

Associations Between the Number of Vaginal Delivery and Urinary Incontinence in Women Before and After Natural Menopause: A Cross-Sectional Study

Xuejiao Bai¹, Guanghui Li¹, Dongxia Yang², Hongli Ma³, Hongxia Zhao⁴, Xinming Yang³

¹Department of First Clinical Medical College, Heilongjiang University of Chinese Medicine, Harbin, Heilongjiang, People's Republic of China;

²Department of Gynecology, The Second Affiliated Hospital of Heilongjiang University of Chinese Medicine, Harbin, Heilongjiang, People's Republic of China;

³Department of Gynecology, The First Affiliated Hospital of Heilongjiang University of Chinese Medicine, Harbin, Heilongjiang, People's Republic of China;

⁴Institute of Basic Theory of Chinese Medicine, China Academy of Chinese Medical Sciences, Beijing, People's Republic of China

Correspondence: Xinming Yang, Department of Gynecology, The First Affiliated Hospital of Heilongjiang University of Chinese Medicine, 26 Heping Road, Xiangfang District, Harbin, Heilongjiang, 150040, People's Republic of China, Tel +8613654540399, Email yangxinming12305@163.com; 420873126@qq.com

Purpose: Urinary Incontinence (UI) associates with menopausal status and vaginal delivery (VD). Existing literature has not confirmed a cumulative effect of an increasing number of VDs on UI incidence, while hormonal changes following natural menopause modulate the risk of UI. This study investigate the association between the number of VDs and UI by stratifying women based on menopausal status.

Patients and Methods: This study examined the relationship between the number of VDs and categories of UI, including Stress Urinary Incontinence (SUI), Urge Urinary Incontinence (UII), and Mixed Urinary Incontinence (MUI) in pre- and postmenopausal women, utilizing publically accessible data from the National Health and Nutrition Examination Survey (NHANES, 2007–2018). Weighted multivariable logistic regression, restricted cubic splines (RCS), and subgroup analyses with interaction tests were used to examine their association.

Results: This study comprised 4,046 premenopausal and 2,698 postmenopausal women. Adjusted multivariable regression analysis (Model 2) revealed that compared to nulliparous women, the risk of SUI significantly increased with ≥ 1 VDs in premenopausal women (1 VD: OR=2.53; 2 VDs: OR=3.73; ≥ 3 VDs: OR=2.94; all $P < 0.001$) and with ≥ 2 VDs in postmenopausal women (2 VDs: OR=1.52; ≥ 3 VDs: OR=1.62, all $P < 0.05$). MUI risk was elevated at 2 VD in both premenopausal (OR=1.78, $P=0.012$) and postmenopausal women (OR=1.57, $P=0.049$). No significant association was detected between the number of VDs and UII in either group. RCS modeling revealed inverse U-shaped relationships between the number of VDs and SUI risk and L-shaped relationships with MUI risk across both groups (nonlinear $P < 0.001$).

Conclusion: The number of VDs exhibits positive associations with incident SUI and MUI, while no significant association was detected with UII. Notably, the additional risk for SUI and MUI associated with the number of VDs was attenuated in postmenopausal women compared to premenopausal women. Prospective studies are warranted to validate the robustness of these associations.

Keywords: Stress Urinary Incontinence, Urge Urinary Incontinence, Mixed Urinary Incontinence, vaginal delivery, natural menopause, NHANES

Introduction

Urinary Incontinence (UI), defined as the complaint of involuntary loss of urine with or without bladder control dysfunction, is influenced by factors such as age, obesity, bladder and pelvic floor function.¹ It is more prevalent in women, with epidemiological studies indicating a progressive increase in prevalence with advancing age.² Clinically, UI is categorized into Stress Urinary Incontinence (SUI), Urge Urinary Incontinence (UII), and Mixed Urinary Incontinence (MUI).¹ A representative survey of US women reported prevalence rates of 45.9%, 31.1%, and 18.1%

for these three categories of UI, respectively.³ As a disease that severely impacts women's health and quality of life, its diagnosis, treatment, and clinical care remain insufficient and impose an economic burden on individuals and society.^{4,5}

A high number of births, vaginal delivery (VD) history and menopause have been identified as independent risk factors for UI.^{6,7} VD has a significant impact on overall pelvic floor function. Women with a history of VD during their reproductive period are at an elevated risk of UI than cesarean section and nulliparous women due to overstretching of the birth canal, weakening of the bladder neck support, and damage to pelvic floor muscle fibrous tissue caused by the natural delivery process.^{8,9} However, no literature has confirmed cumulative pelvic floor injury with increasing VDs.⁶

Menopause and aging are also closely related to UI occurrence in women.¹⁰ Declining estrogen has been recognized to increase the risk of UI, with an overall prevalence of UI of 38% in women over the age of 60 years and increasing with age.¹¹ Estrogen receptors are expressed throughout the lower urinary tract, particularly in structures directly implicated in UI pathogenesis.¹² Estrogen may elevate contractile resistance by increasing adrenergic receptor sensitivity in urethral smooth muscle.^{13,14} Ovarian follicle depletion after natural menopause results in estrogen fluctuations in women.¹⁵ These fluctuations can influence the occurrence of UI by modulating urethral blood flow and mucosal proliferation to affect urethral closure pressure, as well as altering bladder sensory thresholds and urethral pressure transmission, which impairs urinary continence homeostasis.¹⁶ However, these pathophysiological associations require validation through prospective mechanistic studies.

This study aimed to investigate whether the number of VDs may exert cumulative effects on UI risk development, while exploring potential differential impacts of VD on UI incidence between premenopausal and naturally postmenopausal women. These investigations may provide novel perspectives on risk assessment and inform future research on UI-related adverse outcomes.

Materials and Methods

Study Population

This cross-sectional study utilized data from the NHANES. Comprehensive datasets and methodologies are publicly accessible at <https://www.cdc.gov/nchs/nhanes/>. NHANES has been collected biennially since 1999, employing a complex stratified multistage sample designed to comprehensively survey and evaluate the US population's health and nutritional status. The National Center for Health Statistics (NCHS) Research Ethics Review Board (ERB) approved the study protocol, and each participant obtained written informed consent. We used data from six NHANES survey cycles (2007–2018). For the current analysis, we utilized data from women with complete records on the number of VDs and UI subtypes, stratified by natural menopausal status. Exclusion criteria: (1) Individuals currently breastfeeding or pregnant to avoid menstrual cycle confounding; (2) Individuals with non-natural menopause due to hysterectomy or oophorectomy; (3) Individuals with a history of cesarean delivery; (4) Individuals with undocumented menopausal status; (5) Extreme outliers in the number of VDs (defined as >10 deliveries based on the NHANES Reproductive Health Questionnaire 2017–2018, which caps vaginal births at 0–10); (6) Missing data about three groups of covariates. Following these exclusion criteria, the final analytical cohort comprised 4046 premenopausal women and 2698 naturally postmenopausal women (Figure 1).

Study Variables

Exposure Variable Assessment

Natural menopausal status and the number of VD were operationally defined using data from the NHANES "Reproductive Health" questionnaire. Women were classified as naturally postmenopausal if they responded "No" to the question {Have you/Has SP} had at least one menstrual period in the past 12 months? (Please do not include bleedings caused by medical conditions, hormone therapy, or surgeries.) (RHQ031), followed by reporting "Hysterectomy" or "Menopause/change of life" as the reason for amenorrhea (RHD043/RHD042), while simultaneously denying a history of hysterectomy ({Have you/Has SP} had a hysterectomy, including a partial hysterectomy, that is, surgery to remove {your/her} uterus or womb?" RHD280: "No") and bilateral oophorectomy ("{Have you/Has SP} had both of {your/her} ovaries removed (either when {you/she} had {your/her} uterus removed or at another time)?" RHQ305: "No"). Premenopausal status was assigned to those affirming recent menstruation via a "Yes" response to RHQ031.

