A Systematic Review of Overweight and Obesity as Risk Factors and Targets for Clinical Intervention for Urinary Incontinence in Women

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Aims: To review the epidemiological literature of urinary incontinence with respect to overweight and obesity as a risk factor, and how the findings eventually fulfill general criteria for being a causal factor for the condition. Likewise to review all interventional studies assessing the effect of weight reduction on incontinence.

Methods: Systematic searches until June 2008 for publications of community based prevalence studies with bivariate or multivariate analyses of the association between urinary incontinence in women and overweight/obesity. In addition an attempt was made to identity and assess all relevant longitudinal studies, prospective case series, and trials, whatever design. Results: There is evidence 3 and some evidence 2 level data to support that in addition to BMI, waist-hip ratio and thus abdominal obesity may be an independent risk factor for incontinence in women. Only a few interventional studies have been carried out to assess the effect of weight reduction on incontinence. Five studies report effect on incontinence after surgical weight reduction procedures, and one study after a weight reduction program, thus giving some level 2 documentation. There are three RCTs which all show reducing incontinence by weight loss (level of evidence 1). Conclusions: Epidemiological studies document overweight and obesity as an important risk factor for urinary incontinence. There is now valid documentation for weight reduction as a treatment for urinary incontinence in women.

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Key words: obesity; overweight; risk factor; urinary incontinence; weight loss; women

INTRODUCTION

Epidemiological and clinical studies conducted in various populations reveal a number of variables associated with urinary incontinence, including several possible risk factors or contributing variables (Table I). Some factors, like age, parity, overweight and obesity, have been rather rigorously studied, and are often referred to as “established” risk factors. The understanding of epidemiology as the study of the distribution and determinants of disease, is critical not only in the search for the risk factors, but ultimately should lead us to primary or secondary disease prevention and effective treatments. Overweight or obesity is a potentially modifiable risk factor for urinary incontinence, and weight reduction may therefore be a treatment option. Obesity is an increasing health problem all over the world, and may be a contributing factor for an increased incidence of urinary incontinence.

In this paper I review the epidemiological literature of urinary incontinence with respect to overweight and obesity as a risk factor. I also review the current level of evidence for efficacy of weight reduction as a clinically feasible treatment option.

OVERWEIGHT AND OBESITY: A MAJOR HEALTH ISSUE OF THE WORLD

The prevalence of obesity now exceeds 25% of the adult population in countries in Europe, Australia, and the United States. According to the US Surgeon General, the primary concern of overweight and obesity is one of health and not appearance. Obesity in adulthood is associated with an increased risk of disability throughout life and a reduction in the length of time spent free of disability. The health consequences of overweight and obesity ranges from a general increase in premature death, heart disease and type 2 diabetes to an increased risk for some types of cancer. Several chronic conditions are also increased by weight gain, including sleep apnea, asthma, arthritis, and depression. The US Surgeon General also states that overweight and obesity are associated with increased risks of incontinence, but adequate references to the risk of incontinence are not found among the 769 references cited.

Measurements of Obesity

Overweight and obesity is usually measured and classified by the body mass index (BMI) (Table II). BMI is a practical...
TABLE I. Suggested Risk Factors for Urinary Incontinence in Women

<table>
<thead>
<tr>
<th>Age</th>
<th>Pregnancy</th>
<th>Parity</th>
<th>Obstetrical factors</th>
<th>Menopause and reproductive hormones</th>
<th>Hysterectomy</th>
<th>Obesity</th>
<th>Lower urinary tract symptoms</th>
<th>Functional impairment</th>
<th>Cognitive impairment</th>
<th>Smoking</th>
<th>Family history and genetics</th>
<th>Other factors</th>
</tr>
</thead>
</table>

Cited from Hunskaar et al.2

and easy measure that requires only two things: accurate measures of the person’s weight and height. It is calculated as weight in kilograms divided by the square of the height in meters (kg/m²), alternatively weight in pounds divided by the square of the height in inches, multiplied by 703.

BMI may overestimate body fat in persons who are very muscular, and it may underestimate body fat in persons who have lost muscle mass, such as many elderly, but for epidemiological purposes BMI is a very convenient variable, with a linear association to weight and usually an even distribution in the population.

MATERIALS AND METHODS

The source publications for this review were intended to be an adequate, but not complete sample of community based prevalence studies with bivariate or multivariate analyses of the association between urinary incontinence in women and overweight/obesity. In addition an attempt was made to identify and assess all relevant longitudinal studies, prospective case series, and trials, whatever design.

