Pelvic Floor Disorders, Diabetes, and Obesity in Women

Findings from the Kaiser Permanente Continence Associated Risk Epidemiology Study

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OBJECTIVE — We examined associations between obesity and diabetes and female pelvic floor disorders (PFDs), stress urinary incontinence (SUI), overactive bladder (OAB), and anal incontinence (AI) in community-dwelling women.

RESEARCH DESIGN AND METHODS — Women were screened for PFD using a validated mailed survey. Diabetes status, glycemic control, and diabetes treatment were extracted from clinical databases, while other risk factors for PFDs were obtained through self-report. Women were categorized hierarchically as nonobese/nondiabetic (reference), nonobese/diabetic, obese/nondiabetic, and obese/diabetic.

RESULTS — Of 3,962 women, 393 (10%) had diabetes. In unadjusted analyses, women with diabetes and women who were obese had greater odds of having PFDs. Among women with diabetes, being obese was associated with SUI and OAB. After adjusting for confounders, we found that obese/diabetic women were at the highest likelihood of having SUI (odds ratio 3.67 [95% CI 2.48–5.43]) and AI (2.09 [1.48–2.97]). The odds of having OAB among obese women was the same for obese/diabetic women (2.97 [2.08–4.36]) and obese/nondiabetic women (2.93 [2.33–3.68]). Nonobese/diabetic women had higher odds of SUI (1.90 [1.15–3.11]) but did not differ significantly in their OAB (1.45 [0.88–2.38]) and AI (1.33 [0.89–2.00]) prevalence from nonobese/nondiabetic women.

CONCLUSIONS — Given the impaired quality of life experienced by women with PFDs, health care providers should counsel women that obesity and diabetes may be independent modifiable risk factors for PFDs.

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Diabetes, obesity, and incontinence are all common health problems for women in the U.S. It has been estimated that 9.7 million, or 8.8%, of all women aged ≥20 years had diabetes in 2005 (1), while almost 50% may experience urinary incontinence in their lifetime (2). In 2003–2004, 28.6% of women were overweight and 33.2% were obese (3). Urinary incontinence alone accounts for the expenditure of up to 19.5 billion dollars annually in the U.S. (4) and can have a significant impact on the quality of womens' lives (5).

Studies (6–12) have demonstrated the association between urinary incontinence and diabetes, and some (11,12) have found that women who used insulin were more likely to be incontinent than women with diabetes who did not require insulin, but the mechanisms are unclear. It has been suggested that the most likely reason for the increase in risk is microvascular compromise, leading to damage to the urethral sphincter mechanism and bladder sensitivity, and that stricter glycemic control may reduce the risk or severity of urinary incontinence (13). Studies (14,15) of the relationship between anal incontinence and diabetes have had conflicting results.

Strong associations between obesity and both urinary and fecal incontinence have been reported (16–24). The pathophysiologic basis posited for this relationship lies in the significant correlation between BMI and intra-abdominal pressure, suggesting that obesity may stress the pelvic floor secondary to a chronic state of increased pressure (25). Weight loss has been shown to improve incontinence in obese women (26–28).

In this secondary analysis of data from the KP CARES (Kaiser Permanente Continence Associated Risk Epidemiology Study) study, we examined associations between female pelvic floor disorders (PFDs) (stress urinary incontinence [SUI], overactive bladder [OAB], and anal incontinence [AI]) and diabetes and obesity. Pelvic organ prolapse was excluded from these analyses due to insufficient power to assess the associations of interest for this condition. We sought to evaluate the relative importance of the associations between diabetes and obesity in their contributions to PFDs.

RESEARCH DESIGN AND METHODS

Kaiser Permanente is a large, prepaid, managed health care plan that serves >3 million residents in southern California. The Epidemiology of Prolapse and Incontinence Questionnaire (EPIQ) was developed to assess the prevalence of PFDs in a sample of women from this racially and ethnically diverse population. Survey development, pilot testing, and survey methods have been described

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Abbreviations: AI, anal incontinence; EPIQ, Epidemiology of Prolapse and Incontinence Questionnaire; KPSC, Kaiser Permanente Southern California; PFD, pelvic floor disorder; SUI, stress urinary incontinence; OAB, overactive bladder.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

elsewhere (29–31). Briefly, the EPIQ was developed and validated in English and Spanish to assess the presence or absence of AI, OAB, SUI, and pelvic organ prolapse in a community-dwelling population.