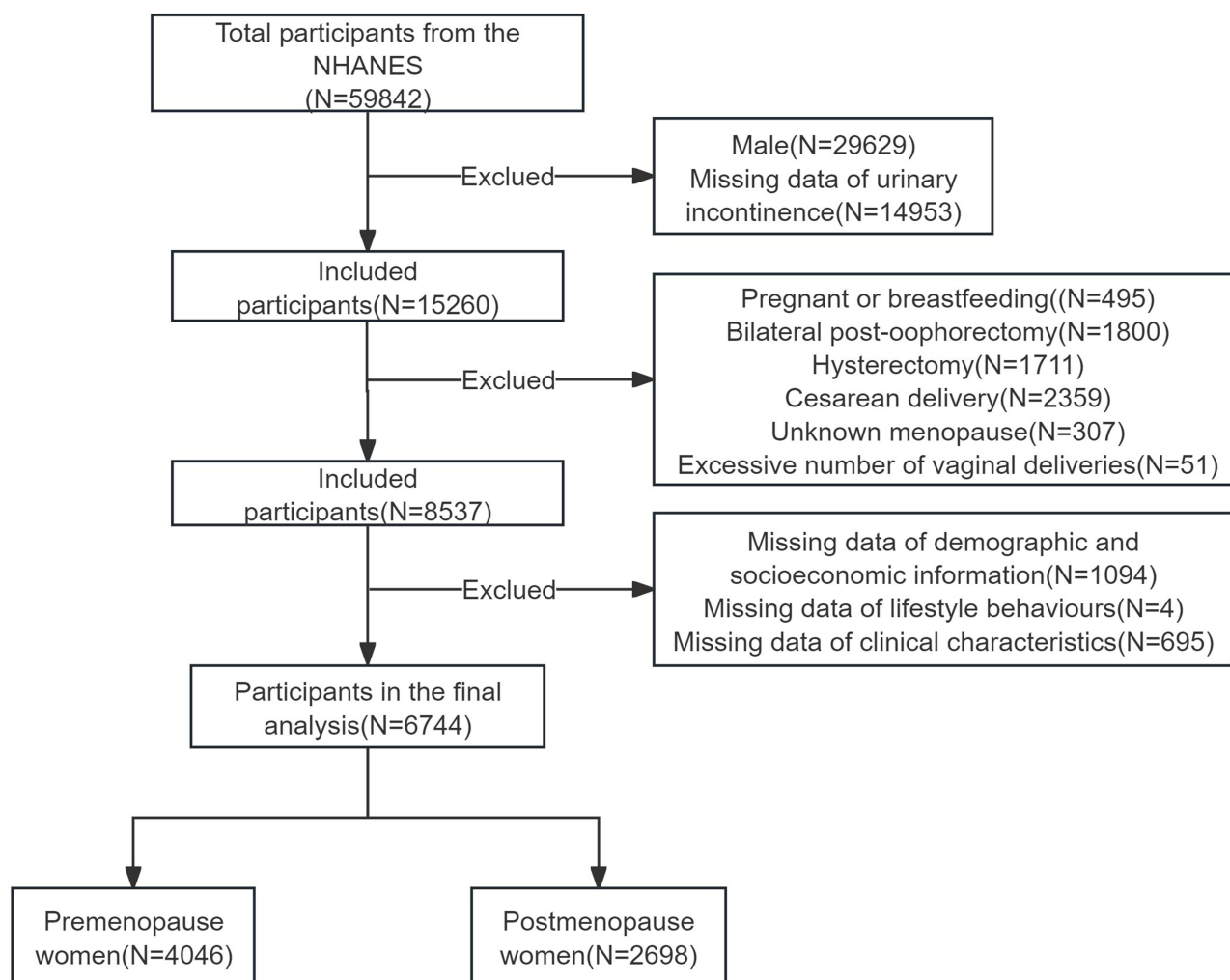


Figure 1 Flow chart of subject inclusion.

VD counts were derived from the question “How many vaginal deliveries {have you/has SP} had? {Please count stillbirths as well as live births}” (RHQ166), with nulliparous women defined as respondents reporting 0 vaginal delivery on RHQ162 or with a “No” answer to The next questions are about {your/SP’s} pregnancy history. The question is: {Have you/Has SP ever been pregnant? Please include (current pregnancy,) live births, miscarriages, stillbirths, tubal pregnancies and abortions. (RHQ131). Detailed variable specifications and questionnaire protocols are publicly accessible through the NHANES documentation portal.

Outcome Assessment

Two questions in the NHANES “Kidney Condition” questionnaire were used to evaluate the occurrence of UI. Participants who responded “Yes” to the question “During the past 12 months, {have you/has SP} leaked or lost control of even a small amount of urine with an activity like coughing, lifting or exercise?”(KIQ042) were classified as SUI. While UUI was considered as a question: During the past 12 months, have you leaked or lost control of even a small amount of urine with an urge or pressure to urinate and you couldn’t get to the toilet fast enough?(KIQ044) participants answered “Yes”. In addition, if participants answered “Yes” to the above questions, they were set as MUI.

Covariate Assessment

Covariates were selected based on clinical insights into potential UI risk factors. Previous studies indicate some risk factors, such as vitamin D deficiency, obesity, depression, and smoking, are significant for UI incidence among women.^{17–22} Based

on the availability of NHANES data, the following covariates were included in this study: (a) demographic and socioeconomic information, including age, age at menarche, race (Non-Hispanic white, Non-Hispanic black, Mexican American, other Hispanic or Other race), education level (Less than 9th grade, 9th-11th grade, High school graduate/GED, Some college/AA, College graduate or above), marital status (Married, Widowed, Divorced, Separated, Never married, Living with a partner), family poverty-income ratio (PIR); (b) lifestyle behaviours, including smoking status (more or less than 100 cigarettes in a lifetime categorized as “yes” or “no”); (c) clinical characteristics, including body mass index (BMI, kg/m²), waist circumference (cm), serum vitamin D (nmol/L), depression (PHQ-9 scores of 10 or more categorized as “yes”, otherwise “no”), diabetes (indicated as yes or no), hypertension (indicated as “yes” or “no”).

Statistical Analysis

This study utilized R software (version 4.3.1), with statistical significance set at P (two-sided) < 0.05. Weighted variables were introduced to account for the intricate sample design of the NHANES and to assure data representativeness of the US population. In baseline analyses, categorical variables were summarized as frequencies (percentages), whereas continuous variables were expressed as mean \pm standard deviation (SD). To characterize the study population, normally distributed continuous variables were analyzed using one-way ANOVA, skewed continuous variables were assessed with the Kruskal–Wallis test, and categorical variables were compared using the chi-square test. Weighted multivariable logistic regression models were utilized to analyze the association between the number of VDs and UI infections, with results reported as odds ratios (ORs) and 95% confidence intervals (95% CIs). Three models were constructed: (1) Crude Model, unadjusted to evaluate original associations; (2) Model 1, adjusted for age, race, PIR, marital status, and education level to control for demographic confounders; (3) Model 2, adjusted for age, race, PIR, marital status, education level, smoking status, BMI, depression, waist circumference, Age at Menarche, Vitamin D, hypertension, and diabetes to control all confounders.

Additionally, weighted restricted cubic spline (RCS) curves were applied to investigate possible nonlinear associations between exposure (number of VDs) and outcomes (UI). To validate the stability of the findings, subgroup analyses were stratified by age, race, marital status, education level, smoking status, diabetes, and depression. These analyses aimed to identify whether distinct correlations between the number of VDs and UI incidence existed within specific population subgroups.

Results

Participants Characteristics

This study included 6744 participants, with demographic and clinical characteristics of premenopausal ($n=4046$) and postmenopausal ($n=2698$) women. Among premenopausal women (Table 1), 1,399 (36%), 738 (17%), and 432 (10%) reported SUI, UUI, and MUI, respectively. Corresponding figures for postmenopausal women (Table 2) were 1,255 (49%), 1,043 (37%), and 579 (21%). To maximize data balance, the number of VDs was categorized into four groups: 0, 1, 2, and ≥ 3 deliveries. Premenopausal women with 0 or 2 VDs exhibited higher family income, elevated educational levels, and serum vitamin D concentrations compared to those with other numbers of VDs. They also demonstrated lower BMI, waist circumference, and reduced likelihood of diabetes and depression. In contrast, postmenopausal women showed no significant associations between VD numbers and BMI, depressive symptoms, or smoking status. However, postmenopausal women with 0 or 2 VDs generally displayed higher family income, serum vitamin D concentrations, and lower probabilities of diabetes and hypertension compared to other parity groups.

Association Between the Number of VDs and UI in Pre- and Postmenopausal Women

Three multivariate logistic regression models were constructed to evaluate the effects of the number of VDs on three UI subtypes. Analyses treating VD count as a continuous variable showed a significant positive association between VDs and SUI and MUI in crude Model for premenopausal women ($P < 0.001$). This association persisted for SUI (Table 3) ($P < 0.001$) and MUI (Table 4) ($P = 0.039$) in Model 2 (fully adjusted). However, no significant associations were observed between the number of VDs and any UI subtypes in postmenopausal women. When VDs were assessed categorically in