Systematic searches were done using Medline (1966 onwards) with the terms epidemiology, risk factor, urinary incontinence, overweight, obesity, body weight, body mass index, weight reduction, weight loss, female, and surgery, in different combinations, in an effort to balance sensitivity and specificity of the literature output. Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, and the Database of Abstracts of Reviews of Effects were also searched. There was no systematic attempt to search gray literature (conferences, abstracts, theses, and unpublished trials). The reference lists in national and international guidelines were checked against subsequent searches to identify missing evidence. Searches were updated and thereby including evidence published and included in the databases up to June 2008.

All retrieved papers were first reviewed by title and abstract, yielding a material of 424 papers. Thereafter full text articles were examined. For cross-sectional surveys only a sample could be included. The selection was made based on subjectively assessed quality, size and power of study, and the scientific level of analysis.

Criteria for Risk Factors and Causation

There is neither uniform definitions of how to define an established risk factor nor is there a uniform definition of causality or causation. A classical and much used set of criteria was presented by Austin Bradford Hill (1897–1991) (Table III).6 Later, criticism and counterarguments have been broaden by others.7,8 However, the usefulness of the criteria is that they describe what aspects of an association that need to be considered when deciding whether the likely interpretation of any association is causation.

Levels of Evidence, and Quality and Reporting of Epidemiological Studies

In this paper I review evidence by the established hierarchical system used by the UK National Institute for Health and Clinical Excellence (NICE),7 shown in Table IV. For issues of therapy or treatment, the highest possible evidence level (EL) is a well-conducted systematic review or meta-analysis of randomized controlled trials (RCTs; EL = 1++) or an individual RCT (EL = 1+).

In 2007 the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Initiative published recommendations on what should be included in an accurate and complete report of an observational study.10 Only a few and the most advanced epidemiological studies of urinary incontinence report their research according to the STROBE statement. Specifications for reporting of meta-analyses of observational studies in epidemiology have also been published.11 However, such meta-analyses on risk factors of urinary incontinence are not available.

Even in many of the recent studies reviewed analyses are very simple. Often only proportions or percentages are used to describe differences in different subgroups. Many analyses do not control for confounders (by stratification or multivariate analysis techniques). There is an obvious need for more advanced epidemiological analyses of risk factors and comorbidity, and strength of associations should be determined by relative risks and odds ratios.2

EPIDEMIOLOGICAL EVIDENCE FOR OVERWEIGHT AND OBESITY AS RISK FACTOR FOR URINARY INCONTINENCE

Cross-Sectional Surveys (Level of Evidence 3)

Data from a large number of studies indicate that urinary incontinence in women is associated with higher body mass index and greater weight, the references given are examples of cross-sectional studies of moderate to good quality.12–37 Table V shows the findings from a sample of the cross-sectional studies. It seems that there is a common picture of a clear dose-response effect, that the odds ratios seldom exceed a value of 2.0 except for the group of obese or very obese women, and that the maximum effect rarely exceeds an odds ratio greater than 4–5 in well controlled analyses.

When it comes to type, there seems to be a stronger association between increasing weight and stress incontinence (including mixed incontinence), than for urge incontinence and overactive bladder syndrome (Table V and Fig. 1).37 There seems to be only a small increase in risk for incontinence by increasing age (Fig. 2).14

TABLE II. WHO Classification of Overweight and Obesity and the Subsequent Risk of Comorbidity

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m²)</th>
<th>Risk of comorbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy weight</td>
<td>18.5–24.9</td>
<td>Average</td>
</tr>
<tr>
<td>Overweight</td>
<td>25–29.9</td>
<td>Some increased risk</td>
</tr>
<tr>
<td>Obesity I</td>
<td>30–34.9</td>
<td>Moderately increased risk</td>
</tr>
<tr>
<td>Obesity II</td>
<td>35–39.9</td>
<td>Severely increased risk</td>
</tr>
<tr>
<td>Obesity III</td>
<td>40 or more</td>
<td>Very severely increased risk</td>
</tr>
</tbody>
</table>

Cited from Hunskaar et al.2

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We have still very little knowledge of the absolute and relative importance of obesity, and almost no informations about the attributable risk of the factor in the society. Based on data from the EPINCONT study which comprised a rather slim population with about 18% of the women being obese, it can be calculated that if all these obese women reduced their BMI to less than 30, this would reduce the prevalence of incontinent women with a maximum of 10%, that is from 25 to about 22%. Given the same scenario, a obesity rate of 30% would reduce the prevalence of incontinence with about 20% (from 28 to 22), and obesity rate of 50% would reduce the prevalence of incontinence with about 30% (from 31 to 22).

**Case Series of Very Obese Patients (Level of Evidence 3)**

Such studies may be seen as prevalence studies in selected and biased populations, most often samples of patients planning for weight reduction surgery. These studies, however, often include a larger number of very obese persons than found in population based studies, and may therefore give a better estimate of the prevalence level. Even in the EPINCONT study with almost 28,000 participants included, only 288 women had BMI $\geq 40$.

