Berberine lowers blood glucose in type 2 diabetes mellitus patients through increasing insulin receptor expression.


Source
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Abstract
Our previous work demonstrated that berberine (BBR) increases insulin receptor (InsR) expression and improves glucose utility both in vitro and in animal models. Here, we study the InsR-up-regulating and glucose-lowering activities of BBR in humans. Our results showed that BBR increased InsR messenger RNA and protein expression in a variety of human cell lines, including CEM, HCT-116, SW1990, HT1080, 293T, and hepatitis B virus-transfected human liver cells. Accordingly, insulin-stimulated phosphorylations of InsR beta-subunit and Akt were increased after BBR treatment in cultured cells. In the clinical study, BBR significantly lowered fasting blood glucose (FBG), hemoglobin A(1c), triglyceride, and insulin levels in patients with type 2 diabetes mellitus (T2DM). The FBG- and hemoglobin A(1c)-lowering efficacies of BBR were similar to those of metformin and rosiglitazone. In the BBR-treated patients, the percentages of peripheral blood lymphocytes that express InsR were significantly elevated after therapy. Berberine also lowered FBG effectively in chronic hepatitis B and hepatitis C patients with T2DM or impaired fasting glucose. Liver function was improved greatly in these patients by showing reduction of liver enzymes. Our results confirmed the activity of BBR on InsR in humans and its relationship with the glucose-lowering effect. Together with our previous report, we strongly suggest BBR as an ideal medicine for T2DM with a mechanism different from metformin and rosiglitazone.

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PMID:
19800084