Table 1 Baseline Weighted Characteristics of Premenopausal Women

Characteristic	Overall N = 4046 ¹	0 Delivery N = 1609 ¹	1 Delivery N = 634 ¹	2 Deliveries N = 851 ¹	≥ 3 Deliveries N = 952 ¹	p-value ²
Year	34.51± (9.76)	29.55± (8.53)	34.65± (9.63)	39.25± (8.67)	40.26± (7.53)	<0.001
Race						<0.001
Mexican American	634 (9.2%)	185 (6.9%)	83 (8.4%)	124 (8.6%)	242 (16%)	
Other Hispanic	405 (6.4%)	150 (6.1%)	70 (7.7%)	89 (6.0%)	96 (6.4%)	
Non-Hispanic White	1622 (64%)	680 (67%)	224 (59%)	375 (68%)	343 (57%)	
Non-Hispanic Black	800 (12%)	304 (10%)	153 (15%)	145 (9.2%)	198 (14%)	
Other Race	585 (8.8%)	290 (9.8%)	104 (9.7%)	118 (8.1%)	73 (6.6%)	
Education level						<0.001
Less than 9th grade	245 (3.6%)	56 (2.0%)	28 (3.3%)	51 (3.6%)	110 (7.4%)	
9-11th grade	499 (9.3%)	111 (5.3%)	81 (11%)	99 (8.9%)	208 (18%)	
High school graduate/GED or equivalent	788 (19%)	252 (16%)	148 (24%)	189 (20%)	199 (21%)	
Some college or AA degree	1327 (33%)	542 (32%)	218 (33%)	282 (34%)	285 (31%)	
College graduate or above	1187 (36%)	648 (45%)	159 (29%)	230 (33%)	150 (23%)	
Marital status						<0.001
Married	1927 (50%)	573 (36%)	310 (53%)	506 (66%)	538 (62%)	
Widowed	91 (2.1%)	38 (2.0%)	15 (2.7%)	19 (1.7%)	19 (2.1%)	
Divorced	348 (8.8%)	90 (5.8%)	55 (9.3%)	99 (11%)	104 (12%)	
Separated	162 (3.2%)	31 (1.8%)	27 (4.0%)	39 (3.0%)	65 (6.2%)	
Never married	1028 (24%)	676 (41%)	143 (18%)	95 (8.0%)	114 (8.3%)	
Living with partner	490 (12%)	201 (13%)	84 (13%)	93 (9.8%)	112 (9.4%)	
PIR	2.90± (1.68)	3.17± (1.66)	2.77± (1.63)	3.03± (1.70)	2.25± (1.57)	<0.001
BMI	28.41± (7.80)	27.85± (8.09)	28.86± (7.98)	28.25± (6.97)	29.53± (7.73)	<0.001
Waist circumference	93.58± (17.70)	91.57± (18.50)	94.98± (18.68)	93.98± (15.58)	96.61± (16.77)	<0.001
Vitamin D (nmol/L)	67.51± (27.78)	68.31± (29.45)	65.86± (26.54)	70.43± (26.74)	63.68± (25.43)	<0.001
Depression						0.001
Yes	415 (9.4%)	126 (7.4%)	81 (14%)	87 (9.3%)	121 (11%)	
No	3631 (91%)	1483 (93%)	553 (86%)	764 (91%)	831 (89%)	
Age at menarche (years)	12.70± (1.69)	12.63± (1.66)	12.61± (1.81)	12.83± (1.63)	12.79± (1.72)	0.014
Smoking status						<0.001
Yes	1255 (34%)	391 (27%)	226 (42%)	300 (38%)	338 (37%)	
No	2791 (66%)	1218 (73%)	408 (58%)	551 (62%)	614 (63%)	
Diabetes						0.002
Yes	156 (3.2%)	42 (1.9%)	31 (5.1%)	33 (3.1%)	50 (4.4%)	
No	3890 (97%)	1567 (98%)	603 (95%)	818 (97%)	902 (96%)	
Hypertension						<0.001
Yes	399 (8.9%)	94 (5.9%)	57 (8.7%)	107 (10%)	141 (15%)	
No	3647 (91%)	1515 (94%)	577 (91%)	744 (90%)	811 (85%)	
SUI						<0.001
Yes	1399 (36%)	292 (19%)	230 (40%)	415 (54%)	462 (49%)	
No	2647 (64%)	1317 (81%)	404 (60%)	436 (46%)	490 (51%)	
UUI						<0.001
Yes	738 (17%)	231 (14%)	107 (17%)	174 (20%)	226 (22%)	
No	3308 (83%)	1378 (86%)	527 (83%)	677 (80%)	726 (78%)	
MUI						<0.001
Yes	432 (10.0%)	97 (5.6%)	55 (9.0%)	129 (15%)	151 (15%)	
No	3614 (90%)	1512 (94%)	579 (91%)	722 (85%)	801 (85%)	

Note: ¹Mean± (SD); n (unweighted) (%). ²Significant p-values (p < 0.05) are shown in bold.

Abbreviations: PIR, family poverty-income ratio; BMI, body mass index; SUI, stress urinary incontinence; UUI, urge urinary incontinence; MUI, mixed urinary incontinence.

Table 2 Baseline Weighted Characteristics of Postmenopausal Women

Characteristic	Overall N = 2698 ¹	0 Delivery N = 409 ¹	1 Delivery N = 321 ¹	2 Deliveries N = 673 ¹	≥ 3 Deliveries N = 1295 ¹	p-value ²
Year	63.62± (9.25)	61.28± (8.29)	61.27± (8.46)	62.82± (8.84)	66.11± (9.64)	<0.001
Race						<0.001
Mexican American	340 (4.4%)	39 (3.2%)	19 (2.3%)	53 (2.7%)	229 (6.9%)	
Other Hispanic	335 (4.5%)	47 (3.9%)	37 (3.7%)	80 (4.1%)	171 (5.3%)	
Non-Hispanic White	1267 (77%)	222 (82%)	156 (80%)	362 (81%)	527 (71%)	
Non-Hispanic Black	472 (7.8%)	55 (5.3%)	67 (7.7%)	102 (6.0%)	248 (10%)	
Other Race	284 (6.0%)	46 (5.4%)	42 (6.5%)	76 (5.8%)	120 (6.3%)	
Education level						<0.001
Less than 9th grade	322 (5.7%)	32 (4.1%)	16 (2.5%)	51 (3.2%)	223 (9.3%)	
9-11th grade	364 (10%)	29 (3.9%)	30 (6.2%)	83 (11%)	222 (14%)	
High school graduate/GED or equivalent	663 (24%)	83 (19%)	87 (27%)	167 (24%)	326 (27%)	
Some college or AA degree	723 (29%)	118 (29%)	99 (30%)	190 (30%)	316 (28%)	
College graduate or above	626 (31%)	147 (44%)	89 (34%)	182 (33%)	208 (22%)	
Marital status						<0.001
Married	1294 (54%)	155 (44%)	145 (48%)	361 (62%)	633 (55%)	
Widowed	503 (16%)	58 (11%)	49 (14%)	112 (14%)	284 (20%)	
Divorced	455 (16%)	59 (16%)	71 (21%)	128 (16%)	197 (16%)	
Separated	102 (2.3%)	7 (1.9%)	6 (2.0%)	17 (1.3%)	72 (3.4%)	
Never married	264 (8.3%)	116 (23%)	37 (8.0%)	41 (5.2%)	70 (3.7%)	
Living with partner	80 (3.1%)	14 (4.2%)	13 (6.4%)	14 (1.3%)	39 (2.7%)	
PIR	3.17± (1.63)	3.39± (1.57)	3.28± (1.69)	3.53± (1.57)	2.78± (1.60)	<0.001
BMI	28.87± (6.95)	29.38± (7.67)	28.70± (7.27)	28.30± (6.57)	29.11± (6.73)	0.109
Waist circumference	98.48± (15.41)	100.06± (16.74)	97.40± (16.33)	97.13± (14.72)	99.14± (14.86)	0.046
Vitamin D (nmol/L)	80.76± (32.50)	87.49± (35.97)	79.78± (30.92)	82.10± (33.12)	77.05± (30.35)	0.003
Depression						0.814
Yes	252 (7.5%)	46 (8.3%)	34 (8.7%)	50 (7.1%)	122 (7.0%)	
No	2446 (93%)	363 (92%)	287 (91%)	623 (93%)	1173 (93%)	
Age at menarche (years)	12.85± (1.69)	12.88± (1.73)	12.66± (1.78)	12.77± (1.57)	12.97± (1.71)	0.029
Smoking status						0.156
Yes	1033 (42%)	164 (44%)	149 (48%)	265 (42%)	455 (38%)	
No	1665 (58%)	245 (56%)	172 (52%)	408 (58%)	840 (62%)	
Diabetes						0.003
Yes	485 (14%)	54 (9.1%)	55 (14%)	91 (11%)	285 (18%)	
No	2213 (86%)	355 (91%)	266 (86%)	582 (89%)	1010 (82%)	
Hypertension						0.031
Yes	1373 (46%)	179 (40%)	165 (44%)	330 (43%)	699 (51%)	
No	1325 (54%)	230 (60%)	156 (56%)	343 (57%)	596 (49%)	
SUI						0.045
Yes	1255 (49%)	164 (42%)	145 (45%)	323 (51%)	623 (52%)	
No	1443 (51%)	245 (58%)	176 (55%)	350 (49%)	672 (48%)	
UUI						0.180
Yes	1043 (37%)	149 (35%)	101 (31%)	262 (39%)	531 (40%)	
No	1655 (63%)	260 (65%)	220 (69%)	411 (61%)	764 (60%)	
MUI						0.057
Yes	579 (21%)	74 (17%)	55 (16%)	139 (23%)	311 (23%)	
No	2119 (79%)	335 (83%)	266 (84%)	534 (77%)	984 (77%)	

Note: ¹Mean± (SD); n (unweighted) (%).²Significant p-values (p < 0.05) are shown in bold.

Abbreviations: PIR, family poverty-income ratio; BMI, body mass index; SUI, stress urinary incontinence; UUI, urge urinary incontinence; MUI, mixed urinary incontinence.

Table 3 Logistics Regression for the Associations Between the Number of VDs and SUI

		Crude Model OR (95% CI)	Minimally-Adjusted Model (Model 1) OR (95% CI)	Fully-Adjusted Model (Model 2) OR (95% CI)
Premenopausal Women	Number of VDs	1.50(1.42,1.59) <0.001	1.29 (1.20,1.38) <0.001	1.31(1.22,1.41) <0.001
	Number of VDs			
	0	Ref.	Ref.	Ref.
	1	2.88(2.20,3.79) <0.001	2.52(1.86,3.43) <0.001	2.53(1.85,3.47) <0.001
	2	4.96(3.98,6.17) <0.001	3.44(2.69,4.40) <0.001	3.73(2.92,4.77) <0.001
	≥3	4.14(3.32,5.17) <0.001	2.73(2.03,3.68) <0.001	2.94(2.18,3.97) <0.001
	P for trend	<0.001	<0.001	<0.001
Postmenopausal Women	Number of VDs	1.05(0.99, 1.12) 0.088	1.06(1.00, 1.13) 0.064	1.07(1.00,1.14) 0.056
	Number of VDs			
	0	Ref.	Ref.	Ref.
	1	1.17(0.77,1.80) 0.5	1.13(0.72,1.77) 0.6	1.17(0.76,1.81) 0.5
	2	1.49(1.04,2.13) 0.032	1.44(0.98,2.11) 0.061	1.52(1.05,2.20) 0.028
	≥3	1.52(1.10,2.11) 0.012	1.55(1.10,2.18) 0.013	1.62(1.16,2.27) 0.006
	P for trend	0.007	0.007	0.003

Notes: Model 1: adjust age, race, PIR, marital status, education level. Model 2: adjust age, race, PIR, marital status, education level, smoking status, BMI, depression, waist circumference, Age at Menarche, Vitamin D, hypertension, diabetes. Significant p-values ($p < 0.05$) are shown in bold.

