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-κB nuclear translocation by 2-hydroxycinnamaldehyde in RAW 264.7 cells

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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 cinnamalgehyde. 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 IC_{50} value was 1.3μM. Using gel shift assay, we showed that HCA inhibit activation of the transcription factor NF-κB, 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-κB 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

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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 trichostaitn 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]

Biodistribition of [^{125}I]-labeled biotinylated dendrimer derivatives for antibody pretargeting