

drugs. We could also determine the plasma concentration of AS even 5 hours after administration to rats. Conclusion: These results show that the longer duration of AS in blood has a possibility to interact with coagulation factors.

[PA1-61] [2003-10-10 14:30 - 17:30 / Grand Ballroom Pre-function]

gInhibition effect of nitric oxide production and NF- κ B nuclear translocation by 2-hydroxycinnamaldehyde in RAW 264.7 cells

Lee Seung Ho^o, Lee Sun Young, Park Hye Ji, Lee Yoot Mo, Lee Heesoon, Song Sukgil, Yoo Hwan Soo, Oh Ki Wan, Kwon Byoung Mog, Hong Jin Tae

College of Pharmacy, Chungbuk National University and KRIBB

Cinnamaldehyde is the main component of cinnamon bark oil and show several biological activities such as anti-tumor, anti-fungal, anti-mutagenic and anti-inflammatory effects. A couple of studies have investigated how the natural compound exerts its anti-inflammatory effect. In despite of numerous investigations, the biological mechanism of effects belong to cinnamaldehyde remain unclear. We isolated 2-hydroxycinnamaldehyde(HCA) from the bark of Cinnamomun cassia Blume and reported a various of biological activities of HCA. HCA also exert several biological effects as much as that of cinnamaldehyde. In this study, we investigated anti-inflammatory effects of 2-hydroxycinnamaldehydes and putative mechanisms of its action in Raw 264.7 cells. HCA inhibited Nitric Oxide(NO) production in RAW 264.7 cells, which IC₅₀ value was 1.3 μ M. Using gel shift assay, we showed that HCA inhibit activation of the transcription factor NF- κ B, a central regulator of NOS and inflammatory response of body. We are also investigating of other molecular mechanism of HCA; Whether HCA can inhibit COX-2 expression, and thereby inhibit prostaglandin E2 production, another important inflammatory mediator through interfering NF- κ B activation. We provide evidence that HCA is a potent anti-inflammatory agent and could serve as lead compounds for the development of pharmaceutically used anti-inflammatory remedies.

[PA2-1] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Histone deacetylase inhibitor Trichostatin A enhanced the efficiency of adenovirus mediated gene transfer into non-small cell lung cancer cells

Park Misun^o, Kang Hoil, Lim Sinae, Jee Seungwan, Eom Miok, Ryeom Taikyung, Kim Okhee

KFDA, National Institute of Toxicological Research, Genetic Toxicology Division

One of the major limitations in using adenoviral vector for gene therapy is inefficient infection of host cells. The presence of coxsackievirus and adenovirus receptor (CAR) and α -integrin on cell surfaces is required for efficient adenovirus infection. In this study, we investigated the effect of trichostatin A, a histone deacetylase inhibitor, on transfection efficiency after transduction of adenovirus mediated p16^{INK4a} gene transfer. In our previous study, p16^{INK4a} tumor suppressor gene transfer in the non-small cell lung cancer cells (A549 cells) by transduction of recombinant adenovirus (Ad5CMV-p16) resulted in significant inhibition of cancer cell proliferation. We found that A549 cells treated with trichostatin A prior to adenoviral vector (Ad5CMV-LacZ) infection had an increase in expression of β -galactosidase. p16^{INK4a} gene expression was also increased in A549 cells after combination treatment of trichostatin A and Ad5CMV-p16 by RT-PCR. On the other hand, there was only weak combination effect of trichostatin A and Ad5CMV-p16 in normal lung cell lines (CCD-16, MRC-9). Currently, we are investigating the effect of trichostatin A on CAR expression level. These studies suggest that trichostatin A increases the efficiency of adenoviral transgene expression in cancer cells and this combination therapy may be useful in cancer gene therapy.

[PA2-2] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Biodistribution of [¹²⁵I]-labeled biotinylated dendrimer derivatives for antibody pretargeting