such an association, but they could not find an association between urge type and parity [143].

Several obstetric factors have been explored as possible contributors to the development of incontinence. Evidence has emerged that vaginal delivery may predispose a woman to incontinence, more so than a Cesarean section. One study of incontinence in pregnancy investigated the role of prior deliveries and found that having had a prior vaginal delivery increased by 5.7 the risk of incontinence in a subsequent pregnancy compared to having no prior deliveries, but having had a Cesarean section did not increase the risk [149]. Similarly, vaginal delivery at older ages (> 30) were associated with increased risk of stress incontinence (OR=3.1) in one investigation [143] However, Cesarean section carried an increased risk of 1.7 and there were too few respondents to make a valid comparison.

Data on other obstetric techniques and complications are inconsistent. Thom and colleagues [125] found an increased risk of UI with increased exposures to oxytocin (OR = 1.9 for one exposure, 3.1 for two exposures). Foldspang et al [129] found strong associations between incontinence and forceps delivery/vacuum extraction, episiotomy, and perineal suturing in bivariate analyses. However, the effects of these variables were insignificant in multivariate analyses possibly suggesting that they may simply have been markers for having had a vaginal delivery. In one study, 11 obstetric variables were explored [149]. Only birthweight > 4,000g and mediolateral episiotomy were associated with incontinence in subsequent pregnancy (OR=1.9 and 1.9). All other variables including perineal laceration, vaginal laceration, vacuum extraction, oxytocin stimulation, and length of 2nd stage labor were not significant. Other studies have reported no relationship between incontinence and a number of obstetric variables, including episiotomy, sphincter lesion, forceps delivery, lacerations, epidural anesthesia, long labor, and fetal birth weight [125, 145, 150, 151].

Vesico-vaginal fistula is an important cause of disastrous UI in developing countries, usually due to obstetric trauma [152]. Differences in birthing practices world wide (including route of delivery and availability of obstetric care) should be investigated to determine potential relationship to continence status.

d) Menopause

Clinically, it has long been understood that urinary symptoms are an integral part of the transition from the premenopausal to the post-menopausal state. The atrophic changes increase susceptibility to urinary tract infections and can cause storage symptoms (such as urinary frequency and urgency), dysuria, vaginal dryness, and dyspareunia. Given the evidence that atrophy of these tissues can be reversed with estrogen, and that estrogen replacement reduces UI in some cases, it seems reasonable to propose that estrogen loss contributes to the problem.

However, the literature is inconsistent in describing the role of menopause and estrogen loss as significant contributors to UI. Positive findings were reported by Rekers and colleagues, who compared premenopausal women (N=355) with postmenopausal women (N=858) and found no significant difference in the prevalence of UI between the two groups (25% versus 26%) [126]. However, there were significant differences in the frequency of incontinence episodes, indicating that postmenopausal women had more severe incontinence. Postmenopausal women were more likely to have UI on a daily basis or more frequently (7 %), compared to the premenopausal women (3 %). They were also much more likely to have urgency and nocturia. Postmenopausal women were less likely, however, to have large volume accidents, and there were no differences in the types of UI. These investigators also examined the time frame between menopause and the onset of UI. A significant increase in the incidence of UI occurred 10 years before the menopause, and an even larger increase was found at menopause. Among postmenopausal women with UI, 28% had onset before menopause, 18% around the time of menopause, and 54 % after menopause. Finally, women who experienced a surgical menopause had a higher rate of UI (36 %) compared to those who experienced a natural menopause (22 %).

Other studies have found no significant differences between postmenopausal and premenopausal women in the prevalence or the frequency of incontinence [89, 130, 143]. Some studies have even reported significantly lower prevalence rates among postmenopausal women than among premenopausal women [92, 136, 143], though in one, the effect was significant for stress incontinence but not for urge incontinence [143]. Further, recent studies of risk factors have found that incontinence is not associated with the number of years since menopause [123], age at menopause [76], nor mean age of natural menopause [125].

Related to menopause is the issue of estrogen replacement therapy. While one might expect lower rates of incontinence in women taking hormone replacement therapy, three studies have reported a two to three times increased risk of incontinence in older women taking estrogen [96, 125, 145]. In the HERS study the estrogen/progestin group worsened their UI compared to the controls [153].

e) Hysterectomy

When asked about the onset of UI, many women will report that it began immediately following hysterectomy. A hysterectomy with oophorectomy puts a woman into surgical menopause. This may imply a hormonal mechanism a cause of UI. Recently, with the development of neurophysiological investigations to measure neurologic impairment of the pelvic floor, the question has been raised whether the development of post-hysterectomy UI might be caused by nerve damage during the procedure. It may also be due to disturbances of musculo-fascial attachments of the bladder to the surrounding pelvic wall [154].

Well controlled prospective studies investigating the role of hysterectomy are scarce and results are mixed. In studies utilizing univariate analyses, some have shown significant associations between UI and hysterectomy [79, 127], as well as oophorectomy [79]. Milsom and colleagues, in a survey of 3,896 women, reported that those who had a hysterectomy were more likely to report UI than those who had not (21% vs. 16 %), and this trend occurred across five birth cohorts from 1900 to 1920 [127].

Some studies have reported no association between hysterectomy and UI in general [90] and stress incontinence in particular [143]. Others have found a significant relationship in univariate analyses but nonsignificant results after controlling for other variables in multivariate analyses [125, 143] Studies using multivariate analysis models also yielded inconsistent results. Two studies found a positive association between hysterectomy and incontinence with odds ratios of 1.5 and 2.4 [151, 155]. In another, there was no association between UI and hysterectomy with an odds ratio of 1.07 [76].

In a review of the clinical literature on the effects of hysterectomy, Thom and Brown [156] concluded that most studies did not find an increase in UI in the first two years after surgery. Also, UI was decreased after surgery in some studies. A subsequent study of the clinical effects of hysterectomy examined the prevalence of stress and urge incontinence before and one year after hysterectomy and found no detrimental changes [157].

Thus, there is evidence to suggest that hysterectomy may place a woman at risk for UI and contradictory information to suggest that hysterectomy may not contribute to the development of UI. Thus, the role of hysterectomy remains controversial [155, 158]. Further study of the relationship of hysterectomy to UI could clarify the issue and potentially yield surgical techniques that would minimize the risks of UI.

f) Obesity

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 preganancy, may bear down on pelvic tissues causing chronic strain, stretching and weakening of the muscles, nerves, and other structures of the pelvic floor. Anecdotally, patients are known to report improvement in symptoms of UI associated with weight loss and increased severity with weight gain. In addition, there is clear epidemiological support for the role of obesity in UI.

Data from several studies indicate that UI in women is associated with higher body mass index and greater weight [67, 76, 79, 93, 123, 125, 128, 137, 143, 151, 155, 159, 160]. In one study, a significant relationship was found between UI and body mass index such that women with regular UI had the highest mean body mass index and those who had never been incontinent had the lowest mean body mass [161, 93]. Dwyer and colleagues found that obesity was significantly more common among women with detrusor instability, as well as among those with stress incontinence, compared to continent women [161]. Similarly, in several other studies, increased body mass index was found to be an independent risk factor for incontinence [76, 103, 123, 125, 143, 149, 160]. One study found a positive ”almost linear association” between BMI and both stress incontinence and urge incontinence [151]. The highest BMI quartile had a 4.2 times greater risk of stress incontinence and a 2.2 times greater risk of urge incontinence. Two studies found that increased BMI was a risk factor for stress incontinence but not for urge incontinence [123, 143].

Other investigators found no associations between UI and obesity [150, 162, 163]. One of these did find that women with a positive stress test (i.e., clinically demonstrated loss of urine with physical stress (coughing) had a higher body mass index than those who had a negative stress test [162]. The other was restricted to the study of postpartum incontinence and found that antenatal BMI did not predict postpartum UI [150]. Fischer et al [163] found a significant relationship on univariate but not on multivariate analyses. This study was limited in that there was a small range of BMI values and only 5 respondents had a BMI > 30. The possibility that the effect was dependent on a threshold value was reflected in the results of another study that found an association between UI during pregnancy and prepregnancy BMI > 35 (OR=2.5), but a lack of significance with BMI >30 (OR=1.7) [149].

