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论著

宫内发育迟缓对大鼠肝糖异生关键酶的影响

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摘要：

目的：通过检测宫内发育迟缓(IUGR)仔鼠肝组织中糖异生关键酶磷酸烯醇丙酮酸羧激酶(PEPCK)和葡萄糖-6-磷酸酶(G-6-Pase)的mRNA表达变化，探讨IUGR个体发生胰岛素抵抗的机制。方法：通过孕期全程给予孕鼠10%低蛋白饲料建立IUGR仔鼠模型，对照组给予孕鼠21%正常蛋白饲料建立正常出生体质量仔鼠模型。每组仔鼠出生1周、3周、8周时测定其体质量、空腹血糖、血清胰岛素水平及胰岛素抵抗指数，并采用反转录-聚合酶链反应(RTPCR)法检测仔鼠肝组织中PEPCK和G-6-Pase的mRNA表达。结果：IUGR组仔鼠出生体质量明显低于对照组(P<0.001)，1周、3周、8周时亦低于对照组(P<0.05)。各时间点IUGR仔鼠空腹血糖、血清胰岛素水平及胰岛素抵抗指数与对照组无明显差异(P>0.05)。IUGR仔鼠各时间点肝组织PEPCK和G-6-Pase mRNA的表达水平均高于对照组(P<0.01)。结论：IUGR仔鼠肝糖异生关键酶PEPCK和G-6-Pase的表达明显增高，可能增加肝糖异生，是IUGR个体发生胰岛素抵抗和糖尿病的重要机制之一。

关键词： 宫内发育迟缓 胰岛素抵抗 肝 糖异生关键酶

Effect of intrauterine growth retardation on gluconeogenic enzymes in rat liver

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Abstract:

Objective: To investigate the expression of gluconeogenic enzymes phosphoenolpyruvate carboxykinase (PEPCK) and G-6-Pase mRNA of hepatic tissue in rats with intrauterine growth retardation (IUGR) and to explore the molecular mechanism of insulin resistance in IUGR rats.
Methods: Pregnant rats were randomly divided into 2 groups: a normal group and a model group. The normal group were fed with 21% protein forage and the model group with 10% low protein forage to obtain IUGR pup rats. The pup rats were introduced to the normal group and the IUGR group prospectively. At 1, 3 and 8 weeks, the body weight, blood glucose, insulin concentration and insulin resistance index of the pup rats were measured. Expression of PEPCK and G-6-Pase mRNA were detected by RT-PCR.
Results: The birth weight of the IUGR group was significantly lower than that of the normal group (P<0.001). The weight of the IUGR group was still lower than that of the normal group at 1, 3 and 8 weeks. There was no significant difference in the blood glucose, insulin level and

insulin resistance index between the 2 groups (P>0.05). The hepatic expression of PEPCK and
G-6-Pase mRNA in the IUGR group was significantly higher than that of the normal group at 1,
3 and 8 weeks (P<0.01).
Conclusion: The significantly increased expression of PEPCK and G-6-Pase mRNA of hepatic tissue in IUGR rats may increase gluconeogenesis, which is probably one of the molecular mechanisms of insulin resistance and diabetes in IUGR.

Keywords: intrauterine growth retardation insulin resistance liver gluconeogenic enzyme
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