After approval by the institutional review board, 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 Kaiser Permanente Southern California (KPSC) membership who had an address on file with the health plan. Surveys in English and Spanish were mailed with a cover letter, small incentive, and postcard to opt-out or request additional information, followed by a second survey mailing, a reminder telephone call, and a third survey mailing to women in the youngest age strata (31). Of 12,200 surveys mailed, 4,458 (37%) were returned. Data were collected from April 2004 through January 2005

Assessment of PFDs

Women were screened for PFDs based on their responses to stem questions plus their degrees of bother, as indicated on a visual analog scale. Positive and negative predictive values and 95% CIs for the detection of specific PFDs were 88% (75–95) and 87% (76–93) for SUI, 77% (59–88) and 90% (81–95) for OAB, and 61% (48–73) and 91% (80–96) for AI, respectively. AI included flatal, solid, and/or liquid incontinence (30).

Assessment of diabetes, treatment, and complications

We linked survey respondents to the KPSC Diabetes Case Identification Database, which uses an algorithm to identify members who have a high probability of having diabetes (32,33) based on at least one of the following criteria: 250.XX ICD-9 hospital diagnosis, a prescription for insulin or other oral hypoglycemic agents, A1C ≥6.7%, or a fructosamine test result ≥280 μmol/l. Women with gestational diabetes (ICD-9 code 648.8) and no other criteria were not included. For this analysis, women who were identified as having diabetes based only on the A1C threshold had to meet or exceed 7.0% to increase the sensitivity of the algorithm. We assumed that the majority of these women have type 2 diabetes, as it comprises 85–95% of all adults with diabetes (34).

To characterize the women with diabetes, information about current treatment (insulin and/or oral hypoglycemic agents), based on the most recent prescription(s) filled before survey completion and the results of the A1C measured closest to the time of survey completion (±6 months), were extracted from the pharmacy and laboratory databases, respectively. All laboratory tests were conducted at a single laboratory operated by KPSC.

Self-reported variables

Age was calculated in completed years on the date of survey completion, and BMI was calculated as weight in kilograms divided by the square of height in meters and dichotomized into nonobese (<30 kg/m^2) or obese (≥30 kg/m^2). Smoking was categorized as never smoked, past smoker, or current smoker. Chronic lifting was defined as repetitive lifting of >9 kg regularly for >1 year. Caffeine consumption was defined as more than one cup of caffeinated beverage per day. Presence or absence of neurological disease, lung disease or asthma, history of depression, hysterectomy, menopause status (yes/no/don't know), and hormone exposure (never/past/present) were assessed using survey data.

To adjust for the known associations between pregnancy, mode of delivery, and PFD as previously described (31), we defined the nulliparous group as those women who had never been pregnant or only delivered a baby ≤2 kg. The cesarean birth group was defined as having been delivered by one or more cesarean births and no vaginal births exceeding 2 kg. Vaginally parous women were defined as having one or more vaginal deliveries exceeding 2 kg birth weight regardless of history of cesarean births. Parity was modeled as a continuous variable.

Statistical analysis

Of 4,458 EPIQ surveys returned, we excluded women sequentially for the following reasons: insufficient data to categorize into one of the three birth groups (n = 289), insufficient information to assess at least one of the PFDs (n = 66), and insufficient information to calculate BMI (n = 141), for a final sample size of 3,962 subjects.

Statistical analyses were performed with SAS version 8.02 (SAS Institute, Cary, NC). Power and sample size calculations were based on the primary study objectives to assess the prevalence of each PFD and to identify the risk of vaginal delivery compared with cesarean births (31). We assessed the differences between groups of women using χ^2 tests for categorical variables and Student's t tests for continuous variables.

Each PFD (SUI, OAB, or AI) was expressed dichotomously as "present" or "absent." Women for whom we did not have information to assess presence or absence were excluded from the models for that outcome. Among women with information to assess the presence or absence of at least one of these PFDs, we created a summary variable labeled "any PFD." Significance was evaluated using a two-sided P value of <0.05. Logistic regression analysis was used to calculate the odds ratios and 95% CIs for the associations between diabetes and obesity and each and any PFD.