Deitel et al. registered incontinence among 138 morbidly obese women who lost more than half of their excess weight with bariatric surgery. Mean age was 35 years and mean weight 124 kg (BMI $\sim$40–45) before surgery. Prevalence of stress incontinence was found to be 61%. In another study of 1,976 patients undergoing gastric bypass-induced weight, mean age was 44 years and mean preoperative BMI was 61. Severe urinary incontinence (self report of pad use) was found in only 23%. In a series of 180 women with BMI $>$40 that underwent evaluation for laparoscopic weight loss surgery, mean age was 40 years and mean BMI was 50 (range 40–81). The prevalence of incontinence was 67%. In a recent study of 101 women, aged 20–55 years with BMI $\geq 40$ who had laparoscopic gastric bypass, preoperative urinary incontinence was found in 67%. The prevalence of pure stress type was 28%, pure urge type 4%, and mixed type 32%.

These few case series give an impression of a very high prevalence of incontinence in morbidly obese women, especially of stress type.

**Prospective Cohort Studies (Level of Evidence 2)**

In prospective cohort studies it is possible to link overweight and obesity to the subsequent new onset of urinary incontinence, thus giving documentation for a temporal relationship between a possible cause and an outcome. Ideally, incident urinary incontinence should be associated with an increase in BMI in the period of study.

| **TABLE IV. Levels of Evidence for Intervention Studies (NICE)** |
|-------------------|--------------------------|
| **Level** | **Source of evidence** |
| 1++ | High-quality meta-analyses, systematic reviews of randomized controlled trials (RCTs) or RCTs with a very low risk of bias |
| 1+ | Well-conducted meta-analyses, systematic reviews of RCTs or RCTs with a low risk of bias |
| 2++ | High-quality systematic reviews of case–control or cohort studies; high-quality case–control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal |
| 2+ | Well-conducted case–control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal |
| 2− | Case–control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal |
| 3 | Non-analytical studies (e.g., case reports, case series) |
| 4 | Expert opinion, formal consensus |

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A retrospective cohort study of 104 female American Olympians who competed between 1960 and 1976, recorded the prevalence of stress and urge incontinence symptoms 20–35 years later. The prevalence of any stress incontinence symptoms was 45% (regular 10%) and any urge incontinence symptoms 26% (regular 9%). When age, BMI, parity, sport group, and recalled incontinence during sport were entered into stepwise logistic regression analyses, only BMI was significantly associated with regular stress or urge incontinence symptoms (ORs 1.21 and 1.32) (Evidence level 2—).42

In a prospective cohort study based on a random sample of women from Leicester UK, 6,424 were followed for 1 year in order to investigate the role of diet and other lifestyle factors in the incidence of overactive bladder and stress incontinence. A dose-response relationship was found in univariate analyses between weight groups and onset of stress incontinence and overactive bladder 1 year after. In a multivariate model for the onset of overactive bladder and stress incontinence, adjustments were made for age, physical functioning, energy and fluid intake, and stress incontinence/overactive bladder, respectively. There were significantly increased risks

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### TABLE V. Cross-Sectional Studies With Published Associations Between Urinary Incontinence in Women and Overweight/Obesity as Measured by Body Mass Index (BMI) and Using Adjusted or Multivariate Analyses

