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Review Article
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The role of obesity on urinary incontinence and anal incontinence in women: a review

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Obesity prevalence is increasing worldwide, with significant healthcare implications. We searched PubMed/MEDLINE, Embase and the Cochrane Library for articles registered until June 2020 to explore the relationship between obesity and urinary (UI) and anal incontinence (AI). Obesity is associated with low-grade, systemic inflammation and proinflammatory cytokine release, producing reactive oxygen species and oxidative stress. This alters collagen metabolism and, in combination with increased intra-abdominal pressure, contributes to the development of UI. Whereas in AI, stool consistency may be a factor. Weight loss can reduce UI and should be a management focus; however, the effect of weight loss on AI is less clear.

Keywords Anal incontinence, obesity, pelvic floor, urinary incontinence, women.

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Background

Up to 39% of the global population are classified as being overweight or obese,[1] and to date no country has been able to reverse the obesity epidemic.[2] Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health. A body mass index (BMI) of greater than 25 kg/m^2 is considered overweight and a BMI of over 30 kg/m^2 is classed as obese.[1] The prevalence of overweight and obesity has been steadily increasing since 1999 (Figure 1), and is becoming a significant health and financial burden worldwide .[2]

In 2015 excess weight contributed to 4.0 million (2.7–5.3 million) deaths and 120 million (84–158 million) disability-adjusted life years (DALYs), representing 4.9% (3.5–6.4%) of all-cause DALYs among adults globally.[4]

Obesity has been linked to the pathophysiology of several health conditions, including urinary incontinence (UI) and anal incontinence (AI).

The EPICONT study (Epidemiology of Incontinence in the County of Nord-Trøndelag) analysed data from 34,755 women and demonstrated that obesity had a significant impact on stress, urge and mixed UI (Table 1).[5]

Previous evidence suggests that the odds ratio for the presence of UI is 1.6 per 5-unit increase in BMI[6], and the prevalence of UI in women in the 'morbidly obese' category seeking weight-loss surgery is as high as 67%.[7]

Urinary incontinence (UI) is a common condition, with a prevalence of 25% in the general population.[8] It has a significant impact on individuals and the wider society.[9] Several different aetiological factors have been identified in epidemiological studies, such as previous hysterectomy, pregnancy and operative vaginal birth.[10] In younger women the prevalence is lowest, increasing towards menopause and then rising more steadily from the age of 60 years.[9]

There is a significant economic burden associated with UI. The direct costs of UI care in the USA has been estimated to be greater than $16 billion per year.[11] A systematic review by Milsom et al. in 2014 demonstrated that there is an increasing burden in the USA as well as in several other countries.[12] Annual cost-of-illness estimates for urge urinary incontinence (UUI), including direct and indirect costs, have been reported as being up to €7 billion for six western countries, including Canada and the UK.[13]

Studies on quality of life (QoL) have highlighted a potentially detrimental impact, regardless of age,[14,15] with