In addition to the associations found between obesity, body mass index, and UI, confirmatory results have been reported for intervention studies. Bariatric surgery was used in one study to drastically reduce weight in a group of morbidly obese women [164]. As a group, the women had both subjective and objective resolution of stress as well as urge incontinence. In another study, weight reduction by bariatric surgery resulted in reduction of stress incontinence from 61 % to 12 % of the group [165]. Thus, there is strong evidence to support the causal role of excess weight in the development of UI. A link between body mass and UI supports the concept that weight gain may increase susceptibility to incontinence and suggests that weight loss may decrease incontinence.

g) Lower urinary tract symptoms (LUTS)

Irrespective of whether due to disease or normal aging, urinary tract symptoms such as blood in urine, cloudiness or foul smell in urine, burning during urination, trouble starting urine flow, inability to shut off urine flow, needing to push and strain while urinating, or needing to urinate more than once to empty bladder emerge as one of the most critical set of correlates and potential precursors of UI from the analysis of the MESA data [74]. UI is further related to fecal incontinence [74, 80]. Others have also found a strong correlation between LUTS and UI [151], and the transition from overactive bladder without incontinence to the form with incontinence should be studied.

h) Functional impairment

Another set of health-related correlates that have been substantiated in several studies are functional impairments, particularly mobility limitations [71, 74, 77, 80, 81, 103, 160, 166, 167]. Odds ratios from a recent study demonstrated the increasing likelihood of incontinence with worsening level of mobility from requiring no help with mobility to needing support (1.8), depending on care providers (5.63), and being wheelchair or bedbound (7.38) [80]. 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=2.15), inability to move heavy objects (OR=1.89) and inability to climb stairs (OR=1.66) [103]. In addition improvement in ADLs was associated with 3-year remission rates of urge incontinence (OR=0.5). Another large study of older adults 70+ years of age showed that UI was associated not only with functional impariment, but also with sensory impariment, which may contribute to mobility limitations [160]. Mobility problems include having experienced a fall during the last 12 months, being diagnosed with arthritis, currently using equipment to get around, being restricted from going out, and several performance measures of lower body physical functions.

The exact interpretation of the relationship between functional impairment and UI, however, 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 UI is a predictor of frailty as shown in a study of 1531 older women [71]. Longitudinal information is needed to sort out the direction of causation between functional impairments and UI.

Alternatively, mobility limitations and UI may both be consequences of general frailty in older age or of an underlying systemic illness such as a stroke. There is evidence of an association between UI and stroke [79], [103], [160] as well as Parkinson's disease [71] [103]. UI may be a direct consequence of neurological damage caused by these diseases or an indirect result of the physical limitations imposed by these diseases.

i) Cognitive impairment

Studies in nursing homes have suggested a link between dementia and UI [80, 81, 84, 168 - 169]. Two recent studies of UI in nursing home residents have shown an association between UI and cognitive impairment using multivariate analyses. In one, patients lacking mental orientation had a 3.6 times greater risk of being incontinent than those with normal mental status [80]. In the second, the presence of dementia increased the odds of UI by 2.3 [81]. However, in a community sample, no relationship was found between mental status and difficulty holding urine [170].

In the Canadian Study of Health and Aging it was found a strong association between severity of dementia and UI in elderly women [77]. Odds ratios were 1.2, 4.0,and 12.6 for mild, moderate and severe dementia, respectively, after controling for age, residence, and ambulatory function.

A systematic review of 11 studies examining the rate of UI in persons with dementia [171] concluded that UI is common in patients with dementia and is more prevalent in demented than in nondemented older individuals.

j) Occupational risks

Currently there is a dearth of knowledge to aid medical providers in advising their patients about occupational factors that promote either the onset or recurrence of urinary incontinence.

A study of 274 female aircrew revealed that 26% experienced UI [163]. Most (89%) reported that incontinence occurred off duty, and 18% reported UI on duty while flying. Crew position affected the risk of UI, but no other occupational variables were associated with UI, including flying high performance military aircraft, the number of hours flown, or type of aircraft (high gravity vs low gravity).

k) Family history and genetics

Parallel to the developments in genetics and molecular biology, there is an interest for investigating many medical conditions for genetic components. Little is known about a genetic component for UI.

Twin studies are the best genetic studies. Only one has been identified in the literature [172]. The inheritability was significant for urge, but not for stress UI in a study of 161 MZ and 249 DZ twins aged 75+. Data suggests that about half the risk for urge incontinence is inherited in this age group.

Family studies may find evidence for familial clustering. One study found that family history was associated with stress UI [173]. Relative risks for mothers, sisters, and daughters of women with stress incontinence were 2.8, 2.9, and 2.3, respectively.

l) Other factors

Other published articles have reported correlations between UI and several other variables, including history of cystitis or urinary tract infections [80, 123, 143, 159, 174], diabetes [79, 103, 123], previous gynecological surgery [76, 92, 136] constipation [79, 151, 175], fecal incontinence [80], use of diuretics [128, 151], other drugs [103, 176], caffeine comsumption [177] perineal suturing [136], exercise [178], genital prolapse [90, 145], radiation [179] impaired function of the levator muscles [76, 90, 145] childhood enuresis [35, 143] current and former cigarette smoking [145, 180], respiratory problems [71] and nighttime awakening [71]. A recent observational study [88] reviewed the medical records of 5986 members aged 65+ of a large health maintenence organization in California. There was an increased risk of UI associated to the diagnoses of Parkinson's disease, dementia, stroke, depression, and congestive heart failure.

Summary points

- The prevalence estimates of at least some degree of UI show a wide range. The variability may be explained by factors such as variations in the definition of UI, in collection of the study samples, or variation in survey procedures, or effects of intervention.
- The median level of prevalence estimates gives a picture of increasing prevalence during young adult life (prevalence 20-30%), a broad peak around middle age (prevalence 30-40%), and then a steady increase in the elderly (prevalence 30-50%).

- Proportions of types of UI are difficult to estimate, and estimates vary considerably. Approximately half of all incontinent women are classified as stress incontinent. A smaller proportion is usually classified as mixed incontinent, and urge incontinence is the smallest category. Little is known about the risk factors and demographic correlates of the different types, but the types probably reflect different pathologies and etiologies.
- The prevalence of severe or “significant” incontinence (depending on frequency and amount of leakage, soaking of clothes, use of pads etc.), is rather consistent, and ranges between 3% and 17%, with most studies reporting between 6% and 10%.
- Incidence studies are scarce and should be encouraged. Remission can occur, but we know little about the rates of remission or its predictors. Such studies will also give a better understanding of the dynamics between risk factors and the onset of UI.
- There remains a paucity of published information regarding UI in non-caucasian women world wide. The data suggests higher prevalence of UI among Caucasian women that may be due primarily to racial differences in the prevalence of stress UI.
- A number of medical correlates of UI have been identified, but need to be investigated in a prospective, longitudinal, well controlled design in order to establish the temporal ordering between risk factors and onset of UI.
- Well controlled analyses of potential risk factors and predictors are limited. Age, childbearing, urinary symptoms, and functional impairment remain the best established risk factors.

V. EPIDEMIOLOGY OF UI IN MEN

1. 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 the 3 last years. In almost all 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 are much different between the sexes, and risk factors, although less investigated in men, seem to be different. We have found no reviews of the epidemiological studies of male UI.

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 such as weak stream, hesitancy, dribbling or impotence exist.

An area in male UI that is being studied and reported with increasing regularity in the last few years is post-prostatectomy incontinence. Since radical prostatectomy is being performed with increased frequency in recent years, the incidence of post-prostatectomy UI is now being scrutinized in greater detail.

2. PREVALENCE

Some of the major reviews also discuss the prevalence of UI in men [52, 53, 55], ranging from 3% reported by Feneley [181], 5% by Schulman [182], 9% by Thomas [141] and Malmsten [183], and 11% by Yarnell [184].

There are no studies reporting prevalences for men according to the ICS definition. But for any definition, there is a steadily increase in prevalence with increasing age (Table 11), than for women.

Due to differences in pathological anatomy and pathophysiology of UI in men, 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%) [55, 69, 74, 111, 182]. The increasing prevalence of UI as age increases observed in men is largely due to the contribution of the 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 group respectively [111]. The recent NOBLE telephone survey found an overall prevalence of overactive bladder with urge incontinence of 2.6% in men 18+, increasing with age from less than 1% in age group 18-44 to 9% in age group 65+ [97].

Most studies have a large fraction of other/unclassified types. One recent study found that constant dribbling was reported by 7.4% of their respondents [111]. Terminal dribbling or postvoid dribbling is another type of leakage in men that is difficult to assign to the conventional subtypes of UI. In a recent Australian survey, 12% of respondents reported frequent terminal dribbling [185].

When it comes to severity, the sex differences do not seem to be different from those for any incontinence. Estimates for severe UI in older women tend to be about twice as high as for older men [55].

