THE MOLECULAR MECHANISM OF THE ANTI-OXIDATIVE EFFECTS OF CATECHIN ON PALMITIC ACID-INDUCED CYTOTOXICITY IN ASTROCYTES

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Aims: Metabolic syndrome (MS) encompasses a group of problems which will put a person at a high risk of developing cardiovascular diseases, including heart attack and stroke. Effective prevention or treatment of MS significantly reduces the risk for developing serious complications. Palmitic acid (PA) is a saturated fatty acid, when being excessive, is a significant risk factor for development of MS or stroke. However, damage by MS to astrocytes is relatively unexplored. Catechin is an effective antioxidant which would be beneficial to neurons subjected to reactive oxygen species (ROS) damage as well as on cardiovascular diseases. This study was to identify the mechanism(s) of PA-induced cytotoxicity in rat astrocytes and also to assess the protective effects of catechin.
Methods: Cell apoptosis assessed by TUNEL assay. Cytosolic Ca2+ in astrocytes was measured with Fura-2 method. Intracellular ROS was detected by fluorescence spectrophotometry. Mitochondria membrane potential(MMP) measured by MMP Assay Kit. The $p < 0.05$ were considered significant (ANOVA).
Results: Exposure of astrocytes to PA (100 μM) for 24 h resulted in approximately 50 % cell death. Cell death was apoptotic (TUNEL) and unrelated to endoplasmic reticulum(ER) stress and cytosolic Ca2+ elevation. Exposure of astrocytes to PA for 30 min to 5 h was associated with significant mitochondria membrane potential(MMP) collapse and ROS production. Co-treatment of astrocytes with catechin (300 μM) significantly prevented PA-induced MMP collapse, ROS production and cell death.
Conclusions: Our results suggest that PA-induced cytotoxicity in astrocytes may involve MMP collapse and ROS production, which can be prevented by catechin.