Multiple logistic regression models were constructed for all women in the study sample. We assessed the contributions of diabetes and obesity to the likelihood of having each and any PFD after controlling for other known risk factors. Women were categorized hierarchically as nonobese/nondiabetic (reference), nonobese/diabetic, obese/nondiabetic, and obese/diabetic.

Once all of the variables were entered into the model, we removed covariates that were no longer significant in the multivariate model and had no impact on the primary variable of interest except for age (modeled as a continuous variable), race/ethnicity, mode of delivery, and parity, which remained in every model.

RESULTS

Characteristics of the study population by diabetes status

The median age of the women studied was 56.6 years, and the racial/ethnic distribution was 62% white, 19% Hispanic, 10% black, 8% Asian/Pacific Islanders, and 1% other or unknown race (Table 1). Ten percent (n = 393) of the women in the sample had diabetes. Compared with women without diabetes, we found that women with diabetes were significantly more likely to be older, African American or Hispanic, obese, parous, postmenopausal, and to have had a hysterectomy, a history of depression, a neurological condition, or lung disease. The prevalence of the PFDs was 15% SUI, 13% OAB, and 25% AI, and 35% had any PFD (Table 2).

Prevalence of PFDs among women with diabetes

Women with diabetes were significantly more likely to have each or any PFD than women without diabetes (Table 2). Of the

Table 1—Characteristics of 3,962 female survey respondents aged 25–84 years with and without diabetes

	All women	Nondiabetic women	Diabetic women	*P* value
n	3,962	3,569	393	
Age (years)	56.6 ± 15.8	55.8 ± 15.9	64.4 ± 12.5	<0.0001
Race/ethnicity				<0.005
Non-Hispanic white	2,444 (61.7)	2,227 (62.4)	217 (55.2)	
Hispanic	760 (19.2)	674 (18.9)	86 (21.9)	
Black	382 (9.6)	327 (9.2)	55 (13.4)	
Asian/Pacific Islander	323 (8.2)	298 (8.3)	27 (6.9)	
Other/unknown race	53 (1.3)	45 (1.3)	8 (2.0)	
BMI	27.8 ± 6.2	26.9 ± 5.9	32.1 ± 7.3	<0.0001
BMI category				<0.0001
Average (<25.0 kg/m^2)	1,643 (41.5)	1,586 (44.4)	57 (14.5)	
Overweight (25.0–29.9 kg/m^2)	1,229 (31.0)	1,114 (31.2)	115 (29.3)	
Obese (≥30 kg/m^2)	1,090 (27.5)	869 (24.4)	221 (56.2)	
Mode of delivery				0.0057
Nulliparous	755 (19.1)	702 (19.7)	53 (13.5)	
Any vaginal birth	2,837 (71.6)	2,543 (71.3)	294 (74.8)	
Cesarean births only	370 (9.3)	324 (9.1)	46 (11.7)	
Parity	2.1 ± 1.6	2.1 ± 1.6	2.6 ± 1.9	<0.0001
Postmenopausal	2,611 (66.0)	2,275 (63.9)	336 (85.5)	<0.0001
Hormone use				NS
None	2,101 (53.8)	1,900 (53.9)	201 (53.0)	
Past	1,234 (31.6)	1,108 (31.4)	126 (33.3)	
Current	572 (14.6)	520 (14.7)	52 (13.7)	
Hysterectomy	1,104 (28.0)	956 (26.9)	148 (37.9)	<0.0001
Cigarette smoker				<0.0005
Never	2,403 (61.4)	2,181 (61.9)	222 (57.2)	
Past	1,150 (29.4)	1,005 (28.5)	145 (37.4)	
Current	360 (9.2)	339 (9.6)	21 (5.4)	
Any caffeine use	2,205 (56.0)	1,979 (55.8)	226 (57.7)	NS
History of depression	756 (20.2)	663 (19.4)	93 (28.0)	<0.0005
Neurological disease	96 (2.6)	74 (2.2)	22 (6.8)	<0.0001
Lung disease or asthma	512 (13.6)	433 (12.6)	79 (23.3)	<0.0001

