Xanthorrhizol inhibits cell proliferation, cellular cholesterol uptake in HT29 colon cells and adipogenesis in 3T3-L1 adipocytes

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Abstract

Hyperlipidemia is defined as the presence of either hypertriglyceridemia or hypercholesterolemia, which could cause atherosclerosis, cardiovascular diseases and certain cancers. Although hyperlipidemia can be treated by hypolipidemic drugs, they are limited due to lack of effectiveness and safety. Since flavonoids and tannins have been reported to possess antihyperlipidemic activity, it is believed that phytochemicals isolated from plants may decrease the lipid levels with lower side effects. Previous studies demonstrated that xanthorrhizol (XNT) isolated from *Curcuma xanthorrhizza* reduced the levels of free fatty acid and triglyceride *in vivo*. However, antiproliferative activity of XNT and its ability to inhibit cholesterol uptake and adipogenesis are yet to be reported. In this study, the IC_{50} values of XNT were 15.12 ± 0.68 µg/mL in HT29 cells and 35.07 ± 0.24 µg/mL in 3T3-L1 adipocytes, respectively. Cholesterol uptake inhibition study was conducted in HT29 colon cells using fluorescent cholesterol analogue NBD. The result showed that XNT (15 µg/mL) siginificantly inhibited the cholesterol uptake by up to 37.6 ± 1.53 % relative to control. On the other hand, higher concentration of XNT (50 µg/mL) significantly suppressed the growth of 3T3-L1 adipocytes (5.9 ± 0.58 %) compared to 3T3-L1 preadipocytes (80.42 ± 8.29 %). It was found that XNT (3.125 µg/mL) impeded adipogenesis by reducing lipid content of 3T3-L1 adipocytes for 25.37 ± 3.24 % comparable to positive control (quercetin), 20.07 ± 8.78%. We postulate that inhibition of cholesterol uptake, adipocyte number and adipogenesis may be utilized as treatment modalities to reduce the prevalence of lipidemia. To conclude, XNT could be a potential hypolipidemic agent and further studies could be done on its mechanism of action.

Keywords: Adipogenesis, cell proliferation, cholesterol, xanthorrhizol.

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