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Hydroxytyrosol prevents diet-induced metabolic syndrome and attenuates mitochondrial abnormalities in obese mice.

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Abstract

A Mediterranean diet rich in olive oil has profound influence on health outcomes including metabolic syndrome. However, the active compound and detailed mechanisms still remain unclear. Hydroxytyrosol (HT), a major polyphenolic compound in virgin olive oil, has received increased attention for its antioxidative activity and regulation of mitochondrial function. Here, we investigated whether HT is the active compound in olive oil exerting a protective effect against metabolic syndrome. In this study, we show that HT could prevent high-fat-diet (HFD)-induced obesity, hyperglycemia, hyperlipidemia, and insulin resistance in C57BL/6J mice after 17 weeks supplementation. Within liver and skeletal muscle tissues, HT could decrease HFD-induced lipid deposits through inhibition of the SREBP-1c/FAS pathway, ameliorate HFD-induced oxidative stress by enhancing antioxidant enzyme activities, normalize expression of mitochondrial complex subunits and mitochondrial fission marker Drp1, and eventually inhibit apoptosis activation. Moreover, in muscle tissue, the levels of mitochondrial carbonyl protein were decreased and mitochondrial complex activities were significantly improved by HT supplementation. In db/db mice, HT significantly decreased fasting glucose, similar to metformin. Notably, HT decreased serum lipid, at which metformin failed. Also, HT was more effective at decreasing the oxidation levels of lipids and proteins in both liver and muscle tissue. Similar to the results in the HFD model, HT decreased muscle mitochondrial carbonyl protein levels and improved mitochondrial complex activities in db/db mice. Our study links the olive oil component HT to diabetes and metabolic disease through changes that are not limited to decreases in oxidative stress, suggesting a potential pharmaceutical or clinical use of HT in metabolic syndrome treatment.

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