Data are means ± SD or *n* (%). Women with missing data are excluded from these analyses. NS, not significant.

women with diabetes, over half (56%) were obese; 17% were on insulin, 63% were treated with oral hypoglycemic agents only, and 20% were not on any diabetes medications. Over two-thirds (n = 271) had an A1C test in the 6 months before or after their survey completion, with a mean value of 7.0%. Of these women, 24% were in borderline control (7.0–8.5%) and 12% were in poor control (>8.5%). Women with diabetes were 90% more likely to have SUI or OAB, 50% were more likely to have AI, and 68% were more likely to have any PFD than women without diabetes (Table 3).

Women with obesity and prevalence of PFDs

Obese women were over twice as likely to experience SUI and OAB, >40% were more likely to have AI, and 92% more likely to have any PFD than women who were not obese (Table 3). When we restricted our analysis to women with diabetes, as shown at the bottom of Table 3, we found that being obese was positively associated with all conditions, but the relationship with AI was not significant.

Other risk factors associated with PFDs under study

When we examined the associations between other common risk factors for PFDs (shown in Table 1) and each and any PFD, we found that age, race/ethnicity, smoking status, mode of delivery, parity, hormone use, menopause, previous hysterectomy, history of depression, neurological disease, lung disease, and caffeine consumption were significantly associated with each and any PFD, with the following exception: caffeine consumption was not associated with OAB (data not shown).

Unadjusted and adjusted odds ratios for contributions of diabetes and obesity

When diabetes and obesity were combined hierarchically into a four-category exposure variable (nonobese/nondiabetic [reference], nonobese/diabetic, obese/nondiabetic, and obese/diabetic), we found that the unadjusted odds of having SUI, OAB, AI, or any PFD progressively increased with each category (Table 3). There was no statistical interaction between having diabetes and being obese for any of the four outcomes (data not shown).

After controlling for age, race/ethnicity, mode of delivery, and other known risk factors for PFDs that were significant in the bivariate analysis, we found that women categorized as obese/diabetic had the highest probability of having SUI, AI, and any PFD, whereas women who were obese/nondiabetic were as likely as obese/diabetic women to have OAB (Table 4). Women categorized as nonobese/diabetic did not differ significantly in their prevalence of OAB, AI, or any condition than nonobese/nondiabetic women (reference), whereas nonobese/diabetic women were significantly more likely to have SUI than nonobese/nondiabetic women.

CONCLUSIONS— In our sample of community-dwelling women, we found that being obese, regardless of having di-

Table 2—Prevalence of PFDs in 3,962 women aged 25–84 years with and without diabetes

	All women	Nondiabetic women	Diabetic women	*P* value
n	3,962	3,569	393	
SUI (n = 3,912)	589 (15.1)	497 (14.1)	92 (23.8)	<0.0001
OAB (n = 3,877)	518 (13.4)	438 (12.5)	80 (21.4)	<0.0001
AI (n = 3,823)	959 (25.1)	839 (24.3)	120 (32.5)	<0.0005
Any PFD (n = 3,785)	1,324 (35.0)	1,157 (33.8)	167 (46.1)	<0.0001

Data are *n* (%). Women with missing data are excluded from these analyses.

Table 3—*Crude odds ratios (95% CI) for the associations between obesity and diabetes and PFDs*

	SUI	OAB	AI	Any PFD
All women (n = 3,962)				
n	589	518	959	1,324
Diabetes				
Yes	1.91 (1.48–2.46)	1.90 (1.46–2.49)	1.50 (1.19–1.89)	1.68 (1.35–2.09)
No	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Obese				
Yes (BMI ≥30 kg/m^2)	2.58 (2.15–3.09)	2.67 (2.20–3.22)	1.46 (1.25–1.71)	1.92 (1.66–2.22)
No (BMI <30 kg/m^2)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Obesity and diabetes				
Obese/diabetic	3.24 (2.36–4.45)	3.31 (2.37–4.63)	1.90 (1.41–2.56)	2.42 (1.82–3.22)
Obese/nondiabetic	2.56 (2.10–3.11)	2.65 (2.16–3.27)	1.40 (1.17–1.66)	1.87 (1.59–2.19)
Nonobese/diabetic	1.77 (1.17–2.68)	1.78 (1.15–2.75)	1.33 (0.93–1.90)	1.50 (1.07–2.09)
Nonobese/nondiabetic	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Women with diabetes (n = 393)				
n	98	90	132	184
Obese				
Yes (BMI ≥30 kg/m^2)	1.83 (1.12–2.99)	1.86 (1.11–3.14)	1.43 (0.92–2.23)	1.62 (1.06–2.47)
No (BMI <30 kg/m^2)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)

