HHS Public Access
Author manuscript
Int Urogynecol J Pelvic Floor Dysfunct. Author manuscript; available in PMC 2016 July 13.

Published in final edited form as:
Int Urogynecol J Pelvic Floor Dysfunct. 2009 March ; 20(3): 289–294. doi:10.1007/s00192-008-0765-x.

Prevalence and degree of bother from pelvic floor disorders in obese women

Emily L. Whitcomb,
Division of Urogynecology and Reconstructive Pelvic Surgery, Department of Reproductive Medicine, University of California, San Diego, CA, USA. 9350 Campus Point Drive, #0974, La Jolla, CA 92037, USA

Emily S. Lukacz,
Division of Urogynecology and Reconstructive Pelvic Surgery, Department of Reproductive Medicine, University of California, San Diego, CA, USA. 9350 Campus Point Drive, #0974, La Jolla, CA 92037, USA

Jean M. Lawrence,
Department of Research and Evaluation, Kaiser Permanente Southern California, 100 S. Los Robles, 2nd Floor, Pasadena, CA 91101, USA

Charles W. Nager, and
Division of Urogynecology and Reconstructive Pelvic Surgery, Department of Reproductive Medicine, University of California, San Diego, CA, USA. 9350 Campus Point Drive, #0974, La Jolla, CA 92037, USA

Karl M. Luber
Department of Female Pelvic Medicine and Reconstructive Surgery, Kaiser Permanente San Diego Medical Center, 4647 Zion Ave., San Diego, CA 92120, USA

Emily S. Lukacz: elukacz@ucsd.edu

Abstract

We aimed to determine the prevalence and bother from pelvic floor disorders (PFD) by obesity severity, hypothesizing that both would increase with higher degrees of obesity. We performed a secondary analysis of 1,155 females enrolled in an epidemiologic study that used a validated questionnaire to identify PFD. Prevalence and degree of bother were compared across three obesity groups. Logistic regression assessed the contribution of degree of obesity to the odds of having PFD. Prevalence of any PFD was highest in morbidly (57%) and severely (53%) obese compared to obese women (44%). Regression models demonstrated higher prevalence of pelvic organ prolapse, overactive bladder, stress urinary incontinence, and any PFD in morbidly compared to obese women and higher prevalence of stress urinary incontinence in severely obese compared to obese women. Degree of bother did not vary by degree of obesity. Prevalence of PFD increases with higher degrees of obesity.

Correspondence to: Emily S. Lukacz, elukacz@ucsd.edu.

Conflicts of interest None.

Keywords

Obesity; Pelvic floor disorders

Introduction

Obesity and pelvic floor disorders (PFD), including pelvic organ prolapse (POP), stress urinary incontinence (SUI), overactive bladder (OAB), and anal incontinence (AI), are increasingly common and have significant quality of life, health-related, and economic burden [1–4]. Prevalence estimates for both PFD and obesity exceed 30% of the adult population [5, 6]. In the US, the prevalence of obesity, defined as a body mass index (BMI) of ≥30 kg/m^2, was estimated by the National Health and Nutrition Examination Survey in 2005–2006 to be 34.3% of the adult population, representing an increase in prevalence of 50% over the past decade [5, 7]. Obesity has emerged as a risk factor for PFD [8–10] with numerous epidemiological studies describing the associations between obesity and the prevalence of urinary [10–15] and fecal incontinence [10, 14, 15]. In addition, weight loss, achieved both medically [16, 17] and surgically [18–20], has been shown to reduce incontinence in obese women. Despite the well-described associations between obesity and PFD, data on the prevalence and associated bother for the spectrum of PFD in obese women are relatively sparce.

Using data from the Kaiser Permanente Continence-Associated Risk Epidemiology Study (KP CARES), the objectives of this study were to determine the prevalence and degree of bother from PFD across three levels of obesity (obese, severely obese, and morbidly obese). Our hypotheses were that the prevalence of PFD and the degree of bother from PFD symptoms would increase with higher degrees of obesity.

