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SECOND TRIMESTER SERUM RELAXIN CONCENTRATIONS ARE ASSOCIATED WITH PELVIC ORGAN PROLAPSE FOLLOWING CHILDBIRTH

Hypothesis / aims of study

Relaxin is a hormone produced exclusively by the corpus luteum[1] and typically peaks in the 1st trimester,[2] Serum levels then gradually decrease through the third trimester[3], remain stable in labour, and then decrease below the sensitivity of the assay three days post-partum.[1] Relaxin has been hypothesized to facilitate connective tissue remodelling through its effect on collagenase while reducing levels of tissue inhibitor of metalloproteinase (TIMP).[4] Lower relaxin concentrations have been observed in early pregnancy in women who develop urinary incontinence later in pregnancy.[5] We sought to determine the relationship between relaxin concentrations at the end of the 2nd trimester with the development of post-partum pelvic organ prolapse (POP) or stress urinary incontinence (SUI).

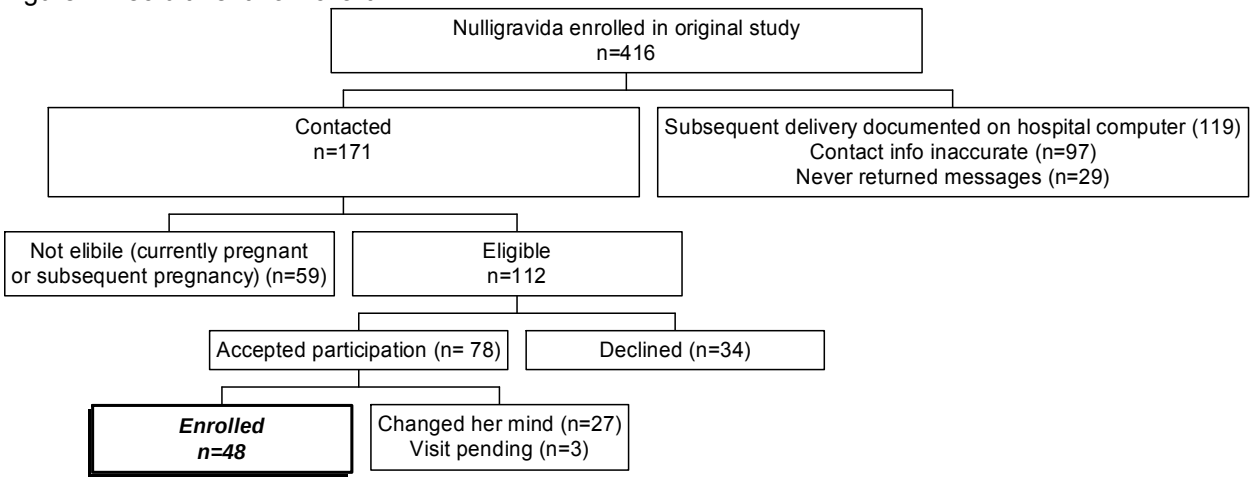
Study design, materials and methods

Primigravida who had participated in a previous study on preterm labour were approached for enrolment.[6] Women who had a subsequent pregnancy or were pregnant were excluded. Evaluations included pelvic organ prolapse quantification system (POPQ), Urogenital Distress Inventory short form (UDI-6), and a cough stress test. Presence of POP was defined as POPQ stage ≥2; subjective stress urinary incontinence (SUI) as a positive response on question #3 of the UDI-6 (i.e. score ≥ 1); and objective SUI as a positive cough stress test with a bladder volume ≥150cc, as documented on voided volume immediately following stress test.

Results

Forty-eight women (mean age 31yr) were evaluated at least one year post-partum (mean 1 year, 8 ½ months). All but one (34 5/7wks) delivered at term (>37 weeks). Mean body mass index was 28 (SD 6.8). Most (73%) were non-smoker, 15% were ex-smoker, 4% smoked less than 10 cig/day and only 8% smoked > 10cig/day. All were White. Recruitment flow chart is shown in Figure 1.

Figure 1 Recruitment flow chart



Obstetrical variables are in Table 1. One third (35%) of the population nursed for 6 months or more, 31.3% between 3-6 months. Only 16.7% of the population chose not to nurse their infant. Outcome measures are presented in Table 2.

Serum relaxin concentrations were available for at least one time point in 45 women (Table 3). Relaxin tended to be higher but to decline significantly faster from 24 to 28 weeks in women with pelvic floor dysfunction (PFD), i.e. with either POP or SUI (Student t-test for unequal variance).

Using a cut-off of 1200 pg/ml (typical peak according to Bell et al[3]), women with PFD were more likely to have elevated relaxin concentrations at 24 and 28 weeks (x^2, P<0.02 for all).

Table 1 Obstetrical parameters of study population (n=48)

Delivery route	SVD: 47.9% Forceps: 8.3% Vacuum: 22.9% Caesarean: 16.7%
Episiotomy	33.3%
Epidural/spinal	79%
Foetal weight (gm, SD)	3609 (497)
Foetal head circumference (cm, SD)	35 (1.5)
2nd stage of Labour duration (hh:mm) (SD)	2:22 (1:41)
Breastfeeding duration (months)	5.6 (5.5)

SVD: spontaneous (unassisted) vaginal delivery, SD: standard deviation

Table 2 Study outcomes (n=48)

Prolapse (POPQ stage >2)	33.3%
Subjective SUI	45.8%
Objective SUI (n=25)	24%
UDI-6 total score (0-3)	0.42
Question #3 score (0-3)	0.58

Table 3 Relaxin level amongst women with or without prolapse (POP), and with or without subjective stress urinary incontinence (SUI).

Relaxin (pg/ml) (mean, standard error)	n	24 weeks	P	n	28 weeks	P	n	Change (28-24w)	P
Overall	45	866 (650)		43	824(527)		43	-42 (205)	
POP	15	1111 (233)	0.2	15	968 (193)	0.3	15	-143 (63)	**0.04**
No POP	30	744 (82)		28	747 (67)		28	13 (30)	
SUI	20	1014 (196)	0.2	19	924 (161)	0.3	19	-143 (51)	**0.005**
No SUI	25	746 (73)		24	744 (67)		24	38 (31)	

(Student t-test for unequal variance)

Interpretation of results

These data demonstrate that post-partum PFD is associated with both higher (albeit not significantly) but significantly more rapidly decreasing relaxin concentrations at the end of the 2nd trimester. These finding are at variance with Kristiansson, who measured relaxin level in the 1st trimester. It is possible that either an abrupt fall in relaxin concentration or a delayed mid-gestational peak in women with PFD may account for this discrepancy.

Concluding message

The role of relaxin in protecting the pelvic floor appears complex and time sensitive, but relevant to the pathogenesis of pelvic floor dysfunction.

References

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