abetes, increased the likelihood of having a PFD compared with nonobese women. The prevalence of SUI, AI, and any PFD increased in the following manner: nonobese/nondiabetic (lowest), nonobese/diabetic, obese/nondiabetic, and obese/diabetic (highest), while women who were obese, regardless of whether they had diabetes, were most likely to have OAB.

Our approach to these analyses differed from others, as we directly examined the associations between PDFs and diabetes with or without obesity using women with neither condition as the reference group instead of examining the association between one of these conditions while controlling for the other (9–11). We were able to examine these associations across three different conditions, whereas many reports (7,9,10,12,28) limit their analysis to one condition, and unlike some studies (7,12), we were able to include premenopausal women in our cohort. As with most other studies, we found an association between PFDs and both diabetes and obesity.

While studies (14,15) of the relationship between AI and diabetes have had conflicting results, we found that AI was associated with having diabetes among obese women only, whereas the relationship between AI and diabetes in women who were not obese was not statistically significant.

The strength of this study includes using a carefully validated instrument to assess a spectrum of PFDs in a large, racially and ethnically diverse population distributed across a wide age range including obese and nonobese women. In addition, we were able to characterize the women in our sample with diabetes by linking clinical information about glycemic control and diabetes treatment regimen to the survey responses closest to the time of the survey.

Our response rate was lower than anticipated despite considerable effort to increase it, particularly among younger health plan members. We found that younger members were hardest to reach; the likelihood of not having a valid address on file decreased with age, from 11% of 25- to 39-year-old subjects to 3% of 70- to 84-year-old subjects. When we compared women in the final analytic sample (n = 3,962) with all other women originally surveyed (n = 8,238), 10% of the women in the sample and 11% of the remaining women had diabetes ($P <$ 0.05). Among women with diabetes, there was no difference in mean A1C percent (P = 0.76) nor a difference in the racial/ethnic distribution (P = 0.26) when women in the analytic sample were

Table 4—*Adjusted odds ratios (95% CI) for the associations between obesity and diabetes-related factors and PFDs*

	SUI*†	OAB*‡	AI*§	Any PFD*‖
Obesity and diabetes				
Obese/diabetic	3.67 (2.48–5.43)	2.97 (2.03–4.36)	2.09 (1.48–2.97)	2.62 (1.87–3.67)
Obese/nondiabetic	2.62 (2.09–3.30)	2.93 (2.33–3.68)	1.45 (1.20–1.76)	1.83 (1.54–2.18)
Nonobese/diabetic	1.81 (1.09–3.00)	1.45 (0.88–2.38)	1.33 (0.89–2.00)	1.32 (0.90–1.94)
Nonobese/nondiabetic	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
n in adjusted model	3,353	3,574	3,478	3,446
n with PFD	484	453	866	1,257

*All models are adjusted for age, race/ethnicity, mode of delivery, and parity (base model). †Base model plus hormone therapy use, menopause status, hysterectomy, smoking, caffeine use, history of depression, lung disease/asthma, and neurological disease. ‡Base model plus hysterectomy and lung disease/asthma. §Base model plus hormone therapy use, menopause status, and history of depression. ‖Base model plus hormone therapy use, menopause status, hysterectomy, and history of depression. Obese is considered BMI ≥30 kg/m^2. Any PFD = one or more of the three PFDs (SUI, OAB, or AI).

compared with all others originally surveyed. Data on the prevalence of obesity were not available for comparison. Given that our overall prevalence of obesity and diabetes was consistent with what we would have anticipated given national estimates, we do not believe that our response rate biased the result of this study.