<table>
<thead>
<tr>
<th>References</th>
<th>Country</th>
<th>N</th>
<th>Age (years)</th>
<th>Results from of multivariate analyses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alling Moller et al. 20</td>
<td>Denmark</td>
<td>487 cases, 564 controls</td>
<td>40–60</td>
<td>BMI assessed by quartiles. Stress incontinence: Dose–response relationship with ORs 1.4, 2.6, 4.2, compared with lowest quartile. Urge incontinence: Dose–response relationship with ORs 0.8, 1.9, 2.2, compared with lowest quartile. Urge/OAB: Dose–response relationship with ORs 0.8, 1.8, 2.6, compared with lowest quartile.</td>
</tr>
<tr>
<td>Bortolotti et al. 22</td>
<td>Italy</td>
<td>2,767</td>
<td>40+</td>
<td>Stress incontinence: Dose–response relationship with ORs from 1.5 (BMI 21.6–23.8), to 2.5 (BMI &gt;23.7) compared with BMI &lt;21.6. Analysis adjusted for age only</td>
</tr>
<tr>
<td>Brown et al. 21</td>
<td>USA</td>
<td>7,949</td>
<td>65+, mean 77</td>
<td>OR 1.6 per 5 units of BMI increase. Attributable risk fraction of BMI &gt;30: 16%</td>
</tr>
<tr>
<td>Brown et al. 16</td>
<td>USA</td>
<td>2,763</td>
<td>Mean 67</td>
<td>OR per 5 units of BMI increase: 1.13 for stress, 1.09 for urge, 1.26 for mixed. Significant dose dependant relationship between increasing frequency of incontinence (from never to daily) and BMI. No effect of type of incontinence</td>
</tr>
<tr>
<td>Burgio et al. 15</td>
<td>USA</td>
<td>541</td>
<td>42–50</td>
<td>OR 1.07 per unit BMI increase</td>
</tr>
<tr>
<td>Burgio et al. 64</td>
<td>USA</td>
<td>759</td>
<td>Mean 28</td>
<td>OR 1.12 for overweight and 1.24 for obesity, not statistically significant</td>
</tr>
<tr>
<td>Buchsbaum et al. 65</td>
<td>USA</td>
<td>149</td>
<td>Mean 68</td>
<td>Stress incontinence: Dose–response relationship between increasing BMI and any incontinence in all age groups, see Figure 2</td>
</tr>
<tr>
<td>Chiarelli et al. 14</td>
<td>Australia</td>
<td>41,724</td>
<td>18–23, 45–50, 70–75</td>
<td>OR 1.15 per unit BMI increase</td>
</tr>
<tr>
<td>Danforth et al. 67</td>
<td>USA</td>
<td>83,355</td>
<td>37–54</td>
<td>Stress incontinence: Dose–response relationship with ORs from 1.5 (BMI 21.6–23.8), to 2.5 (BMI &gt;23.7) compared with BMI &lt;21.6. Analysis adjusted for age only</td>
</tr>
<tr>
<td>Elia et al. 66</td>
<td>USA</td>
<td>553</td>
<td>Mean 54</td>
<td>OR 1.95 for BMI &gt;26 compared with ≤26</td>
</tr>
<tr>
<td>Fritel et al. 70</td>
<td>France</td>
<td>2,625</td>
<td>49–61</td>
<td>OR for severe stress incontinence were 1.4 for overweight and 2.6 for obesity</td>
</tr>
<tr>
<td>Han et al. 47</td>
<td>Korea</td>
<td>769</td>
<td>Mean 54</td>
<td>Stress incontinence: Dose–response relationship with ORs from 1.67 (BMI 23–24), to 2.43 (BMI 25–26) and 2.97 (BMI 27+) compared with BMI &lt;21. Relationship and significance lost after adjusting for waist circumference</td>
</tr>
<tr>
<td>Hannestad et al. 37</td>
<td>Norway</td>
<td>27,936</td>
<td>20+</td>
<td>Dose–response relationship with any, stress, mixed, and urge. See Figure 1</td>
</tr>
<tr>
<td>Jackson et al. 48</td>
<td>USA</td>
<td>1,584</td>
<td>70–79, mean 74</td>
<td>OR per 5 units of BMI increase: 1.33 for stress, no association for urge</td>
</tr>
<tr>
<td>Janssen 79</td>
<td>USA</td>
<td>2,001</td>
<td>65+</td>
<td>OR 1.12 for overweight and 1.24 for obesity, not statistically significant</td>
</tr>
<tr>
<td>Kock et al. 72</td>
<td>Turkey</td>
<td>1,012</td>
<td>18+, mean 44</td>
<td>Not statistically significant association with BMI&lt;25 versus ≥25</td>
</tr>
<tr>
<td>Kuh et al. 31</td>
<td>UK</td>
<td>1,333</td>
<td>All 48</td>
<td>Stress incontinence: Dose–response relationship with ORs 1.1 for stress, 1.2 for urge, 1.3 for severe incontinence</td>
</tr>
<tr>
<td>Larrieu et al. 71</td>
<td>France</td>
<td>8,966</td>
<td>65–101, mean 77</td>
<td>Stress incontinence: Dose–response relationship with ORs from 1.1 (BMI 27–30), to 1.3 (BMI 30–35) and 2.3 (BMI 35+) compared with BMI 23–27. Analyzed for associations between incontinence, obesity and diabetes-related factors. ORs for stress incontinence in nonobese/diabetic, obese/nondiabetic, obese/diabetic women were 1.81, 2.62, and 3.67. ORs for overactive bladder syndrome in nonobese/diabetic, obese/nondiabetic, obese/diabetic women were 1.45, 2.93, and 2.97</td>
</tr>
<tr>
<td>Lawrence et al. 73</td>
<td>USA</td>
<td>3,962</td>
<td>Median 57</td>
<td>Stress incontinence: Dose–response relationship with ORs from 1.1 (BMI 27–30), to 1.3 (BMI 30–35) and 2.3 (BMI 35+) compared with BMI 23–27. Analyzed for associations between incontinence, obesity and diabetes-related factors. ORs for stress incontinence in nonobese/diabetic, obese/nondiabetic, obese/diabetic women were 1.81, 2.62, and 3.67. ORs for overactive bladder syndrome in nonobese/diabetic, obese/nondiabetic, obese/diabetic women were 1.45, 2.93, and 2.97</td>
</tr>
<tr>
<td>Melville et al. 74</td>
<td>USA</td>
<td>3,536</td>
<td>30–90, mean 53</td>
<td>Stress incontinence: Dose–response relationship with ORs 1.21 and 1.32 (Evidence level 2—).42</td>
</tr>
<tr>
<td>Mommsen and Foldspang 55</td>
<td>Denmark</td>
<td>2,589</td>
<td>30–59</td>
<td>OR 1.07 per unit BMI increase</td>
</tr>
<tr>
<td>Nygaard et al. 75</td>
<td>USA</td>
<td>5,701</td>
<td>50–69</td>
<td>OR 1.05 for any incontinence and OR 1.04 for moderate/severe incontinence per BMI unit</td>
</tr>
<tr>
<td>Sampselie et al. 27</td>
<td>USA</td>
<td>3,302</td>
<td>42–52, mean 46</td>
<td>OR 1.25 for mild incontinence and OR 1.4 for severe incontinence per BMI unit</td>
</tr>
<tr>
<td>Song et al. 76</td>
<td>China</td>
<td>4,684</td>
<td>20+, mean 40</td>
<td>BMI assessed as ≥75 percentile compared with under. OR 1.8 for stress incontinence and OR 1.5 for urge incontinence</td>
</tr>
<tr>
<td>Teleman et al. 77</td>
<td>Sweden</td>
<td>2,682</td>
<td>55–64</td>
<td>Obesity was associated with both overactive bladder (OR 1.6) and stress incontinence (OR 1.5). Independent risk factor for stress incontinence was also elevated BMI by 25% or more since age of 25 (OR 1.69)</td>
</tr>
<tr>
<td>Waetjen et al. 45</td>
<td>USA</td>
<td>3,301</td>
<td>40–55, mean 46</td>
<td>BMI was not significantly associated with any, frequent, stress, urge, or mixed urinary incontinence, but multivariate analyses included waist circumference, which was associated with any (OR 1.03), frequent (OR 1.05), stress (OR 1.04) and mixed (OR 1.04) incontinence (per cm increase)</td>
</tr>
</tbody>
</table>