We have found no studies adressing racial or ethnic dif-

Table 11 : Examples of prevalence of UI across age spectrum in men

First author	Ref	Year	Distribution by age	Prevalence (%)
Yarnell	184	1979	65	9
			70 - 80	8
			80+	22
Thomas	141	1980	45 - 54	5
			55 - 64	9
			65 - 74	15
			75+	18
Diokno	74	1986	60+	19
Malmsten	183	1997	45	4
			50	4
			60	5
			70	7
			75	10
			80	20
			90+	28
Schulman	182	1997	45 - 49	2
			50 - 54	5
			60 - 64	6
			70+	14
Bartolotti	67	2000	51 – 60	2
			61 – 70	3
			70+	7
Ueda	111	2000	40 – 49	1
			50 – 59	2
			60 – 69	4
			70+	4

ferences in the prevalence of UI in men. However, unpublished data from the MESA study do not indicate differences in prevalence among white male respondents compared to African American respondents.

Literature on incidence of male UI is very scarce. The MESA study [102] found a one year incidence rate for elderly men of 9%. Substantial remission rates were also obtained, rates that were higher among men (27%) than women (11%). one likely explanation of the relative instability of male Ui focuses on the predominance of urge type incontinence among men. Urge UI is often caused by prostate gland disease, infections, or bowel dysfunction, all of which are relatively amenable to treatment or improve even without treatment.

Malmsten [183] analysed the age of onset for each age cohort. Mean debut age for all men was 63 years, mean duration was about 8-10 years in the cohorts.

3. POTENTIAL RISK FACTORS

There is relative little research concerning conditions and factors that may be associated with UI in men, and clear risk factors are more seldom scientifically documented (Table 12). However, a few available studies have identified potential risk factors which are described below.

Table 12 : Risk factors for UI in men reviewed in this text

- Age
- Lower urinary tract symptoms (LUTS)
- Functional and cognitive impairment
- Neurological disorders
- Prostatectomy
- Other Factors

a) Age

As for women increasing age is correlated with increasing prevalence of UI. There seems to be a more steadily increase in prevalence with increasing age, than for women.

b) Lower Urinary Tract Symptoms (LUTS)

In a telephone survey of 150 community-living incontinent men aged 20 years and over, about 70% had experienced a variety of other medical conditions, many of which may cause or aggravate UI [186]. About half of the men reported symptoms of bladder outlet obstruction, and almost a third had undergone prostatectomy.

In a study of 805 noninstitutionalized elderly men, Diokno and coworkers found that a variety of problems, conditions and symptoms are more prevalent among those with UI than among those without. UI was reported by only 15% of men without voiding symptoms, frequency or urgency and by 34% of those with symptoms [74]. When outlet obstruction is established, this may lead to increasing post-void residual urine which may lead to chronic retention and manifest as overflow incontinence. Established outlet obstruction may cause detrusor instability and manifest as urgency, frequency, and urge incontinence.

Recent studies reported that urinary tract infections and cystitis are strongly associated with male UI [69, 111]. Men reporting cystitis were at increased risk for UI with an odds ratio of 3.65 [111].

c) Functional and cognitive impairment

Mobility problems such as use of a wheelchair or aids to walking, as well as diagnosed arthritis or rheumatism or having a fall the last year, were significantly greater among incontinent than continent men [69, 175]. A recent report noted that UI are 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 [111]. 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 normal ambulatory function [77]. In general, most studies find similarities between men and women (see above) for functional and cognitive impairment as risk factors for UI.

d) Neurological disorders

Many specific neurological diseases may lead to UI [187]. Detrusor hyper-reflexia is seen commonly in mengingo-myelocele patients and in spinal injuries, Parkinson's disease and multiple sclerosis. Areflexic bladder dysfunction due to a cauda equina lesion or diabetes might cause overflow or a paralysed pelvic floor and h[111]stress incontinence. Men who had suffered a stroke were at increased risk for incontinence with an odds ratio of 7.12. Similar history of stroke did not seem to significantly increase the risk among women [111].

e) Prostatectomy

A well known iatrogenic cause of male incontinence is prostatectomy. The reported incidence of stress incontinence following TURP is about 1%. Figures from radical prostatectomy technique are much higher. As the past decade has witnessed an increased number of radical prostatectomies, and incontinence is one of the main complications of the procedure, we decided to do an indepth analysis of the postprostatectomy patient population. In addition to epidemiological studies, we included clinical trials if the report had relevant data on incontinence. Minimum follow-up and time of assessment for continence should be at 12 months from the time of surgery.

We have found overall prevalences of post-prostatectomy incontinence from 5 to well over 60%. Table 13 shows examples of recent studies on post-prostatectomy UI. The very wide range of prevalence rates may be explained by many factors, including differences in study characteristics, population characteristics, study site, and the definition used.

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% [195 - 197].

Incontinence rates post-prostatectomy are observed to progressively decline with time and plateau 1 to 2 years postop [189, 191, 198]. Thus, it is important to emphasize that studies presenting incontinence rates should have a minimum follow-up of one year to establish true and reliable rates.

Modifications in the technique of radical prostatectomy have been developed primarily to minimize complications. Numerous studies demonstrate the impact of such modifications on postoperative incontinence rates. The variations associated with lower incontinence rates include the perineal approach [194], [199] and preservation of neurovascular bundle [200]. Bladder neck preservation affords earlier return to continence compared

Table 13 : Examples of prevalence of UI after radical prostatectomy

First author	Year	Ref	N	Prevalence (%)
Poon	2000	[188]	140	5.0
Walsh	2000	[189]	59	7.0
Catalona	1999	[190]	1 325	8.0
Goluboff	1998	[191]	480	8.2
Mettlin	1997	[192]	1 304 1 076	19.7 20.2
Benoit	2000	[193]	25 651	21.7
Bishoff	1998	[194]	907	44.7

to bladder neck resection although continence rates at one year post-op do not significantly differ between the two techniques [188].

Older age at time of surgery has been found to be a significant factor associated with a higher prevalence of post-prostatectomy incontinence [190, 191, 199, 200]. Catalona's work has shown that the risk for incontinence doubles for every 10 years of age beginning at age 40 years. Horie [198] further noted that rather than absolutely affecting final continence prevalence, age determined the rate at which continence would be achieved so that elderly men would take a longer time to achieve continence. Two studies however found no relation between age and incontinence prevalence [196, 201].

Other factors have been found to be associated with a higher prevalence of post-prostatectomy, although not consistently. These include : prior TURP, preoperative lower urinary tract symptoms, weight (obesity) and advanced clinical stage [200].

f) Other factors

As for women (see above) several other factors may be involved.

Summary points:

- The epidemiology of UI in men has not been investigated to the same extent as for females.
- 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.
- 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.
- Literature on incidence of male UI is still very scarce.
- Clear risk factors are more seldom scientifically documented, but several medical correlates have been reported. Risk factors reviewed in this text include age, lower urinary tract symptoms (LUTS), functional and cognitive impairment, neurological disorders, and prostatectomy.
- UI after radical prostatectomy is frequent, and should attain more attention.
- More world wide data are needed.

VI. WHY DO PREVALENCE ESTIMATES DIFFER?

1. 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 epidemiologic research. Herzog and Fultz [202], 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 [52, 58, 203, 204]. It is clear that there are large methodological challenges to rigorous research in this field. In general, quality of recent large studies has undoubtly improved, but the scientific community must continous deal with methodological challenges in order to achieve progress.

2. 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 ICS definition of UI – a condition in which involuntary loss of urine is a social or hygienic problem and is objectively demonstrable – includes objective demonstration of urine loss as one critical component. This aspect limits the ICS definition for community-based epidemiologic 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, the social or hygienic aspect of the ICS definition might be problematic in epidemiologic studies because it adds a subjective aspect to an objectively defined condition and therefore confounds the investigation of prevalence, incidence, and risk factors. By the same token, the subjective definition of a problem or "bother" might be relevant in the investigation of care seeking for UI and the prediction of who is likely to become a patient for this condition.

Studies use different severity levels for defining UI. Whereas some classify any involuntary loss at all as UI, others require at least monthly, weekly og even daily loss for UI classification. Moreover, different time frames for the occurence of involuntary urine loss have been used in existing studies. Loss during the previous week, month, 6 of 12 months have been used; sometimes the time period is left unspecified. 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 [203]. 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. Diokno et al. [131] invited both continent and incontinent respondents from a community survey for extensive clinical investigations and found 83% agreement between self-reports of UI and the clinical assessment. Two Swedish studies have reported that 5% and 6% of self-reported UI could not be verified in the clinic [75, 127]. Another study [98] revealed less than satisfactory predictive validity of self-reported types of UI compared with urodynamic investigations.

Holtedahl [76] 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 full ICS definition for 19%. Another study found prevalences of 69% and 30% for any UI and ICS defined UI, respectively [64] The results indicate that the ICS definition is rather restrictive and yields prevalence estimates that are lower than many other definitions used in epidemiologic studies.

