Pre-gestational overweight in guinea pig sows induces fetal vascular dysfunction and increased rate of large and small fetuses

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Resumen

In humans, obesity before and during pregnancy is associated with both fetal macrosomia and growth restriction, and long-term cardiovascular risk in the offspring. We aimed to determine whether overweighted pregnant guinea pig sows results in an increased fetal weight at term and the effects on the vascular reactivity in fetal systemic and umbilical arteries. Pregnant guinea pigs were classified as control (n = 4) or high weight (HWS, n = 5) according to their pre-mating weight, and their fetuses extracted at 0.9 gestation (similar to 60 days). Segments of fetal femoral and umbilical arteries were mounted in a wire myograph, where the contractile response to KCl (5-125 mM), and the relaxation to nitric oxide synthase-dependent agents (insulin, 10(-10)-10(-7) and acetylcholine, 10(-10)-10(-5)) and nitric oxide [sodium nitroprusside (SNP), 10(-10)-10(-5)] were determined. Fetuses from HWS (HWSF) were grouped according to their body weight as low (<76 g) or high (>85 g) fetal weight, based on the confidence interval (76.5-84.9 g) of the control group. No HWSF were observed in the normal range. Umbilical arteries from HWSF showed a lower response to KCl and insulin compared with controls, but a comparable response with SNP. Conversely, femoral arteries from HWSF showed an increased response to KCl and acetylcholine, along with a decreased sensitivity to SNP. These data show that overweight sows have altered fetal growth along gestation. Further, large and small fetuses from obese guinea pig sows showed altered vascular reactivity at umbilical and systemic vessels, which potentially associates with long-term cardiovascular risk.

Palabras clave

Palabras clave de autor:cardiovascular; fetus; maternal pregnancy; obesity
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