Materials and methods

Data for these analyses were derived from KP CARES, the details of which have been previously published [21]. In brief, after approval by the Kaiser Permanente Southern California (KPSC) Institutional Review Board, random samples of 3,050 women in each of four age strata (25–39, 40–54, 55–69, and 70–84 years) were selected from the KPSC membership, a large prepaid managed health care plan that serves over three million residents in southern California. English and Spanish versions of the Epidemiology of Prolapse and Incontinence Questionnaire (EPIQ) were mailed with a cover letter describing the voluntary nature of the study, with a small gift card ($5) and a postcard to opt-out or request additional information. The Institutional Review Board waived written documentation of informed consent, which was assumed by survey completion. Reminder telephone calls and a second survey mailing were made to all nonrespondents. Data were collected from April 2004 through January 2005.

The EPIQ was developed and validated to ascertain the prevalence and risk factors for PFD in women from this racially and ethnically diverse community-dwelling population. Survey development, pilot testing, and survey methods have been described elsewhere [22]. Briefly, the EPIQ includes questions related to the presence or absence of POP, SUI, OAB, and AI based on current symptoms and associated degree of bother. For example, for SUI, respondents are asked: "Do you experience urine leakage related to activity, coughing, or sneezing?" For POP, respondents were asked: "Do you have a sensation that there is a bulge in your vagina or that something is falling out from your vagina?" Frequency or duration of symptoms was not queried. The EPIQ also includes demographic questions such as age, race and ethnicity, and marital status and questions about height, weight, smoking, chronic lifting, caffeine intake, obstetric history, menopause, hormone use, depression, diabetes, pulmonary disease, neurologic disease, and pelvic surgery. The questionnaire was validated using focus group testing, test–retest reliability testing, and construct validation in women with and without PFD using a priori clinical criteria, physical examination measures, and bowel and bladder diaries. The positive and negative predictive values for the detection of the specific conditions have been previously reported and are: 76% and 97% for POP, 88% and 87% for SUI, 77% and 90% for OAB, and 61% and 91% for AI, respectively [22].

The prevalence of each PFD was characterized by degree of obesity, and the associated current degree of bother for each PFD was assessed using a 100-mm visual analog scale (VAS) [23]. For each of the conditions, respondents were asked: "How much are you bothered by" followed by the particular symptom(s) that characterize the condition. For example, for SUI, respondents were asked: "How much are you bothered by urine leakage related to activity, coughing, or sneezing?" The prevalence of PFD and degree of bother were compared across three categories of obesity: obese ≥30 kg/m^2 and <35 kg/m^2, severely obese ≥35 kg/m^2 and <40 kg/m^2, and morbidly obese ≥40 kg/m^2. Chi-squared and Kruskal–Wallis analyses were used to compare the prevalence of each PFD by degree of obesity. Analysis of variance (ANOVA) was used to compare mean VAS scores across the three obesity groups. Partially adjusted regression models including age, mode of delivery, and parity were used to assess the relative impact of degree of obesity on the presence of each and any PFD. Comorbid conditions were compared across obesity groups using chi-squared tests of proportions. Those variables that were different across degrees of obesity were entered into multivariable logistic regression analyses, along with age, mode of delivery, and parity. These fully adjusted models were explored to confirm the independent contribution of increasing degree of obesity to the odds of having any PFD. Crude and adjusted odds ratios (OR) and 95% confidence intervals (CI) were reported for each and any PFD as the outcome. Associations with a two-sided p value <0.05 were considered significant. Statistical analyses were performed using SPSS 11.0 (SPSS Inc., Chicago, IL, USA). Power calculations and sample size were based on the primary study objectives to assess the prevalence of each PFD and identify the risk of mode of delivery on development of PFD [14]. However, post hoc power calculations were conducted for these analyses. The current sample size had greater than 99% power to detect the differences in prevalence of any one or more PFD identified between the three obesity groups.

Results

The mean age (±standard deviation) of the 1,155 obese women was 56.4±14.8 years, and the mean BMI was 35.4±5.3 kg/m^2. Six hundred and ninety women were obese (mean BMI 32 kg/m^2); 284 women were severely obese (mean BMI 37 kg/m^2), and 181 women were morbidly obese (mean BMI 45 kg/m^2). The race–ethnicity of these women was 58% non-

Hispanic white, 23% Hispanic, 14% African–American, 3% Asian–Pacific Islander, and 2% other or unknown race. The demographic and clinical characteristics of the cohort are shown in Table 1. The prevalence (95% CI) of PFD in all women with a BMI ≥30 kg/m^2 was: POP 9% (7–10%), SUI 24% (22–26%), OAB 22% (19–24%), AI 29% (27–32%), and any one or more PFD 46% (43–49%). In comparison, the prevalence of PFD in the 3,238 nonobese women (BMI< 30 kg/m^2) from the original study population was: POP 6% (5–7%), SUI 12% (10–13%), OAB 10% (9–11%), AI 22% (21–24%), and any one or more PFD 32% (30–34%; all p values <0.05 when compared to obese women, data not shown).

