Berberine reducing insulin resistance by up-regulating IRS-2 mRNA expression in nonalcoholic fatty liver disease (NAFLD) rat liver.


Source

Institute of Digestive Disease, Longhua Hospital, Shanghai University of Traditional Chinese Medicine, China.

Abstract

This study was performed to investigate the molecular mechanism and the therapeutic effect of berberine on nonalcoholic fatty liver disease (NAFLD). Rat models were given a high-fat diet (42% kcal) until they developed NAFLD, then were given normal saline (n=10), berberine (n=10) at 187.5mg/kg/day, or pioglitazone (n=10) at 10.0mg/kg/day intragastrically for 4 weeks, respectively, and evaluated by hyperinsulinemic euglycemic clamping for insulin sensitivity. Serum biochemical markers and liver triglyceride (TG) were analyzed, real-time RT-PCR for mRNA expression and western blotting for protein expression of insulin receptor (IR) and insulin receptor substrate-2 (IRS-2) in liver tissues were performed, and hepatic histopathology in the rat models with NAFLD at the end of treatment was compared with normal controls (n=10). The NAFLD rats developed insulin resistance, showing increased fasting blood glucose and insulin levels, decreased glucose infusion rate, increased weight of epididymal fat (g/100g body weight), obvious hepatic steatosis and inflammation, and down-regulated IRS-2 mRNA and protein levels compared with normal controls (all P<0.05). In comparison with those treated with saline, model rats treated with berberine or pioglitazone underwent significant recovery, including up-regulated IRS-2 mRNA and protein (all P<0.05). Our results indicate that berberine may improve insulin resistance of NAFLD by up-regulating mRNA and protein levels of IRS-2, a key molecule in the insulin signaling pathway, suggesting that berberine may be used to treat NAFLD.

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