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Epigallocatechin Gallate inhibits Prostagladins Generation by Suppression of cPLA2 Activity on Arachidonic Acid Metabolism in LPS-Stimulated RAW264.7 Cells

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Green tea contains several antioxidants including polyphenols of the catechin, which have been shown to act *in vitro* and *in vivo* as anti-inflammatory, anti-viral and anti-tumor drugs. Prostaglandins (PGs) are a family of intercellular and intracellular messengers derived from arachidonic acid(AA) by phospholipase(PL) and cyclooxygenase(COX). These mediators exert a wide range of effects on processes such as smooth muscle tone, vascular permeability, cellular proliferation, and inflammatory/immune function. In this study, Epigallocatechin gallate(EGCG), a major compound of green tea catechins, reduced the generations of PGE_2 and PGD_2 in RAW264.7 cells stimulated by lipopolysaccharide(LPS) in a dose-dependent manner when added to the culture media at the time of stimulation. In order to elucidate the mechanism involved in the anti-inflammatory activity of EGCG, we investigated its effects on the AA metabolism and enzyme activity such as $cPLA_2$-, $sPLA_2$- and COX-activity, and protein expression such as $cPLA_2$- and COX_2-expression. In the results, LPS stimulated the generations of PGE_2 and PGD_2 in RAW264.7 cells in a dose- and time-dependent manner. EGCG inhibited $cPLA_2$ activity, but did not suppress the $sPLA_2$-, or COX-activity in LPS-stimulated RAW264.7 cells. Furthermore, EGCG did not affect the $cPLA_2$-, or COX_2-expression. These results suggest that EGCG may inhibit the generations of PGE_2 and PGD_2 through the suppression of the $cPLA_2$ activity in LPS-stimulated RAW264.7 cells.

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Antidiabetic effect and mechanisms of SPH-2 in db/db mice

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SPH-2 is a herbal medicine composing oriental prescription. We have studied the antidiabetic effect and mechanism of SPH-2 in insulin-resistant diabetic db/db mice. Mice were grouped and treated for 3 weeks as follows: control group was administrated with tap water orally; treated group was administrated with SPH-2 orally at dose of 500 mg/kg. SPH-2 lowered plasma glucose level by 43% as compared to the diabetic control. Total cholesterol, triglyceride and free fatty acid were all reduced in SPH-2 treated group. The control group showed hyperinsulinemia, whereas SPH-2 treatment decreased insulin level at the end of treatment. SPH-2 treated mice also exhibited low urinary glucose and albumin level as compared to the diabetic control, in parallel to the plasma glucose concentration. In the mechanism study, PPARγ mRNA expression in epididymal fat were increased in SPH-2 treated group. GLUT4 mRNA expressions in skeletal muscle was also increased in SPH-2 treated group. We have also investigated glucose-6-phosphatase, phosphoenolpyruvate carboxykinase, and glucokinase activi- ties in liver. There were significant differences between control and treatment group in these parameters. From these result we may conclude that SPH-2 showed the excellent antidiabetic activity probably due to improvement of insulin resistance.

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