Different influences of physiological and medicamentous hyperprolactinemia on calcium metabolism in rats – experimental study

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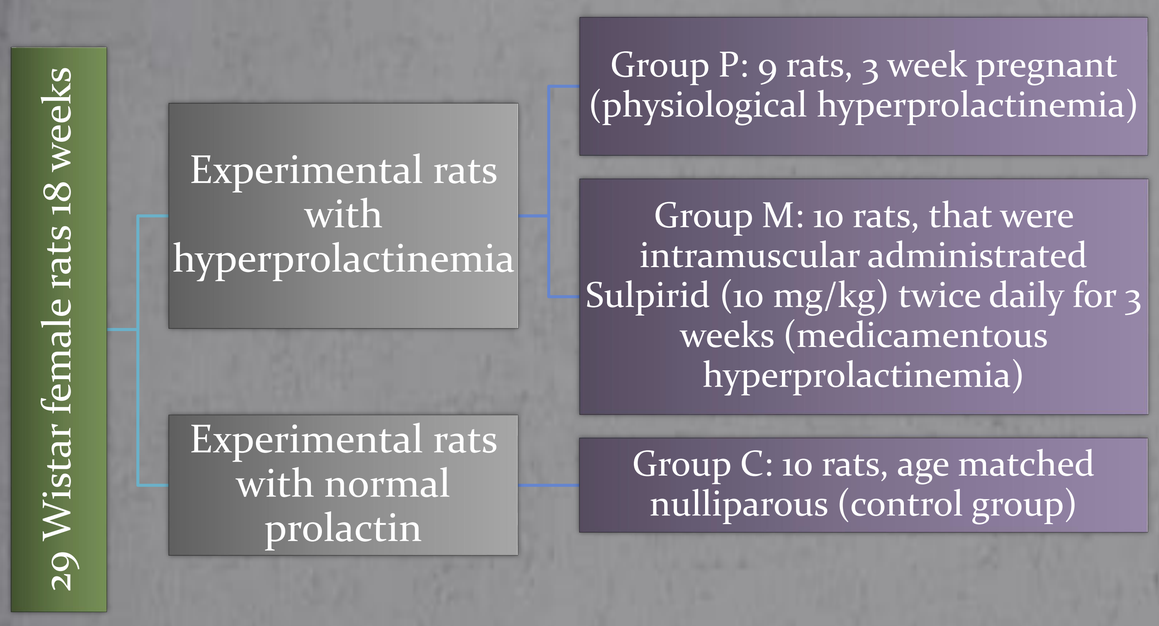
Introduction

The mechanism by which hyperprolactinemia in pregnancy leads to mild and reversible changes in maternal skeletal system and medicamentous hyperprolactinemia causes more detrimental effects, is not completely clarified.

The aim of the study

We conducted the experimental study to compare prolactin receptor gene (*Prlr)* expression in the duodenum, vertebra and kidney, during physiological and medicamentous hyperprolactinemia which could influence calcium homeostasis.

Experimental design

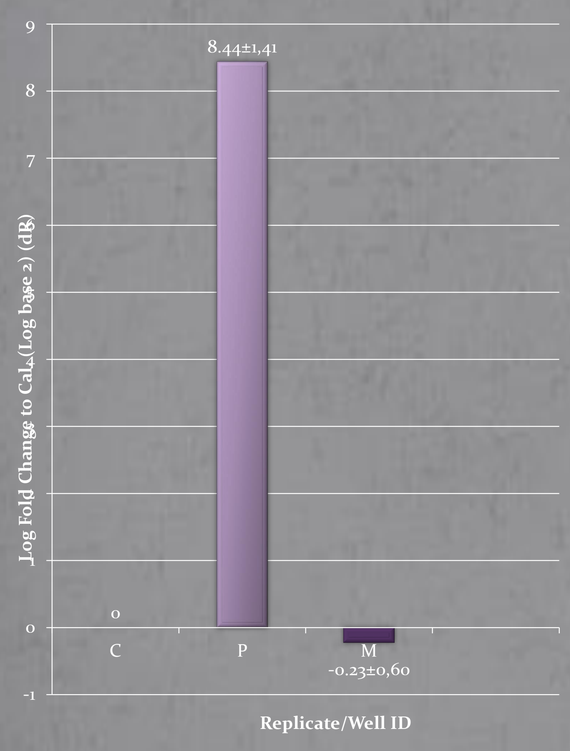


Laboratory results

	P-group	M-group	C-group
PRL (pg/mL) X±SD	181,80±29,65 [a]	182,03±57,80 [a]	105,38±28,34
s-Ca++ (mmol/L) X±SD	0,5±0,2 [a]	1,15±0,04 [a]	1,12±0,04
s-P (mmol/L) X±SD	2,42±0,46 [c]	2,14±0,48	2,05±0,19
u-Ca (mmol/24h) X±SD	3,90±0,46 [a]	4,31±1,11 [b]	3,05±0,58
u-P (mmol/24h) X±SD	141,15±20,65 [a]	50,58±9,77	45,54±7,99
TP1NP (pg/mL) X±SD	489,22± 46,77 [a]	309,60±36,74 [c]	361,90±53,01

