

Title: Mechanism of Benzyl isothiocyanate Inhibits TNF-α Driven Lipolysis in 3T3-L1 Adipocytes

Abstract

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Background: It has been found that both obesity-induced chronic inflammation and cancer cachexia lead to increased serum level. of tumor necrosis factor alpha (TNF-α) TNF-α stimulates lipolysis in differentiated adipocytes, which releases free fatty acids (FFAs) and excessive serum level of FFAs termed lipotoxicity. Lipotoxicity induces numerous adverse effects, including impairment of insulin sensitivity and activation of Kupffer cells in liver leading to in the development of non-alcoholic fatty liver disease (NAFLD). Benzyl isothiocyanate (BITC) has been shown to possess anti-inflammatory activity and improvement of insulin-resistance in previous studies. In our laboratory, BITC was shown to attenuate IL-1β-induced insulin resistance in 3T3-L1 adipocytes and palmitate-induced insulin resistance in hepatocytes.
Objective: Elevated blood FFA levels play a key role in the disturbance of glucose and lipid metabolism in liver and skeletal muscles, which leads to insulin resistance. Since it has been demonstrated that BITC protects the development of insulin resistance induced by inflammatory cytokines and FFAs, the aim of this study was to investigate whether BITC owns the activity to ameliorate TNF-α-induced lipolysis and subsequent lipotoxicity.
Methods: Differentiated 3T3-L1 adipocytes were used as the study model. Oil Red O staining assay was used to measure lipid accumulation. Gene expression was measured by RT-qPCR and protein expression was measured by Western blotting. Glycerol was determined by using glycerol release kit.
Results: In current r study, it was found that BITC can effectively inhibit the lipolysis induced by TNF-α in 3T3-L1 adipocytes and the underlying mechanisms are still under investigation.

Biography
I graduated from Providence University at the age of 24 years, and then study at China Medical University for master degree.