Abbreviations: SUI, stress urinary incontinence; VDs, vaginal deliveries.

Table 4 Logistics Regression for the Associations Between the Number of VDs and MUI

		Crude Model OR (95% CI)	Minimally-Adjusted Model (Model 1) OR (95% CI)	Fully-Adjusted Model (Model 2) OR (95% CI)
Premenopausal Women	Number of VDs	1.35(1.25,1.45) <0.001	1.09(0.99,1.21) 0.087	1.11(1.01,1.22) 0.039
	Number of VDs			
	0	Ref.	Ref.	Ref.
	1	1.66(1.09,2.53) 0.019	1.13(0.72,1.76) 0.5	1.07(0.68,1.69) 0.8
	2	2.88(2.05,4.05) <0.001	1.66(1.05,2.62) 0.031	1.78(1.14,2.79) 0.012
	≥3	3.02(2.17,4.19) <0.001	1.34(0.87,2.07) 0.2	1.44(0.94,2.21) 0.090
	P for trend	<0.001	0.836	0.026
Postmenopausal Women	Number of VDs	1.08(1.03, 1.14) 0.005	1.03(0.97, 1.10) 0.3	1.04(0.98, 1.10) 0.2
	Number of VDs			
	0	Ref.	Ref.	Ref.
	1	0.93(0.55,1.55) 0.8	0.89(0.53,1.51) 0.7	0.91(0.55,1.53) 0.7
	2	1.49(0.92,2.43) 0.11	1.46(0.92,2.34) 0.11	1.57(1.00,2.46) 0.049
	≥3	1.47(1.03,2.10) 0.036	1.25(0.86,1.80) 0.2	1.32(0.92,1.87) 0.13
	P for trend	0.009	0.073	0.029

Notes: Model 1: adjust age, race, PIR, marital status, education level. Model 2: adjust age, race, PIR, marital status, education level, smoking status, BMI, depression, waist circumference, Age at Menarche, Vitamin D, hypertension, diabetes. Significant p-values ($p < 0.05$) are shown in bold.

Abbreviations: MUI, mixed urinary incontinence; VDs, vaginal deliveries.

Model 2, the risk of SUI was elevated in premenopausal women with 1, 2, or ≥ 3 deliveries compared with that of nulliparous women, with ORs of 2.53, 3.73, and 2.94, respectively; whereas among postmenopausal women, having 2 or ≥ 3 VDs was positively associated with SUI risk with ORs of 1.52 and 1.62, respectively. A significant association between the number of VDs and MUI risk was observed at 2 deliveries in both premenopausal (OR = 1.78) and postmenopausal women (OR = 1.57). No significant association was detected between the number of VDs and UUI risk in either group ($P > 0.05$) (Table 5).

Table 5 Logistics Regression for the Associations Between the Number of VDs and UUI

		Crude Model OR (95% CI)	Minimally-Adjusted Model (Model 1) OR (95% CI)	Fully-Adjusted Model (Model 2) OR (95% CI)
Premenopausal Women	Number of VDs	1.17(1.09,1.26) <0.001	0.99 (0.90,1.08)0.8	0.99(0.90,1.09) 0.9
	Number of VDs			
	0	Ref.	Ref.	Ref.
	1	1.20(0.90,1.61) 0.2	0.88(0.64,1.20) 0.4	0.83(0.60,1.13) 0.2
	2	1.50(1.16,1.94) 0.002	0.97(0.70,1.36) 0.9	1.00(0.72,1.41) 0.9
	≥3	1.68(1.26,2.24) <0.001	0.86(0.59,1.24) 0.4	0.89(0.61,1.29) 0.5
	P for trend	<0.001	0.562	0.794
Postmenopausal Women	Number of VDs	1.06(1.01, 1.12) 0.024	1.01(0.95, 1.07) 0.9	1.01(0.95, 1.07) 0.7
	Number of VDs			
	0	Ref.	Ref.	Ref.
	1	0.83(0.53,1.29) 0.4	0.82(0.52,1.29) 0.4	0.82(0.52,1.29) 0.4
	2	1.19(0.79,1.80) 0.4	1.16(0.76,1.76) 0.5	1.21(0.81,1.83) 0.3
	≥3	1.22(0.90,1.66) 0.2	0.99(0.71,1.36) 0.9	1.02(0.75,1.40) 0.9
	P for trend	0.725	0.640	0.513

Notes: Model 1: adjust age, race, PIR, marital status, education level. Model 2: adjust age, race, PIR, marital status, education level, smoking status, BMI, depression, waist circumference, Age at Menarche, Vitamin D, hypertension, diabetes.

Abbreviations: UUI, urge urinary incontinence; VDs, vaginal deliveries.

To further explore potential nonlinear relationships between the number of VDs and SUI/MUI in women before and after natural menopause, we employed RCS plots. Solid lines represent smoothed curve fits between variables, with shaded areas indicating 95% CIs. Stratified analyses by menopausal status revealed, in fully adjusted covariate models, an inverted U-shaped (Figure 2) association between the number of VDs and SUI and an L-shaped (Figure 3) pattern with MUI across both groups (P for nonlinearity < 0.001).

Subgroup Analysis

To elucidate the potential heterogeneity of the relationship between the number of VDs and SUI/MUI in subgroups of women with distinct characteristics before and after menopause, we conducted subgroup analyses and interaction tests

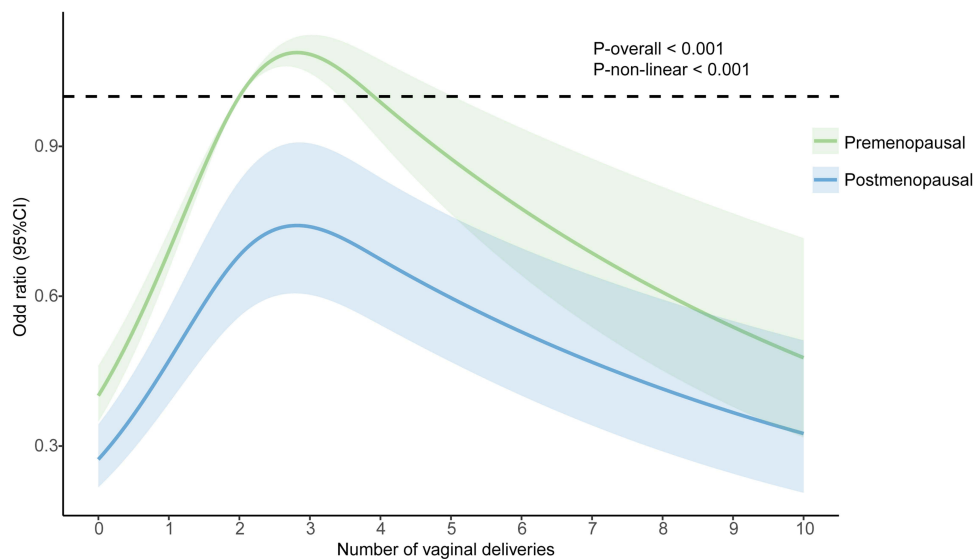


Figure 2 RCS curve analysis for relationships between the number of VDs and SUI in pre- and postmenopausal women.

Note: adjust age, race, PIR, marital status, education level, smoking status, BMI, depression, waist circumference, Age at Menarche, Vitamin D, hypertension, diabetes.

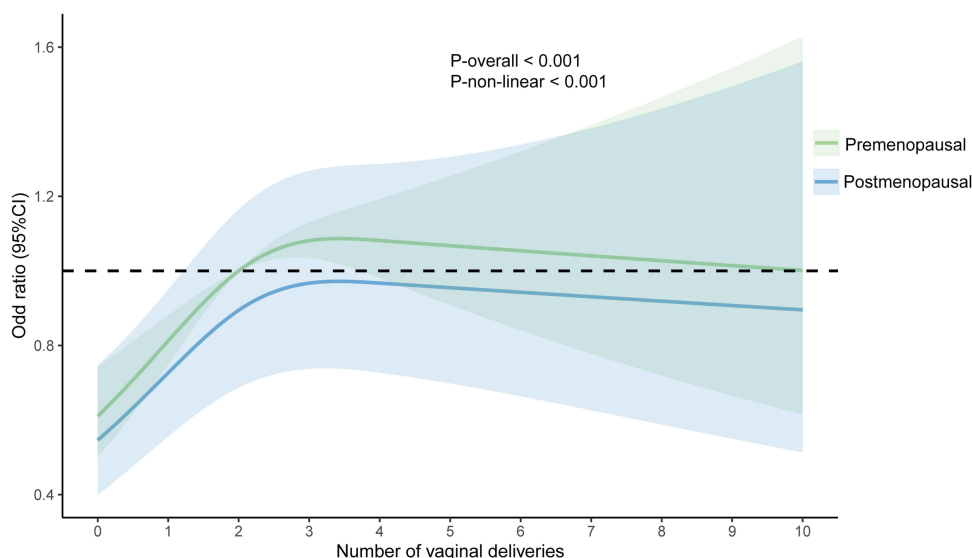


Figure 3 RCS curve analysis for relationships between the number of VDs and MUI in pre- and postmenopausal women.