The prevalence of any one or more PFD was highest in morbidly (57%) and severely obese (53%) women compared to obese women (44%; Table 2). A significant increase in the prevalence of POP and SUI was found in morbidly obese compared to obese women and SUI in severely obese compared to obese women. Although not statistically significant, a trend toward increasing prevalence of OAB and AI was also seen with higher degree of obesity. There were no significant differences in the prevalence of any individual or combined PFD between morbidly and severely obese women. Degree of bother related to each PFD did not vary significantly by degree of obesity (Table 2). Mean VAS scores for overactive bladder only were significantly higher in women with a BMI≥30 kg/m^2 compared to women with BMI<30 kg/m^2, p<0.01 (data not shown).

Partially adjusted logistic regression models controlling only for age, mode of delivery, and parity demonstrated increased odds of POP, OAB, SUI, and any one or more PFD in the morbidly obese compared to obese women and increased odds only for SUI in the severely obese compared to obese women (Table 3). Degree of obesity was not associated with AI. In an analysis of covariates that could potentially be associated with both obesity and PFD (parity, mode of delivery, race, recurrent urinary tract infection (UTI), diabetes, depression, lung disease–asthma, neurologic disease, hormone–menopause status, and pelvic surgery), degree of obesity was significantly associated with parity, recurrent UTI, diabetes, depression, and lung disease–asthma (data not shown). Fully adjusted multivariable logistic regression models controlling for age, parity, mode of delivery, and the above confounders determined that morbidly obese women had the highest odds of having any one or more PFD compared to obese women (OR 1.56, 1.06–2.29), and severely obese women also had higher odds of having any PFD compared to obese women (1.46, 1.06–2.02). In fully adjusted models, any PFD was also significantly associated with age (1.85, CI 1.16–2.95 for ages 70–84 years compared to ages 25–39 years), recurrent UTI (OR 2.20, CI 1.54–3.14), and depression (OR 1.90, CI 1.36–2.64). Interestingly, mode of delivery (OR 1.08, CI 0.67–1.73 for vaginally parous compared to nulliparous), parity (OR 1.12, CI 1.00–1.25), and other comorbid conditions were not associated with presence of any PFD while controlling for degree of obesity.

Discussion

In this sample of women from a managed health care population in which obese women had been shown to have a significantly higher prevalence of PFD compared to nonobese women [15], there was a consistent trend toward increasing prevalence of PFD with increasing degree of obesity. This trend was significant for POP, SUI, and any one or more PFD and was nearly significant for OAB and AI. When we adjusted for covariates, we found that morbidly obese women had the highest odds of having POP, OAB, and any one or more PFD compared to obese women, whereas severely obese women had the highest odds of having SUI compared to obese women. Interestingly, mode of delivery and parity were not associated with any one or more PFD in adjusted models, suggesting the primary importance of the modifiable risk factor of obesity. Associated bother for any of the PFD did not increase with the degree of obesity.

Obesity has emerged as a risk factor for PFD [8–10] with numerous epidemiological studies describing the impact of obesity on the prevalence of urinary [10–15] and fecal incontinence [10, 14, 15]. Scant data exist on the effects of obesity on POP or OAB. In a cross-sectional study undertaken to understand the possible role of obesity in the etiology of adult female incontinence, Foldspang and Mommsen [24] reported a positive association between increasing BMI and increasing prevalence of urinary incontinence. In addition, Moller and colleagues [25] reported a nearly linear relationship between increasing BMI and the the presence of urinary incontinence. The present finding of increasing prevalence of PFD, particularly SUI, with higher degree of obesity confirms these findings. Others have also identified a ceiling effect where additional weight over 40 kg/m^2 had no impact on the prevalence of PFD [14]. Our findings suggest that there may be an even lower threshold effect on prevalence of PFD above a BMI of 35 kg/m^2 because no significant differences in prevalence could be found between the severely obese and morbidly obese groups for any PFD.

