MODIFICATION OF PRESSURE AND PLACENTAL PROTEIN EXPRESSION IN PREGNANT SPONTANEOUSLY HYPERTENSIVE RATS (SHR)

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Introduction. During pregnancy, several physiological modifications occur to substain fetal well-being through protective mechanisms. In spontaneously hypertensive rats (SHR) the behaviour of blood pressure during pregnancy has been a matter of controversy in literature (1, 2). The aim of this study was to evaluate, in SHR and their normotensive Wistar Kyoto (WKY) counterparts, the mutual effect between pregnancy and hypertension, and for the first time, the modifications of the nitric oxide synthase isoforms (eNOS, iNOS) and angiotensin receptor (AT1) in placenta.
Materials amd Methods. WKY and SHR were divided into four groups (n=6): pregnant (WKY-P and SHR-P), and age-matched unmated females (WKY-NP and SHR-NP). Body weight, food intake, frequency and systolic blood pressure were evaluated on days 0 (before breeding), 6, 14 and 20 of pregnancy. On day 20 bioelectrical impedance analysis was applied for body composition assessment, then all rats were sacrificed and blood was collected for metabolic parameter determination (glucose, HDL, LDL, triglycerides and cholesterol). Placenta, kidney, and aorta were excised for the evaluation of AT1, eNOS, iNOS expression by Western blot analysis.
Results. Pregnancy led to a significant increase of body weight ($p<0,001$) related to a variation of food intake and fat mass ($p<0,001$). SHR-P displayed a significant decrease in blood pressure and frequency from day 14 ($p<0,05$): a definite fall was observed in late pregnancy (20 day). At the end of gestation a significant increase in AT1 and iNOS was evidenced by Western blot analysis in SHR-P placenta, whereas no significant differences in eNOS expression was observed in both strains.
Conclusions. This animal model offer the opportunity to explore the modification of cardiovascular protein expression and these data may provide new insights into the placental adaptive mechanisms that take place during pregnancy in SHR.

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