3. SAMPLE AND DATA COLLECTION METHODOLOGY

Some surveys have selected their samples from lists of patients in general practice or in health organizations [88, 92, 101, 136, 141]. These surveys usually get good response rates, but unless such lists include all persons in the population, they are biased sampling frames. Some population based studies have included institutionalized patients [75, 127] some have excluded them [59, 65, 74] while other authors have not provided any information on this issue. The differences will obviously account for some of the variation in prevalence estimates observed among older women, as specificly shown in one study [77].

Low response rates may further bias prevalence estimates [203]. 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 how these factors may affect the comparision between incontinent and continent women.

Data on UI are often collected by post, while some authors have conducted personal interviews or telephone interviews. Personal interviews allow to explore issues in greater detail and they achieve generally higher response rates than postal questionnaires. There is also the possibility that responses elicited by interview are more susceptible to social desirability bias than those elicited by post.

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.
- Amajor problem is the use of different definitions of incontinence. The ICS definition might be problematic in epidemiological studies because it adds a subjective aspect to an objectively verifiable definition.
- 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.

VII. HELP SEEKING BEHAVIOUR

A majority of people with UI have not sought help [59, 60, 65, 126, 182, 205 - 209]. 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 [210]. Increasing age, increasing severity, increasing duration, and urge/mixed type of UI were shown to be associated with consulting a doctor [211].

The major method of actively managing UI among community residents is the use of absorbent products [182, 205, 211 - 213]. Only a small proportion of incontinent community-residing women have used surgery, medication, or exercise regimens [205, 206]. The only factor that appears reliably related to whether any treatment or management is sought is the severity of the condition [205, 214] (for review see [52]. It is also probable that many primary health care providers lack confidence in managing UI, and that this contribute to undertreatment in those seeking help [215].

It is obvious that millions of men and women suffer from their UI, and that for many of them good treatment can be offered. However, for many persons with very mild or occasional UI it is probably adequate not to seek help. 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 also shown that simple information and advice was adequate "treatment" for 23% of the women seeking an open access incontinence clinic [216]. A recent Swedish study found that among 136 women with UI, 36% wanted clinical evaluation, and only 24% subsequently started treatment [96].

It is important to realize that many incontinent persons have never talked to a health care professional about the condition. Both epidemiological and qualitative research in this field should be encouraged in order to understand cultural, religious, and other factors for help seeking behaviour world wide [209, 214, 217].

Summary points:

- A majority of people with UI have not sought help.
- Only a small proportion of incontinent community-residing people have used surgery, medication, or exercise regimens.
- Increasing severity, increasing duration, and urge/mixed type of UI are related to consulting a health care provider.
- Health care personnell should be encouraged to approach persons at risk for UI. People with UI should be assessed so services and treatment can be offered and targeted. The patient's view of management, even denial, should be respected.

VIII. EPIDEMIOLOGY AND CLINICAL WORK: FROM RESPONDENT TO PATIENT

In this review we have emphasized 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 with 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 a serious bias into research and hampers our ability to generalize hospital based research back to general practice 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 [218]. 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 emphasize use of tests or equipment that are not appropriate or relevant for general practice and primary health care, thus leading to overutilization 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.

One study provides substantial empirical evidence to support the existence of selection bias for UI [218]. 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 we have given examples of how the prevalence estimates change dramatically when botherness and severity are considered. In addition, there is also selection bias through the health care system. Taken together, this emphasizes 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 medicalization, 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.

Summary points:

- The spectrum of severity of UI 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 referrral process may introduce bias into research and hamper the ability to generalize 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.

IX. RECOMMENDATIONS FOR FURTHER RESEARCH

It is recommended to perform more sustained research on measurement of UI, 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 as regarding 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 informations about the attributable risk of the factors in the society.

Some potential risk and protective factors deserve more attention. For example, the role of childbirth in the development of UI must be studied in a fashion that links population-based methods to clinical assessment of the birth trauma and follows women over many years. Such a design is necessary because the effect of 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 and episiotomy, etc. Other potential risk factors include overweight, various forms of stress of the pelvic floor, and smoking. Physical exercise may play protective roles. There should be more emphasis on the associations between UI and specific diseases like stroke, diabetes 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.

The committee will again emphasize that there is merit to reconsider the definition of UI, and to move towards a standardization of measurement instruments of UI in community surveys that can be used worldwide. Developing a new definition 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. Clearly, the core of the definition is "any involuntary loss of urine". However, elaboration of this core definition may be required depending on the purpose of its use.

Table 14 : 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

In addition, it is recommended to include validated measures of bother/quality of life and urinary symptoms other than UI.

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 as age, parity, and weight.

For the purpose of epidemiological research, it is recommended to include the following elements in the definition:

- the individual’s statement of any involuntary urine loss

- the frequency of urine loss

- the quantity of urine loss

- the duration of the condition

Consistent with standard epidemiological practices, it is not recommended to include quality of life or bother in the definition of UI for epidemiological purposes. One reason to not include the patients’ perceptions of the condition is that it confounds or distorts our estimates of prevalence and incidence of UI. A second reason is that it could seriously limit the detection of risk factors. Quality of life or bother may be important elements of a definition of UI for clinical purposes

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 characterize 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 14), including elements of screening question, frequency measure, quantity of urine loss, duration, type, and severity. In addition, it is recommended to include validated measures of bother/quality of life and urinary symptoms other than UI. We here also refer to the chapter from the committee on symptom and quality of life assessment.

ACKNOWLEDGEMENTS

We wish to acknowledge the valuable contributions of dr. Hogne Sandvik and dr. Catherine McGrother who acted as consultants for the committee. We thank several other colleagues for help, comments and suggestions.

REFERENCES

1. Hunskaar, S., Arnold, E.P., Burgio, K., Diokno, A.C., Herzog, A.R. And Mallett, V.T.: Epidemiology and natural history of urinary incontinence. In: Abrams P, Khoury S, Wein A.: Incontinence, 197-226. Plymouth: Scientific International, 1999.
2. Nørgaard, J.P., Van Gool, J.D., Hjälmås, K., Djurhuus, J.C. And Hellström, A.-L.: Standardization and definitions in lower urinary tract dysfunction in children. Br. J. Urol. Suppl., 81: 1, 1998.
3. Hjälmas, K.: Urinary incontinence in children: suggestions for definitions and terminology. Scand. J. Urol. Nephrol. Suppl., 141: 1, 1992.
4. Holmdahl, G., Hanson, E., Hanson, M., Hellström, A.-L., Hjälmås, K. And Sillen, U.: Four-hour voiding observation in healthy infants. J. Urol., 156: 1809, 1996.
5. Meadow, S.R.: Day wetting. Pediatr. Nephrol., 4: 178, 1990.
6. Krantz, I., Jylkäs, E., Ahlberg, B.M. And Wedel, H.: On the epidemiology of nocturnal enuresis: A critical review of methods used in descriptive epidemiological studies on nocturnal enuresis. Scand. J. Urol. Nephrol. Suppl., 163: 75, 1994.
7. Mcgee, R., Mackinson, T., Williams, S., Simpson, A. And Silva, P.A.: A longitudinal study of enuresis from 5-9 years. Aust. Paediatr. J., 20: 39, 1984.
8. Swithinbank, L.V., Carr, J.C. And Abrams, P.H.: Longitudinal

study of urinary symptoms in children. Scand. J. Urol. Nephrol. Suppl., 163: 67, 1994.

9. Fergusson, D.M., Horwood, L.J. And Shannon, F.T.: Factors related to the age of attainment of nocturnal bladder control: an 8-year longitudinal study. Pediatrics, 78: 884, 1986.

10. Hellström, A.-L., Hanson, E., Hansson, S., Hjalmas, K. And Jodal, V.: Micturition habits and incontinence in 7 year old Swedish school entrants. Eur. J. Pediatr., 149: 434, 1990.

11. Jarvelin, M.R., Vikevainen-Tervonen, L., Moilanen, J. And Huttenen, N.P.: Enuresis in seven year old children. Acta Paediatr., 77: 148, 1988.

12. Swithinbank, L.V., Brookes, S.T., Shepherd, A.M. And Abrams, P.: The natural history of urinary symptoms during adolescence. Br. J. Urol. Suppl., 81: 90, 1998.

13. Bower, W.F., Moore, K.H., Shephard, R. And Adams, R.: The epidemiology of childhood enuresis in Australia. Br. J. Urol., 78: 602, 1996.

14. Watanabe, H. And Kawauchi, A.: Nocturnal enuresis: Social aspects and treatment perspectives in Japan. Scand J. Urol. Nephrol. Suppl., 163: 29, 1994.

15. Lottmann, H.: Enuresis treatment in France. Scand. J. Urol. Nephrol. Suppl., 33: 66, 1999.

16. Neveus, T., Hetta, J., Cnattingius, S., Tuvemo, T., Läckgren, G., Olsson, U. And Stenberg, A.: Depth of sleep and sleep habits among enuretic and incontinent children. Acta Paediatr., 88: 748, 1999.