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associated with BMI for both symptoms, ORs for overweight and obesity were 1.25 and 1.74 for stress symptoms and 1.24 and 1.46 for overactive bladder. In a new analysis using the same dataset, the multivariate models were expanded and adjusted for a large number of comorbidities and chronic illnesses. There were again increased risks associated with BMI for both symptoms, ORs for overweight and obesity were 1.4 and 2.3 for stress symptoms and 1.3 and 1.2 for overactive bladder, but only the trend for stress incontinence reached statistical significance. The differences indicate a weaker association between BMI and overactive bladder than with stress incontinence (Evidence level 2+).

In a 5-year annual follow up of a cohort study of women aged 42–52 at inclusion (n = 3,301), 2,702 women could be analyzed for 5-year incidence of urinary incontinence. Among the continent women at baseline, incident incontinence was found in 55.7% during the follow up period, giving an annual incidence of 11.1% per year, most developed mild incontinence. Multivariate analyses that included waist circumference, showed that BMI per unit increase was associated with any (OR 1.05), frequent (OR 1.12), stress (OR 1.06), urge (OR 1.03), and mixed (OR 1.09) urinary incontinence. The results show that most incident incontinence is mild, but still overweight and obese women have a significantly higher risk of developing incontinence (Evidence level 2+).

In a recent report from the American Nurses’ Health Study II, a prospective subcohort was defined to describe the relations between BMI, weight gain, and incident urinary incontinence in middle-aged women (37–54 years old). The women recalled and reported their weight at age 18 years, and then again current weight in 2001. BMI was calculated based on height given in 1989. In the period 2001–2003 more than 4,000 incident cases with at least monthly incontinence were identified among 30,982 women who reported no incontinence in 2001. Increasingly higher BMI was related to increasing odds of developing incontinence for all severity groups, the reference group being BMI 21–22.9. Comparing women with BMI ≥35, the OR for at least monthly incontinence was 2.11, for at least weekly incontinence 3.85, and for severe incontinence 5.52. The same pattern was seen for the association to weight gain, with ORs of 1.91, 4.04, and 4.96 for the same incontinence groups, respectively. The study also analyzed incontinence types. Compared to the BMI 21–22.9 group, women with BMI ≥35 had OR for stress incontinence of 3.42, for urge incontinence of 6.10 and mixed incontinence 5.60, that means smallest risk increase for stress incontinence. When weight gain was analyzed, however, stress incontinence had the highest OR (5.92) compared with urge (2.33) and mixed (2.53) incontinence (Evidence level 2+).