Note: adjust age, race, PIR, marital status, education level, smoking status, BMI, depression, waist circumference, Age at Menarche, Vitamin D, hypertension, diabetes.

according to age, race, marital status, education level, smoking status, diabetes and depression. Among premenopausal women, there was an interaction effect of age, race, and education level between the number of VDs and SUI (Figure 4). Among postmenopausal women, interactions between the number of VDs and MUI (Figure 5) were observed for education level, diabetes. No interaction effects were observed (Figures 6 and 7).

Discussion

According to standardized definitions by the International Urogynecological Association (IUGA) and the International Continence Society (ICS), SUI is characterized by involuntary urine leakage during coughing, sneezing, or physical exertion. Its primary etiology involves dysfunction of the urethral closure function due to anatomical support loss and increased intra-abdominal pressure. UUI is defined as an idiopathic condition characterized by an uncontrollable urge to urinate and a sudden, compelling need to void, primarily driven by detrusor overactivity. It is commonly observed in patients with systemic neurological disorders. MUI manifests as a combination of both SUI and UUI symptoms.¹

Childbirth represents a unique phase in women's lives, with VD established as a significant risk factor for UI—a condition that profoundly impairs quality of life, mental health, and social engagement.²³ Despite most women are troubled by this condition, they often delay seeking treatment for several years, and healthcare providers typically undervalue UI's clinical significance.^{24,25} This cross-sectional study investigated the associations between the number of VDs and UI subtypes among 4,046 premenopausal and 2,698 naturally postmenopausal women. Our analyses detected inverted U-shaped relationships between the number of VDs and SUI in both menopausal groups, alongside an L-shaped association with MUI in both them. Notably, premenopausal women exhibited stronger relations for SUI and MUI with the increase in numbers of VDs compared to postmenopausal counterparts, while no significant correlations emerged between the number of VDs and UUI in either group.

The biomechanical mechanisms underlying these nonlinear patterns remain speculative. Based on current evidence, we propose the following potential mechanisms: As VDs accumulate before reaching the peak risk threshold, cumulative mechanical trauma progressively exacerbates pelvic floor dysfunction through three primary pathways: 1) Overstretching of pelvic floor muscles leads to diminished elastic recoil; 2) Ligamentous laxity substantially reduces structural support for bladder and urethral positioning; 3) Pudendal nerve damage compromises neuromuscular coordination essential for urinary continence.^{26,27} These synergistic effects culminate in significant deterioration of urethrovesical support systems. Furthermore, multiparity may induce permanent anatomical alterations, including periurethral ligament rupture and

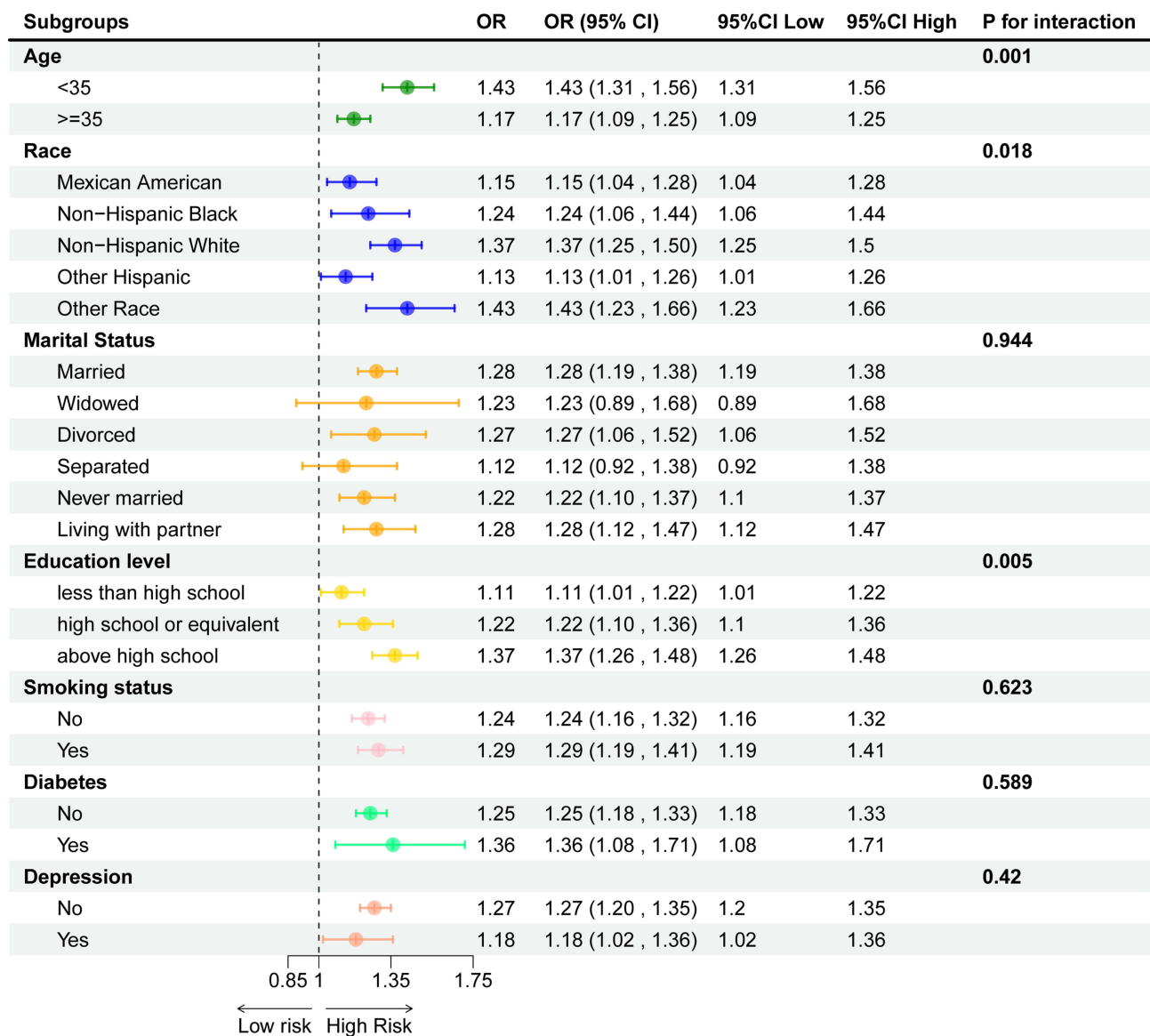


Figure 4 Subgroup analysis for associations between the number of VDs and SUI in premenopausal women.

caudal displacement of the bladder neck.²⁸ This pathophysiological cascade may ultimately trigger UI through anatomical and functional impairment of the urinary continence apparatus.

Nonetheless, when the number of VDs rises, the body may activate compensatory mechanisms to maintain pelvic floor function.²⁹ Studies have shown that women with UI demonstrate enhanced activation of auxiliary muscle groups, such as abdominal and gluteal muscles, due to the progressive deterioration of pelvic neuromuscular structures, suggesting potential compensatory participation in pelvic floor support.³⁰ Simultaneously, repeated VDs may induce adaptive structural and functional remodelling of pelvic floor tissues in response to recurrent pulling and injury, thus potentially reducing UI incidence to a certain extent.²⁷ With increasing parity, women are advised to adopt preventive strategies to mitigate pelvic floor dysfunction, including lifestyle modifications such as weight management to reduce intra-abdominal pressure, avoiding prolonged standing, scheduling voiding, and restricting fluid intake.^{31,32} Alongside these measures, pelvic floor muscle training interventions like Kegel exercises can enhance muscular support for the bladder neck and proximal urethra, especially during times of elevated intra-abdominal pressure, thereby reducing the risk of UI.³³

