عنوان مقاله:

Gestational diabetes leads to down-regulation of CDK۴-pRB-E۲F۱ pathway genes in pancreatic islets of rat offspring

محل انتشار:

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خلاصه مقاله:

Objective(s): The link between a hyperglycemic intrauterine environment and the development of diabetes later in life has been observed in offspring exposed to gestational diabetes mellitus (GDM), but the underlying mechanisms for this phenomenon are still not clear. Reduced β-cells mass is a determinant in the development of diabetes (type ۱ and type ۲ diabetes). Some recent studies have provided evidence that the CDK۴-pRB-E۲F۱ regulatory pathway is involved in β-cells proliferation. Therefore, we postulated that GDM exposure impacts the offspring's β-cells by disruption in the CDK۴-pRB-E۲F۱ pathway. Materials and Methods: Adult Wistar rats were randomly allocated in control and diabetic group. The experimental group received ۴۰ mg/kg/body weight of streptozotocin (STZ) on day zero of gestation. After delivery, diabetic offspring of GDM mothers and control dams at the age of ۱۵ week were randomly scarified and pancreases were harvested. Langerhans islets of diabetic and control groups were digested by collagenase digestion technique. After RNA extraction, we investigated the expressions of the kir ۶.۲ and CDK۴-pRB-E۲F۱ pathway genes by quantitative real-time PCR. Results: GDM reduced the expression of CDK۴-pRB-E۲F۱ pathway genes in Langerhans islets cells of offspring. CDK۴, pRB and E۲F۱ pathway genes were downregulated in diabetic islets by ۵۱%, ۳۵% and ۸۴%, respectively. Also, the expression of Kir ۶.۲ was significantly decreased in diabetic islets by ۸۸%. Conclusion: We suggest that the effect of gestational diabetes on offspring's β-cells may be primarily caused by the suppression of CDK۴-pRB-E۲F۱ pathway.

کلمات کلیدی:

Gene expression, Gestational Diabetes Mellitus, Langerhans islets, Offspring

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