Investigations in cohorts of women undergoing surgical weight reduction have shown consistent reductions in incontinence. Richter and colleagues [14] reported a prevalence of 66.9% for urinary incontinence, predominantly mixed stress, and urge incontinence and 32% for AI in a cohort of 180 morbidly obese women (mean BMI 49.5 kg/m^2) undergoing evaluation for laparoscopic weight loss surgery. The higher prevalence of urinary incontinence in their study compared to ours may be attributable to the higher mean BMI and degree of comorbid conditions (including PFD) prompting care seeking in their study; however, the prevalence of AI in that study was comparable to our own. Wasserberg and colleagues [26] also examined the prevalence of fecal incontinence in morbidly obese women (mean BMI 49.3 kg/m^2) attending a bariatric surgery seminar and found that 63% reported fecal incontinence, a prevalence estimate that well exceeds our own and may be due to the care-seeking population studied. In addition, weight loss achieved both medically and surgically has been shown to improve incontinence in obese women [16–20]. Burgio and colleagues [19] found the prevalence of urinary incontinence and fecal incontinence decreased after laparoscopic bariatric surgery in morbidly obese women. Kuruba and colleagues [18] found that surgically induced weight loss resulted in an 82% improvement or resolution of urinary incontinence.

Other studies have correlated BMI with severity of urinary incontinence [8, 18–20]. In the present study, severity of PFD was assessed with use of VAS bother scores. Associated bother for any of the PFD did not increase with the degree of obesity. This finding supports previous studies that have found lower impact, as measured by IIQ-7 and UDI-6 scores, of urinary incontinence in morbidly obese women compared to those seeking urogynecologic care, in spite of matching for urinary incontinence severity [27]. Similarly, in an attempt to determine the impact of excess body mass on the prevalence of PFD, Wasserberg and colleagues [28] demonstrated significantly higher scores on two validated condition-specific quality-of-life questionnaires assessing impact in morbidly obese females compared to age-matched nonobese controls; however, additional increases in BMI>35 kg/m^2 did not show increased adverse impacts on pelvic floor symptoms. Our findings confirm those of others and suggest that a threshold effect on bother above a BMI≥30 kg/m^2 may be operative in this population. That is, the threshold bother effect of urinary incontinence and other PFD may vary depending on the population studied and may be overwhelmed by other comorbidities known to be associated with obesity.

The strengths of this study include its large racially and ethnically diverse cohort of community-dwelling women distributed across a wide range of both age and BMI. The use of a validated instrument that underwent psychometric and criterion validation to identify the presence of a variety of PFD including not only SUI and AI, but also POP and OAB, lends credence to our findings. The use of the validated questionnaire, inclusion of racially diverse sample with a broad range of BMI, and the large sample size distinguish our findings. The limitations of our study are that BMI was based on self-report and thereby prone to inaccuracy in reporting. However, the correlation between self-reported and measured BMI has been shown to be very high (0.90–0.95) in the National Health and Nutrition Education Study III, and we have no reason to believe our population is any different [29]. Additionally, our response rate was lower than anticipated despite considerable effort to increase it. Other limitations of the present study are those common to population-based surveys, such as response bias that may have overestimated or underestimated degree of bother related to PFD, and the potential inaccuracy of self-reported pelvic floor symptoms on a questionnaire. Limitations of the present findings also include the inability to determine causal relationships. However, the pathophysiologic basis posited for the relationship between obesity and PFD lies in the correlation between BMI and intra-abdominal pressure, suggesting that obesity may stress the pelvic floor secondary to a chronic state of increased pressure [30]. In addition, neurologic and neuromuscular disease may contribute to pelvic floor and urethral dysfunction in obese women. Finally, our findings may not be generalizable to an uninsured population.

Given the increasing prevalence of obesity in the population, the present work advances our understanding of the epidemiology of all PFD in an obese, severely obese, and morbidly obese population. These data help to establish prevalence estimates and may inform future studies evaluating the impact of interventions such as major weight reduction on the prevalence of PFD. Our findings delineate the burden of a spectrum of PFD in obese, severely obese, and morbidly obese women and provide a comparison against which postintervention (i.e., bariatric surgery or major weight reduction achieved by alternate means) prevalence may ultimately be compared. Finally, health care providers caring for obese women should recognize the coexistence of obesity and pelvic floor dysfunction, and future studies should evaluate whether obesity-associated PFD can be reduced through successful weight reduction interventions.

