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Urogynaecology

Vitamin D and stress urinary incontinence in pregnancy: a cross-sectional study

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Objective To assess the association between levels of vitamin D and urinary incontinence (UI) in pregnancy.

Design A cross-sectional study. Secondary analysis of a randomised controlled trial.

Setting Two university hospitals in Norway.

Population A total of 851 healthy, pregnant women >18 years in gestational weeks 18–22 with a singleton live fetus.

Methods Data on UI were collected from a questionnaire at inclusion and serum analysis of 25-hydroxy vitamin D (25(OH)D) was performed. Univariable and multivariable logistic regression analyses were applied to study associations between exposure and outcomes.

Main outcome measures Prevalence of self-reported UI, stress (SUI) and urge (UUI) or mixed UI.

Results In total, 230/851 (27%) of the participants were vitamin D insufficient (25(OH)D <50 nmol/l) and 42% reported to have any UI. Women with 25(OH)D <50 nmol/l were more likely to report any UI (P = 0.03) and SUI (P < 0.01) compared with women with 25(OH)D ≥50 nmol/l. In a univariable logistic regression analysis, serum levels of 25(OH)D <50 nmol/l was associated with increased risk of any UI (odds ratio [OR] 1.5 with 95% CI 1.0–2.1), SUI only (OR 1.7, 95% CI 1.2–2.4), but not mixed UI or UUI only (OR 0.8, 95% CI 0.5–1.5). In a multivariable logistic regression model, serum levels of 25(OH)D <50 nmol/l were associated with a higher risk of experiencing SUI only (OR 1.5, 95% CI 1.1–2.2).

Conclusions Serum 25(OH)D <50 nmol/l was associated with increased risk of any UI, and SUI in particular.

Keywords 25-hydroxy vitamin D, pregnancy, stress urinary incontinence, urinary incontinence, vitamin D insufficiency.

Tweetable abstract Low levels of vitamin D are associated with increased risk of urinary incontinence in pregnancy.

Linked article This article is commented on by M Huebner, p. 1712 in this issue. To view this mini commentary visit https://doi.org/10.1111/1471-0528.16398

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Introduction

Pelvic floor disorders (PFDs) are prevalent, affecting one in three women, and increase with age.[1,2] The main function of the pelvic floor is to support pelvic organs and so maintain continence. Weakness of pelvic floor muscles is associated with urinary incontinence (UI), which is the most frequently reported symptom among PFDs. Pregnancy and vaginal birth are major risk factors for developing UI. The aetiology is complex, including both hormonal and mechanical pregnancy-related changes.[3] In the Norwegian Mother and Child Cohort study, 58% of 43 279 women reported UI at any frequency and 35% reported UI weekly or more in pregnancy week 30.[4]

The vitamin D has an essential role in regulation of calcium and bone homeostasis.[5,6] Parathyroid hormone (PTH) interacts with vitamin D in this regulation, and serum levels of PTH reflect vitamin D status. The vitamin

D receptor, as well as the enzyme necessary for conversion of vitamin D to its active form 1,25-dihydroxyvitamin D, is present in tissues throughout the body, and vitamin D seems to have health effects beyond stimulation of calcium uptake and calcification of the skeleton.[7] There is evidence suggesting the presence of vitamin D receptor in skeletal muscle.[8] Accordingly, numerous studies have found that vitamin D affects muscle strength and function, and low serum levels of vitamin D are associated with reduced muscle mass, strength and performance as well as increased risk of falls in the elderly.[9] Vitamin D status is assessed by measuring the level of circulating 25-hydroxvitamin D (25(OH) D). An optimal 25(OH)D target concentration is lacking; however, there is general agreement that a serum level <50 nmol/l is classified as insufficiency and <30 nmol/l as deficiency.[10,11] Hypovitaminosis D is epidemic and in people of all ages, including pregnant women.[12]

Vitamin D insufficiency has emerged as a risk factor for PFDs. In non-pregnant women low vitamin D level is found to be associated with an increased risk of UI.[13–15] Moreover, a positive correlation between antepartum vitamin D levels and postpartum pelvic floor muscle strength and endurance has been observed.[16] Identification of modifiable risk factors is of high importance to reduce the prevalence, severity and negative consequences of UI. The primary objective of this study was to assess the association of vitamin D and UI in healthy pregnant women. The secondary objective was to examine the associations of calculated free 25(OH)D and PTH with UI.

