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ABSTRACT

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Ameliorative effect of astragalus polysaccharide on hepatic insulin resistance in the KKAy mice model

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Insulin signaling in liver is critical in regulating glucose homeostasis and maintaining normal hepatic funtion. To investigate the potential effect of astragalus polysaccharide(APS) on the hepatic insulin resistance, twelve weeks old female KKAy mice(an animal model of T2DM) and C57BL/6J mice(the age/gender matched non – diabetic mice controls) were respectively randomized into APS treated and untreated groups. The diabetic KKAy mice responded to 8 – week APS therapy with a significant decrease in the level of blood glucose, plasma insulin, body weight, the content of visceral fat and improved glucose tolerance by comprehensive analysis of oral glucose tolerance test (OGTT) and calculated HOMA – IR index. The increases of hepatic GSK3 gene transcription and translation were measured by Western blotting and RT – PCR. And the pathological features of typical hepatic steatosis were presented through microscope and TEM. In this study we observed the significant amelioration of hepatic insulin resistance after astragalus polysaccharide therapy. These results indicate that APS enables insulin – sensitizing and can alleviate the extent of hepatic fatty degeneration, at least partly by inhibiting the expression and activity of the hepatic GSK3.

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Repglinide can correct the first phase of insulin secretion in persons with prediabetes

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INTRODUCTION: The progression from normal to impaired glucose homeostasis (IGH) and, finally diabetes is associated with a reduction in insulin sensitivity and a progressive decrease of the acute insulin response to glucose (the first phase of insulin secretion), which is lost at the onset of diabetes. Repaglinide is a short – acting, insulinotropic antidiabetic agent which stimulates insulin secretion and which has a novel insulin release profile. **AIM**: The aim of this study was to investigate whether a single use of Repaglinide can correct the first phase of insulin secretion in persons with IGH. **METHODS**: Five patients with IGH were included in the study. Insulin secretion was assessed by the frequently sampled intravenous glucose tolerance test (FSIVGTT). Each one underwent two FSIVGTT performed on two successive days, one day regular FSIVGTT and the day after, FSIVGTT modified by premedication of 1.0 mg of Repaglinide. **RESULTS**: It was demonstrated that Repaglinide increases the first phase of insulin secretion: area under the curve of insulin of the plasma insulin concentration in the first 10 min after the administration of glucose (before 82.96 ± 77.04 *vs* after 377.88 ± 374.33, $P = 0.043$) and the total sum of insulinemia in the first and the third minutes of FSIVGTT (before 53.54 ± 39.64 *vs* after 127.04 ± 100.60, $P = 0.043$). Repaglinide does not influence the second phase of insulin secretion in persons with IGH. Also, in these patients, Repaglinide doesn't change significantly the metabolism of glucose (assessed by area under the curve of glucose during FSIVGTT, half – time of glucose disappearance rate ($T_{1/2}$) and Conard's constant). **CONCLUSION**: Repaglinide increases the first (but not the second) phase of insulin secretion in persons with IGH. The metabolism of glucose was not affected by Repaglinide premedication.