17. Chiozza, M.L., Bernardinelli, L., Caione, P., Delgado, R., Ferrara, P., Giorgi, P.L. Et Al.: An Italian epidemiological multicentre study of nocturnal enuresis. Br. J. Urol. Suppl., 81: 86, 1998.

18. Spee-Van Der Wekke, J., Hirasing, R.A., Meulmeester, J.F. And Radder, J.J.: Childhood nocturnal enuresis in The Netherlands. Urology, 51: 1022, 1998.

19. Serel, T.A., Akhan, G., Koyuncouglu, H.R., Öztürk, A., Dogruer, K., Ünal, S. Et Al.: Epidemiology of enuresis in Turkish children. Scand. J. Urol. Nephrol., 31: 537, 1997.

20. Gümüs, B., Vurgun, N., Lekili, M., Iscan, A., Müezzinoglu, T. And Büyüksu, C.: Prevalence of nocturnal enuresis and accompanying factors in children aged 7-11 years in Turkey. Acta Paediatr., 88: 1369, 1999.

21. Kalo, B.B. And Bella, H.: Enuresis: prevalence and associated factors among primary school children in Saudi Arabia. Acta Paediatr., 85: 1217, 1996.

22. Lee, S.D., Sohn, D.W., Lee, J.Z., Park, N.C. And Chung, M.K.: An epidemiological study of enuresis in Korean children. B.J.U. Int., 85: 869, 2000.

23. Yeung, C.K.: Nocturnal enuresis in Hong Kong: different Chinese phenotypes. Scand. J. Urol. Nephrol. Suppl., 31: 17, 1997.

24. Eiberg, H.: Nocturnal enuresis is linked to a specific gene. Scand. J. Urol. Nephrol. Suppl., 173: 15, 1995.

25. Arnell, H., Hjälmås, K., Jägervall, M., Lackgren, G., Stenberg, A., Bengtsson, B. Et Al.: The genetics of primary nocturnal enuresisi: inheritance and suggestion of a second major gene on chromosome 12q. J. Med. Genet., 34: 360, 1997.

26. Von Gontard, A., Eiberg, H., Hollman, E., Rittig, S. And Lehmkuhl, G.: Molecular genetics of nocturnal enuresis: clinical and genetic heterogeneity. Acta Paediatr. 87: 571, 1997.

27. Feehan, M., Mcgee, R., Stanton, W. And Silva, P.A.: A 6 year follow-up of childhood enuresis: Prevalence in adolescence and consequences for mental health. J. Paediatr. Child Health, 26: 75, 1990.

28. Schulpen, T.W.J.: The burden of nocturnal enuresis. Acta Paediatr., 86: 923, 1997.

29. Fergusson, D.M., And Horwood, L.J.: Nocturnal enuresis and behavioural problems in adolescence: a 15-yearlongitudinal study. Pediatrics, 94: 662, 1994.

30. Moilanen, I., Tirkkonen, T., Järvelin, M.R., Linna, S.L., Almqvist, F., Piha, J. Et Al.: A follow-up of enuresis from childhood to adolescence. Br. J. Urol. Suppl., 81: 94, 1998.

31. Rittig, S., Knudsen, U.B., Norgaard, J.P., Pedersen, E.B. And Djurhuus, J.C.: Abnormal diurnal rhythm of plasma vasopressin and urinary output in patients with enuresis. Am. J. Physiol., 25: 664, 1989.

32. Wolfish, N.M., Pivik, R.T., And Busby, K.A.: Elevated sleep arousal thresholds in enuretic boys: clinical implications. Acta Paediatr. 86: 381, 1997.

33. Kawauchi, A., Imada, N., Tanaka, Y., Minami, M., Watanabe, H. And Shirakawa, S.: Changes in the structure of sleep spindles and delta waves on electroencephalography in patients with nocturnal enuresis. Br. J. Urol. Suppl., 81: 72, 1998.

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

35. Foldspang, A. And Mommsen, S.: Adult female urinary incontinence and childhood bedwetting. J. Urol., 152: 85, 1994.

36. Moore, K.H., Richmond, D.H. And Parys, B.T.: Sex distribution of adult idiopathic detrusor instabilty in relation to childhood bedwetting. Br. J. Urol., 68: 479, 1991.

37. Baruzzi, A., Riva, R., Cirignotta, F., Zucconi, M,. Capelli, M. And Lugaresi, E.: Atrial natriuretic peptide and catecholamines in obstructive sleep apnoea. Sleep, 14: 83, 1991.

38. Weider, D.J., Sateia, M.J., And West, R.P.: Nocturnal enuresis in children with upper airway obstruction. Otolaryngol. Head Neck Surg., 105: 427, 1991.

39. Loening-Baucke, V.: Urinary incontinence and urinary tract infection and their resolution with treatment of chronic constipation of childhood. Pediatrics, 100: 228, 1997.

40. Lunsing, R.J., Hadders-Algra, M., Touwen, B.C. And Huisjes, H.D.: Nocturnal enuresis and minor neurological dysfunction at 12 years: a follow-up study. Dev. Med. Child Neurol., 33: 439, 1991.

41. Robson, W.L.M., Jackson, H.P., Blackhurst, D. And Leung, A.K.: Enuresis in children with attention deficit hyperactivity disorder. South. Med. J., 90: 503, 1997.

42. Forbes, F.C.: Children with enuresis. Nowadays, a strong suspicion of sexiual abuse would prompt full investigation. BMJ., 316: 777, 1998.

43. Forsythe, W.I. And Redmond, A.: Enuresis and spontaneous cure rate. Arch. Dis. Child., 49: 259, 1974.

44. Hirasing, R.A., Van Leerdam, F.J.M., Bolk-Bennink, L. And Janknegt, R.A.: Enuresis nocturna in adults. Scand. J. Urol. Nephrol., 31: 533, 1997.

45. Hellström, A.-L., Hanson, E., Hansson, S., Hjälmås, K. And Jodal, U.: Micturition habits and incontinence at age 17 – reinvestigation of a cohort studied at age 7. Br. J. Urol., 76: 231, 1995.

46. Hjalmas, K., Hoebeke, P.B. And De Paepe, H.: Lower urinary tract dysfunction and urodynamics in children. Eur. Urol., 38: 655, 2000.

47. Bloom, D.A., Seeley, W.M., Ritchey, M.L. And Mcguire, E.J.: Toilet habits and continence in children: an opportunity sampling in search of normal parameters. J. Urol., 149: 1087, 1993.

48. Järvelin, M.R., Moilanen, I., Kangas, P., Moring, K., Vikevainen-Tervonen, L., Huttunen, N.P. Et Al.: Aetiological and precipitating factors for childhood enuresis. Acta Paediatr., 80: 361, 1991.

49. Hansson, S., Hjalmas, K., Jodal, U. And Sixt, R.: Lower urinary tract dysfunction in girls with asymptomatic or covert bacteriuria. J. Urol., 143: 333, 1990.

50. Nguyen, H.T. And Peters, C.A.: The long-term complications of posterior urethral valves. B.J.U. Int. Suppl., 83: 23, 1999.

51. De Jonge, G.A.: Epidemiology of enuresis: a survey of the literature. In Kolvin I, MacKeith RC, Meadow SR (eds),: Bladder Control and Enuresis, Spastics International Medical Publications, 39-46. London: William Heinemann Medical Books Ltd., 1973

52. Fultz, N.H. And Herzog, R.: Epidemiology of urinary symptoms in the geriatric population. Geriatr. Urol., 23: 1, 1996.

53. Hampel, C., Wienhold, D., Benken, N., Eggersmann, C. And Thüroff, J.W.: Prevalence and natural history of female incontinence. Eur. Urol. Suppl., 32: 3, 1997.

54. Hampel, C., Wienhold, D., Benken, N., Eggersmann, C. And Thüroff, J,W.: Definition of overactive bladder and epidemiology of urinary incontinence. Urology, 50: 4, 1997.

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

56. Cheater, F.M., And Castleden, C.M.: Epidemiology and classification of urinary incontinence. Clin. Obstet. Gynaecol., 14: 183. 2000.

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

58. Thom, D.: Variation in estimates of urinary incontinence prevalence in community: Effects of differences in definition, population characteristics, and study type. J. Am. Geriatr. Soc., 46: 473, 1998

59. Yarnell, J.W., Voyle, G.J., Richards, C.J. And Stephenson, T.P.: The prevalence and severity of urinary incontinence in women. J. Epidemiol. Community Health, 35: 71, 1981.

