β-conglycinin regulates gene expression involved in cholesterol and triacylglycerol homeostasis in rats fed a hypercholesterolemic diet

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β-conglycinin has been shown to reduce triglyceride and cholesterol concentrations in plasma. However, the mechanisms of action are not yet clear and deserve further study. We investigated the effect and mechanism of β-conglycinin on hyperlipidemia through expression of genes involved in cholesterol and triacylglycerol homeostasis in rats. Twenty-seven male rats were randomly assigned to three (n=9) fed Nath’s hypercholesterolemic diets which following: (HC) hypercholesterolemic diet plus vehicle; (HC+β-conglycinin) HC diet plus 300 mg.kg⁻¹.day⁻¹ of β-conglycinin, and (HC+SVT) HC diet plus 50 mg.kg⁻¹.day⁻¹ of simvastatin. β-conglycinin decreased plasma cholesterol (TC), non-HDL-c and triglycerides (TG) 22, 39 and 21%, respectively. There are also an increased HDL-C fraction (49%) and lipoprotein lipase activity (67%). Hepatic TC and TG concentrations were lower 10 and 16% in rats that received to protein compared to HC group. Additionally, β-conglycinin downregulated mRNA expression levels of fatty acid synthase (-23%), HMG-CoA reductase (-13%), HMG-CoA syntetase (-81%), SREBP-1c (-41%), SREBP-2 (-52%) and upregulated expression of LDL-receptor (+27%). Opposite, PPAR-α (-57%) and CYP7A1 (-57%) expression was downregulated. Our data suggest that the hypolipidemic effects from β-conglycinin are likely to be mediated by up or down-regulating genes involved in uptake and lipogenesis.

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