Methods

This is a secondary analysis of a randomised controlled trial designed to study the effect of exercise during pregnancy on pregnancy-related diseases, and the primary outcome was gestational diabetes mellitus.[17] In this present paper, we used data collected at inclusion in mid-pregnancy (gestational weeks 18–22), before the randomisation procedure. For this reason, we are considering the present study design as a cross-sectional study. Recruitment details are described elsewhere.[17] We included healthy, pregnant, white European women over 18 years of age. Women were included after a routine ultrasound scan at 18–20 weeks of gestation to ensure that participating women were carrying only one live fetus. Exclusion criteria were high-risk pregnancies and/or conditions in which exercise training is contraindicated.[18]

Outcome variables

The main outcome was prevalence of UI at gestational weeks 18–22. UI was self-reported, using Sandvik's severity index.[19,20] Women replying 'Do not have urinary leakage' were classified as continent, and women reporting UI at any frequency were classified as incontinent. Further, UI was classified according to the definitions given in the standardised International Urogynecological Association/International Continence Society terminology of lower urinary tract symptoms.[21] Leakage reported only with activities that increase abdominal pressure was classified as stress urinary incontinence (SUI only), leakage with urge was classified as urge urinary incontinence (UUI only). The combination of both SUI and UUI was classified as mixed urinary incontinence (mixed UI). As a result of low numbers, UUI only and mixed UI were merged into one group in the statistical analyses.

Exposure variables

Blood samples were collected after overnight fasting and sera were stored at −80°C. The following analyses were conducted at Trondheim University Hospital: 25(OH)D and PTH were analysed by electrochemiluminescence immunoassay, calcium was measured using a colorimetric method, and albumin was measured by photometric methods. All assays were delivered by Roche Diagnostics Ltd (Basel, Switzerland). Total calcium was corrected for the albumin concentration. Vitamin D-binding protein was analysed at the Hormone Laboratory, Oslo University Hospital, using an in-house competitive radioimmunoassay with GC-globulin (Sigma-Aldrich Corp, St Louis, MO, USA) and polyclonal anti-GC-globulin antibodies (Dako-Cytomation, Glostrup, Denmark). Reference range, limit of detection and coefficient of analytical variation for the different analyses have been presented elsewhere.[22] Calculation of free 25(OH)D was performed according to Bikle et al.,[23] as reported previously.[22] The definition of vitamin D insufficiency in the present study was based on recommendations by the US Institute of Medicine, and Nordic Nutrition Recommendations, with serum 25(OH)D levels <50 nmol/l classified as insufficiency.[10,11]

A self-administered Food Frequency Questionnaire[24,25] containing around 180 food items was used to collect information about vitamin D and calcium intake at inclusion (gestational weeks 18–22). Women were instructed to provide information about their dietary intake during the last 4 weeks.

Confounding variables

Potential confounding variables were age, body mass index (kg/m^2) in gestational weeks 18–22 and parity. Based on self-reports, modes of delivery in multiparous women were categorised into caesarean section/-s only (n = 29, 8%), uncomplicated vaginal delivery/-ies (n = 273, 75%) and at least one instrumental delivery (n = 61, 17%).

Ethics

Study procedures followed the Helsinki declaration. All women received written information and signed informed consent forms before participation. Participants did not receive any financial compensation. The Regional Committees for Medical and Health Research Ethics approved the study (REK 4.2007.81), and the trial was registered in Clinical trial.gov (NCT 00476567).