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

61. Sommer, P., Bauer, T., Nielsen, K.K., Kristensen, E.S., Hermann, G.G., Steven, K. Et Al.: Voiding patterns and prevalence of incontinence in women. A questionnaire survey. Br. J. Urol., 66: 12, 1990.

62. Brocklehurst, J.C.: Urinary incontinence in the community-Analysis of a MORI poll. BMJ., 306: 832, 1993.

63. Sandvik, H., Hunskaar, S., Seim, A., Hermstad, R., Vanvik, A. And Bratt, H.: Validation of a severity index in female urinary incontinence and its implementation in an epidemiological survey. J. Epidemiol. Community Health, 47: 497, 1993.

64. Swithinbank, L.V., Donovan, J.L., Du Heaume, J.C., Rogers, C.A., James, M.C., Yang, Q. Et Al.: Urinary symptoms and incontinence in women: Relationships between occurrence, age, and perceived impact. Br. J. Gen. Pract., 49: 897, 1999

65. Hannestad, Y.S., Rortveit, G., Sandvik, H. And Hunskaar, S.: A community-based epidemiological survey of female urinary incontinence: The Norwegian EPINCONT study. J. Clin. Epidemiol., 53: 1150, 2000.

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

67. Bortolotti, A., Bernardini, B., Colli, E., Dibenedetto, P., Nacci, G.G., Landoni, M. Et Al.: Prevelance and risk factors for urinary incontinence in Italy. Eur. Urol., 37: 30, 2000.

68. Moller, L.A., Lose, G. And Jorgensen, T.: The prevalence and bothersomeness of lower urinary tract symptoms in women 40-60 years of age. Acta Obstet. Gynecol. Scand., 79: 298, 2000.

69. 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.

70. Gavira Iglesias, F.J., Caridad Y Ocerin, J.M., 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. Biol. Sci., 55: M207, 2000.

71. Maggi, S., Minicuci, N., Langlois, J., Pavan, M., Enzi, G. And Crepalid, G.: Prevalence rate of urinary incontinence in community-dwelling elderly individuals: The Veneto Study. J. Gerontol. Med. Sci., 56A: M14, 2001.

72. Nakanishi, N., Atara, K., Naramura, H., Takashima, Y. And Fukada, H.: Urinary and fecal incontinence in a community-residing older population in Japan. J. Am. Geriatr. Soc., 45: 215, 1997.

73. Tseng, I.J., Chen, Y.T., Chen, M.T., Kou, H.Y. And Tseng, S.F.: Prevalence of urinary incontinence and intention to seek treatment in the elderly. J. Formos. Med. Assoc., 99: 753, 2000.

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

75. Molander, U., Milsom, I., Ekelund, P. And Mellstrom, D.: An epidemiological study of urinary incontinence and related urogenital symptoms in elderly women. Maturitas, 12: 51, 1990.

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

77. Hunskaar, S., Østbye, T. And 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.

78. Roberts, R.O., Jacobsen, S.J., Rhodes, T., Reilly, W.T., Girman, C.J., Talley, N.J. Et Al.: Urinary incontinence in a community-based cohort: Prevalence and healthcare-seeking. J. Am. Geriatr. Soc., 46: 467, 1998.

79. Chiarelli, P., Brown, W. And Mcelduff, P.: Leaking urine: prevalence and associated factors in Australian women. Neurourol. Urodyn.,18:567, 1999.

80. Aggazzotti, G., Pesce, F., Grassi, D., Fantuzzi, G., Righi, E., De Vita, 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.

81. Brandeis, G.H., Baumann, M.M., Hossain, M., Morris, J.N. And Resnick, N.M.: 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.

82. Sgadari, A., Topinkova, E., Bjornson, J. And Bernabei, R.: Urinary incontinence in nursing home residents: A cross-national comparison. Age Ageing, 26: 49, 1997.

83. Toba, K., Ouchi, H., Iimura, O., Sasaki, H., Nakamura, Y., Takasaki, M. Et Al.: Urinary incontinence in elderly inpatients in Japan: A comparison between general and geriatric hospitals. Aging, 8: 47, 1996.

84. Borrie, M. And Davidson, H.: Incontinence in institutions: costs and contributing factors. Can. Med. Assoc. J., 147: 322, 1992.

85. Ouslander, J.G., Palmer, M.H., Rovner, B.W. And German, P.S.: Urinary incontinence in nursing homes: incidence, remission and associated factors. J. Am. Geriatr. Soc., 41: 1083, 1993.

86. Fonda, D. And Victorian Geriatricians Peer Review Group.: Improving management of urinary incontinence in geriatric centres and nursing homes. Aust. Clin. Rev., 10: 66, 1990.

87. Peet, S.C. And Castleden, C.M.: The prevalence of urinary and faecal incontinence in hospitals and residential and nursing homes for older people. BMJ., 311: 1063, 1995.

88. Thom, D.H., Haan, M.N. And Van Den Eeden, S.: Medically

recognized urinary incontienence and risks of hospitalization, nursing home admission and mortality. Age Aging, 26: 367, 1997.

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

90. H ording, U., Pedersen, K.H., Sidenius, K. And Hedegaard, L.: Urinary incontinence in 45-year-old women. An epidemiological survey. Scand. J. Urol. Nephrol., 20: 183, 1986.

91. Elving, L.B., Foldspang, A., Lam, G.W. And Mommsen, S.: Descriptive epidemiology of urinary incontinence in 3 100 women age 30-59. Scand. J. Urol. Nephrol. Suppl., 125: 37, 1989.

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

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

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

95. Sandvik, H., Hunskaar, S., Vanvik, A., Bratt, H., Seim, A. And Hermstad, R.: Diagnostic classification of female urinary incontinence: an epidemiologic survey corrected for validity. J. Clin. Epidemiol., 48: 339, 1995.

96. Samuelsson, E., Victor, A. And 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.

97. Stewart, W., Herzog, A.R., Wein, A., Abrams, P., Payne, C., Corey, R. Et Al.: Prevalence of overactive bladder in the US: results from the NOBLE program. 2nd International Consultation on Incontinence, abstract no 79, Paris 2001.

98. Kirschner-Hermanns, R., Scherr, P.A., Branch, L.G., Wetle, T. And Resnick, N.M.: Accuracy of survey questions for geriatric urinary incontinence. J. Urol., 159: 1903, 1998.

99. Sandvik, H., Seim, A., Vanvik, A. And Hunskaar, S.: A severity index for epidemiological surveys of female urinary incontinence: Comparison with 48-hour pad-weighing tests. Neurourol. Urodyn., 19: 137, 2000.

100. Hanley, J., Capewell, A. And Hagen, S.: Validity study of the severity index, a simple measure of urinary incontinence in women. BMJ., 322: 1096, 2001.

101. Vetter, N.J., Jones, D.A. And Victor, C.R.: Urinary incontinence in the elderly at home. Lancet, ii: 1275, 1981.

102. Herzog, A.R., Diokno, A.C., Brown, M.B., Normolle, D.P. And Brock, B.M.: Two-year incidence, remission, and change patterns of urinary incontinence in noninstitutional older adults. J. Gerontol., 45: M67, 1990.

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

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

105. Ju, C.C., Swan, L.K., Merriman, A., Choon, T.E. And Viegas, O.: Urinary incontinence among the elderly people of Singapore. Age Ageing, 20: 262, 1991.

106. Shershah, S. And Ansari, R.L.: The frequency of urinary incontinence in Pakistani women. J.P.M.A., 39: 16, 1989.

107. Lee, K.S., Chan, C.J., Merriman, A., Tan, E.C. And Osborn, V.: Clinical profile of elderly urinary incontinence in Singapore: a community based study. Ann. Acad. Med. Singapore, 20: 736, 1991.

108. Brieger, G.M., Mongelli, M., Hin, L.Y. And Chung, T.K.H.: The epidemiology of urinary dysfunction in Chinese women. Int. Urogynecol. J. Pelvic Floor Dysfunct., 8: 191, 1997.

109. Kato, K. And Kondo, A.: Prevalence of urinary incontinence in working women. Jap. J. Urol., 77: 1501, 1986.

110. Fukui, J.: Urinary incontinence in women: results of questionnaires. Jap. J. Urol., 77:, 707, 1986.

111. 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

112. Miles, T.P., Palmer, R.F., Espino, D.V., Mouton, C.P., Lichtenstein, M.J. And Markides, K.S.: New-onset incontinence and markers of frailty: Data from the Hispanic Established Populations for Epidemiologic Studies of the Elderly. Journal of Gerontology: Medical Sciences, 56A: M19, 2001.