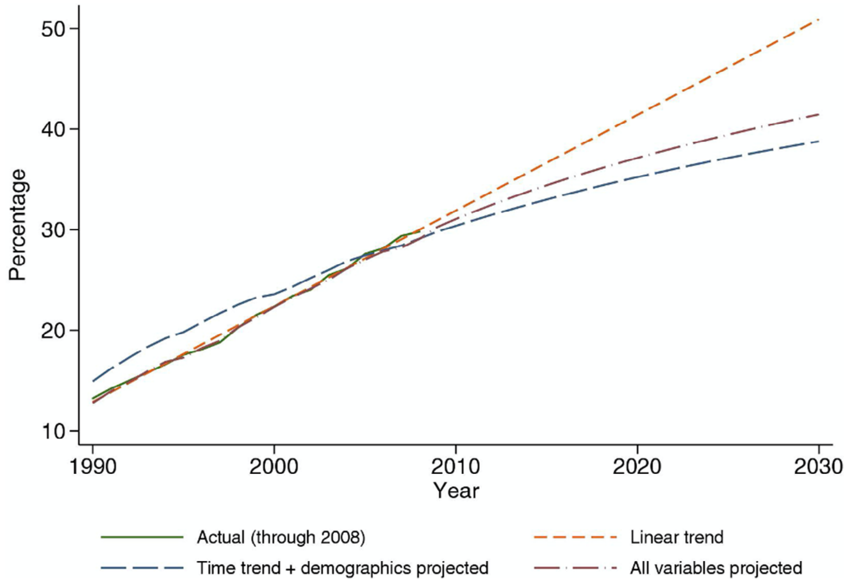

Figure 1. Actual and predicted prevalence of obesity through 2030. Reprinted.[3] Copyright 2020 Elsevier.

Table 1. Multiple regression analysis of the association between BMI and UI[5]

	Incontinence		Stress-type incontinence		Mixed-type incontinence		Urge-type incontinence	
	All	Severe	All	Severe	All	Severe	All	Severe
Body mass index								
<25 kg/m^2	1	1	1	1	1	1	1	1
25–29 kg/m^2	1.4 (1.3–1.5)	2.0 (1.7–2.3)	1.4 (1.2–1.5)	1.9 (1.5–2.4)	1.7 (1.5–1.9)	2.3 (1.9–2.8)	1.1 (0.9–1.3)	1.6 (1.1–2.4)
30–34 kg/m^2	1.9 (1.7–2.1)	3.1 (2.6–3.7)	1.7 (1.6–2.0)	2.8 (2.1–3.6)	2.3 (2.0–2.7)	3.5 (2.8–4.3)	1.5 (1.2–1.9)	3.0 (1.9–4.6)
35–39 kg/m^2	2.4 (2.1–2.8)	4.2 (3.3–5.3)	2.0 (1.7–2.5)	3.2 (2.1–4.8)	3.5 (2.9–4.3)	5.5 (4.1–7.4)	1.4 (0.9–2.1)	2.4 (1.2–4.9)
40+ kg/m^2	2.7 (2.1–3.5)	5.0 (3.4–7.3)	2.4 (1.7–3.3)	4.2 (2.2–7.9)	3.7 (2.7–5.2)	6.0 (3.7–9.6)	1.8 (0.9–3.5)	3.8 (1.3–11.1)

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anxiety, depression and sexual dysfunction commonly reported.[16-18] Pace et al. found an inverse correlation for the total female sexual function index in the domains arousal, orgasm, lubrication and satisfaction with increased BMI in postmenopausal women.[19]

The joint International Urogynecological Association–International Continence Society (IUGA–ICS) report on the terminology for female pelvic organ prolapse has defined AI as the involuntary loss of flatus or faeces,[20] where the symptom of faecal incontinence (FI) is defined as the involuntary loss of solid and/or liquid faeces and the symptom of flatal incontinence is the involuntary loss of flatus (gas).[21,22]

There is patient reluctance to seek medical help for AI, potentially through embarrassment, but this is compounded by the infrequent screening provided by healthcare providers. Subsequently, there are limited epidemiological data for AI and the magnitude of the condition remains largely unknown,[23] especially in comparison with UI. In a recent study of 457 women presenting for benign gynaecological care, only 17% of women with FI were questioned about these symptoms by their clinician[24]. Johansen et al. reported that less than a third of patients with AI had disclosed this to a healthcare provider.[23]

Response rates to questionnaires highlight this difference in willingness to disclose symptoms of AI, compared with UI, with only 60–70% of participants responding to AI-related questionnaires, whereas a review of epidemiological studies with 230 000 respondents reported a median response rate for UI questionnaires of 80%.[10,25]

Barriers in disclosing incontinence have been reported, including social expectations, the lack of a ‘trusted space’ for disclosure, confusion surrounding the medical terminology and meaning, and emotional, social and psychological consequences.[26]