In summary, there is sound evidence from a few, but well performed longitudinal studies that high BMI is associated with the later new onset of urinary incontinence. The associations seems strongest for stress and mixed incontinence, somewhat weaker for urge incontinence and overactive bladder syndrome.

**OTHER MEASURES OF OVERWEIGHT: WAIST—HIP RATIO**

Little is known about whether the fat distribution is of importance for incontinence. BMI does not give a picture of this, while measuring abdominal obesity (waist circumference) or central obesity as expressed by a high waist-hip ratio, will.

Data from a randomized trial of hormone therapy in postmenopausal women were used to analyze risk factors for incontinence. In multivariable analyses including also BMI, waist-hip ratio was found to be an independent risk factor for stress incontinence (OR 1.18 per 0.1 Unit), but not for urge and mixed incontinence. In a study of 769 Korean women (mean age 54 years) it was found that in comparison with women in the lowest quartile of waist circumference, the ORs for stress incontinence increased significantly in a dose dependant relationship (1.79, 3.50, and 6.07) for the next quartiles, respectively, after adjustments for BMI. Among women aged 54–79 years in the Nurses’ Health Study there were highly significant trends of increasing risk of incontinence with both increasing BMI and waist circumference. When BMI and waist circumference were included in models simultaneously, BMI was associated with urge and mixed incontinence, but not stress incontinence. Waist circumference was associated only with stress incontinence.

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**Fig. 1.** The association between BMI and urinary incontinence types, adjusted for age, number of children, coughing and dyspnoea. Values are expressed as odds ratios. Data from Hannestad et al. A Any incontinence. B Severe incontinence.

**Fig. 2.** The association between BMI and urinary incontinence by age groups. Values are expressed as odds ratios. Data from Chiarelli et al.
In an analysis from the EPINCONT study (n = 27,936) with 6,876 incontinent women (24.6%), waist-hip-ratio was calculated.44 A waist–hip ratio of >0.80–0.85 in women is taken to indicate a so-called android fat distribution and central obesity. The frequency of incontinence increased with increasing waist-hip ratio for all types and severity of incontinence in the bivariate unadjusted analyses. The results from multivariable analyses with adjustment for age and parity showed a statistically significant effect for any incontinence, severe symptoms and mixed type. The association weakened but remained significant for any incontinence and mixed incontinence (OR 1.1 for both) after additional adjustment for body mass index. The study indicates that there is an association between central obesity and the presence of urinary incontinence. Women with urinary incontinence and a high waist-hip ratio may benefit more from weight-reduction than the women with a “pear” body shape.

In accordance with the above postulate it is interesting to learn results from a cohort study of 40 women followed for 6 months after a randomized controlled study of diet based weight reduction.50 Twelve of 19 women who lost 3% or greater in waist circumference experienced 50% or greater reduction in incontinence frequency compared with 5% one of 21 achieving less waist circumference loss. In a 2-predictor logistic model of this end point, reduction in waist circumference appeared to explain the effect of reduction in BMI.

There is thus evidence 3 and some evidence 2 level data to support that in addition to BMI, waist-hip ratio and thus abdominal obesity may be an independent risk factor for incontinence in women.

**BIOLOGICAL EVIDENCE FOR AN ASSOCIATION BETWEEN OBESITY AND INCONTINENCE**

Many of the epidemiological studies that claim obesity to be a well established factor for causing incontinence or contributing to the severity of the condition, also often suggest the mechanism. A common statement is that obesity acts through the added weight, like pregnancy, that bear down on pelvic tissues, causing chronic strain, stretching and weakening of the muscles, nerves, and other structures of the pelvic floor. The scientific basis for these mechanisms are, however, rather weak. The link between obesity and increased intraabdominal pressure is supported by some studies. Increased abdominal wall weight should in turn increase intraabdominal pressure and also intravesical pressure in addition to inducing changes of urethral mobility.

In a much cited study52 13 women underwent a comprehensive evaluation of lower urinary tract function before and 1 year after surgically induced weight loss. Statistically significant changes were seen in measures of vesical pressure, the magnitude of bladder pressure increases with coughing, bladder-to-urethra pressure transmission with cough, and urethral axial mobility.

