Can berberine enhance glucose-stimulated insulin secretion in rat islets?

November 3 2008

The therapeutical actions of berberine on diabetes have been well studied. Previous researches show that berberine modulates cholesterol through increasing low-density lipoprotein receptor mRNA stability, reduces body adiposity and increases insulin sensitivity partly through activating AMP-activated protein kinase and improves glucose metabolism via induction of glycolysis. Yet, the underlying mechanism for berberine promoting insulin release remains unclear.

A research article to be published on October 21, 2008 in the *World Journal of Gastroenterology* addresses this question. The research team was directed by Prof. Lu from Institute of integrative traditional and western medicine of Tongji medical college.

Using glucose-stimulated insulin secretion assay, the authors first examined the action of berberine on insulin secretion. Further, the authors detected the expression of HNF4 alpha on both gene and protein levels to examine if HNF4 alpha a candidate target of berberine. The role of HNF4 alpha in mediating berberine's insulinotropic effect was further elucidated by determining the activity of Glucokinase, the "Glucose Sensor" in pancreatic beta cell, which is also deemed as one of the downstream targets of the HNFs transcription regulatory circuit in pancreatic islets.

The results demonstrate that berberine only exerts insulinotropic effect in the high glucose condition, and berberine could enhance HNF4 alpha expression and GK activity dose-dependently.

The data of the research strongly suggested a correlation among insulin secretion, HNF4α expression and GK activity. The article proposes that berberine might facilitate glucose-stimulated insulin secretion in a pathway involving HNF4 alpha, which subsequently modulats the downstream targets such as glucokinase, resulting in increased sensitivity of beta cell to glucose challenge and insulin release.

Source: World Journal of Gastroenterology