Statistical analysis

Analyses were performed using SPSS statistical package version 25 (IBM Corp., New York, NY, USA). Descriptive data are presented as mean with standard deviation (SD) and frequencies (%) when appropriate. The association between primary outcome variables; any UI, SUI only, or mixed UI or UUI only in gestational weeks 18–22 and selected independent background variables, were assessed using univariable logistic regression analyses. Variables found to have an association with the primary outcome variables with a P value <0.20 in the univariate analyses were included in a multivariable logistic regression model. Multivariable logistic regression analyses were performed using backwards selection to evaluate the independent strength of the association between risk factors for experiencing UI symptoms in mid-pregnancy. In each step the variable with the highest P value was removed from the model until all variables were statistically significant with $P < 0.05$. None of the variables in the multivariable logistic regression model were highly correlated (Variance Influencing Factor <2). A 5% level of significance was used throughout.

Core outcome sets

The core outcome set for evaluating maternal care previously suggested[26] was not used when the present trial was designed in 2005 to 2007.

Patient involvement

Patients were not involved when designing the original trial in 2005–2007.

Funding

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Results

In total, 855 women were included in the study and assessed at study entry. Complete data were available for 851 women (Figure 1).

Baseline characteristics of the participants are shown in Table 1. Two in five women (351/851) reported any UI, with 73% (256/351) reporting SUI only and 27% (95/351) reporting UUI or mixed UI. One in three reported onset of UI before the present pregnancy, whereas two in three reported onset during the first 20 weeks of gestation.

Mean (±SD) serum level of 25(OH)D for the whole population was 66.2 ± 24.8 nmol/l. In total, 230/851 (27%) of the participants were vitamin D insufficient (25(OH)D <50 nmol/l). Forty women (4.7%) had vitamin D deficiency (25(OH)D <30 nmol/l), and only five had PTH levels above the upper reference limit (>6.9 pmol/l).

Among women reporting SUI only, 34% (88/256) were vitamin D insufficient, and 25% (24/95) of women reporting UUI only or mixed UI were vitamin D insufficient. Compared with those with 25(OH)D 50–74 nmol/l and ≥75 nmol/l, women with vitamin D insufficiency reported higher occurrence of any UI ($P = 0.03$) and SUI only ($P < 0.01$) (Table 2).

In a univariable logistic regression analysis, serum levels of 25(OH)D <50 nmol/l were associated with increased risk of any UI (odds ratio [OR] 1.5 with 95% CI 1.0–2.1), SUI only (OR 1.7, 95% CI 1.2–2.4), but not mixed UI or UUI only (OR 0.8, 95% CI 0.5–1.5). In a multivariable logistic regression model, there was no increased risk for any UI, or mixed UI or UUI only with serum levels of 25(OH)D <50 nmol/l, but a higher risk of experiencing SUI only (OR 1.5, 95% CI 1.1–2.2). Being multiparous increased the risk of any UI, SUI only and mixed UI or UUI only in both the univariable and multivariable logistic models (Table 3). In an explorative analysis, previous normal vaginal delivery and instrumental delivery increased the OR for UI compared with being nulliparous and previous caesarean delivery only (see Supplementary material, Table S1). Calculated free 25(OH)D was significantly associated with any UI and SUI only in the univariable analysis, but not in the multivariable analysis (Table 3). PTH was not associated with any of the outcome variables in either the univariable or multivariable analyses (Table 3).

Mean total daily intake of vitamin D was 10.4 ± 7.0 µg. Two out of five followed the recommendations of a total daily intake of vitamin D (≥10 µg) and half met the recommendations for intake of fish (≥300 g/week) and calcium (≥900 mg/day). There was no difference in vitamin D nutritional status between continent and incontinent women (Table 1).