Acknowledgments

Funding NICHD #R01 HD4113-01A1

Abbreviations

POP	pelvic organ prolapse
SUI	stress urinary incontinence
OAB	overactive bladder
AI	anal incontinence
PFD	pelvic floor disorders
BMI	body mass index
KP CARES	Kaiser Permanente Continence-Associated Risk Epidemiology Study
EPIQ	Epidemiology of Prolapse and Incontinence Questionnaire
VAS	visual analog scale

References

1. Subak LL, Brown JS, Kraus SR, Brubaker L, Lin F, Richter HE, et al. The “costs” of urinary incontinence for women. Obstet Gynecol. 2006; 107:908–916. [PubMed: 16582131]
2. Grimby A, Milsom I, Molander U, Wiklund I, Ekelund P. The influence of urinary incontinence on the quality of life of elderly women. Age Ageing. 1993; 22:82–89. [PubMed: 8470564]
3. Norton PA, MacDonald LD, Sedgwick PM, Stanton SL. Distress and delay associated with urinary incontinence, frequency, and urgency in women. BMJ. 1988; 297:1187–1189. [PubMed: 3144344]
4. Varma MG, Brown JS, Creasman JM, Thom DH, Van Den Eeden SK, Beattie MS, et al. Fecal incontinence in females older than aged 40 years: who is at risk? Dis Colon Rectum. 2006; 49:841–851. [PubMed: 16741640]
5. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. Jama. 2006; 295:1549–1555. [PubMed: 16595758]
6. Lawrence JM, Lukacz ES, Nager CW, Hsu JW, Luber KM. Prevalence and co-occurrence of pelvic floor disorders in community-dwelling women. Obstet Gynecol. 2008; 111:678–685. [PubMed: 18310371]
7. Ogden, CL.; Carroll, MD.; McDowell, MA.; Flegal, KM. Obesity among adults in the United States-no change since 2003–2004. National Center for Health Statistics; Hyattsville: 2007. NCHS data brief no 1
8. Richter HE, Burgio KL, Brubaker L, Moalli P, Markland AD, Mallet V, et al. Factors associated with incontinence frequency in a surgical cohort of stress incontinent women. Am J Obstet Gynecol. 2005; 193:2088–2093. [PubMed: 16325621]
9. Kapoor DS, Davila GW, Rosenthal RJ, Ghoniem GM. Pelvic floor dysfunction in morbidly obese women: pilot study. Obes Res. 2004; 12:1104–1107. [PubMed: 15292474]
10. Uustal Fornell E, Wingren G, Kjolhede P. Factors associated with pelvic floor dysfunction with emphasis on urinary and fecal incontinence and genital prolapse: an epidemiological study. Acta Obstet Gynecol Scand. 2004; 83:383–389. [PubMed: 15005787]
11. Dwyer PL, Lee ET, Hay DM. Obesity and urinary incontinence in women. Br J Obstet Gynaecol. 1988; 95:91–96. [PubMed: 3342213]