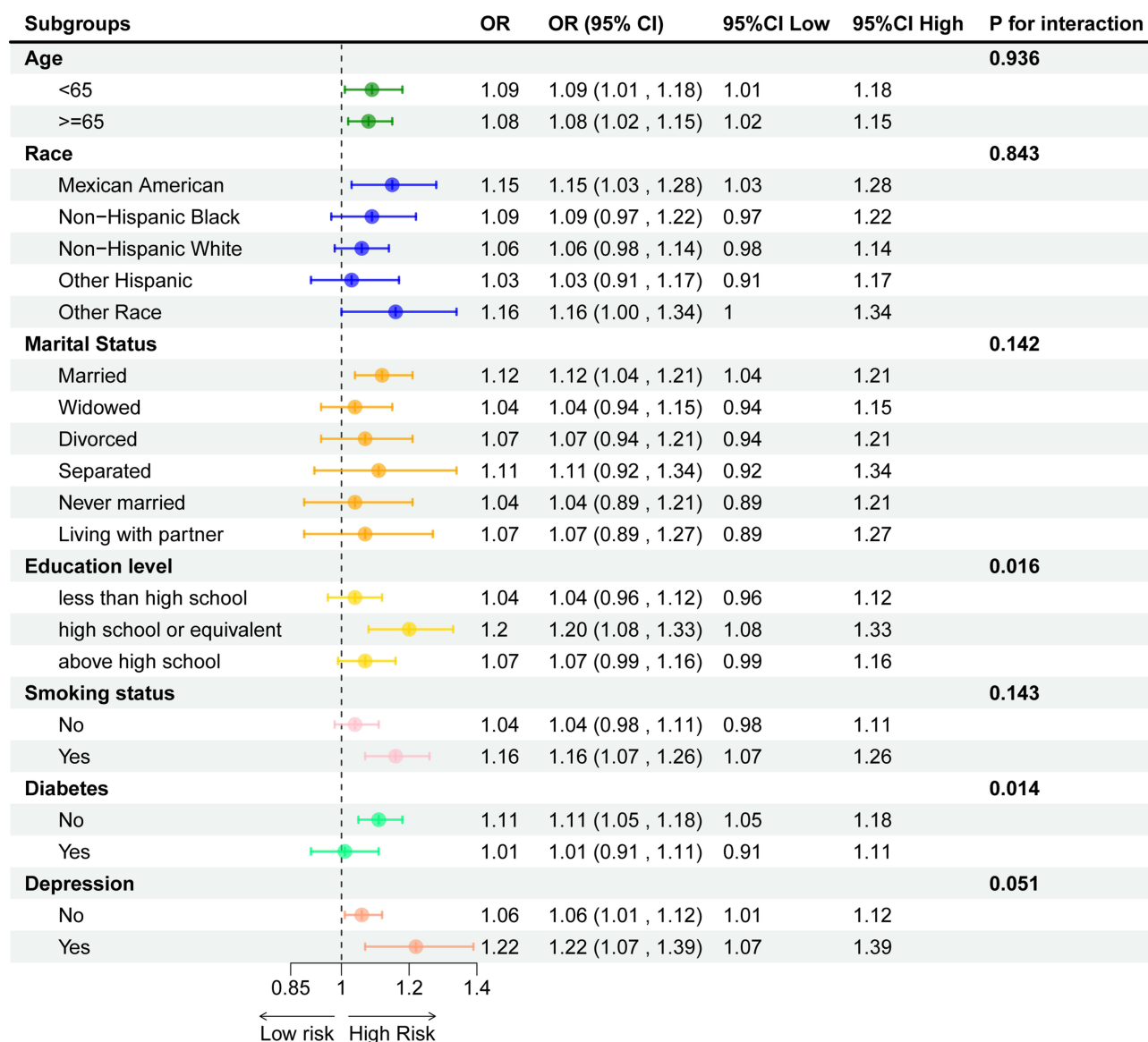


Figure 5 Subgroup analysis for associations between the number of VDIs and MUI in postmenopausal women.

However, we found that the additional risk for SUI and MUI caused by an increased number of VDIs was attenuated in postmenopausal women. This may be closely linked to changes in hormone levels and natural aging of tissues in postmenopausal women.³⁴ The female lower urinary tract, vagina and urogenital fascia originate from the urogenital sinus, which is sensitive to steroid hormones, with estrogen receptors widely distributed throughout the vagina, urethra, bladder, pelvic muscles, and ligaments.^{35–37} Estrogen plays a pivotal role in women's health, and its postmenopausal decline results in thinning of the urethral mucosal epithelium, relaxation of the submucosal venous plexus, reduction of vascular density, and decrease in urethral closure pressure.³⁸ Concurrently, studies indicate that collagen concentration increases multifold in postmenopausal women, while the proteoglycan/collagen ratio decreases. These changes compromise the elasticity and mechanical load-bearing capacity of pelvic floor connective tissue and disrupt the structural integrity and dynamic support functions of the musculofascial system.³⁹ Furthermore, the co-occurrence of frailty and osteosarcopenic obesity (OSO) in elderly postmenopausal women exacerbates SUI and MUI through pelvic floor muscle loss, impaired tissue repair capacity, and elevated intra-abdominal pressure.^{40,41} Consequently, the impact of mechanical trauma from VD is diminished in postmenopausal women, leading to a relatively weaker association between the number

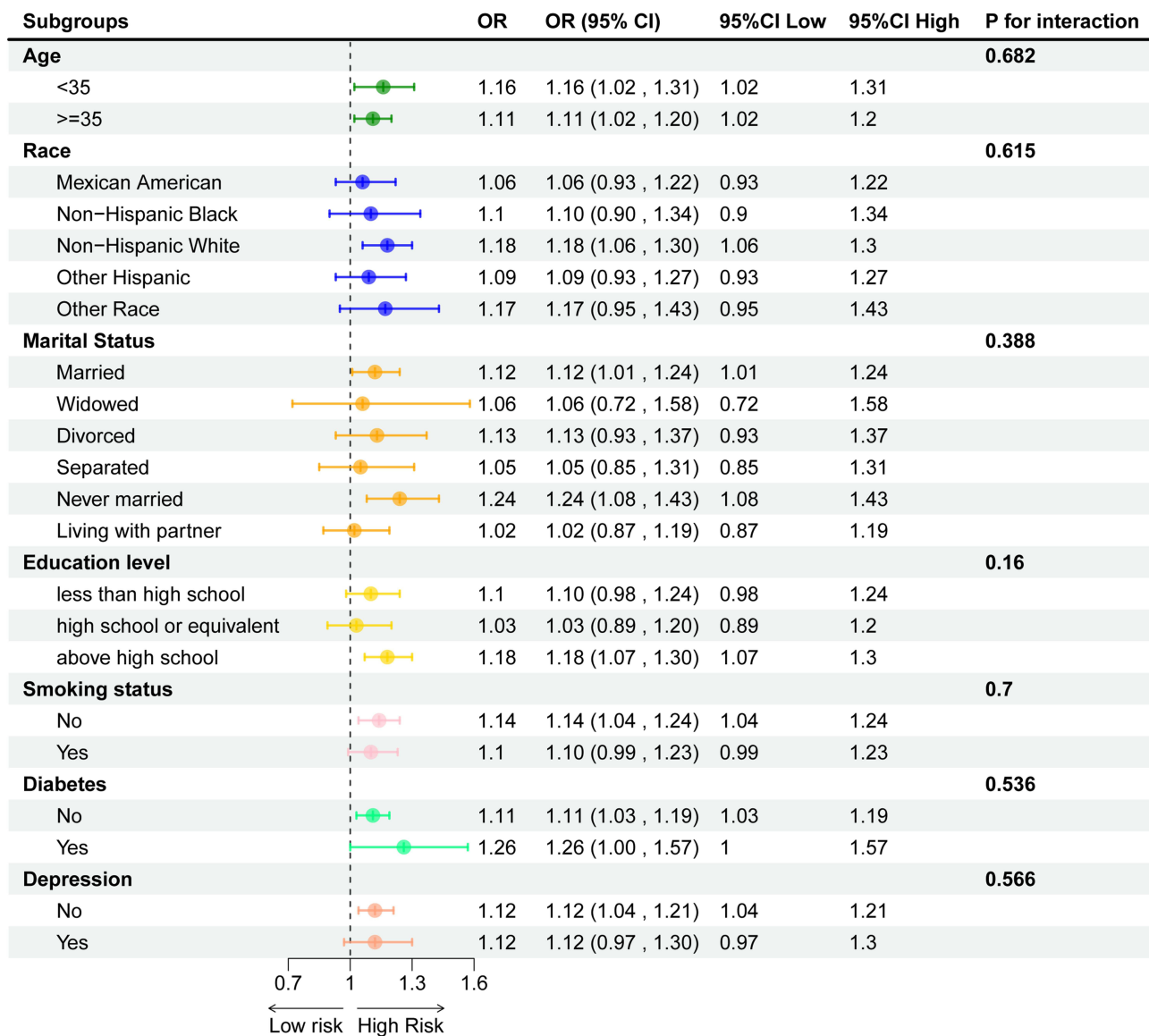


Figure 6 Subgroup analysis for associations between the number of VDs and MUI in premenopausal women.

of VDs and SUI/MUI risk compared to premenopausal women. However, this does not imply that postmenopausal women should disregard the impact of parity on pelvic floor muscles and tissues. For all women, regular pelvic floor muscle exercises and maintaining a healthy lifestyle remain critical measures for preventing SUI and MUI.⁴²

UII is predominantly attributed to bladder overactivity, characterized by involuntary contractions of the detrusor muscle. When these contractions generate sufficient force to overcome urethral resistance, UII occurs.⁴³ Such involuntary activity may correlate with heightened afferent sensory stimuli and increased excitability of bladder smooth muscle.³¹ Epidemiological investigations have demonstrated that VD elevates the prevalence of SUI, while its impact on UII remains limited, consistent with our findings.⁴⁴ VD, as a mechanical insult primarily affecting the pelvic outlet, exerts limited damage to bladder innervation, insufficient to induce enduring neurological alterations in bladder afferent pathways.⁴⁵ Furthermore, studies indicate that nerve injuries during delivery predominantly involve somatic motor fibres, while autonomic fibres (eg, sympathetic and parasympathetic nerves) as well as the central nervous system regulating detrusor muscle function remain unaffected.^{46,47}