As this was a secondary analysis of data gathered primarily to evaluate the associations between pregnancy, mode of delivery, and PFDs (31), we did not have enough power to assess the relationship between glycemic control, diabetes treatment, and PFDs. Finally, we could only examine associations between prevalent PFDs and obesity and diabetes without information on the temporal sequence the onset of these conditions, since this was a cross-sectional study.

The findings from this study suggest that being obese may be a modifiable risk factor for PFDs. Women who are obese, regardless of whether they have diabetes, are more likely to have SUI, OAB, and AI, whereas nonobese/diabetic women had similar odds of each and any PFD as nonobese/nondiabetic women. Other published studies have suggested that weight loss may reduce the prevalence of incontinence among this group of high-risk women. Given the aging of the population, the increased prevalence of obesity, and the concurrent increase in the prevalence of diabetes in the U.S., women and health care professionals should be made aware of the associations between PFDs and obesity and diabetes. Women who are obese, regardless of whether they have diabetes, should be advised that they may be more likely to develop a PFD associated with their weight and should be encouraged to adopt patterns of physical activity and dietary intake to promote healthy weight loss and maintenance of a healthy weight.

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References

1. United States Department of Health and Human Services, Centers for Disease Control and Prevention and Health Promotion Division of Diabetes Translation: Diabetes Public Resource: National Diabetes Fact Sheet. Available from http://www.cdc.gov/diabetes/pubs/estimates05.htm#prev2. Accessed 2 August 2007
2. Waetjen LE, Liao S, Johnson WO, Sampselle CM, Sternfield B, Harlow SD, Gold EB: Factors associated with prevalent and incident urinary incontinence in a cohort of midlife women: a longitudinal analysis of data: study of women's health across the nation. *Am J Epidemiol* 165:309–318, 2007
3. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM: Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* 295:1549–1555, 2006
4. Hu TW, Wagner TH, Bentkover JD, Leblanc K, Zhou SZ, Hunt T: Costs of urinary incontinence and overactive bladder in the United States: a comparative study. *Urology* 63:461–465, 2004
5. Patrick DL, Martin ML, Bushnell DM, Yalcin I, Wagner TH, Buesching DP: Quality of life of women with urinary incontinence: further development of the incontinence quality of life instrument (I-QOL). *Urology* 53:71–76, 1999
6. Brown JS: Urinary incontinence: an important and underrecognized complication of type 2 diabetes mellitus. *J Am Geriatr Soc* 53:2028–2029, 2005
7. Jackson SL, Scholes D, Boyko EJ, Abraham L, Fihn SD: Urinary incontinence and diabetes in postmenopausal women. *Diabetes Care* 28:1730–1738, 2005
8. Lifford KL, Curhan GC, Hu FB, Barbieri RL, Grodstein F: Type 2 diabetes mellitus and risk of developing urinary incontinence. *J Am Geriatr Soc* 53:1851–1857, 2005
9. Sampselle CM, Harlow SD, Skurnick J, Brubaker L, Bondarenko I: Urinary incontinence predictors and life impact in ethnically diverse perimenopausal women. *Obstet Gynecol* 100:1230–1238, 2002
10. Brown JS, Vittinghoff E, Lin F, Nyberg LM, Kusek JW, Kanaya AM: Prevalence and risk factors for urinary incontinence in women with type 2 diabetes and impaired fasting glucose: findings from the National Health and Nutrition Examination Survey (NHANES) 2001–2002. *Diabetes Care* 29:1307–1312, 2006
11. Lewis CM, Schrader R, Many A, Mackay M, Rogers RG: Diabetes and urinary incontinence in 50- to 90-year-old women: a cross-sectional population-based study. *Am J Obstet Gynecol* 193:2154–2158, 2005
12. Jackson RA, Vittinghoff E, Kanaya AM, Miles TP, Resnick HE, Kritchevsky SB, Simonsick EM, Brown JS: Urinary incontinence in elderly women: findings from the Health, Aging, and Body Composition Study. *Obstet Gynecol* 104:301–307, 2004
13. Brown JS, Nyberg LM, Kusek JW, Burgio KL, Diokno AC, Foldspang A, Fultz NH, Herzog AR, Hunskaar S, Milsom I, Nygaard I, Subak LL, Thom DH: Proceedings of the National Institute of Diabetes and Digestive and Kidney Diseases International Symposium on Epidemiologic Issues in Urinary Incontinence in Women. *Am J Obstet Gynecol* 188:S77–S88, 2003
14. Quander CR, Morris MC, Melson J, Bienias JL, Evans DA: Prevalence of and factors associated with fecal incontinence in a large community study of older individuals. *Am J Gastroenterol* 100:905–909, 2005
15. Nelson R, Furner S, Jesudason V: Fecal incontinence in Wisconsin nursing homes: prevalence and associations. *Dis Colon Rectum* 41:1226–1229, 1998
16. Richter HE, Burgio KL, Brubaker L, Moalli PA, Markland AD, Mallet V, Menefee SA, Johnson HW, Boreham MK, Dandreo KJ, Stoddard AM: Factors associated with incontinence frequency in a surgical cohort of stress incontinent women. *Am J Obstet Gynecol* 193:2088–2093, 2005
17. Kapoor DS, Davila GW, Rosenthal RJ, Ghoniem GM: Pelvic floor dysfunction in morbidly obese women: pilot study. *ObesRes* 12:1104–1107, 2004
18. Uustal FE, 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* 83:383–389, 2004
19. Dwyer PL, Lee ET, Hay DM: Obesity and urinary incontinence in women. *Br J Obstet Gynaecol* 95:91–96, 1988
20. 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* 26:312–319, 1999
21. Melville JL, Fan MY, Newton K, Fenner D: Fecal incontinence in US women: a population-based study. *Am J Obstet Gynecol* 193:2071–2076, 2005
22. Yarnell JW, Voyle GJ, Sweetnam PM, Milbank J, Richards CJ, Stephenson TP: Factors associated with urinary incontinence in women. *J Epidemiol Community Health* 36:58–63, 1982
23. Varma MG, Brown JS, Creasman JM, Thom DH, Van Den Eeden SK, Beattie MS, Subak LL: Fecal incontinence in females older than aged 40 years: who is at risk? *Dis Colon Rectum* 49:841–851, 2006
24. Foldspang A, Mommsen S: [Overweight and urinary incontinence in women]. *Ugeskr Laeger* 157:5848–5851, 1995 (article in Danish)
25. Noblett KL, Jensen JK, Ostergard DR: The relationship of body mass index to intra-abdominal pressure as measured by mul-