12. Roe B, Doll H. Lifestyle factors and continence status: comparison of self-report data from a postal survey in England. J Wound Ostomy Continence Nurs. 1999; 26:312–313. 315–319. [PubMed: 10865616]
13. Yarnell JW, Voyle GJ, Sweetnam PM, Milbank J, Richards CJ, Stephenson TP. Factors associated with urinary incontinence in women. J Epidemiol Community Health. 1982; 36:58–63. [PubMed: 7069357]
14. Richter HE, Burgio KL, Clements RH, Goode PS, Redden DT, Varner RE. Urinary and anal incontinence in morbidly obese women considering weight loss surgery. Obstet Gynecol. 2005; 106:1272–1277. [PubMed: 16319252]
15. Lawrence JM, Lukacz ES, Liu IL, Nager CW, Luber KM. Pelvic floor disorders, diabetes, and obesity in women: findings from the Kaiser Permanente Continence Associated Risk Epidemiology Study. Diabetes Care. 2007; 30:2536–2541. [PubMed: 17620443]
16. Subak LL, Johnson C, Whitcomb E, Boban D, Saxton J, Brown JS. Does weight loss improve incontinence in moderately obese women? Int Urogynecol J Pelvic Floor Dysfunct. 2002; 13:40–43. [PubMed: 11999205]
17. Subak LL, Whitcomb E, Shen H, Saxton J, Vittinghoff E, Brown JS. Weight loss: a novel and effective treatment for urinary incontinence. J Urol. 2005; 174:190–195. [PubMed: 15947625]
18. Kuruba R, Almahmeed T, Martinez F, Torrella TA, Haines K, Nelson LG, et al. Bariatric surgery improves urinary incontinence in morbidly obese individuals. Surg Obes Relat Dis. 2007; 3:586–590. discussion 590–591. [PubMed: 17950043]
19. Burgio KL, Richter HE, Clements RH, Redden DT, Goode PS. Changes in urinary and fecal incontinence symptoms with weight loss surgery in morbidly obese women. Obstet Gynecol. 2007; 110:1034–1040. [PubMed: 17978117]
20. Bump RC, Sugerman HJ, Fantl JA, McClish DK. Obesity and lower urinary tract function in women: effect of surgically induced weight loss. Am J Obstet Gynecol. 1992; 167:392–397. discussion 397–9. [PubMed: 1497041]
21. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. Obstet Gynecol. 2006; 107:1253–1260. [PubMed: 16738149]
22. Lukacz ES, Lawrence JM, Buckwalter JM, Burchette RJ, Nager CW, Luber KM. Epidemiology of prolapse and incontinence questionnaire: validation of a new epidemiologic survey. Int Urogynecol J Pelvic Floor Dysfunct. 2005; 16(4):272–284. [PubMed: 15856132]
23. Lukacz ES, Lawrence JM, Burchette RJ, Luber KM, Nager CW, Buckwalter JM. The use of visual analog scale in urogynecologic research: a psychometric evaluation. Am J Obstet Gynecol. 2004; 191(1):165–170. [PubMed: 15295359]
24. Foldspang A, Mommsen S. Overweight and urinary incontinence in women. Ugeskr Laeger. 1995; 157:5848–5851. [PubMed: 7483064]
25. Moller LM, Lose G, Jorgensen T. Risk factors of lower urinary tract symptoms in women aged 40–60 years. Ugeskr Laeger. 2001; 163:6598–6601. [PubMed: 11760543]
26. Wasserberg N, Haney M, Petrone P, Crookes P, Rosca J, Ritter M, et al. Fecal incontinence among morbid obese women seeking for weight loss surgery: an underappreciated association with adverse impact on quality of life. Int J Colorectal Dis. 2008; 23:493–497. [PubMed: 18228028]
27. Gerten KA, Richter HE, Burgio KL, Wheeler TL, Goode PS, Redden DT. Impact of urinary incontinence in morbidly obese women versus women seeking urogynecologic care. Urology. 2007; 70:1082–1085. [PubMed: 18158022]
28. Wasserberg N, Haney M, Petrone P, Ritter M, Emami C, Rosca J, et al. Morbid obesity adversely impacts pelvic floor function in females seeking attention for weight loss surgery. Dis Colon Rectum. 2007; 50:2096–2103. [PubMed: 17899277]
29. McAdams MA, Van Dam RM, Hu FB. Comparison of self-reported and measured BMI as correlates of disease markers in US adults. Obesity (Silver Spring). 2007; 15:188–196. [PubMed: 17228047]
30. Noblett KL, Jensen JK, Ostergard DR. The relationship of body mass index to intra-abdominal pressure as measured by multichannel cystometry. Int Urogynecol J Pelvic Floor Dysfunct. 1997; 8:323–326. [PubMed: 9609328]

Table 1

Demographic and clinical characteristics of obese (BMI≥ 30 kg/m^2) women from KP CARES (*n*=1,155)

Characteristic	*N*=1,155, *n* (%)
Age	56.4±14.8
Race–ethnicity	
Caucasian	676 (59)
Hispanic	270 (23)
African–American	159 (14)
Asian–Pacific Islander	32 (3)
Other	18 (2)
Body mass index (kg/m^2) mean, SD	35.4±5.3
Body mass index category	
Obese ≥30 to <35 kg/m^2	690 (60)
Severely obese ≥35 to <40 kg/m^2	284 (25)
Morbidly obese ≥40 kg/m^2	181 (16)
Parity (mean)	2.3±1.7
Delivery mode	
Vaginally parous	784/1,091 (72)
Cesarean birth only	119/1,091 (11)
Nulliparous	188/1,091 (17)
Pelvic surgery	359/1,038 (35)
Depression	269/1,086 (25)
Diabetes	259 (22)
Neurologic disease	35/1,060 (3)
Lung disease or asthma	188/1,089 (17)
Recurrent urinary tract infections	218/1,090 (20)
Hormone and menopausal status	
Premenopausal	294/1,060 (28)
Post—no hormone therapy	310/1,060 (29)
Post—past hormone therapy	312/1,060 (29)
Post—current hormone therapy	144/1,060 (14)
Smoking	
Nonsmoker	695/1,139 (61)
Past smoker	366/1,139 (32)
Current	78/1,139 (7)
Chronic lifting	447/1,105 (40)
Caffeine use	691/1,147 (60)

Means and percentages are based on all 1,155 women unless otherwise noted. SD = standard deviation.

