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 immunofluorescence 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, NFκB, TNFα expression compared to control. The increased levels of NFκB 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. TNFα 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 TNFα. The optimum dose of quercetin was 50 µM. This inhibition process occurred through inhibition of NFκB signal, but not ERK1/2 MAPK pathways.

Keywords: leptin, quercetin, NFκB, TNFα, endothelial dysfunction