Loss of 69% of excess weight following gastric bypass surgery in 15 patients decreased urinary bladder pressure.52 A Korean study of 98 women who were clinically diagnosed as having stress incontinence and 102 controls, found that the average intraabdominal pressure was significantly increased in the obese group over that in the nonobese group. Stress incontinence did not, however, influence the urodynamic parameters.53

Among 136 female patients with mean age 61 years and mean BMI 28, a strong association between intra-abdominal pressure and BMI was demonstrated, with a Pearson coefficient value of 0.76. A strong correlation between intravesical pressure and BMI was also demonstrated, with a Pearson coefficient correlation value of 0.71. Of the 136 patients, 48% had genuine stress urinary incontinence, 26% also with a low-pressure urethra, and 13% had detrusor instability. The authors conclude that obesity may stress the pelvic floor secondary to chronic state of increased pressure, and may represent a mechanism which supports that obesity is a causal factor in the development of stress incontinence.54

In a similar study it was found that intraabdominal pressure increased by increasing BMI, and was higher in patients with pressure related morbidities, including stress incontinence.55

In a RCT of weight loss as treatment for incontinence with 40 women enrolled56 the groups had similar urodynamic measurements within the normal range at start. Significant correlations between weight change and changes in urodynamic findings were observed, including decreased initial intravesical pressure (Spearman \( r = 0.52, P = 0.01 \)), decreased intravesical pressure at maximum capacity \( r = 0.62, P = 1 \) and increased Valsalva leak point pressure \( r = 0.57, P = 0.03 \). This study therefore also indicate that change of the risk factor changes a biological parameter and interferes with a plausible mechanism.

In a short review from 2000 Cummings and Rodning56 conclude that additional neurophysiologic and urodynamic studies are needed to better define the mechanisms of the association between obesity and incontinence. This statement holds even in 2008.

**RESULTS OF CLINICAL INTERVENTIONS**

Only a few interventional studies have been carried out to assess the effect of weight reduction on incontinence. I have found five studies reporting changes of incontinence after surgical weight reduction procedures, and one study evaluating a weight reduction program, thus giving us some level 2 documentation. There seems to be published only 3 RCTs with the aim of reducing incontinence by weight loss, thus giving us valid level 1 documentation. The interventions were a radical liquid diet program in one, a diet and exercise program in one, and intense lifestyle intervention in pre-diabetic women in one.

**Case Series of Surgically Induced Weight Loss in Morbidly Obese Women (Level of Evidence 3)**

Deitel et al.58 assessed urinary incontinence in morbidly obese women (n = 138) who lost more 50% or more of their excess weight following bariatric surgery. The prevalence of urinary stress incontinence decreased from 61% to 12% \( P < 0.001 \) after stabilization of weight loss after 2–5 years.

In a series of 13 women who underwent a comprehensive evaluation of lower urinary tract function before and 1 year after surgically induced weight loss, Bump et al.51 found significant improvements after weight loss. Of 12 subjects who complained of incontinence before surgery only three (25%) complained of incontinence \( P = 0.004 \) and only one requested treatment after weight loss. Objective and subjective resolution of both stress and urge incontinence was documented. Statistically significant changes were seen in urodynamic parameters, number of incontinence episodes, and the need to use absorptive pads.

In 2003 Sugerman et al.57 published experiences with 1,025 obese patients (78% women) treated with gastric bypass between 1981 and 2000. Mean age was 39 years and mean BMI was 51. Before surgery incontinence (no details given
except leakage) was found in 30% of women and 4% of men. One to two years after surgery BMI had decreased to a mean of 33. Urinary incontinence decreased from 25% to 2%, no results by gender given.

In a group of 195 men and women (83% women) who was investigated before and 1 year after laparoscopic adjustable gastric banding, BMI was reduced from 46 to 32, mean percent excess body weight lost was 46% during the first year. Major improvements occurred in many comorbidities, for stress incontinence 64% reported to be better.

In a prospective cohort study, 101 women (aged 20–55 years) with BMI ≥40 underwent laparoscopic Roux-en-Y gastric bypass and were followed to 6 and 12 months. Presence, severity, and effect of incontinence were assessed. Mean BMI was reduced from 49 presurgery to 35 at 6 months and 30 at 12 months postsurgery. Prevalence of incontinence decreased from 67% presurgery to 41% at 6 months and 37% at 12 months (P < .001). Among incontinent women who lost 18 or more BMI points, 71% regained continence at 12 months.

In another prospective cohort study, 44 women with mean BMI of 48 underwent bariatric surgery and were followed to >6 months. Mean loss of excess body weight was 61%. Prevalence of severe incontinence decreased from 50% presurgery to 13% at 6 months (P < .001) (both genders combined).

