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Nitric oxide (NO) and prostagladins(PGs) produced by inducible nitric oxide synthase(iNOS) and cyclooxygenase(COX-2) are known as inflammatory mediator. Modulation of these enzymes, induced by many stimuli(LPS, IFN-gamma, TNF-alpha, phorbol ester, etc), is a potent strategy as treatment of inflammatory diseases.
Treatment of murine macrophage RAW 264.7 cell line with indole compound(IND-6) markedly reduced lipopolysacchride(LPS) stimulated NO production in a concentration-related manner. In this point of view, we tested the effect of various indole compounds in LPS-stimulated RAW 264.7 murine macrophage cell line. Western blot analysis and RT-PCR showed that IND-6 inhibited of iNOS and COX-2 proteinand mRNA expression through the attenuation of IkappaB-alpha degradation induced by LPS. Moreover, we investigated the effect of this compound on pro-inflammatory cytokine TNF-alpha production.

[PC1-3] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Antioxidative effect of flavonol quercetin and hydrocaffeic acid against a oxidative stress on B16F10 murine melanoma cell of pretreated with hydrogen peroxide

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In this study, we investigated the effect of inhibition of proliferation and antioxidant effect on B16F10 murine melanoma cell. Also, we examined by MTT(3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay and intracellular reactive oxygen intermediate levels and the levels of catalase(CAT), superoxide dismutase (SOD), and glutathione peroxidase(GPX) an adaptive response of oxidative stress on B16F10 murine melanoma cell of pretreated with hydrogen peroxide. Quercetin and hydrocaffeic acid were used 25uM, 50uM, 100uM, 200uM,concentration. From this result, quercerin and hydrocaffeic acid demonstrated a dose-dependent reduction in the effect of inhibition of proliferation and increased enzymic antioxidant levels. It may be useful by reducing or preventing an oxidative stress damage.

[PC1-4] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Inhibitory effects of synthetic isoflavone compounds on IL-5 bioactivity

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Eosinophilic inflammation is the main histological correlate of airway hyperresponsiveness and tissue injury in the pathogenesis of bronchial asthma. Interleukin (IL)-5 appears to be one of main proinflammatory mediators that induce eosinophilic inflammation. Allergic IL-5-deficient mice do not generate eosinophilia in the bone marrow, blood or lung in response to allergen provocation. However, airway instillation of recombinant IL-5 to the allergic IL-5-deficient mice