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GH therapy for IUGR rats enhances Pdx1 expression and improves metabolic parameters

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Aims: Intrauterine growth retardation (IUGR) permanently alters pancreatic beta cells function leading to the development of diabetes in adulthood, and reduces the expression of the pancreatic homeobox transcription factor (Pdx1) that plays an important role during this development. To determine whether early adiminstration of two doses of GH may improve metabolic parameters and beta-cell function in the IUGR rats.

Methods: IUGR was induced by restricting food intake during pregnancy. IUGR or control offspring were randomly assigned to receive low dose of GH (2mg/kg/d, IUGR+HGH, n=8) or high dose of GH (5mg/kg/d, IUGR+LGH, n=8) or normal saline (IUGR+NS, n=8). Daily subcutaneous injections of GH were administered at 21 days untill offspings achieve catch-up body weight. Body weight, length, blood pressure, fasting glucose levels were analyzed at 3 weeks, and 10 weeks. Pdx1 mRNA level are measured at 0, 3 weeks, and 10 weeks.

Results: At 3 weeks, IUGR rats were lighter and shorter than the controls (40.62±0.73

vs. 48.16±0.51g; 10.94±0.12 vs. 12.43±0.10 cm, $p<0.05$), and had higher fasting glucose than the controls (5.82±0.18 vs. 4.96±0.29 mmol/L, $p<0.05$). IUGR received either high or low dose of GH achieved catch-up growth at 5 weeks or 6 weeks respectively. At 10 weeks, IUGR+NS exhibited higher fasting glucose levels than the control groups (5.91±0.69 vs. 5.26±0.35, $p<0.05$), while neither IUGR+LGH (5.38±0.82 mmol/L) nor IUGR+HGH (5.18±0.77 mmol/L) showed no significance in fasting glucose compared with controls. No significance was found between blood pressures of each group at 10 weeks. Pdx1 mRNA expreesion is remarkably reduced in IUGR neonatal rats, and can be rescued in HGH+IUGR rats by the time of adulthood, but can not be rescued in LGH+IUGT rats.

Conclusion: These data suggests that the high dose of GH (5mg/kg/d) treatment may benefit metabolic parameter of IUGR rats in adulthood.

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