

activation at 1–5 μM with minimal prevention of I- κB phosphorylation. Treatment of cells with arctigenin (1 μM) and demethyltaxillagenin (50 μM) inhibited LPS-inducible nitrite/nitrate production by 50%. Both compounds inhibited iNOS expression in a concentration-dependent manner (IC₅₀=1 and 50 μM). Suppression of iNOS expression was confirmed by Northern blot analysis. These results showed that arctigenin and demethyltaxillagenin inhibited LPS-inducible iNOS expression via suppression of NF- κB activation with minimal I- κB phosphorylation. Inhibition of LPS-inducible NO production in macrophage cells by dibenzylbutyrolactone lignans may be associated with their anti-inflammatory effects.

[PA1-27] [04/20/2001 (Fri) 10:30 – 11:30 / Hall 4]

Inhibition of Hepatic Stellate Cell Proliferation and Activation by Butein(3,4,2',4'-tetrahydroxychalcone), a Plant Polyphenol, in Cultured Rats.

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Butein(3,4,2',4'-tetrahydroxychalcone), a plant polyphenol that acts as a specific protein tyrosine kinase inhibitor, is a chalcone compound belonging to the flavonoid subclass. The structures of chalcones are similar to curcumin, a known antioxidant. Hepatic stellate cells play an important role in the pathogenesis of hepatic fibrosis. The aim was to examine the inhibitory effect of butein on hepatic stellate cells activation. Hepatic stellate cells were isolated from normal rat livers and cultured on plastic dishes. The cell morphology and actin cytoskeleton were studied with phase contrast and fluorescence microscopy, in cultured hepatic stellate cells, butein inhibited type I collagen production, and the alpha-smooth muscle actin expression and cell proliferation. This finding indicates that the plant polyphenols, butein inhibited activation and proliferation in hepatic stellate cells.

[PA1-28] [04/20/2001 (Fri) 10:30 – 11:30 / Hall 4]

The effect of DKY on intestinal glucose absorption, insulin secretion, and α -glycosidase inhibition

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Dongryongkangdangyoungjung (DKY), composed of 36 herbs, has been used in China for treating diabetes mellitus. We investigated the effect of DKY on intestinal glucose absorption and insulin secretory activity using clonal β cell line RINm5F cell. α -glycosidase inhibitory activity of DKY was also examined *in vivo* and *in vitro*. In the *in situ* intestine circulation method, DKY inhibited glucose absorption from the small intestine in a concentration dependent manner. Release of insulin was stimulated by DKY. DKY inhibited the increase of blood glucose level in an oral administration of glucose in KKAY mice. There was also concentration dependent effect of DKY on α -glycosidase inhibitory activity *in vitro* using p-nitrophenyl- α -D-Glucopyranoside as a substrate. This study indicated that part of the hypoglycemic activity of DKY is based on its inhibitory actions on intestinal glucose absorption, α -glycosidase inhibitory activity and insulin secretory activity.

[PA1-29] [04/20/2001 (Fri) 10:30 – 11:30 / Hall 4]