# Catechin Protected Cortical Astrocytes from Palmitic Acid-Induced Apoptosis

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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.

Catechins, a group of compounds belonging to the polyphenol family, are the major components in green tea extract. Cytoprotective effect of catechins has been shown in neuropathological disease models. The aim of this study was to identify the mechanism(s) of PA-induced cell death in rat cortical astrocytes and examine whether (+)-catechin could offer protection against PA-induced cytotoxicity.

Methods: Palmitic acid (PA), (+)-catechin, salubrinol, rotenone, ascorbic acid, carbonyl cyanide 4-trifluoromethoxy phenylhydrazone 60 (FCCP) and cyclopiazonic acid (CPA) were from Sigma-Aldrich (MO, USA). Cortical astrocytes were prepared from 1 to 2-day-old Sprague Dawley rats purchased from Bio Lasco Co. Ltd (Taiwan). The cells viability was measured using the MTT method. Cell apoptosis assessed by TUNEL assay. The $p < 0.05$ were considered significant (ANOVA).

**Results:** Our study shown the concentration-dependent cytotoxic effect of a 24-h PA exposure; at 100 mM, PA caused approximately 50% cell death. (+)-Catechin (50-300 mM) exhibited a concentration-dependent cytoprotective effect. TUNEL assay revealed that PA-induced cell death was apoptotic, as indicated by the green fluorescence of apoptotic cells. Apoptosis could be prevented by (+)-catechin (300 mM)

**Conclusion:** This is the first report to show that (+)-catechin offers protection against PA-induced lipotoxicity in astrocytes. Whether other green tea components such as epicatechin could protect against PA-induced lipotoxicity will also warrant future examination. Maintaining astrocytic functional integrity and antioxidant may offer a potential therapeutic strategy for neurodegeneration in MS.