The prevalence of AI is estimated to range between 7 and 15% in community-dwelling adults; however, the prevalence in care home residents is reported to be at least three times higher.[27,28]

This difference in prevalence is primarily associated with the increased prevalence of dementia in care home populations, a disorder with direct negative impact on anal sphincter control. Other contributors such as altered stool consistency and immobility are also likely to play a role. There is evidence to suggest that care home residents with AI often have coexisting UI, known as double incontinence.[26] In population-based studies, UI has been found to coexist in 50% of patients with AI.[29]

Risk factors for AI include: acquired structural abnormalities, for example secondary to childbirth and obstetric anal sphincter injuries; functional disorders, such as irritable bowel syndrome and inflammatory bowel disease; and neurological disorders, such as multiple sclerosis and dementia.[30] The impact of AI can be variable and detrimental in some patients, with feelings of social isolation and a loss of dignity.[31]

The impact of obesity on AI has not been widely researched. Women with AI have higher BMIs compared with the general population.[29] In a study by Varma et al.,[32] obesity was independently associated with a 20% higher prevalence of AI per 5-unit increase in BMI.

The World Health Organization has recognised the increasing prevalence of obesity as a global epidemic, with 2.8 million people dying each year as a result of being overweight or obese. Given the associations between obesity and pelvic floor disorders and the socio-economic burden of UI and AI, the aim of this review was to evaluate the current evidence on the effects of obesity on the pelvic floor. An overview of the relationship between obesity and UI and AI, as well as the outcomes of continence surgery treatments and weight loss, is warranted to better inform clinical practice.

Pathophysiology of incontinence

Pathophysiology of urinary incontinence in obese populations

Several studies have investigated the association between obesity and UI. A systematic review by Hunskaar et al.[33] investigated the factors that predispose women who are overweight or obese to develop UI. The study suggested that intra-abdominal pressure increases with obesity, weakening the pelvic muscles and pelvic innervation. The prolonged effects of chronic strain on the pelvic musculature, nerve supply and supporting structures may cause pelvic floor muscle weakness and have a negative impact on pelvic organ function.[34]

This increase in pressure can be demonstrated in urodynamic results, which show an increased maximal intravesical peak pressure during a cough; however, this increase in pressure does not seem to alter urethral sphincter function, as patients who are obese have a similar Valsalva leak point pressure and a comparable maximal urethral closure pressure compared with controls who are not obese.[35-37]

Coexisting excess weight and morbid obesity causes a rise in intra-abdominal pressure, which seems to reach a value of 12 cmH_2O. This significantly differs from the intra-abdominal pressure found in healthy controls who are not obese (BMI < 30 kg/m^2, mean intra-abdominal pressure of 0 ± 1.2 cmH_2O; $P < 0.0001$), and contributes to the development of UI symptoms.[38]

In addition, the associated oxidative stress arising from adipose tissue has been postulated to increase the prevalence and severity of incontinence through alterations in collagen metabolism. Visceral adipose tissue is considered an endocrine organ in itself, and in populations who are overweight or obese the secretion of inflammatory cytokines and factors, such as tumour necrosis factor alpha (TNF-α) and interleukin 6 (IL-6), is dysregulated.[39] Leptin activates nicotinamide adenine dinucleotide phosphate (NADPH) oxidases, stimulating the production of reactive oxygen species such as hydrogen peroxide (H_2O_2), also contributing to the increased oxidative stress resulting from obesity.[39,40]

A study by Liu et al. showed that exogenous H_2O_2 had two-way regulatory effects on collagen metabolism.[41] After incubation for 24 hours in vitro with human uterosacral ligament fibroblasts, lower concentrations of H_2O_2 stimulated the anabolism of collagen type 1 alpha 1 (COL1A1), whereas a higher concentration promoted catabolism. They also noted the upregulation of transforming growth factor beta 1 (TGF-β1) and proteolytic enzymes such as matrix metalloproteinase 2 (MMP-2), which promote collagen catabolism, with increasing oxidative stress. It was concluded that oxidative stress contributed to collagen metabolic disorder in human pelvic fibroblasts.