Discussion

Main findings

In this cross-sectional study including 851 healthy, pregnant women, 40% reported having any UI and 27% were found to have vitamin D insufficiency (25(OH)D <50 nmol/l) in mid-pregnancy. We found that UI in general, and SUI in particular, were more frequent in women with vitamin D insufficiency. The risk of SUI increased by 50% in vitamin-D-insufficient

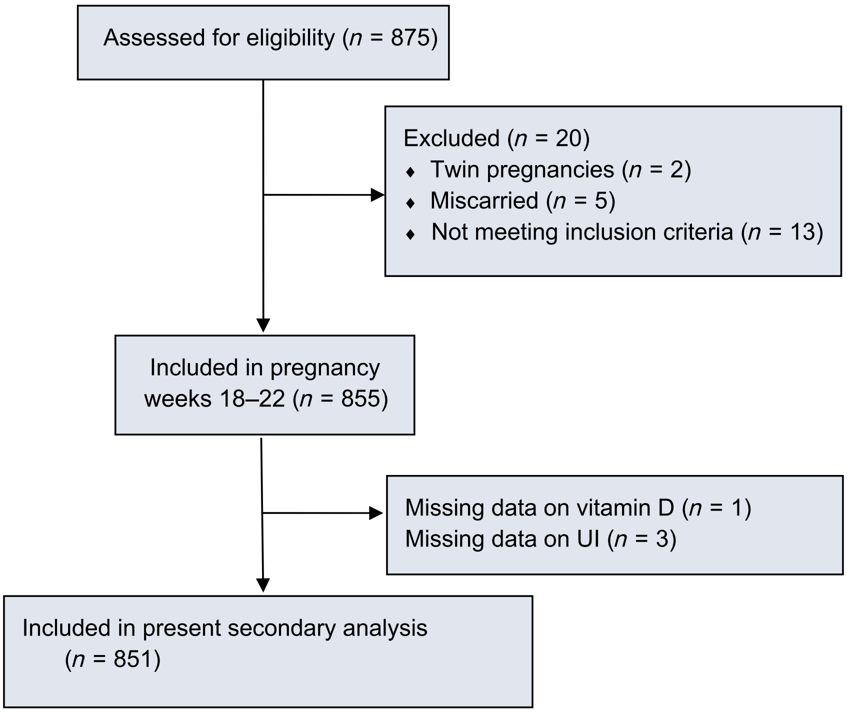

Figure 1. Flow chart of study participants.

pregnant women after adjusting for potential confounders. Free 25(OH)D was associated with any UI and SUI, whereas no association was observed between PTH and UI. Being multiparous was associated with increased risk of any UI, SUI only and mixed UI or UUI only.

Strengths and limitations

Strengths of the present study include assessment of several parameters in the vitamin D endocrine system, a large sample size, the use of a validated questionnaire and standardised procedures for blood sampling and analyses.

The study has some limitations. Liquid chromatography-tandem mass spectrometry is considered the reference standard technique for analysis of 25(OH)D, so the analytic method (electrochemiluminescence immunoassay) applied may be a possible limitation of the study. Calculation of free 25(OH)D may overestimate the level compared with direct measurement.[27,28] Further, the Food Frequency Questionnaire used in this study may overrate the intake of vitamin D.[24] Participants had normal weight and were fair-skinned. Results may therefore not be representative for obese women or other ethnic groups. Prevalence of UI was based on self-reports only. Anatomical reasons for SUI were not explored in this study.

Interpretation

Considering that serum 25(OH)D <50 nmol/l was associated with increased risk of any UI and SUI in the present study, our findings support the hypothesis that vitamin D plays a role in the function of the pelvic floor muscles during pregnancy. Whether vitamin D deficiency (25(OH)D <30 nmol/l) is associated with an even higher risk of UI could not be assessed because of the small number of women in this category.

Some argue that measurement of total 25(OH)D levels may be misleading in conditions, such as pregnancy, where the relationship between total and free 25(OH)D levels is altered.[29] Hence, we assessed the relation between calculated free 25(OH)D and UI and could show associations similar to those for total 25(OH)D. We observed no association between serum levels of PTH and UI. PTH interacts with vitamin D in the regulation of mineral metabolism; it is suppressed by sufficient 25(OH)D levels and increases in a state of vitamin D deficiency. The 25(OH)D threshold above which PTH is maximally suppressed is considered to indicate sufficient levels. Kramer et al. reported this threshold to be 82 nmol/l (95% CI 61–103 nmol/l) in pregnancy.[30] Based on this we would expect that women with 25(OH)D above this threshold displayed the lowest risk for UI. However, no further risk reduction was seen in women with 25(OH)D levels >75 nmol/l, which may be explained by the relationship between 25(OH)D and PTH that differs between the pregnant and non-pregnant state.[30]

