

## Effect of Ginsenoside Rb1 on IL-1 $\beta$ expression in rat microglia

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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,  $\beta$ -amyloid(A $\beta$ ) 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  $\beta$ -amyloid(A $\beta$ ) in AD. However, in a state of already started and progressed AD, microglial cell rather induces chronic inflammation and lastly shows neurotoxicity. In our study, Rb1 was investigated on the release of IL-1 $\beta$  and NO from microglia. As a result, Rb1 showed to suppress the release of IL-1 $\beta$  and NO in 100 $\mu$ M of A $\beta$ -treated group, while normal control group showed a weak increase of IL-1 $\beta$  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- $\alpha$ in rat microglia by ginsenoside Rb1

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Alzheimer'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 (A $\beta$  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 A $\beta$  peptide by its phagocytic function. In our study, we investigated how Rb1 acts in the release of TNF- $\alpha$  and NO from microglial cells, especially in suppressive function. In conclusion, Rb1 suppressed the release of TNF- $\alpha$  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

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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-1 $\beta$  and NO released from microglia. Study groups were separated in two, one for A $\beta$  and the other for non-A $\beta$  group designed for various times and concentrations. As a result, Rg1 showed the highest response both in NO and IL-1 $\beta$  at 48 hour. In non-A $\beta$  group, Rg1 showed increased release of NO and IL-1 $\beta$  at 0.1 $\mu$ M, 100 $\mu$ M. In addition, A