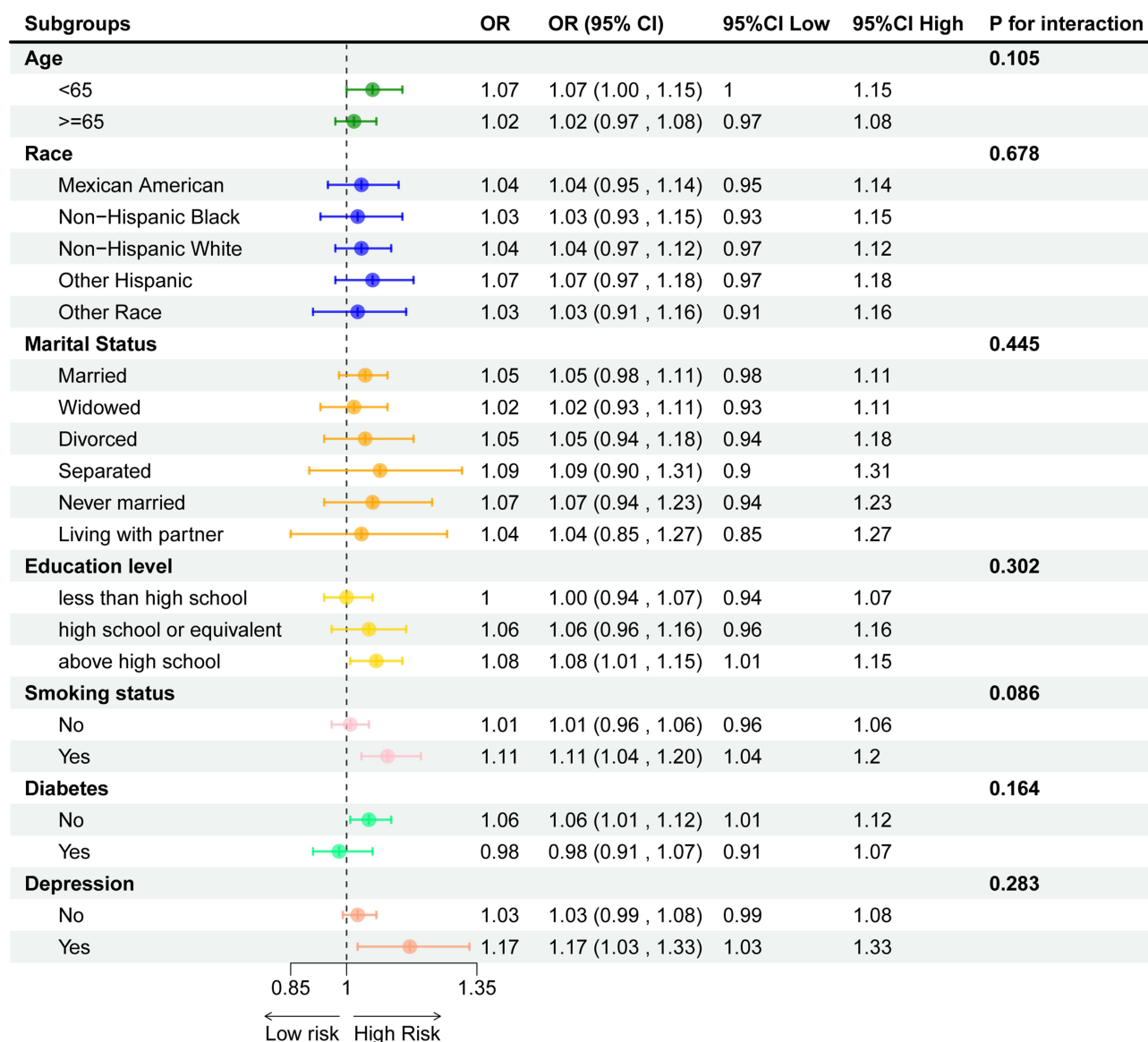


Figure 7 Subgroup analysis for associations between the number of VDIs and SUI in postmenopausal women.

The primary strength of this study lies in its novel exploration of the association between the number of VDIs and the incidence of UI subtypes across premenopausal or naturally postmenopausal women, providing insights into this under-researched relationship. Additionally, leveraging the complex sampling design and sample weights of the NHANES enhanced the generalizability and representativeness of our findings. However, several limitations should be acknowledged. First, the cross-sectional design precludes causal inferences between the number of VDIs and UI. The absence of support from prospective cohort studies or animal models renders our conclusions still speculative. Second, both exposure (number of VDIs) and outcome (UI) variables were self-reported, introducing potential recall bias and compromising data accuracy. Furthermore, unmeasured factors like congenital pelvic floor dysfunction, individual variations in postpartum rehabilitation, and various delivery-assisted interventions (eg, episiotomy, forceps, or vacuum extraction) may cause residual confounding even after adjusting for multiple confounders, and it is worth noting that the effect of various delivery-assisted interventions on UI remains controversial.⁴⁸ Moreover, inherent limitations in the design of the questionnaire on UI in NHANES likely underestimated clinically validated UI cases. In conclusion, our findings are still hypothesis-generating and that further research is required to validate these correlations.

Conclusion

The findings of this investigation detect an inverted U-shaped association between the number of VDs and the occurrence of SUI, an L-shaped relationship was observed with MUI, while no detectable correlation was found with UUI. Notably, the additional risk conferred by increased VDs exhibited attenuated effects on both SUI and MUI in postmenopausal women compared to their premenopausal counterparts. These results elucidate the complex relationship between the number of VDs and UI, providing insights into the disparities in UI risk profiles between pre- and postmenopausal women. Clinicians need to pay special attention to premenopausal women with multiple VDs, performing prenatal and postnatal pelvic floor muscle strength assessments. For women identified with weakened musculature, early intervention may be beneficial. Future studies are needed to validate these observations and elucidate underlying mechanisms to inform early prevention and management strategies that could potentially reduce the UI burden.

Data Sharing Statement

We strongly agree to update our Data Availability to the following: This study analyzed publicly available datasets. These data can be found here: <https://www.cdc.gov/nchs/nhanes/>. Further inquiries can be directed to the corresponding author.

Ethics Approval and Informed Consent

This secondary analysis utilized de-identified public data from the NHANES. The original NHANES protocols (eg, Protocol #2011-17) received approval from the National Center for Health Statistics (NCHS) Ethics Review Board, with written informed consent obtained from all participants during primary data collection. Our study strictly adhered to the ethical principles of the Declaration of Helsinki (2013 revision) for human subject research. Analysis was conducted exclusively on anonymized datasets without access to personally identifiable information. According to Article 32, Items 1 and 2 of the Measures for Ethical Review of Life Science and Medical Research Involving Human Subjects from China (effective February 18, 2023), “those who utilize legally obtained public data” or “those who conduct research using anonymized information data” are exempt from ethical review. Therefore, this investigation would not require additional ethics approval from the First Affiliated Hospital of Heilongjiang University of Chinese Medicine Ethics Committee. For further details, see the NHANES ethics documentation: <https://www.cdc.gov/nchs/nhanes/irba98.htm>.

Acknowledgments

We thank the NHANES participants and staff for their contributions.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study was supported by the following sources: the National Natural Science Foundation of China (No.82474566); The National Natural Science Foundation of China (No.82004403); The Natural Science Foundation of Heilongjiang Province (No. LH2024H048); China Academy of Chinese Medical Sciences (CACMS) Science and Technology Innovation Project (No.CI2021A00404); Cultivation Program of the National Natural Science Foundation of China (No.PYMS202501005).

Disclosure

The authors report no conflicts of interest in this work.

