

Abstract:

Nicotine, the principal alkaloid in tobacco, induces a cellular damage on heart and cardiomyocyte culture. We investigate the protective role of green tea extract (GTE) against nicotine. Male albino rats were treated by injecting nicotine (1 mg/kg b.w. for 2 months) subcutaneously and thereby supplementing GTE 2% orally to them. The levels of plasma lipids, cardiac MDA (malondialdehyde) and catalase activity Mitogen-activated proteins kinases MAPKs were measured. The expression levels of (ERK 1/2, extracellular signal σ regulated kinase 1/2 and P38 MAP kinase), endoplasmic reticulum stress (ERS)-related protein (GRP78 glucose regulated protein-78, HSP70 heat shock protein-70, CHOP C/EBP homologous protein), AIF (apoptosis-inducing factor) and VDAC (voltage-dependant anion channel) were evaluated by Western blot. In the *in vitro* study, the cardiomyocytes were exposed to nicotine (10 μ M) and major GTE polyphenol epigallocatechin gallate EGCG (50 μ M). Data showed that nicotine induced a significant increase on MDA levels, LDH (lactate dehydrogenase) and aminotransferase activity compared with control. The heart sections of nicotine exposed-rats showed severe degenerative changes. Nicotine increased the expression of P38, but not ERK 1/2, ER stressrelated proteins and AIF with no changes of VDAC. Concomitant GTE treatment significantly normalized and/or improved the levels of MDA, enzymatic activity and histological injuries. The proteins expression was attenuated by GTE co-administration without any changes for VDAC. ERK 1/2 expression enhanced in GTETreated groups. Exposure of cardiac cells to nicotine induced the expression of ERS markers and p38; the ERK 1/2 was highly expressed only in the presence of EGCG. It was suggested that green tea beverage can protect against nicotine toxicity by attenuating oxidative stress, endoplasmic reticulum stress and apoptosis. Otherwise, our results have showed that ERK1/2 and p38 are survival signaling pathways activated by GTE and EGCG.