113. Heyns, O.S.: Bantu Gynaecology. Johannesburg: Witwaterstrand University Press, 1956: 98.

114. Heyns, O.S.: Clin. Proc., 2: 311, 1943.

115. Skinner, D.P.: Stress Incontinence: A comparative racial study. Med. Proc., 9: 189, 1963.

116. Knobel, J.: Stress incontinence in the black female. S. Afr. J. Obstet. Gynaecol., 49: 430, 1975.

117. Cox, P.S.V. And Webster, D.: Genital prolapse amongst the Pokot. East Afr. Med. J., 52: 694, 1975.

118. Howard, D., Davies, P.S. Delancey, J.O. And Small, Y.: Differences in perineal lacerations in black and white primiparas. Obstet. Gynecol., 96: 622, 2000

119. White, L.R., Blazer, D.G. And Fillenbaum, G.: Chapter 5: Related health problems. In: Cornoni-Huntley, J., Blazer, D. G., Lafferty, M. E., Everett, D. F., Brock, D. B. and Farmer M. E., eds. Established populations for epidemiologic studies of the elderly, Volume II. National Institute on Aging, U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, NIH Publication, 1986; No. 90-495:70-6.

120. Bump, R.C.: Racial comparisons and contrasts in urinary incontinence and pelvic organ prolapse. Obstet. Gynecol., 81: 421, 1993.

121. Peacock, L.M., Wiskind, A.K. And Wall, L.L.: Clinical features of urinary incontinence and urogenital prolapse in a black inner city population. Am. J. Obstet. Gynecol., 171: 1464, 1994.

122. Burgio, K.L., Locher, J.L., Zyczynski, H., Hardin, J.M. And Singh, K.: Urinary incontinence during pregnancy in a racially mixed sample: characteristics and predisposing factors. Int. Urogynecol. J. Pelvic Floor Dysfunt., 7: 69, 1996.

123. Brown, J.S., Grady, D., Ouslander, J.G., Herzog, A.R., Varner, R.E. And Posner, S.F.: Prevalence of urinary incontinence and associated risk factors in postmenopausal women. Obstet. Gynecol., 94: 66, 1999.

124. Brown, J.S., Jackson, R.A., Kanaya, A.M., Vittinghoff, E., Resnik, H.E., Kritchevsky, S. Et At.: Urinary incontinence in elderly women: The health, aging and body composition study. 2nd International Consultation on Incontinence, Abstract nr 24, Paris 2001.

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

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

127. Milsom, I., Ekelund, P., Molander, U., Arvidsson, L. And Areskoug, B.: The influence of age, parity, oral contraception, hysterectomy and menopause on the prevalence of urinary incontinence in women. J. Urol., 149: 1459, 1993.

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

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

130. Dolan, L.M., Casson, K., Mcdonald, P. And Ashe R.G.: Urinary incontinence in Northern Ireland: a prevalence study. B.J.U. Int. 83: 760, 1998.

131. Diokno, A.C., Brown, M.B., Brock, B.M., Herzog, A.R. And Normolle, D.P.: Clinical and cystometric characteristics of continent and incontinent noninstitutionalized elderly. J. Urol., 140: 567, 1988.

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

133. Mellier, G. And Delille, M.A.: Urinary disorders during pregnancy and post-partum. Rev. Fr. Gynecol. Obstet., 85: 525, 1990.

134. Viktrup, L., Lose, G., Rolff, M. And Farfoed, K.: The symptom of stress incontinence caused by pregnancy or delivery in primiparas. Obstet. Gynecol., 79: 945, 1992.

135. Iosif, C.S. And Ingemarsson, I.: Prevalence of stress incontinence among women delivered by elective cesarian section. Int. J. Gynaecol. Obstet., 20: 87, 1982.

136. Jolleys, J.V.: Reported prevalence of urinary incontinence in women in a general practice. BMJ., 296: 1300, 1988.

137. Yarnell, J.W., Voyle, G.J., Richards, C.J. And Stephenson, T.P.: Factors associated with urinary incontinence in women. J. Epidemiol. Community Health, 36: 58, 1982.

138. Crist, T., Shingleton, H.M., Koch, F. And Koch, G.G.: Stress incontinence and the nulliparous patient. Obstet. Gynecol., 40: 13, 1972.

139. Foldspang, A., Mommsen, S., Lam, G.W. And Elving, L.: Parity as a correlate of adult female urinary incontinence prevalence. J. Epidemiol. Community Health, 46: 595, 1992.

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

141. Thomas, T.M., Plymat, K.R., Blannin, J. And Meade, T.W.: Prevalence of urinary incontinence. BMJ., 281: 1243, 1980.

142. Faundes, A., Guarisi, T. And 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.

143. Kuh, D., Cardozo, L. And 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.

144. Rortveit, G., Daltveit, A.K. And Hunskaar, S.: Age and type dependent effects of parity on urinary incontinence. The Norwegian EPINCONT study. Obstet. Gynecol., 2001 in press

145. Samuelsson, E., Victor, A. And Svardsudd, K. Determinants of urinary incontinence in a population of young and middle-aged women. Acta Obstet. Gynecol. Scand., 79: 208, 2000.

146. Smith, A.R.B., Hosker, G.L. And Warrell, D.W.: The role of pudendal nerve damage in the aetiology of genuine stress incontinence in women. Br. J. Obstet. Gynaecol., 96: 29, 1989.

147. Snooks, S.J., Swash, M., Henry, M.M. And Setchel, M.: Risk factors in childbirth causing damage to the pelvic floor innervation. Int. J. Colorectal Dis., 1: 20, 1986.

148. Persson, J., Wolner-Hanssen, P. And Rydhstroem, H.: Obstetric risk factors for stress urinary incontinence: a population-based study. Obstet. Gynecol., 96: 440, 2000.

149. Hojberg, K.E., Salvig, J.D., Winslow, N.A., Lose, G. And Secher, N.J.: Urinary incontinence: prevalence and risk factors at 16 weeks of gestation. Br. J. Obstet. Gynaecol., 106: 842, 1999.

150. Chaliha, C., Kalia, V., Stanton, S.L., Monga, A. And Sultan, A.H.: Antenatal prediction of postpartum urinary and fecal incontinence. Obstet. Gynecol. 94: 689, 1999.

151. Moller, L.A., Lose, G. And Jorgensen, T.: Risk factors for lower urinary tract symptoms in women 40 to 60 years of age. Obstet. Gynecol., 96: 446, 2000.

152. Danso, K.A., Martey, J.O., Wall, L.L. And Elkins, T.E.: The epidemiology of genitourinary fistulae in Kumasi, Ghana, 1977-1992. Int. Urogynecol. J. Pelvic Floor Dysfunct., 7: 117, 1996.

153. Grady, D., Brown, J.S., Vittinghoff, E., Applegate, W., Varner, E. And Snyder, T.: Postmenopausal hormones and incontinence: The Heart and Estrogen /Progestin Replacement Study. Obstet. Gynecol., 97: 116, 2001

154. Brown, J.S., Sawaya, G., Thom, D.H. And Grady, D.: Hysterectomy and urinary incontinence; a systematic review. Lancet, 356: 535, 2000.

155. Brown, J.S., Seeley, D.G., Fong, J., Black, D.M., Ensrud, K.E. And Grady, D.: Urinary incontinence in older women: Who is at risk? Study of osteoporotic fractures research group. Obstet. Gynecol., 87: 715, 1996

156. Thom, D.H. And Brown, J.S.: Reproductive and hormonal risk factors for urinary incontinence in later life: a review of the clinical and epidemiologic literature. J. Am. Geriatr. Soc., 46: 1411, 1998.

157. Weber, A.M., Walters, M.D., Schover, L.R., Church, J.M. And Piedmonte, M.R.: Functional outcomes and satisfaction after abdominal hysterectomy. Am. J. Obstet. Gynecol., 181: 530, 1999.

158. Thakar, R., Manyonda, I., Stanton, S.L., Clarkson, P. And Robinson, G.: Bladder, bowel and sexual function after hysterectomy for benign conditions. Br. J. Obstet. Gynaecol., 104: 983, 1997.

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

160. Fultz, N.H., Herzog, A.R., Raghunathan, T.E., Wallace, R.B. And Diokno, A.C.: Prevalence and severity of urinary incontinence in older African American and Caucasian women. J. Gerontol. Med. Sci., 54A: M299, 1999.

161. Dwyer, P.L., Lee, E.T.C. And Hay, D.M.: Obesity and urinary incontinence in women. Br. J. Obstet. Gynaecol., 95: 91, 1988.

162. Kolbl, H. And Riss, P.: Obesity and stress urinary incontinence: significance of indices of relative weight. Urol. Int., 43: 7, 1988.

163. Fischer, J.R. And Berg, P.H.: Urinary incontinence in United States Air Force female aircrew. Obstet. Gynecol. 94: 532, 1999.