Table 2

Prevalence and 95% confidence intervals and degree of bother (VAS, mm±SD) for each and any pelvic floor disorder by degree of obesity (*n*=1,155)

Condition	Obese, BMI 30–34.9 kg/m^2, *N*=690	Severely obese BMI 35–39.9 kg/m^2, *N*=284	Morbidly obese BMI≥ 40 kg/m^2, *N*=181	*p*
Prolapse, *N*, % (95% CI)	48, 7.0 (5.3–9.1)	28, 9.9 (6.9–13.9)	23, 12.7 (8.6–18.4)[a]	0.040[b]
VAS	73.2±19.9	72.5±20.1	66.8±18.6	0.422[c]
Stress incontinence, *N*, % (95% CI)	135, 19.7 (16.9–22.9)	91, 32.3 (27.2–38.1)[a]	54, 30.2% (23.9–37.3)[a]	<0.001[c]
VAS	66.3±14.8	65.1±14.2	64.5±14.2	0.699[b]
Overactive bladder, *N*, % (95% CI)	136, 20.2 (17.3–23.4)	71, 26.1 (21.2–31.6)	46, 26.7 (20.7–33.8)	0.054[c]
VAS	78.8±11.4	76.8±10.8	80.5±12.1	0.216[b]
Anal incontinence, *N*, % (95% CI)	188, 27.2% (24.1–30.7)	93, 32.7% (27.6–38.4)	59, 32.6% (26.2–39.7)	0.178[c]
VAS	42.0±17.9	42.4±17.9	41.5±13.6	0.955[b]
Any PFD, *N*, % (95% CI)	292, 44.3 (40.6–48.1)	143, 52.6 (46.6–58.4)[a]	96, 56.8 (49.3–64.0)[a]	0.004[c]

Percentage may vary according to missing data

PFD pelvic floor disorder, *BMI* body mass index, *VAS* visual analog scale, *CI* confidence interval

[a] Mann–Whitney test ($p<0.05$ compared to obese women)

[b] ANOVA between visual analog scale means for obese, superobese, and morbidly obese women

[c] Kruskal–Wallis test

Table 3

Crude and adjusted odds ratios (95% CI) for the associations between degree of obesity (obese, severely obese, and morbidly obese) and each and any pelvic floor disorder

	POP	**OAB**	**SUI**	**AI**	**Any PFD**
Crude OR (95% CI)					
Obese (*N*=690)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Severe (*N*=284)	1.46 (0.89–2.37)	1.38 (0.99–1.92)	1.93 (1.41–2.64)	1.29 (0.95–1.75)	1.38 (1.04–1.84)
Morbidly (*N*=181)	1.95 (1.15–3.31)	1.48 (1.01–2.17)	1.79 (1.24–2.59)	1.32 (0.93–1.89)	1.67 (1.19–2.35)
Adjusted OR (95% CI)					
Obese (*N*=690)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
Severe (*N*=284)	1.55 (0.92–2.60)	1.40 (0.98–1.99)	1.99 (1.43–2.78)	1.32 (0.96–1.82)	1.43 (1.05–1.93)
Morbidly (*N*=181)	2.09 (1.18–3.68)	1.66 (1.11–2.50)	1.87 (1.26–2.76)	1.39 (0.96–2.02)	1.75 (1.22–2.51)

All models are adjusted for age, mode of delivery, and parity. Any pelvic floor disorder = any one or more of the four pelvic floor disorders.

Obese: BMI 30–34.9 kg/m^2, severe obesity: BMI 35–39.9 kg/m^2, and morbid obesity: BMI≥40 kg/m^2

POP pelvic organ prolapse, *OAB* overactive bladder, *SUI* stress urinary incontinence, *AI* anal incontinence