The novelty of this study is that we address the vitamin D endocrine systems association with UI among pregnant women. One previous study has reported a positive

Table 1. Characteristics of women reporting being continent or experiencing UI (SUI only versus UUI only or mixed UI) in gestational weeks 18–22

	Total *n* = 851	Continent *n* = 500	SUI only *n* = 256	UUI or mixed UI *n* = 95
Demographic variables				
Age (years)	30.5 ± 4.4	30.0 ± 4.2	**31.2 ± 4.5****	**31.2 ± 4.6***
Weight (pre-conception) (kg)	65.8 ± 9.8	65.4 ± 9.8	66.5 ± 9.5	66.1 ± 10.5
BMI (pre-conception) (kg/m^2)	23.1 ± 3.2	23.0 ± 3.1	23.4 ± 3.1	23.2 ± 3.5
Weight (gestational weeks 18–22) (kg)	70.6 ± 10.0	70.2 ± 10.1	71.2 ± 9.7	70.6 ± 10.7
BMI (gestational weeks 18–22) (kg/m^2)	24.8 ± 3.2	24.7 ± 3.2	25.1 ± 3.2	24.8 ± 3.6
Parity				
Nulliparous	484 (57)	343 (69)	**100 (39)*****	**41 (43)*****
Multiparous	367 (43)	157 (31)	**156 (61)*****	**54 (57)*****
Married/cohabitant	830 (98)	487 (98)	251 (98)	92 (97)
In paid work	796 (94)	469 (94)	239 (93)	88 (93)
Education				
≤13 years at school	95 (11)	51 (10)	**32 (13)***	12 (13)
≤4 years at university	329 (39)	210 (42)	**84 (33)***	35 (37)
>4 years at university	427 (50)	239 (48)	**140 (55)***	48 (51)
Vitamin D endocrine system				
25(OH)D (nmol/l)	66.2 ± 24.8	67.8 ± 24.7	**62.8 ± 24.7****	66.8 ± 24.9
25(OH)D categorised				
<50 nmol/l	230 (27)	118 (24)	88 (34)	24 (25)
50–74 nmol/l	322 (38)	196 (39)	87 (34)	39 (41)
≥75 nmol/l	299 (35)	186 (37)	81 (32)	32 (34)
Calculated free 25(OH)D (pmol/l)	15.3 ± 5.9	15.8 ± 6.0	**14.4 ± 5.7****	15.1 ± 5.8
PTH (pmol/l)	2.78 ± 1.09	2.72 ± 1.05	2.84 ± 1.07	2.92 ± 1.33
Albumin (g/l)	36.68 ± 2.04	36.65 ± 1.98	36.64 ± 2.04	36.93 ± 2.33
DBP (μmol/l)	5.8 ± 0.8	5.7 ± 0.8	5.8 ± 0.8	**5.9 ± 0.8***
Calcium (mmol/l)	2.268 ± 0.069	2.270 ± 0.067	2.267 ± 0.072	2.265 ± 0.074
Corrected calcium (mmol/l)	2.336 ± 0.061	2.338 ± 0.058	2.335 ± 0.067	2.329 ± 0.063
Vitamin D nutritional status				
Daily total vitamin D intake (μg)	10.4 ± 7.0	10.1 ± 6.9	10.9 ± 7.1	10.8 ± 7.3
Daily total vitamin D intake <10 μg	506 (60)	305 (61)	147 (57)	54 (57)
Daily vitamin D from supplements (μg)	5.5 ± 6.5	5.4 ± 6.5	5.7 ± 6.6	5.5 ± 6.4
Daily vitamin D from supplements ≥10 μg	156 (18)	83 (17)	52 (20)	21 (22)
Daily intake of calcium (mg)	972.5 ± 370.6	966.0 ± 361.5	972.5 ± 353.9	1007.1 ± 455.3
Daily intake of calcium <900 mg	394 (46)	238 (48)	111 (43)	45 (47)
Daily intake of fish (g)	54.4 ± 38.1	52.3 ± 38.1	57.5 ± 36.9	57.6 ± 40.9
Intake of fish <300 g/week	387 (46)	239 (48)	105 (41)	43 (45)

