Indra R . Dual effect of Quercetin, the Antimetabolic Syndrome is Mediated by Adipocyte Activation

Abstract

Backgrounds. The objective of this study was to elucidate the role of flavon quercetin in influencing TNFα expressions via MAPK (ERK1/2) and NFκB pathways in high dose leptin-induced endothelial cells in vitro.

Methods. Human umbilical vein endothelial cells (HUVECs) cultures were exposed with high dose leptin (500 ng/mL) and quercetin in four doses (0, 50, 125 and 625 uM). The experimental groups of this study were: no leptin and no quercetin; 500 ng/mL leptin and no quercetin; no leptin and 50 μM quercetin; no leptin and 125 μM quercetin; no leptin and 625 μM quercetin; 500 ng/mL leptin and 50 μM quercetin; 500 ng/mL leptin and 125 μM quercetin; 500 ng/mL leptin 625 μM quercetin. After six hour incubation, ERK1/2 MAPK, NFκB, and TNFα expression were determined by ELISA and immunofluoresence methods. Data obtained was statistically analyzed using one way ANOVA followed by Tukey's test.

Results. The results showed that 500 ng/mL leptin increases ERK1/2, NFkB, TNFa expression compared to control. The increased levels of NFkB on high dose of leptin were reduced by the addition of 50, 125 and 625 μM quercetin, respectively. Notably, there was no significant difference in ERK1/2 expression between control and quercetin-treated groups. TNFa level was significantly decreased post 50 μM quercetin treatment, which interestingly was increased after increasing the quercetin dosages to 125 and 625 μM.

Conclusions. Low dose of quercetin inhibited the high levels of leptin-induced TNFa . The optimum dose of quercetin was 50 μM. This inhibition process occurred through inhibition of NFkB signal, but not ERK1/2 MAPK pathways.

Keywords: leptin, quercetin, NFkB, TNFa, endothelial dysfunction