164. Bump, R.C., Sugerman, H.J., Fantl, F.A. And Mcclish, D.K.: Obesity and lower urinary tract function in women: Effect of surgically induced weight loss. Am. J. Obstet. Gynecol., 166: 392, 1992.

165. Deitel, M., Stone, E., Kassam, H.A., Wilk, E.F. And Sutherland, D.J.A.: Gynecologic-obstetric changes after loss of massive excess weight following bariatric surgery. J. Am. Coll. Nutr., 7: 147, 1988.

166. Herzog, A.R., Fultz, N.H., Brock, B.M., Brown, M.B. And Diokno, A.C.: Urinary incontinence and psychological distress among older adults. Psychol. Aging, 3: 115, 1988.

167. Tinetti, M.E., Inouye, S.K., Gill, T.M. And Doucette, J.T.: Shared risk factors for falls, incontinence, and functional depen-

dence - unifying the approach to geriatric syndromes. JAMA., 273: 1348, 1995.

168. Ouslander, J.G., Morishita, L., Blaustein, J., Orzeck, S., Dunn, S. And Sayre, J.: Clinical, functional, and psychosocial characteristics of an incontinent nursing home population. J. Gerontol., 42: 631, 1987.

169. Ouslander, J.G., Uman, G.C., Urman, H.N. And Rubenstein, L.Z.: Incontinence among nursing home patients: clinical and functional correlates. J. Am. Geriatr. Soc., 35: 324, 1987.

170. Wetle, T., Scherr, P., Branch, L.G., Resnick, N.M., Harris, T., Evans, D. Et Al.: Difficulty with holding urine among older persons in a geographically defined community: Prevalence and correlates. J. Am. Geriatr. Soc., 43: 349, 1995.

171. Skelly, J. And Flint, A.J.: Urinary incontinence associated with dementia. J. Am. Geriatr. Soc., 43: 286, 1995.

172. Rohr, G., Kragstrup, J. And Christensen, K.: Geners indflydelse på udviklingen af urininkontinens, undersøgt blandt ældre kvindelige tvillinger. 11. Nordic Congress in General Practice, Abstract F11.143, Copenhagen 2000.

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

174. Molander, U., Arvidsson, L., Milsom, I. And Sandberg, T.: A longitudinal cohort study of elderly women with urinary tract infections. Maturitas, 34: 127, 2000.

175. Diokno, A., Brock, B., Herzog, A. And Bromberg, J.: Medical correlates of urinary incontinence in the elderly. Urology, 36: 129, 1990.

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

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

178. Nygaard, I., Nygaard, I.E., Thompson, F.L. Svengalis, S.L. And Albright, J.P.: Urinary incontinence in elite nulliparous athletes. Obstet. Gynecol., 84: 183, 1994.

179. Parkin, D.E., Davis, J.A. And Symonds, R.P.: Urodynamic findings following radiotherapy for cervical carcinoma. Br. J. Urol., 61: 213, 1988.

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

181. Feneley, R.C., Sheperd, A.M., Powell, P.H. And Blannin, J.: Urinary incontinence: prevalence and needs. Br. J. Urol., 51: 493, 1979.

182. Schulman, C., Claesm, H. And Mattijs, J.: Urinary incontinence in Belgium: a population-based epidemiological survey. Eur. Urol., 32: 315, 1997.

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

184. Yarnell, J.W.G. And St Leger, A.S.: The prevalence, severity and factors associated with urinary incontinence in a random sample of the elderly. Age Ageing, 8: 81, 1979.

185. Sladden, M.J., Hughes, A.M., Hirst, G.H. And Ward, J.E.: A community study of lower urinary tract symptoms in older men in Sydney, Australia. Aust. N.Z. J. Surg., 70: 322, 2000.

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

187. Resnick, N.M. And Yalla, S.V.: Detrusor hyperactivity with impaired contractile function: an unrecognised but common cause of incontinence in elderly patients. JAMA., 257: 3076, 1987.

188. Poon, M., Ruckle, H., Bamshad, B.R., Tsai, C., Webster, R. And Lui, P.: Radical retropubic prostatectomy: bladder neck preservation versus reconstruction. J. Urol.,163: 194, 2000.

189. Walsh, P.C., Marschke, P., Ricker, D. And Burnett, A.L.: Patient-reported urinary continence and sexual function after anatomic radical prostatectomy. Urology, 55: 58, 2000.

190. Catalona, W.J., Carvalhal, G.F., Mager, D.E. And Smith, D.S.: Potency, continence and complication rates in 1870 consecutive radical retropubic prostatectomies. J. Urol., 162: 433, 1999.

191. Goluboff, E.T., Saidi, J.A., Mazer, S., Bagiella, E., Heitjan, D.F., Benson, M.C. Et Al.: Urinary continence after radical prostatectomy: The Columbia experience. J. Urol., 159: 1276, 1998.

192. Mettlin, C.J., Murphy, G.P., Sylvester, J., Mckee, R.F., Morrow, M. And Winchester, D.P.: Results of hospital registry surveys by the American College of Surgeons : outcomes of prostate cancer treatment by radical prostatectomy. Cancer, 80: 1875, 1997.

193. Benoit, R.M., Naslund, M.J. And Cohen, J.K.: Complications after radical retropubic prostatectomy in the medicare population. Urology, 56: 116, 2000.

194. Bishoff, J.T., Motley, G., Optenberg, S.A., Stein, C.R., Moon, K.A., Browning, S.M. Et Al.: Incidence of fecal and urinary incontinence following radical perineal and retropubic prostatectomy in a national population J. Urol., 160: 454, 1998.

195. McCammon, K.A., Kolm, P., Main, B. And Schellhammer, P.F.: Comparative quality of life analysis after radical prostatectomy or external beam radiation for localized prostate cancer. Urology, 54: 509, 1999.

196. Donnellan, S.M., Duncan, H.J., MacGregor, R.J. And Russel, J.M.: Prospective assessment of incontinence after radical retropubic prostatectomy: objective and subjective analysis. Urology, 49: 225, 1997.

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

198. Horie, S., Tobisu, K.I, Fujimoto, H., Doi, N. And Kakizoe, T.: Urinary incontinence after non-nerve-sparing radical prostatectomy with neoadjuvant androgen deprivation. Urology, 53: 561, 1999.

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

200. 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.

201. Kao, T.C., Garner, D., Foley, J., Seay, T., Friedrichs, P., Thrasher, J.B. Et Al.: Multicenter patient self-reporting questionnaire on impotence, incontinence and stricture after radical prostatectomy. J. Urol., 163: 858, 2000.

202. Herzog, A.R. And Fultz, N.H.: Epidemiology of urinary incontinence: prevalence, incidence, and correlates in community populations. Urology Suppl., 36: 2, 1990.

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

204. Barry, M.J., Fowler, F.J., O'Leary. M.P., Bruskewitz, R.C., Holtgrewe, H.L. And Mebust, W.K.: Correlation of the American Urological Association symptom index with self-administered versions of the Madsen-Iversen, Boyarsky and Maine medical assessment program symptom indexes. J. Urol., 148: 1558, 1992.

205. Herzog, A.R., Fultz, N.H., Normolle, D.P., Brock, B.M. And Diokno, A.C.: Methods used to manage urinary incontinence by

older adults in the community. J. Am. Geriatr. Soc., 37: 339, 1989.

206. Seim, A., Sandvik, H., Hermstad, R. And Hunskaar, S.: Female urinary incontinence - consultation behavior and patient experiences: an epidemiological survey in a Norwegian community. Fam. Pract., 12: 18, 1995.

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

208. Burgio, K.L., Ives, D.G., Locher, J.L., Arena, V.C. And Kuller, L.H.: Treatment seeking for urinary incontinence in older adults. J. Am. Geriatr. Soc., 42: 208, 1994.

209. Rizk, D.E., Shaheen, H., Thomas, L., Dunn, E. And Hassan, M.Y.: 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.

210. Seim, A., Sivertsen, B., Eriksen, B.C. And Hunskaar, S.: Treatment of urinary incontinence in women in general practice: observational study. BMJ., 312: 1459, 1996.

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

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

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

214. Dugan, E., Roberts, C.P., Cohen, S.J., Preisser, J.S., David, C.C., Bland, D.R. Et Al.: Why older community-dwelling adults do not discuss urinary incontinence with their primary care physicians. J. Am. Geriatr. Soc., 49: 462, 2001.

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

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

217. Shaw, C., Tansey, R., Jackson, C., Hyde, C. And Allan, R.: Barriers to help seeking in people with urinary symptoms. Fam. Pract. 18: 48, 2001.

218. Hunskaar, S., Seim, A. And Freeman, T.: The journey of incontinent women from community to university clinic; implications for selection bias, gatekeeper function, and primary care. Fam. Pract., 13: 363, 1996.