



The cardiovascular effects of salidroside in the Goto-Kakizaki diabetic rat model

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Many factors, including hyperglycemia, hypertension, obesity, dyslipidemia, and a sedentary lifestyle, contribute to a high prevalence of cardiovascular disease. Specific vascular impairment treatments in the context of diabetes and vascular risk need to be improved. Salidroside is the primary active component of *Rhodiola rosea* and has documented antioxidative, cardioprotective, and vasculoprotective properties. The aim of this study was to test the hypothesis that salidroside has protective effects against hyperglycemia, hypertension, and vasodilation impairment in the Goto-Kakizaki (GK) rat model of diabetes. We evaluated cardiovascular parameters (e.g., daytime/nighttime systolic and diastolic blood pressure, heart rate, and activity), metabolic parameters (e.g., body weight, food and water consumption, serum fructosamine level, glucose tolerance), eNOS / phospho-eNOS expression level and in vitro vascular reactivity of aorta and second-order mesenteric arteries in Wistar-Kyoto (control) and GK (diabetic) rats treated with salidroside (40 mg/kg) or placebo (water) for 5 weeks. GK rats showed hypertension, marked glucose intolerance, and impaired endothelium-dependent and endothelium-independent vasodilation capacity. Salidroside showed beneficial effects on endothelial and non-endothelial vasodilation and likely acts on the endothelium and smooth muscle cells through the soluble guanylyl cyclase pathway. Despite its vascular effects, salidroside had no effect on blood pressure and heart rate in GK and control rats, it did not improve glucose metabolism or limit hypertension in the GK model of type 2 diabetes.

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