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