### Case Series From Weight Reduction Programs (Level of Evidence 3)

A prospective cohort study evaluated the effect of weight reduction on urinary incontinence in only 10 moderately obese women. The women had a mean BMI of 38 and a mean of 13 incontinence episodes per week. The mean BMI reduction was 5. Among women achieving a weight loss of ≥5%, 6/6 had ≥50% reduction in incontinence frequency compared to 1 in 4 women with <5% weight loss (P < .003). Incontinence episodes decreased to 8 per week following weight reduction (P < 0.07).

Sixty-four incontinent women were offered a weight reduction program with a target loss of 5–10% based on low-calorie diet, exercise and an anti-obesity drug. Weight loss was associated with significant reduction in pad test loss (median difference 19 g). The results suggest that weight reduction of 5% of initial body weight can improve urinary incontinence severity and its effects on quality of life in obese women.

### Randomized Controlled Trials of Weight Reduction as Treatment for Urinary Incontinence (Level of Evidence 1)

In a randomized, controlled clinical trial among overweight and obese women experiencing at least four incontinence episodes per week, women were randomly assigned to a 3-month liquid diet weight reduction program (n = 24) or a wait-list delayed intervention group (n = 24), with follow-up for 6 months. A total of 40 women were assessed after 3 months. Women in the intervention group had a mean of 16 kg weight reduction (from 97 kg) compared with 0 kg in the control group. There was a 60% reduction in weekly leakage episodes compared with 15% in the control group (P < 0.0005). Both stress (P = 0.003) and urge (P = 0.03) incontinence episodes decreased. Following the weight reduction program the wait-list control group experienced a similar median reduction in weekly incontinence episodes (71%). The result was maintained for 6 months. Among women achieving a weight loss of 5% or greater, 58% experienced a reduction in incontinence frequency of 50% or greater. The authors concluded that weight loss of 5–10% has an efficacy similar to that of other nonsurgical treatments and should be considered a first line therapy for incontinence.

The Diabetes Prevention Program (DPP study) was a RCT over mean 2.9 years of 1,957 overweight pre-diabetic women. A total of 660 (34%) were randomized to intensive lifestyle therapy, 636 (32%) to metformin, and 661 (34%) to placebo with standard lifestyle advice. The main outcome measure was incontinence symptoms by frequency and type by a validated questionnaire completed at the end-of-trial visit. The prevalence of total (stress or urge) weekly incontinence was lower among women in the intensive lifestyle group (38%) than those randomized to metformin (48%) or placebo (46%). This difference was most apparent among women with stress incontinence (31% for intensive lifestyle group vs. 40% for metformin vs. 37% for placebo, P = 0.006). Change in weight accounted for almost all of the incontinence effect explained (25%), with change in exercise and incident diabetes each explaining only 5%.

The first large scale RCT on the effect of weight reduction and incontinence was the recently published Program to Reduce Incontinence by Diet and Exercise (PRIDE) study, conducted at two US sites, including 338 overweight or obese women reporting ≥10 episodes of incontinence weekly. Participants were randomized in a 2:1 ratio to an intensive 6-month lifestyle and behavior change weight intervention program (intervention; N = 226) or to a structured education group (4 health information sessions; control; N = 112). Both groups were given an instructional booklet describing standard behavioral therapy for incontinence. Mean BMI was 36 and mean weekly total incontinent episodes were 25. Women in the intervention group achieved a mean weight loss of 8% (8 kg) compared to 2% (2 kg) in the education group (P < .0001). Mean total incontinent episodes per week decreased by 46% in the intervention group compared to 25% in the control group (P = .04). Compared to control, women in the intervention group had a decrease in stress incontinence episodes (57% vs. 33%, P = 0.02), and also urge incontinent episodes, the latter not statistically significant though (41% vs. 29%, P = 0.23). The proportion of women achieving a clinically significant decrease in weekly frequency of incontinent episodes of at least 70% was greater in the intervention (41%) than the control (22%) group (P < 0.003).

### CONCLUSIONS

There is clear evidence to support that in addition to BMI, waist-hip ratio and thus abdominal obesity may be an independent risk factor for incontinence in women. The strength of the association is not very large, but the effect may be significant in a society because of the large prevalence of obesity. For the individual incontinent women, the exact causality will always be undetermined, but for higher obesity grades the probability that the weight plays a role will be high. The findings from cross sectional studies seem to be consistent, specific and also showing a dose-response relationship. There is some, but not very strong data on the biological mechanisms.

Only a few interventional studies have been carried out to assess the effect of weight reduction on incontinence, but they all show that incontinence is reduced by weight loss. Moderate weight loss seems to be an adequate first line therapy for urinary incontinence in women. The interventional and experimental evidence is also seen as a strong support for the causation hypothesis.
REFERENCES