Bold indicates significant findings.
BMI, body mass index; DBP, vitamin D-binding protein.
Data are mean ± standard deviation or *n* (%).
Continent versus SUI only or continent versus UUI or mixed UI: $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

correlation between antepartum vitamin D levels and pelvic floor muscle strength and endurance 8 weeks postpartum, and women with antenatal vitamin D levels ≥15 ng/ml (≥37.5 nmol/l) reported fewer postpartum urinary symptoms.[16] However, these findings were not statistically significant, and Aydogmus et al. based their measurement of UI on the quality of life measure Urinary Distress Inventory short form (UDI-6).[16] Furthermore, vitamin D level was assessed in pregnancy and pelvic floor muscle strength was assessed 8–10 weeks postpartum.[16] However, our findings comply with studies showing that non-pregnant women with UI have lower levels of vitamin D compared with continent women.[13–15] Two of these studies included women referred to the hospital with gynaecological disorders,[14,15] and one study used data from a national health survey.[13]

Strong and well-functioning pelvic floor muscles are important to obtain continence. Vitamin D is increasingly recognised to play an important role in normal muscle function. Whether vitamin D affects muscle function directly via vitamin D receptor in skeletal muscles or indirectly via systemic changes in calcium and phosphate levels is still a subject of debate.[8] Findings indicate that vitamin

Table 2. Prevalence of any UI, SUI only and mixed UI or UUI only according to serum level of 25(OH)D

	25(OH)D <50 nmol/l *n* = 230	25(OH)D 50–74 nmol/l *n* = 322	25(OH)D ≥75 nmol/l *n* = 299	*P*-value
Any UI	112 (49)	126 (39)	113 (38)	0.03
SUI only	88 (38)	87 (27)	81 (27)	<0.01
Mixed UI or UUI only	24 (10)	39 (12)	32 (11)	0.79

D affects the diameter and number of type II (fast twitch) muscle fibres, and that myopathy is caused by type IIA muscle fibre atrophy.[31] Type II fibres, which are the first to be recruited, predominantly generate energy anaerobically for a quick and powerful contraction, and exert 20% more force than type I (slow-twitch) fibres.[32] Hence, atrophy of fast type II muscle fibres may impede efficient closing of the urethra during activities with increased intra-abdominal pressure, resulting in SUI.[33]

During pregnancy, physiological changes such as increased intra-abdominal pressure and pregnancy-related hormonal changes may lead to reduced strength and reduced supportive and sphincter functions of the pelvic floor muscles.[3] Pregnancy contributes to PFDs later in life,[34] and both a history of UI before pregnancy and incident antenatal UI significantly increase the risk for persistent postpartum UI.[34–41] In line with this, we observed that being multiparous was the strongest predictor for all types of UI.

A general consensus for an optimal level of 25(OH)D is lacking both in the pregnant and non-pregnant state with definition of vitamin D insufficiency ranging from 25–30 nmol/l up to 100 nmol/l.[42] We classified serum 25(OH)D levels <50 nmol/l as insufficiency.[10,11] In the present population, 27% were vitamin D insufficient (25(OH)D <50 nmol/l) in mid-pregnancy (gestational week 18–22). The numbers are in concurrence with two Scandinavian studies reporting vitamin D insufficiency in 24% of women of European heritage in first trimester[43] and 65% of fair-skinned women in the third trimester.[44]