It has therefore been demonstrated that both the physical and the biochemical stresses of obesity on the pelvic floor neuromusculature seem to predispose an individual to the development of UI.

Pathophysiology of anal incontinence in obese populations

There is limited evidence on AI and obesity, but some studies indicate a higher prevalence of AI in individuals who are obese: one study reported a 67% prevalence of AI in 256 women who were morbidly obese.[42] The aetiology of this is likely to be multifactorial. Altered stool consistency is one of the proposed contributors to AI, as obesity has been associated with increased intestinal motility and diarrhoea.[43] In an observational study of patients who were obese and undergoing evaluation for weight loss, conducted

Table 2. Anal manometry parameters[46]

Characteristic	Overall (*n* = 407)	Normal (*n* = 124)	Overweight (*n* = 123)	Obese (*n* = 160)	*P*
Resting pressure, mmHg	36.2 ± 19.6	31.2 ± 18.1	32.5 ± 17.1	43.0 ± 20.6	<0.0001
Squeeze pressure, mmHg	74.0 ± 36.5	68.0 ± 32.3	67.6 ± 31.5	83.6 ± 41.0	<0.0001
Capacity, cc	114.9 ± 55.0	104.3 ± 46.9	117.5 ± 59.5	121.1 ± 56.4	0.031

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by Pares et al., symptoms of AI were found in 32.7%, and those with incontinence reported significantly higher percentages of altered bowel habits with non-formed stools (35.2%, $P = 0.004$).[44] A prospective case-matched study by Brochard et al. reported similar findings.[45] They compared patients with AI who were obese with age- and sex-matched patients with AI who were not obese. The authors of this study suggested that diarrhoea was significantly associated with obesity in patients with FI (OR 2.94, 95% CI 1.22–7.19, $P = 0.0158$), and recommended a focus on stool consistency when managing AI in these patients.

When reviewing evidence from anorectal manometry investigations, patients who are obese have higher upper- and lower-part resting pressures, higher intra-abdominal pressure during effort and increased maximum tolerable volume.[45]

A study by Ellington et al. also showed that baseline resting and squeeze pressures in multivariable analyses of anal manometry were increased in women with FI who were obese, compared with women of normal weight and women who were overweight (Table 2).[46]

A recent study on asymptomatic women showed that age, BMI and parity influences anorectal motion. Using a magnetic resonance imaging (MRI) defecating proctogram, the authors showed that in younger women an increased BMI was associated with a more obtuse median anorectal resting angle: 107° in women with a BMI of ≥35 kg/m^2 and 97° in women with a BMI of <25 kg/m^2.[47] This is perhaps because of the increased intra-abdominal pressure associated with increased BMI that may lead to greater perineal descent at rest. Perineal descent has long been associated with chronic straining and constipation but is also found in those with AI and pudendal nerve neuropathy, which may be caused by chronic stretch and pressure forces on the pelvic floor.[48]

Risk factors for incontinence and obesity

Age, obesity and incontinence

Data are limited regarding the cumulative effect of age on symptom severity in individuals who are obese. Given that most data are derived from community-dwelling study participants, the generalisation of findings is not possible. Obesity in childhood is associated with functional constipation and functional non-retentive AI;[49] however, its effect specifically on AI is not as clear. A recent large-scale population study of 6803 children and adolescents reported on the prevalence of AI, daytime urinary incontinence (DUI), nocturnal enuresis and nocturia.[49] Faecal incontinence was reported in 11.2% of the school-entry group (mean age 6.45 years) and in 2.1% of adolescents (mean age 13.9 years). Obesity was found to be associated with FI in first-grade boys (OR 1.86, 95% CI 1.10–3.15).