References

- Haylen BT, de Ridder D, Freeman RM, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. *Int Urogynecol J*. 2010;21(1):5–26. doi:10.1007/s00192-009-0976-9
- Bardsley A. An overview of urinary incontinence. *Br J Nurs*. 2016;25(18):S14–S21. doi:10.12968/bjon.2016.25.18.S14
- Abufaraj M, Xu T, Cao C, et al. Prevalence and trends in urinary incontinence among women in the United States, 2005–2018. *Am J Obstet Gynecol*. 2021;225(2):166.e1–166.e12. doi:10.1016/j.ajog.2021.03.016
- Minassian VA, Yan X, Lichtenfeld MJ, Sun H, Stewart WF. The iceberg of health care utilization in women with urinary incontinence. *Int Urogynecol J*. 2012;23(8):1087–1093. doi:10.1007/s00192-012-1743-x
- Wu JM, Matthews CA, Conover MM, Pate V, Jonsson Funk M. Lifetime risk of stress urinary incontinence or pelvic organ prolapse surgery. *Obstet Gynecol*. 2014;123(6):1201–1206. doi:10.1097/AOG.0000000000000286
- Tähtinen RM, Cartwright R, Tsui JF, et al. Long-term impact of mode of delivery on stress urinary incontinence and urgency urinary incontinence: a systematic review and meta-analysis. *Eur Urol*. 2016;70(1):148–158. doi:10.1016/j.eururo.2016.01.037
- Rekers H, Drogendijk AC, Valkenburg HA, Riphagen F. The menopause, urinary incontinence and other symptoms of the genito-urinary tract. *Maturitas*. 1992;15(2):101–111. doi:10.1016/0378-5122(92)90244-X
- Peschers U, Schaer G, Anthuber C, Delancey JO, Schuessler B. Changes in vesical neck mobility following vaginal delivery. *Obstet Gynecol*. 1996;88(6):1001–1006. doi:10.1016/S0029-7844(96)00338-9
- Mahajan NN, Gaikwad NL, Mahajan KN, et al. The effect of vaginal and cesarean delivery on lower urinary tract symptoms: what makes the difference? *Int Urogynecol J Pelvic Floor Dysfunct*. 2007;18(8):977. doi:10.1007/s00192-007-0344-6
- Davis SR, Lambrinoukaki I, Lumsden M, et al. Menopause. *Nat Rev Dis Primers*. 2015;1:15004. doi:10.1038/nrdp.2015.4
- Anger JT, Saigal CS, Litwin MS. Urologic Diseases of America Project. The prevalence of urinary incontinence among community dwelling adult women: results from the national health and nutrition examination survey. *J Urol*. 2006;175(2):601–604. doi:10.1016/S0022-5347(05)00242-9
- Robinson D, Toozs-Hobson P, Cardozo L. The effect of hormones on the lower urinary tract. *Menopause Int*. 2013;19(4):155–162. doi:10.1177/1754045313511398
- Lin AD, Levin R, Kogan B, et al. Estrogen induced functional hypertrophy and increased force generation of the female rabbit bladder. *Neurourol Urodyn*. 2006;25(5):473–479. doi:10.1002/nau.2025
- Aikawa K, Sugino T, Matsumoto S, Chichester P, Whitbeck C, Levin RM. The effect of ovariectomy and estradiol on rabbit bladder smooth muscle contraction and morphology. *J Urol*. 2003;170(2 Pt 1):634–637. doi:10.1097/01.ju.0000068723.05004.ca
- Greendale GA, Lee NP, Arriola ER. The menopause. *Lancet*. 1999;353(9152):571–580. doi:10.1016/S0140-6736(98)05352-5
- Losordo DW, Isner JM. Estrogen and angiogenesis: a review. *Arterioscler Thromb Vasc Biol*. 2001;21(1):6–12. doi:10.1161/01.ATV.21.1.6
- Markland AD, Tangpricha V, Mark Beasley T, et al. Comparing vitamin d supplementation versus placebo for urgency urinary incontinence: a pilot study. *J Am Geriatr Soc*. 2019;67(3):570–575. doi:10.1111/jgs.15711
- Vaughan CP, Tangpricha V, Motahar-Ford N, et al. Vitamin D and incident urinary incontinence in older adults. *Eur J Clin Nutr*. 2016;70(9):987–989. doi:10.1038/ejcn.2016.20
- Shao FX, Luo WJ, Lou LQ, et al. Associations of sarcopenia, obesity, and metabolic health with the risk of urinary incontinence in U.S. adult women: a population-based cross-sectional study. *Front Nutr*. 2024;11:1459641. doi:10.3389/fnut.2024.1459641
- Felde G, Engeland A, Hunskar S. Urinary incontinence associated with anxiety and depression: the impact of psychotropic drugs in a cross-sectional study from the Norwegian HUNT study. *BMC Psychiatry*. 2020;20(1):521. doi:10.1186/s12888-020-02922-4
- Lee HY, Rhee Y, Choi KS. Urinary incontinence and the association with depression, stress, and self-esteem in older Korean Women. *Sci Rep*. 2021;11(1):9054. doi:10.1038/s41598-021-88740-4
- Batmani S, Jalali R, Mohammadi M, Bokaei S. Prevalence and factors related to urinary incontinence in older adults women worldwide: a comprehensive systematic review and meta-analysis of observational studies. *BMC Geriatr*. 2021;21(1):212. doi:10.1186/s12877-021-02135-8
- Rortveit G, Daltveit AK, Hannestad YS, Hunskar S, Study NEPINCONT. Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med*. 2003;348(10):900–907. doi:10.1056/NEJMoa021788
- Helfand BT, Evans RM, McVary KT. A comparison of the frequencies of medical therapies for overactive bladder in men and women: analysis of more than 7.2 million aging patients. *Eur Urol*. 2010;57(4):586–591. doi:10.1016/j.eururo.2009.12.025
- Nappi RE, Palacios S. Impact of vulvovaginal atrophy on sexual health and quality of life at postmenopause. *Climacteric*. 2014;17(1):3–9. doi:10.3109/13697137.2013.871696
- Lowder JL, Debes KM, Moon DK, Howden N, Abramowitch SD, Moalli PA. Biomechanical adaptations of the rat vagina and supportive tissues in pregnancy to accommodate delivery. *Obstet Gynecol*. 2007;109(1):136–143. doi:10.1097/01.AOG.0000250472.96672.6c
- Hoff Brækken I, Majida M, Engh ME, K B. Morphological changes after pelvic floor muscle training measured by 3-dimensional ultrasonography: a randomized controlled trial. *Obstet Gynecol*. 2010;115(2 Pt 1):317–324. doi:10.1097/AOG.0b013e3181cbd35f
- Johnson JD, Lamensdorf H, Hollander IN, Thurman AE. Use of transvaginal endosonography in the evaluation of women with stress urinary incontinence. *J Urol*. 1992;147(2):421–425. doi:10.1016/S0022-5347(17)37256-7
- Smith MD, Coppieters MW, Hodges PW. Postural response of the pelvic floor and abdominal muscles in women with and without incontinence. *Neurourol Urodyn*. 2007;26(3):377–385. doi:10.1002/nau.20336
- Navarro Brazález B, Sánchez B, Prieto Gómez V, De La Villa Polo P, McLean L, Torres Lacomba M. Pelvic floor and abdominal muscle responses during hypopressive exercises in women with pelvic floor dysfunction. *Neurourol Urodyn*. 2020;39(2):793–803. doi:10.1002/nau.24284
- Sigurdardottir T, Steingrimsdottir T, Geirsson RT, Halldorsson TI, Aspelund T, K B. Can postpartum pelvic floor muscle training reduce urinary and anal incontinence?: an assessor-blinded randomized controlled trial. *Am J Obstet Gynecol*. 2020;222(3):247.e1–247.e8. doi:10.1016/j.ajog.2019.09.011
- Meyer S, Hohlfeld P, Ahtari C, De Grandi P. Pelvic floor education after vaginal delivery. *Obstet Gynecol*. 2001;97(5 Pt 1):673–677. doi:10.1016/s0029-7844(00)01101-7
- Russo E, Caretto M, Giannini A, et al. Management of urinary incontinence in postmenopausal women: an EMAS clinical guide. *Maturitas*. 2021;143:223–230. doi:10.1016/j.maturitas.2020.09.005

34. Monteleone P, Mascagni G, Giannini A, Genazzani AR, Simoncini T. Symptoms of menopause - global prevalence, physiology and implications. *Nat Rev Endocrinol.* 2018;14(4):199–215. doi:10.1038/nrendo.2017.180
35. Hirai K, Tsuda H. Estrogen and urinary incontinence. *Int J Urol.* 2009;16(1):45–48. doi:10.1111/j.1442-2042.2008.02164.x
36. Stenberg A, Heimer G, Ulmsten U. The prevalence of urogenital symptoms in postmenopausal women. *Maturitas.* 1995;22:S17–S20. doi:10.1016/0378-5122(95)00958-2
37. Bhatia NN, Bergman A, Karram MM. Effects of estrogen on urethral function in women with urinary incontinence. *Am J Obstet Gynecol.* 1989;160(1):176–181. doi:10.1016/0002-9378(89)90114-2
38. Fantl JA, Bump RC, Robinson D, McClish DK, Wyman JF. Efficacy of estrogen supplementation in the treatment of urinary incontinence. the continence program for women research group. *Obstet Gynecol.* 1996;88(5):745–749. doi:10.1016/0029-7844(96)00281-5
39. Verelst M, Maltau JM, Ørbo A. Computerised morphometric study of the paraurethral tissue in young and elderly women. *Neurourol Urodyn.* 2002;21(6):529–533. doi:10.1002/nau.10089
40. Sahiner Z, Mangır N, Güner M, et al. The relationship between urinary incontinence and abdominal muscle thickness in community-dwelling older women undergoing comprehensive geriatric assessment. *Eur Geriatr Med.* 2023;14(6):1319–1325. doi:10.1007/s41999-023-00874-y
41. Gullo G, Etrusco A, Cucinella G, et al. Ovarian tissue cryopreservation and transplantation in menopause: new perspective of therapy in postmenopausal women and the importance of ethical and legal frameworks. *Eur Rev Med Pharmacol Sci.* 2022;26(24):9107–9116. doi:10.26355/eurrev_202212_30660
42. Woodley SJ, Lawrenson P, Boyle R, et al. Pelvic floor muscle training for preventing and treating urinary and faecal incontinence in antenatal and postnatal women. *Cochrane Database Syst Rev.* 2020;5(5):CD007471. doi:10.1002/14651858.CD007471.pub4
43. Nygaard I. Clinical practice. Idiopathic urgency urinary incontinence. *N Engl J Med.* 2010;363(12):1156–1162. doi:10.1056/NEJMcp1003849
44. Chang SR, Lin WA, Chang TC, Lin HH, Lee CN, Lin MI. Risk factors for stress and urge urinary incontinence during pregnancy and the first year postpartum: a prospective longitudinal study. *Int Urogynecol J.* 2021;32(9):2455–2464. doi:10.1007/s00192-021-04788-w
45. Ashton-Miller JA, Delancey JO. On the biomechanics of vaginal birth and common sequelae. *Annu Rev Biomed Eng.* 2009;11:163–176. doi:10.1146/annurev-bioeng-061008-124823
46. Lien KC, Morgan DM, Delancey JO, Ashton-Miller JA. Pudendal nerve stretch during vaginal birth: a 3D computer simulation. *Am J Obstet Gynecol.* 2005;192(5):1669–1676. doi:10.1016/j.ajog.2005.01.032
47. Fowler CJ, Griffiths D, de Groat WC. The neural control of micturition. *Nat Rev Neurosci.* 2008;9(6):453–466. doi:10.1038/nrn2401
48. Langrová P, Vrublová Y. Relationship between episiotomy and prevalence of urinary incontinence in women 2-5 years after childbirth. *small.* 2014;90:98.

International Journal of Women's Health

Publish your work in this journal

The International Journal of Women's Health is an international, peer-reviewed open-access journal publishing original research, reports, editorials, reviews and commentaries on all aspects of women's healthcare including gynecology, obstetrics, and breast cancer. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit <http://www.dovepress.com/testimonials.php> to read real quotes from published authors.

Submit your manuscript here: <https://www.dovepress.com/international-journal-of-womens-health-journal>

Dovepress
Taylor & Francis Group