

The purpose of this research was to investigate effects of *Phellius linteus* methanol extract (PL-M) on nitric oxide production from peritoneal macrophage in normal and cyclophosphamide (CY)-treated mice. PL-M was administered p.o. single (400, 800, 1600 mg/kg) or once a day for 5 days to normal and CY (20mg/kg, i.p.)-treated mice. Peritoneal macrophages were obtained from mice after 2 days and cultured for 48 hours with LPS. Also, normal mouse peritoneal macrophages (1×10^6 cells/ml) were incubated in the presence of various concentration of PL-M (0.5, 1.0, 2.5, 5mg/ml) and/or CY (6mM/ml) for 48 hours. Nitric oxide production was affected by treatment PL-M in vitro and in vivo. And this effect was dependent on the dose of PL-M. PL-M showed inhibition of suppression of nitric oxide production induced by CY.

[PA3-6] [10/19/2000 (Thr) 10:00 – 11:00 / [Hall B]]

Screening of natural product inhibitors on the UVA phototoxicity of Chlorpromazine

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15 Natural products known to contain antiinflammatory effect were screened whether they have UVA phototoxicity inhibitory effect or not by two methods – RBC photohemolysis and MTT assay. Samples were obtained by the process of 80% methanol extraction and then concentration under vacuum. And we made these concentration powder with freeze-dryer at -50°C ~ -60°C . In RBC photohemolysis method, effects of the test compounds on RBCs were monitored with a spectrophotometer by the method of Kahan et al. And in MTT assay we used human fibroblast cell. UVA 3 J/cm^2 and 2.5 J/cm^2 were irradiated respectively.

[PA3-7] [10/19/2000 (Thr) 10:00 – 11:00 / [Hall B]]

Vasorelaxation and Antiproliferation of Apigenin

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Apigenin, a flavone, has showed hypotensive effect in vivo and relaxant effect in vitro. However, the mechanisms remain incompletely understood. This study aimed to determine whether the relaxant effect of apigenin was endothelium-dependent and to examine the possible antiproliferative effect of apigenin on cultured aortic smooth muscle cells. Apigenin relaxed the 65.4 mM K^+ and 10^{-7} M phorbol 12,13-dibutyrate-induced contraction of endothelium-denuded rat aortic rings with respective IC₅₀ values of $12.0 \pm 1.1 \times 10^{-6} \text{ M}$ and $34.6 \pm 1.2 \times 10^{-6} \text{ M}$. Apigenin also relaxed phenylephrine-precontracted endothelium intact aortic rings with IC₅₀ values of $3.7 \pm 0.5 \times 10^{-6} \text{ M}$ and removal of a functional endothelium significantly attenuated the apigenin-induced relaxation (IC₅₀ = $8.2 \pm 0.9 \times 10^{-6} \text{ M}$) without affecting the maximum relaxant response. Pretreatment with 30 and 100 μM N-nitro-L-arginine methyl ester (L-NAME) reduced the relaxant effect of apigenin and the IC₅₀ values of apigenin were decreased to $6.9 \pm 1.2 \times 10^{-6} \text{ M}$ and $8.7 \pm 0.6 \times 10^{-6} \text{ M}$, respectively. Pretreatment of apigenin significantly potentiated relaxant effect of acetylcholine on phenylephrine-induced contraction. In addition, apigenin significantly inhibited [³H]thymidine incorporation into primary cultured rat aortic smooth muscle cell DNA in a dose-dependent manner. These findings suggest that nitric oxide from endothelium may account primarily for the apigenin-induced endothelium-dependent relaxation and mechanisms related to protein kinase C activation may also be involved in hypotensive activity. Both vasorelaxant and antiproliferative effects may contribute to a benefit of apigenin in the vascular system.