beta-cell development of their weanling offspring. In contrast, offspring from dams fed a HFD throughout gestation showed no significant changes in islet cell development. Switching the maternal diet during gestation appears to programme weanling progeny inducing diabetogenic effects characterized by compromised beta-cell development and reduced GK expression.

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Effects of maternal obesity on metabolic syndrome in the offspring

A. C. M. Heinsbroek, G. van Dijk;
Animal Physiology, Unit Neuroendocrinology, University of Groningen, Haren, The Netherlands.

Background and Aims: Epidemiological studies have shown that offspring born to women that have been starved during pregnancy in the Second World War in the Netherlands, often had a low birth-weight and higher risk of developing metabolic syndrome (diabetes, obesity, high blood-pressure). This suggests that there are prenatal environmental factors involved in development of the metabolic syndrome.
Not only starvation, but also maternal obesity may be important for the predisposition to the metabolic syndrome in the offspring. In ethnic groups with a high incidence of diabetes and obesity, like the Pima indians, children from diabetic mothers have a higher risk of becoming diabetic compared with children from non-diabetic mothers. This effect is much stronger with maternal diabetes compared with paternal diabetes, suggesting a non-genetic mode of transfer to the offspring.In this project, the effects of maternal obesity on the development of the metabolic syndrome in the offspring were investigated in rats.
Materials and Methods: Maternal obesity was induced by i3vt infusion of the melanocortin 34 antagonist SHU9119 during the last two weeks of pregnancy and the first two weeks of lactation. Male offspring rats from these pregnancies were used at the age of 3–4 months to assess the risk of metabolic syndrome.
Results: Body weight was increased in offspring from SHU mothers, starting from 68 days of age. Food intake was not changed, but water intake was increased in SHU offspring. Respirometric measurements showed increased RQ and a trend to decreased energy expenditure in the dark phase. This was associated to decreased locomotor activity in the dark phase. Oral glucose tolerance tests showed an increase in basal glucose, with no difference in insulin levels, suggesting glucose intolerance in SHU offspring.
Conclusion: We conclude that maternal obesity increases the risk of the metabolic syndrome in the offspring in a non-genetic way. This is mainly due to changes in fat and glucose metabolism, and decreased physical activity, resulting in high body weight and glucose intolerance.
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Intrauterine growth retardation leads to the functional change of insulin secretion in the newborn rat

Q. X. Yuan[1], C. Liu[1], W. De[2];
[1]Department of Endocrinology, The First Affiliated Hospital of Nanjing Medical University, [2]Department of Biochemistry and Molecular Biology, Nanjing Medical University, China.

Background and Aims: Embryonic pancreatic development had a close relation to adult diseases. Intrauterine growth retardation (IUGR) is a common complication of pregnancy and a risk factor for both perinatal disease and disorders of later life, especially impaired glucose tolerance and diabetes. The aim of this study is to investigate the functional change of insulin secretion in IUGR newborn rat and explore the mechanism of reduced blood insulin level.
Materials and Methods: SD pregnant rats were divided into two groups: control group and intrauterine energy restriction group. The animal model of IUGR in rats was made by 50% calorie restriction in pregnant rats from gestational day 15 until term. Pancreata of control and IUGR newborn rats were dissected respectively. After abstraction of totle RNA, RT-PCR was used to study the expression of genes related to insulin synthesis and exocytosis. Intraperitoneal glucose tolerance tests(IPGTT) and ELISA were done to detect the function of pancreatic islet.
Results: Birth weight and pancreas mass of IUGR newborn rats were significantly lower than those of controls. Although no significant differences were observed in genes expression of insulin and PDX-1, the expressions of genes related to insulin exocytosis such as munc13-1, vamp-2, syntaxin1a, rab3a were reduced markedly in IUGR newborn rats. The blood insulin level and insulin secretion response to glucose challenge were also reduced simultaneously in IUGR newborn rats compared with normal newborn rats.