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Label-free proteomics of the fetal pancreas identifies deficits in the peroxisome in rats with intrauterine growth restriction

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Aim: The objective of the present study was to identify differentially expressed proteins (DEPs) in the pancreas of a fetus with intrauterine growth restriction (IUGR) and to investigate the molecular mechanisms leading to adulthood diabetes in IUGR.

Methods: The IUGR rat model was induced by maternal protein malnutrition. The fetal pancreas was collected at embryonic day 20 (E20). Protein was extracted, pooled and subjected to label-free quantitative proteomics analysis. Bioinformatics analysis (GO and IPA) was performed to define the pathways and networks associated with DEPs. LC-MS results were confirmed by western blotting and/or quantitative PCR (q-PCR).The principal parameters of oxidative stress-superoxide dismutase (SOD) were determined in blood samples of fetal rats.

Results: A total of 57 DEPs (27 upregulated, 30 downregulated) were identified with a 1.5-fold change threshold and a p-value≤0.05 between the IUGR and control pancreas. Bioinformatics analysis revealed that these proteins play important roles in peroxisome biogenesis and fission, fatty acid beta oxidation (FAO), mitotic cell cycle and histone modification. The peroxin Pex14 was downregulated in the IUGR pancreas as confirmed by western blotting and q-PCR.Pmp70, a peroxisomal membrane protein involved in the transport of fatty acids, was upregulated. Hsd17b4 and Acox1/2, which catalyze different steps of peroxisomal FAO, were dysregulated. SOD plasma concentrations in the IUGR fetus were higher than those in the control, suggesting partial compensation for oxidative stress.

Conclusion: The present study identified DEPs in the fetal pancreas of IUGR rats by proteomics analysis. Down regulation of pancreas peroxins and dysregulation of enzymes involved in peroxisomal FAO may impair the biogenesis and function of the peroxisome and may underlie the development of T2 diabetes mellitus in adult IUGR rats. The present data provide new insight into the role of the peroxisome in the development of the pancreas and may be valuable in furthering our understanding of the pathogenesis of IUGR-induced diabetes.