tichannel cystometry. *Int Urogynecol J Pelvic Floor Dysfunct* 8:323–326, 1997
26. 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* 13:40–43, 2002
27. Subak LL, Whitcomb E, Shen H, Saxton J, Vittinghoff E, Brown JS: Weight loss: a novel and effective treatment for urinary incontinence. *J Urol* 174:190–195, 2005
28. Brown JS, Wing R, Barrett-Connor E, Nyberg LM, Kusek JW, Orchard TJ, Ma Y, Vittinghoff E, Kanaya AM: Lifestyle intervention is associated with lower prevalence of urinary incontinence: the Diabetes Prevention Program. *Diabetes Care* 29:385–390, 2006
29. Lukacz ES, Lawrence JM, Burchette RJ, Luber KM, Nager CW, Buckwalter JG: The use of Visual Analog Scale in urogynecologic research: a psychometric evaluation. *Am J Obstet Gynecol* 191:165–170, 2004
30. Lukacz ES, Lawrence JM, Buckwalter JG, Burchette RJ, Nager CW, Luber KM: Epidemiology of prolapse and incontinence questionnaire: validation of a new epidemiologic survey. *Int Urogynecol J Pelvic Floor Dysfunct* 16:272–284, 2005
31. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM: Parity, mode of delivery, and pelvic floor disorders. *Obstet Gynecol* 107:1253–1260, 2006
32. Petitti DB, Contreras R, Ziel FH, Dudl J, Domurat ES, Hyatt JA: Evaluation of the effect of performance monitoring and feedback on care process, utilization, and outcome. *Diabetes Care* 23:192–196, 2000
33. Selby JV, Ray GT, Zhang D, Colby CJ: Excess costs of medical care for patients with diabetes in a managed care population. *Diabetes Care* 20:1396–1402, 1997
34. International Diabetes Federation: *Diabetes Atlas*. 3rd ed. Brussels, Belgium, International Diabetes Federation, 2006