Compared with normal weight, however, no association was found with increasing BMI in adolescents. A possible reason for this may be that obesity-associated constipation, and subsequent incontinence, has spontaneously resolved by adolescence or has been successfully treated. In this study, 21.8% of children and 4.5% of adolescents reported DUI, and no significant association was found with obesity. It may be that the impact of weight on pelvic floor function becomes evident after a longer period of obesity.

Varma et al. reported a 24.2% (511/2106) prevalence of FI in community-dwelling women who were older than 40 years.[32] A positive correlation of age with FI rates was observed after stratification by age. The multivariable analysis revealed a not statistically significant trend (OR 1.1, 95% CI 1.0–1.2, $P = 0.15$), whereby increments of age (per 5 years) increased the odds of developing FI by approximately 10%. It remains unclear, however, whether there is interplay between obesity and age on risk of AI, as obesity was not controlled for in this cross-sectional study. The study authors did recognise the limitations of a cross-sectional study in determining casual associations.

Parity, obesity and incontinence

Childbirth is well known to be associated with pelvic floor dysfunction, AI and UI, and pelvic organ prolapse. The effects are most pronounced with increasing parity and vaginal birth, and are thought to result from antepartum and intrapartum neuromuscular effects and perineal trauma at the time of delivery.[50-53]

Obesity has a positive association with gestational diabetes mellitus (GDM) and cephalopelvic disproportion (CPD), and therefore instrumental delivery rates, incidence of prolonged second stage and rates of obstetric anal

Table 3. Association between BMI and third- and fourth-degree perineal tears[54]

Factor	Odds ratio (95% CI)	*P*
BMI		
<25 kg/m^2	1	–
25 to <30 kg/m^2	1.14 (0.99–1.30)	0.058
30 to <35 kg/m^2	0.89 (0.71–1.10)	0.275
≥35 kg/m^2	0.88 (0.63–1.23)	0.446

sphincter injury (OASIS) could be higher, with increased rates of UI and AI as associated sequelae in women who are obese.

A large retrospective study of 45 557 births showed that perineal trauma rates were actually reduced among women who were obese and that OASIS was not significantly associated with BMI (Table 3).[54]

This is consistent with the findings from Lindholm and Altman, who studied all singleton vaginal deliveries in Sweden in the period 2003–2008 (n = 210 678). The rate of OASIS was 4.25%, and they concluded that increasing BMI showed a significant near-dose-response type of protective effect against third- and fourth-degree lacerations.[55]

Outcomes of continence interventions

Outcomes of urinary incontinence interventions in women who are obese

The majority of existing research into outcomes of continence surgery in the obese population is from studies evaluating mid-urethral slings. In contrast, research concerning the efficacy of bulking agents is more limited.

A meta-analysis by Xia et al. demonstrated that the objective success rates after mid-urethral sling were lower in women with BMIs of >25 kg/m^2, compared with normal BMI, although no significant difference was found between women who were overweight (BMI 25–30 kg/m^2) and women who were obese (BMI > 30 kg/m^2).[56] There was no significant difference in subjective cure between BMI groups.

Studies, often with short- to mid-term follow-up have shown a trend towards favourable results and increased cure rates in patients with a lower BMI, although statistical significance was not reached.[57,58]

Secondary analysis of a randomised controlled trial evaluating mid-urethral slings reported that at 5 years, women who were not obese reported higher rates of objective and subjective cure, with a 76.7% subjective cure rate, compared with 53.6% in women who were obese (P = 0.002, RD 23.2%, 95% CI 8.0–38.3%).[59]

Operative outcomes are also negatively influenced by the severity of obesity, with patients who are morbidly obese being twice as likely to report failure following mid-urethral sling.[60] The incidence of UUI was comparable in both groups; however, bothersome symptoms were more likely to persist in women who were obese (58.9 versus 42.1%).

Since 2018 the use of mesh has been paused during a period of high-vigilance restriction. The use of mesh for stress UI (SUI) has been effectively halted, except in cases where there is no alternative and a delay is unacceptable,[61] and has been replaced by alternative SUI surgical treatments, such as urethral bulking and colposuspension.

