[PB4-2] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Effects of Ginsenoside Rg1 on the Expression of TNF-α from Rat microglia

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Microglial cell can act for phagocytosis against abnormal particles in brain, which means that beta-amyloid produced from APP(amyloid precursor protein) can be phagocytosed by microglia when released. In contrast, when senile plaque has already been formed in brain cortex and hippocamphal region, microglia can also accelerate the AD pathogenesis due to chronic inflammatory action, which lead to neuron cell cytotoxicity. In our study, we investigated the degree of activation of microglia by using Rg1. For the study, we selected TNF-α released from microglia. Experimental groups were separated in two, one for beta-amyloid and the other for non-beta-amyloid group. As a result, Rb1 showed highest release of TNF-α at 48 hour. In non-beta-amyloid group, Rg1 showed increased release of TNF-α at 0.1μM, 100μM. In addition, Aβ group showed that Rg1 suppresses TNF-α at 100μM. In conclusion, Rg1 may play a certain role in treatment and prevention of AD in a way to suppress the immune reaction in microglia adjacent around the neuron cell.

[PB4-3] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Effect of Ginsenoside Rg3 in Mouse Hematopoietic Cells

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Rg3 is a derivative of triterpenoid dammarane, which originally extracted from Red Ginseng, which have been known to have neuroprotective, vasodilator, antioxidative, antimetastasis, and direct anticancer effects. These various backgrounds of Rg3 can provide an additional interest in respect to the "hematopoiesis" in bone marrow and spleen cells. We, therefore, have investigated what effects and correlates of Rg3 (e.g. suppression and side effects) are affected in relation with the bone marrow and spleen cells of mouse. For this study, we designed to know how Rg3 affects the process of hematopoiesis in stem cell level. From the study, we concluded that Rg3 controls the growth and differentiation of the immune cells through increase of the hematopoietic cells, whereas Rg3 reduced and rather mostly controled the side effect of cyclophosphamide (CTX) in over a studied concentration. In conclusion, Rg3 can reduce the cytotoxicity affecting hematopoietic system inducible from anticancer agents as well as direct anticancer effects. IN addition, good potential of hepatopoiesis is expected to give an breakthrough information in clinical use when use Rg3 as an adjuvant agent both in radio- and chemotherapy.

[PB4-4] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Korean Propolis enhances both the presentation of DC and macrophage activation

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Calcineurin inhibitors, cyclosporine A (CsA) and tacrolimus (FK506), have been studied extensively regarding their effects on T lymphocytes, but their effects on dendritic cells (DC) are relatively unknown. DC can really capture Ag from dead and dying cells for presentation to MHC class I-restricted CTL. The main targets for the immunosuppressive calcinerin inhibitors, FK506 and CsA, have been considered to be activated T cells, but not antigen presenting cells (APCs). Here we demonstrate that CsA and FK506 inhibit cross-presentation of exogenous antigen by DCs. Particulate form of OVA was efficiently captured, processed and presented on class I MHC molecules (cross-presentation) as well as on class II MHC. Addition of FK506 and CsA to the cultures of DCs inhibited both class I MHC estricted presentation and class II MHC estricted presentation of exogenous OVA. In this study, we wished to determine whether presentation of exogenous OVA (10 μg/ml) could be enhanced by one of the potential natural products, Water Extract of Korean Propolis (WEP) (1.2 μg/ml), which have been used for the regulator of immune response as a traditional medicine remedy and had been shown that inhibition of the production, cytokine production, enhancement of surface molecule expression, and cell morphologic antigen expression on LPS-activated RAW 264.7 cells in previous screening study. WEP could also activate macrophages by producing cytokines. The production of the macrophage cytokines, IL-1 and TNF-α by RAW 264.7 treated with WEP was examined from 2.5 μg/ml up to 25 μg/ml with dose dependent manner.

[PB4-5] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Linarin enhances both the presentation of exogenous particulate antigen in association of Class I Major Histocompatibility antigen and macrophage activation

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Calcineurin inhibitors, cyclosporine A (CsA)and tacrolimus (FK506), have been studied extensively regarding their effects on T lymphocytes, but their effects on dendritic cells (DC) are relatively unknown. DC can really capture Ag from dead and dying cells for presentation to MHC class I-restricted CTL. The main targets for the immunosuppressive calcinerin inhibitors, FK506 and CsA, have been considered to be activated T cells, but not antigen presenting cells (APCs). Here we demonstrate that CsA and FK506 inhibit cross-presentation of exogenous antigen by DCs. Particulate form of OVA was efficiently captured, processed and presented on class I MHC molecules (cross-presentation) as well as on class II MHC. Addition of FK506 and CsA to cultures of DCs inhibited both class I MHC estricted presentation and class II MHC estricted presentation of exogenous OVA. In this study, we wished to determine whether presentation of exogenous OVA (10 μg/ml) could be enhanced by one of the potential natural products, linarin (0.6 ~ 10 μg/ml), which have been used for the regulator of immune response as a traditional medicine remedy and had been shown that inhibition of the production, cytokine production, enhancement of surface molecule expression, and cell morphologic antigen expression on LPS-activated RAW 264.7 cells in previous screening study. The production of TNF-α by macrophages treated with linarin was appeared in a dose dependent manner. The production of IL-1, however, was not the case by this natural product. The present study demonstrates the ability of linarin to activate macrophages directly or indirectly and affecting both cytokine production and nitric oxide inhibition, as well as the expression of some surface molecules.

[PB4-6] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]