Department of Pharmacy, Sahmyook University, 26-21 Gonglung-Dong, Seoul, 139-742 South Korea

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

Han ShinHa°, Yun Yunha, Son HanShik, Lee SookYeon, Ha Nam-Joo, Kim KyungJae

Department of Pharmacy, Sahmyook University, 26-21 Gonglung-Dong, Seoul, 139-742 South Korea

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]

Effect of Ginsenoside Rb1 on IL-1β expression in rat microglia

Joo SeongSoo, Kang HeeChul, Lee Dolk[O]

Department of Immunology, College of Pharmacy, Chung-Ang University, Seoul 156-756, Korea

As medicinal knowledge developed, population system has been migrating into the aged society. These aged society evokes the social problem of senile diseases. One of these, Alzheimer's Disease (AD) has been an issue, and its causative, β-amyloid(Aβ) has also been a key target in drug development. In AD, microglial cell affects neuron cells in any way of positive and negative. Particularly, microglial cell can eliminate lots of pathogens including β-amyloid(Aβ) in AD. However, in a state of already started and progressed AD, microglial cell rather induces chronic inflammation and lastly shows neurotocixity. In our study, Rb1 was investigated on the release of IL-1β and NO from microglia. As a result, Rb1 showed to suppress the release of IL-1β and NO in 100μM of Aβ-treated group, while normal control group showed a weak increase of IL-1β and NO. These results provide that Rb1 can be used in AD to reduce and prevent the pathological symptoms of AD.

[PB4-7] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Expression of TNF-α in rat microglia by ginsenoside Rb1

Joo SeongSoo, Kwon HeeSeung, Lee Dolk[O]

Department of Immunology, College of Pharmacy, Chung-Ang University, Seoul 156-756, Korea

Azheimer's Disease (AD) known as senile dementia accounts for 50% of all dementia cases and is in growing status as population goes up. Generally, AD is a progressive neurodegenerative disease and includes much of senile plaque in cerebral hippocampus and cortex in patient's brain. For decades, AD theory is explained by amyloid cascade hypothesis. In process of the hypothesis, amyloid hypothesis forms fibrillar form beta-amyloid peptide (Aβ peptide) and extraordinarily accumulates in brain tissue, and lastly senile plaque is formed, which pathologically affect the brain. In this process, activation of microglial cell is important to prevent the plaque formation out of Aβ peptide by its phagocytic function. In our study, we investigated how Rb1 acts in the release of TNF-α and NO from microglial cells, especially in suppressive function. In conclusion, Rb1 suppressed the release of TNF-α and NO from microglial cell.

[PB4-8] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Immuno-modulation Effects of Ginsenoside Rg1 in Rat microglia

Joo SeongSoo, Won TaeJoon, Lee Dolk[O]

Department of Immunology, College of Pharmacy, Chung-Ang University, Seoul 156-756, Korea

Microglial cell is a monocyte involved in the brain, which acts for a primary immune reaction and phagocytosis. Microglia has also been considered to have a great role in AD pathogenesis due to its intact inflammatory and phagocytic responses against foreign invaders. In the study, we tried to investigate the modulation of activation of microglia using Rg1, a class of ginsenoside from red ginseng, which are known to protect neuron cells. For this experiment, we have chosen a proinflammatory cytokine, IL-1β and NO released from microglia. Study groups were separated in two, one for Aβ and the other for non-Aβ group designed for various times and concentrations. As a result, Rg1 showed the highest response both in NO and IL-1β at 48 hour. In non-Aβ group, Rg1 showed increased release of NO and IL-1β at 0.1μM, 100μM. In addition, A