Bladder neck suspension procedures have provided evidence of long-term efficacy, with success rates comparable to mid-urethral slings,[62] and most recent studies have reported on the efficacy and safety of laparoscopic colposuspension.[63] As synthetic mesh mid-urethral slings continue to face scrutiny, the Burch colposuspension, first described in 1961,[64] is becoming increasingly popular as a primary procedure for stress incontinence or for patients where urethral bulking has failed. However, data concerning the efficacy of Burch colposuspension in women who are overweight or obese, and the impact of BMI on treatment efficacy and longevity, are limited.

Onabotulinumtoxin A is the mainstay of treatment for refractory detrusor overactivity (DO), overactive bladder (OAB) and UUI, with reported success compared with placebo in several randomised controlled trials.[65-67] The most recent randomised trial reported on the higher risk of treatment failure and association with non-response (defined as ‘no change’ or ‘worse’) on the Patient Global Impression of Improvement (PGI-I) scale with increased BMI (OR 1.07, 95% CI 1.0–1.16, P = 0.065).[68]

Outcomes of anal incontinence interventions in women who are obese

Behavioural treatment that aims to reduce stool inconsistency is the primary treatment for AI. Various treatment alternatives have been proposed, including bowel training, biofeedback, anti-diarrhoeal drugs and bulk laxatives (in cases of chronic constipation). None of these methods have been evaluated in obese populations. Sphincteroplasty remains the cornerstone of treatment in cases of damaged anal sphincter. A study on 15 women who were obese and 64 women who were not obese, and followed-up for a median period of 64 months,[69] showed that although the risk of complications was comparable between the two groups, improvement was less evident in patients who were obese. Perianal bulking has shown promising results in patients who are obese and undergoing gastric bypass surgery, who are often affected by incontinence postoperatively.[70] However, robust data to support efficacy in the obese symptomatic populations are lacking, and considering the limited efficacy of bulking agents in the treatment of UI they cannot yet be recommended for the treatment of FI.

Impact of weight loss on incontinence

Urinary incontinence following weight loss

It has been noted that weight loss leads to statistically significant reductions in intravesical pressure, bladder-to-urethra pressure transmission during cough and urethral axial mobility.[71] Bump et al. demonstrated a significant reduction in incontinence episodes and need to use absorptive pads at 1 year after surgically induced weight loss.[71]

A randomised controlled trial by Subak et al. demonstrated similar findings.[72] Following a weight reduction programme, women were more likely to experience statistically significant improvements in continence and quality of life at the 6-month follow-up. A reduction in as little as 5–10% baseline weight could confer a 50% reduction in UI episodes, with benefits in both UUI and SUI. Urodynamics studies also showed a reduction in bladder pressure with weight loss, indicating that the higher bladder pressure in women who are overweight or obese is a contributor to UI.

A Cochrane review in 2015 aimed to determine the effectiveness of specific lifestyle interventions, including weight loss, on adult UI.[73] Four trials with a total of 4701 women reported on the effect of weight loss using a low-calorie diet and an exercise programme, compared with no treatment. All four trials reported that women allocated to the intervention group had a statistically significant reduction in weight from baseline compared with the control groups. There was evidence, albeit of 'low' quality, that weight loss programmes were associated with higher improvement rates based on women's self-report and also with higher cure and improvement rates based on quantifiable symptoms. The outcome measures of these studies were variable, including the number of weekly incontinence episodes, and only one small trial assessed the effect using disease-specific quality-of-life measures (Incontinence Impact Questionnaire, IIQ, and the Urogenital Distress Inventory, UDI). This trial showed that at the 3-month follow-up, women in the intervention group reported that UI had less adverse impact (median IIQ scores, 40 women in analysis, 37 versus 89, $P < 0.01$) and was less distressing (median UDI scores, 40 women in analysis, 104 versus 195, $P < 0.0001$), compared with the control group.

Despite this variation in outcome measures, the positive effect of weight loss was consistently reported, which strongly suggests that weight loss has a beneficial effect in treating UI.

