Journal of
Alzheimer's Disease & Parkinsonism

Research Article Open Access

The Effects of Acetylation of PTEN on Hepatic Gluconeogenesis

Jiao Yang[1] Qiu Chen[1]* and Hongmei Zhu[2]
[1]*Chengdu University of Traditional Chinese Medicine,SiChuan Prov. China*
[2]*Central City Hospital of Ankang,Shaanxi Prov.China*

Abstract

Background: With economic development and lifestyle changes, the prevalence of diabetes increased year by year. Hepatic insulin resistance is one of central link of the pathogenesis of diabetes. And the increased hepatic glucose output that induced by disorder of the liver gluconeogenesis is a critical step in the development of hepatic insulin resistance. So it would be very important for the treatment of Diabetes Mellitus (DM) to reduce endogenous glucose through effectively suppressing excessive gluconeogenesis. Recent studies indicate that phosphatase and tensin homologue deleted on chromosome ten 10(PTEN) plays a role in the development of hepatic insulin resistance. Its enhanced protein expression or activity might be involved in the occurrence of insulin resistance (IR). Studies have shown that PTEN is a mediator of oleate-induced insulin resistance in liver, when the PTEN gene was

Keywords: Acetylation; PTEN; Hepatic insulin resistance; Gluconeogenesis; Liver cells

Introduction

Diabetes Mellitus (DM) is a common endocrine and metabolic disease. With the development of economy, acceleration of population aging process and changes of lifestyle, prevalence of diabetes in China has showing a rapid upward trend. As the latest research reported that China had become a diabetes superpower all over the world for more than 92.4 million Chinese people su er with it and most of them are

CCK-8 method to detect drug's e ect on cell proliferation: To determine the concentration of drug intervention, drug's e ect on cell proliferation were detected in accordance with instructions in CCK-8. BRL-3A cells were seeded in 96-well plates with density of 2×10^4 per L and 200 µl per hole. When cell fusion is about to be 50%, synchronous processing for 24 h by serum-free DMEM (high glucose), then divide them into three groups: (1) Blank group: with no cells, only has CCK 8 and medium; (2) e control group: containing cells CCK-8 and medium but without drugs; (3) e drug group: containing cells CCK 8 and medium with di erent concentration of drugs. Four holes were set for each groups, A er 24 h of incubation, absorbed all medium in the holes and washed with PBS for 2 times, 100 µl basal medium and 10 µl CCK-8 were added into each hole, Within 0.5 to 4 h a er CCK-8 was added, the absorbance of each hole were measured in a microplate reader at 450nm,then comparing OD value to calculate the cell viability of each group. (Note: In order to prevent the e ects of water evaporation

PTEN protein levels of each experimental group

Compared with the control group, ere were no signi cant di erences about PTEN protein expression both in nicotinamide group and resveratrol group (▲P>0.05), but in the Oleic acid group and oleic acid+nicotinamide group, the contents of PTEN were increased (*P<0.05. Compared with oleic acid group, PTEN protein levels did not change signi cantly in which ete cells were incubated with oleic acid before adding nicotinamide,(●P>0.05) (Figure 4).

Acetylated PTEN protein levels of each experimental group

Compared with the control group, there was significant difference in each drug intervention group (*P<0.01), after the nicotinamide treated, the acetylated PTEN protein levels of hepatocyte

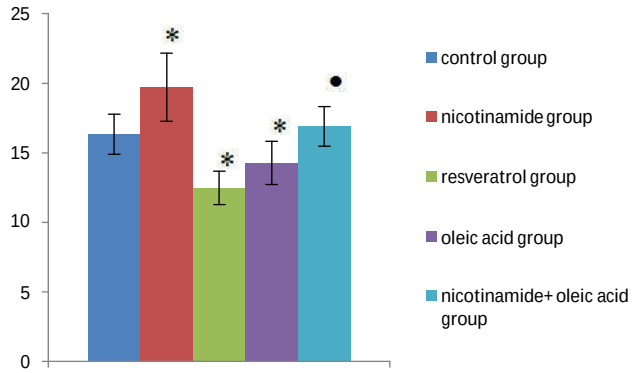

Figure 6: Expression of acetylated PTEN in BRL-3A.

Discussion

Liver as one of a target organ for insulin action, it plays a very important role in maintaining endogenous glucose production and output in the fasting and the glucose uptake, utilization and storage a er eating. Normally, in order to satisfy the demand for energy under di erent physiological state and maintain the plasma glucose level in stability, insulin and glucagon control the glycogen synthesis and output and exogenous glucose absorption utilization and storage through regulating the expression of enzyme related sugar metabolic pathways [5,26]. Abnormal glucose metabolism in liver is the main pathological feature of diabetes, obesity and other metabolic syndrome, while hepatic insulin resistance is the major part of the pathogenesis of these diseases. Increased hepatic glucose output induced by hepatic gluconeogenesis disorders is an important causative factor of the occurrence of hepatic insulin resistance [26,27]. erefore, Reducing hepatic glucose production through regulating di erent aspects of hepatic gluconeogenesis signal path will provide broad prospects for the treatment of hepatic insulin resistance syndrome.

Insulin resistance refers to a signi cant decrease about the physiological e ect of insulin- stimulated glucose uptake and utilization of target cells under a normal situation of insulin secretion. In other words, it means that need extraordinary amount of insulin for target cells in preserving the normal physiological e ects on glucose ingestion and utilization [28]. As the pathophysiological basis of various human metabolic diseases, for example DM, the mechanism of IR is very complex and has not been fully clari ed yet. Studies have

Citation:

6. Moller DE (2001) New drug targets for type 2 diabetes and the metabolic