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Commentary

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Pharmacokinetics of Berberine in Controlling Human Glucose Level

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ABOUT THE STUDY

Diabetes has been steadily increasing in recent years, and it is now the ninth greatest cause of death in the United States, posing a serious threat to human health. Despite the fact that metformin and glucocorticoids have good therapeutic benefits, their use is generally limited due to side effects. As a result, it's critical to locate safe and efficient natural diabetes prevention and treatment drugs with low side effects as soon as possible. Berberine (BBR) has been proven to be a natural medicine in a number of studies. The organic product BBR is an isoquinoline alkaloid that has been used to treat diabetes for thousands of years in medical history and is found in a variety of botanicals and medicinal herbs (e.g., Rhizome Coptidis and Berberidis Cortex). Various pharmacological tests have been undertaken in various cell and animal disease models to examine the effects of antidiabetic BBR and its consequences, with encouraging results. The numerous BBR diabetic treatment approaches are described here.

Antihyperglycemic

Diabetes mellitus is a metabolic disorder characterised by excessive blood sugar levels caused by insufficient insulin secretion or activity. Understanding the source and destination of blood sugar, as well as how to regulate blood sugar concentration, is a requirement for treating diabetes by reducing blood sugar. By suppressing liver gluconeogenesis and increasing glycolysis, BBR can diminish blood sugar sources and increase glucose metabolism. BBR can also control blood glucose levels by improving insulin sensitivity, lowering IR, and boosting insulin secretion.

Insulin sensitivity enhancement : The enzyme AMP-dependent Protein Kinase (AMPK) is essential for maintaining systemic energy balance in the body. Through AMPK activation in numerous tissues, including muscles and adipose tissue, BBR may improve insulin sensitivity and reduce systemic obesity while maintaining glucose homeostasis By activating AMPK, BBR enhance the transfer of Glucose Transporter-4 (GLUT4) to the plasma

membrane and boosted insulin sensitivity in insulin-resistant H9c2 cardiomyocytes. The AMPK pathway is activated by two primary BBR pathways. One is that BBR inhibits mitochondrion respiratory complex I, which stimulates AMPK activity. The other is that BBR activated the AMPK pathway by upregulating the expression of Sirtuin 1 (SIRT1) in adipose tissue. Inhibiting inflammation, alleviating IR, and enhancing insulin sensitivity are eventually achieved.

BBR can stimulate insulin secretion in pancreatic -cells, resulting in a hypoglycaemic effect. The next sentences go over the main mechanism. Furthermore, BBR has the potential to increase insulin secretion by enhancing PARP-1 protein expression and pancreatic cell proliferation. Glucagon-like Peptide-1 (GLP-1) is a hormone secreted by the intestinal tract that has a range of physiological roles, including boosting insulin production, encouraging pancreatic cell proliferation, and regulating glucose metabolism. Furthermore, when blood sugar levels are high, BBR may enhance GLP-1 secretion and synthesis, boosting insulin release in cells and controlling insulin levels in the body.

Increasing glucose absorption: After glucose is absorbed into the bloodstream, glucose transporters allow glucose to enter the cell. GLUT1 is found throughout the human body, while GLUT4 is found mostly in insulin-sensitive heart, skeletal muscle, fat cells, and myocardium. Through the AMPK route, BBR can activate GLUT1 and upregulate GLUT1 expression, as well as boost GLUT4 expression and translocation activity, promoting glucose absorption and increasing glucose availability by tissues and cells of the body. BBR, for example, increased GLUT4 content in the membrane of adipocytes by enhancing the IRS1-PI3-kinase-Akt signal cascade, which was advantageous for glucose uptake enhancement. In addition to the insulin signalling pathways, BBR possessed a method for increasing glucose absorption via the AMP-AMPK-p38 mitogen-activated protein kinase (MAPK) pathway. In H9c2 cells, BBR may enhance glucose uptake and consumption by increasing triacylglycerol synthesis and accumulation while decreasing cellular Di Acyl Glycerol (DAG) levels.

Inducing glycolysis: The main energy sensor, AMPK, was activated when the glucose level increased and deactivated when the glucose level decreased. Experiments revealed that BBR's blood sugar-lowering effect was not always dependent on AMPK activation. In other words, BBR controlled AMPK phosphorylation in both directions. Furthermore, when blood sugar levels rose in vivo, BBR helped to stimulate glycolysis by inhibiting glucose oxidation in the mitochondria, which was linked to glucose oxidation inhibition in the mitochondria. As a result of mitochondrial inhibition, a rise in the AMP/ATP ratio may activate AMPK. In the absence of hypoglycemia, other researchers discovered that BBR increased glycolysis and enhanced sugar metabolism through blocking mitochondrial respiratory chain complex I, which did not require AMPK activation.