Taking into account the modest success rates of behavioural interventions for weight loss, one may expect that the impact of surgical procedures would be more clear. A recent systematic review evaluated the impact of bariatric surgery on women with UI who were obese. Data from 33 cohort studies including 2910 women were included, with a median follow-up of 12 months. Improvement or resolution of any type of UI was reported in 56% (95% CI 48–63%), with larger reductions seen in UUI than in SUI after bariatric surgery. A significant reduction ($P < 0.001$) in symptom questionnaire scores was also reported, with UDI scores reduced by 13.4 points (95% CI 7.2–19.6).[74] However, the quality of evidence was graded as very low and 3% of patients reported the worsening or development of new-onset UI following bariatric surgery.

Another systematic review and meta-analysis measured the effect on incontinence-specific quality of life and incontinence cure rate following bariatric surgery.[75] Analysis of three studies including 3225 women showed that incontinence-specific quality-of-life scores were improved by 14% (weighted mean difference = −14.79; 95% CI =−18.47 to −11.11; I^2 = 87.1%), whereas the cure rates for any type of UI reached 59% (95% CI = 51–66%). Short-term follow-up and study heterogeneity were noted to be limitations to these data.

Anal incontinence following weight loss

By comparison, the impact of bariatric surgery on AI is less encouraging. A systematic review by Montenegro et al. reported the findings from 20 studies (3684 patients), which showed a modest relative risk reduction in FI episodes after bariatric surgery, although the finding was not significant (OR 0.80, 95% CI 0.53–1.21, $P = 0.29$).[76]

The hypothesis for a relationship between weight loss and a reduction in the number of FI episodes assumes that the stool inconsistency associated with obesity is the aetiological basis of the increased prevalence of FI.

A study that investigated pelvic floor dysfunction in 46 women who were obese reported that there was no significant difference in internal or external anal sphincter size or mean anorectal angle during squeeze and during defecation between the BMI groups: <35, 35–40 and >40 kg/m^2.[77] However, FI decreased significantly after bariatric surgery, from 23% preoperatively to 9.2% at the 6-month follow-up and 5.7% at the 12-month follow-up ($P = 0.001$).[77]

Similarly, a systematic review by Poylin et al. also demonstrated a reduction in AI after the Roux-en-Y procedure, although the link between bariatric surgery and diarrhoea was unclear.[78]

Conclusion

Obesity is linked to the development and severity of both UI and AI. Increased intra-abdominal pressure and chronic strain on the pelvic neuromusculature contributes to UI, and oxidative stress from visceral adipose tissue is likely to have a negative effect on the collagen and supportive structures of the pelvic floor, which confer continence. With regards to AI in individuals who are obese, stool consistency seems to be the main contributing factor rather than intra-abdominal pressure.

As a consequence, weight loss through lifestyle changes or bariatric surgery can confer significant improvements in UI, but the effect on AI is less pronounced.

Evidence concerning the surgical treatment of AI in the obese population is scarce and inadequate to inform clinical practice. Studies on urinary continence procedures in the obese population have not been extensive enough to evaluate different surgical interventions in women who are obese, and there is evidence for selected procedures only. Although transobturator tape has superior efficacy compared with single-incision tapes, obesity is associated with poorer long-term subjective and objective surgical outcomes. There is limited evidence regarding the outcomes of urethral bulking and colposuspension in women who are obese, and further research is needed into surgical treatments for AI.

Nevertheless, and in addition to its other numerous beneficial effects promoting overall wellbeing and longevity, weight loss should have a prominent place in treatment pathways for the management of UI and AI.

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Contribution to authorship

SXD: conception of idea, analysis of included articles, and review and editing of the article. JL: review of articles for inclusion, and drafting and editing the article. VP: involved in the conception of the idea, reviewing the included articles and contributed to the initial draft of the article. All authors accept responsibility for the article as published.

Details of ethics approval

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