

Berberine Ameliorates Glucocorticoid-Induced Hyperglycemia: An In-Vitro and In-Vivo Study

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Abstract : Introduction: Berberine (BBR), a bioactive compound isolated from *Coptidis Rhizoma*, possesses diverse pharmacological activities, including anti-bacterial, anti-inflammatory, antitumor, hypolipidemic, and anti-diabetic. However, its role as an anti-diabetic agent in animal models of dexamethasone (Dex)-induced diabetes remains unknown. Studies have shown that natural compounds, including aloe, caper, cinnamon, cocoa, green and black tea, and turmeric, can be used for treating Type 2 diabetes mellitus (DM). Compared to conventional drugs, natural compounds have fewer side effects and are easily available. Herein, we studied the anti-diabetic effects of BBR in a mice model of Dex-induced diabetes. Methods: HepG2 cell line was used for glucose release and glycogen synthesis studies. Cell proliferation was measured by methylthiotetrazole (MTT) assay. For animal studies, mice were treated with Dex (2 mg/kg, i.m.) for 30 days and the effect of BBR at the doses 100, 200, and 500 mg/kg (p.o.) was analyzed. Glucose, insulin, and pyruvate tests were performed to evaluate the development of the diabetic model. An echo MRI was performed to assess the fat mass. Further, to elucidate the mechanism of action of BBR, mRNA expression of genes regulating gluconeogenesis, glucose uptake, and glycolysis were analyzed. Results: In vitro BBR had no impact on cell viability up to a concentration of 50 μ M. Moreover, BBR suppressed the hepatic glucose release and improved glucose tolerance in HepG2 cells. In vivo, BBR improved glucose homeostasis in diabetic mice, as evidenced by enhanced glucose clearance, increased glycolysis, elevated glucose uptake, and decreased gluconeogenesis. Further, Dex treatment increased the total fat mass in mice, which was ameliorated by BBR treatment. Conclusion: BBR improves glucose tolerance by increasing glucose clearance, inhibiting hepatic glucose release, and decreasing obesity. Thus, BBR may become a potential therapeutic agent for treating glucocorticoid-induced diabetes and obesity in the future.

Keywords : glucocorticoid, hyperglycemia, berberine, HepG2 cells, insulin resistance, glucose

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