Exposure in utero to maternal diabetes leads to glucose intolerance and high blood pressure with no major effects on lipid metabolism

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Résumé en anglais	AIM: Recent evidence shows that adult metabolic disease may originate from an adverse fetal environment that can alter organ development and function in postnatal life. This study aimed to analyze the effect of exposure in utero to maternal diabetes on the development of the metabolic syndrome in the offspring. METHODS: Pregnant rats were made diabetic (blood glucose was 20mM) with a single streptozotocin injection on day 0 of gestation. Offspring from diabetic mothers (DMO) and control mothers (CMO) were followed from birth to 12 months of age. In these animals, metabolic parameters, such as glucose tolerance, insulin sensitivity and plasma lipid levels, as well as pancreatic insulin and morphology were studied. RESULTS: Compared with controls, DMO offspring had normal birth weights, but impaired postnatal growth that persisted throughout life. Metabolic tests revealed that DMO offspring also showed impaired glucose tolerance at six months associated with decreased insulin sensitivity and low insulin secretion. In older animals (12 months old), this phenotype persisted, but to a lesser extent. The DMO offspring also presented with high blood pressure and decreased levels of fasting plasma triglycerides, but normal plasma NEFA, and HDL and total cholesterol. CONCLUSION: Altogether, these results show that our model of exposure in utero to maternal diabetes led to normal birth weights, and induced transient glucose intolerance and increased blood pressure with no major effects on lipid metabolism. It also suggests that a hyperglycaemic fetal environment may be able to 'programme' hypertension and glucose intolerance, but not alter lipid metabolism.
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