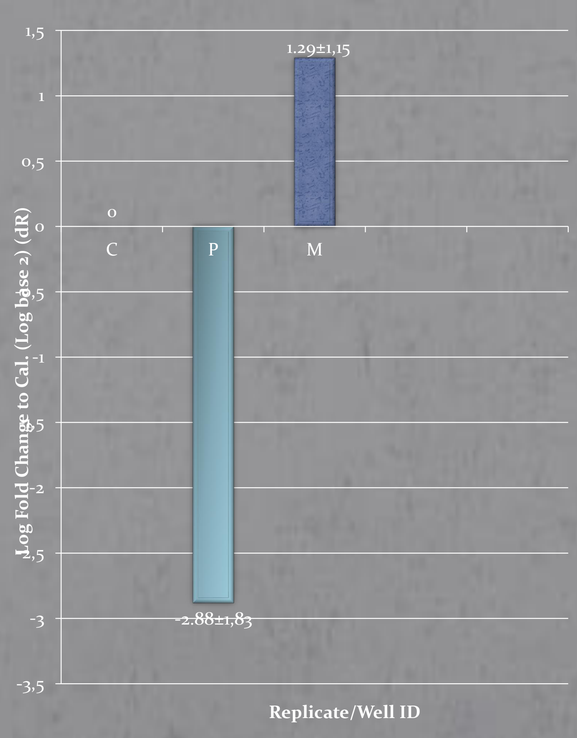
[a] p<0.001; [b] p<0.01 ; [c] p<0.05;

Relative expression of mRNA *Prlr* in duodenum



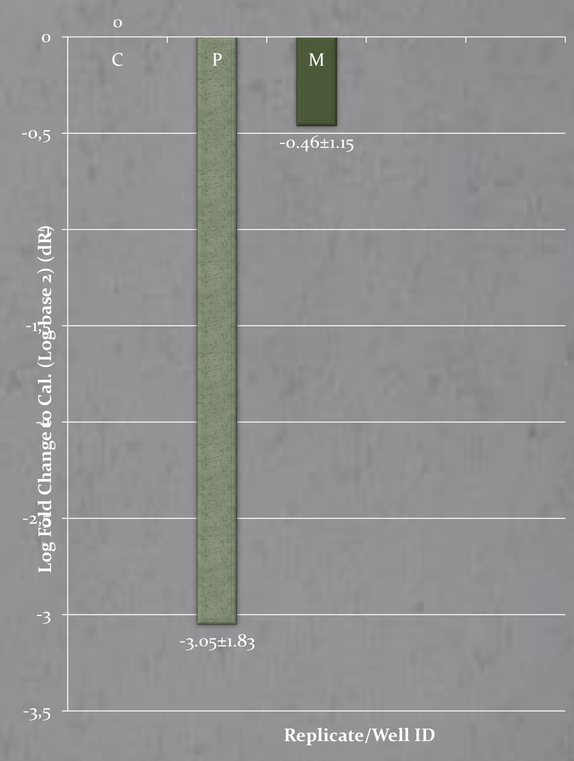
Log2(P/C) vs (M/C) p<0,001

Relative expression of mRNA *Prlr* in vertebra



Log2(P/C) vs (M/C) p<0,001

Relative expression of mRNA *Prlr* in kidney



Log2(P/C) vs (M/C) p<0,01

Conclusion

In medicamentous hyperprolactinemia, down-regulation of *Prlr gene* expression in duodenum could be underlying reason for diminished intestinal calcium absorption. Increased calciumuresis could be partly due to down-regulated *Prlr gene* expression in the kidney. In order to maintain calcium homeostasis, since intestinal absorption is compromised and loosing via kidney elevated, prolactin will rapidly take calcium from skeletal system, thank to increased *Prlr gene* expression in the vertebra, leading to more harmful effect on bone metabolism compering to physiological hyperprolactinemia.

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