enhanced by administration of CK and CKI.

[PB4-13] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Anti-Inflammatory Effect on Rat Microglia by UDCA

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Ursodeoxycholic acid (UDCA) has been known commonly improving hyperbilirubinemia and excretion abnormality of bromsulphalein, which are appeared in the liver, and reducing the release of cholesterol from bile duct. In our study, UDCA was aimed to know if the agent can inhibit the pathogenesis of AD by suppressing the microglial activation when stimulated particularly by Aβ peptide, which is known a major cause of AD. For the study, we selected proinflammatory cytokines such as TNF-α and NO released from the brain microglia. In the study, 1day Sprague Dawley-rat(SD-rat) was used for the culture of microglia. Microglia taken from the isolated period were co-incubated with LPS or beta-amyloid to activate microglia at various concentrations of UDCA during scheduled times. From the experiment, we obtained very interesting results that UDCA plays a role in suppressing the inflammatory parameters of microglia. In conclusion, a new research area for AD by UDCA is worthy of studying by suppressing microglia at early stage of plaque formation in brain, and can be a basic knowledge in a future clinical approach between UDCA and AD.

[PB4-14] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Involvement of Phosphodiesterase Isozymes in Osteoclast Formation

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cAMP acts as a second messenger in the functional responses of various cells to hormones, cytokines and other agents. In turn, this nucleotide also modulates the signal transduction processes regulated by a range of cytokines and growth factors.
The intracellular level of cAMP is regulated by a G protein coupled adenylyl cyclase and degradation is mediated by the phosphodiesterases (PDEs), a superfamily of enzymes that catalyze the hydrolysis of cAMP.
In osteoblast, activation of adenylyl cyclase by parathyroid hormones (PTH) or prostaglandins (PGs) has been found to increase osteoclast formation via expression of TRANCE, a key molecule for osteoclast differentiation and activation. However, whether PDEs in osteoblast regulate osteoclastogenesis is unknown.
In this study, RT-PCR analysis of mouse primary osteoblast revealed the presence of PDE1, PDE2, PDE3, PDE4, PDE5, PDE7, PDE8, and PDE9. Furthermore, the general PDE isozyme inhibitor enhanced osteoclastogenesis in a dose dependent manner in co-culture system. These results indicate that PDE isozymes in osteoblast are involved in the modulation of osteoclast differentiation.

[PB4-15] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Production and characterization of a PPARgamma-specific monoclonal antibody Pγ 48.34A

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Peroxisome proliferator-activated receptor (PPAR), a member of the nuclear hormone receptor superfamily, is a transcription factor activated by specific natural or synthetic ligands. It is involved in various cellular processes including adipogenesis, inflammation, cell cycle progression and carcinogenesis. Here, we report the production and characterization of a PPARgamma subtype-specific monoclonal antibody Pγ48.34A, which was raised against full-length human PPARgamma protein. Characterization of Pγ48.34A has been performed by Western blot analysis using several PPARgamma recombinant proteins, mouse tissue lysates as well as immunoprecipitates obtained from indomethacin-treated 3T3-L1 cell line. Moreover, an ELISA system using Pγ48.34A has been optimized to screen various PPARgamma ligands. Based on these results, Pγ48.34A is considered to be a useful tool for elucidating the role of PPARgamma in the various cellular processes.

[PB4-16] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

An effect of UDCA in production of IL-1β and NO by Microglia in Rat.

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In recent, growing aged people in coupled with the increased senile dementia, Alzheimer's disease, has been a social interests to be cleared out. Alzheimer Disease(AD), first reported by Alios Alzheimer (1864-1915) in 1907, is a neurodegenrative disease. Nothing exact cause of AD is available by now, but in clinical founding β-amyloid peptide(Aβ) and microtubule associated protein(τ protein) is to involved in the disease, and the most important feature in AD is known to induce chronic inflammation to neuron cell. According to drug development fields, Ursodeoxycholic acid(UDCA) in a certain concentration has immunomodulating function, and thus it suppresses the release of IL-2, IL-4, IFN-γ from T-cell and suppresses the production of immunoglobulin. Additionally, UDCA reduces the release of IL-1β in macrophage. In our study, we chosen UDCA as study agent for the suppression of proinflammatory cytokine, IL-1β and NO in microglial cell, a brain immune cell. As a result, UDCA showed outstanding suppressive responses from microglia in the released level of IL-1β and NO stimulated by various active agent including Aβ. These results are expected UDCA to be a potentially promising AD agent preventing and reducing the symptoms of AD.

[PB4-17] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

The protective role of the polysaccharide Ginsan against Staphylococcus aureus : induction of NO and reduction of poinflammatory cytokines

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