Multiple factors affect vitamin D status, including ethnicity, intake of vitamin D, obesity, season of the year and latitude. Given the lack of agreement concerning optimal serum levels of vitamin D, the dosage of supplementation, both in pregnancy and the non-pregnant state, is also debated. The Nordic Nutrition Recommendation regarding vitamin D intake is ≥10 μg/day for adults, including pregnant and breastfeeding women.[10] In our population, 60% reported intake below the recommendations. In comparison, Brembeck et al., found that 39% of the pregnant women had a vitamin D intake >10 μg per day.[44] Previous studies have shown that low circulating 25(OH)D levels in pregnancy have been associated with numerous adverse maternal and offspring outcomes. Developmental origins of disease have gained increasing attention, and maternal hypovitaminosis D during fetal life is one of the factors suggested to be of significance for future disease, including osteoporosis and cardiovascular disease.[45–47]

Table 3. Unadjusted and adjusted OR with 95% CI for women reporting urinary incontinence in gestational week 18–22, *N* = 851

	Any UI *n* = 351		SUI only *n* = 256		Mixed UI or UUI only *n* = 95	
	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
25(OH)D						
<50 nmol/l	1.5 (1.0–2.1)*	–	1.7 (1.2–2.4)**	1.5 (1.1–2.2)*	0.8 (0.5–1.5)	–
50–74 nmol/l	1	–	1	1	1	–
≥75 nmol/l	0.9 (0.7–1.3)	–	1.0 (0.7–1.4)	1.1 (0.8–1.6)	0.9 (0.5–1.4)	–
Calculated free 25(OH)D	1.0 (0.9–1.0)*	–	1.0 (1.0–1.0)*	–	1.0 (1.0–1.0)	–
PTH	1.1 (1.0–1.3)	–	1.1 (0.9–1.2)	–	1.1 (0.9–1.4)	–
Age	1.1 (1.0–1.1)***	–	1.0 (1.0–1.1)**	–	1.0 (1.0–1.1)	–
Body mass index	1.0 (1.0–1.1)	–	1.0 (1.0–1.1)	–	1.0 (0.9–1.1)	–
Parity						
Nulliparous	1	1	1	1	1	1
Multiparous	3.3 (2.4–4.3)***	3.3 (2.4–4.3)***	2.8 (2.1–3.8)***	2.7 (2.0–3.7)***	1.9 (1.2–2.9)**	1.9 (1.2–2.9)**

*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.

Conclusion

In this study of 851 pregnant women 230 (27%) had vitamin D insufficiency in mid-pregnancy. We observed that UI in general, and SUI in particular, was more frequent in those with vitamin D insufficiency. In addition, parity was associated with a three-fold increase in odds of SUI. The low adherence to nutritional recommendations is of concern and should be highlighted when prevention strategies are discussed. Given the high prevalence of UI among pregnant women, and a risk of persistent UI postpartum, further evaluation of the role of vitamin D is warranted. Future research should study the gap between biology and clinical implications of vitamin D, also regarding pelvic floor muscle strength and function in women of all ages. Further, well-designed randomised controlled trials are needed to study the potential complementary effect that combining vitamin D supplementation and pelvic floor muscle training may have on pregnant women with UI.

Disclosure of interests

The authors report no conflict of interest. Completed disclosure of interest forms are available to view online as supporting information.

Contribution to authorships

SNS participated in planning of the main study, coordinated the data collection, organised the training programme, initiated the present paper, participated in analysis of the data, wrote the first draft and finalised the manuscript. SM, the principal investigator, initiated and planned the main study, supervised the training programme and participated in the interpretation of the data as well as finalising the manuscript. MKG initiated and performed the study of vitamin D and related parameters and participated in interpretation of results, revising and finalising of the manuscript. US initiated and performed the study of vitamin D and related parameters, contributed with expertise in endocrinology, in interpretation of results, revising and finalising the manuscript. AKS participated in interpretation of results, revising and finalising the manuscript. KÅS participated in the planning of the main study, interpretation of the results, revising and finalising the manuscript. HHJ participated in the data analyses, interpretation of the results as well as drafting and finalising the manuscript.

Ethics approval

The Regional Committees for Medical and Health Research Ethics approved the study; 1 March 2007 (REK 4.2007.81).

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Unadjusted and adjusted OR with 95% CI for women reporting urinary incontinence in gestational weeks 18–22, n = 851. ■

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