[PB2-1] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Inhibitory Action of Compound-A on Arthus Reaction, Formation of Plaque Forming Cells and Hemagglutination of the Sheep Red Blood Cell

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Effects of Compound-A, a phenylpropanoid isolated from Arctium lappa fruit, on sheep red bood cells (sRBC) - induced arthus reaction (AR) were studied in ICR male mice and determined the plaque forming cells (PFC) numbers and hemagglutinin (HA) titer . Two weeks after sensitization of i.p. injection of sRBC ($4x10^8$ cells), ICR male mice were challenged by i.p. injection of sRBC ($2x10^8$ cells). Five days after the challenge of antigen, paw edema induced three hours after the last challenge by Arthus reaction. Drugs were orally administered one hour before the last challenge of antigen. Spleen cells of the mice were isolated by cytosieve (100 mesh), the viability of spleen cells was determined by trypan blue exclusion test immediately before used. HA titer to sRBC were carried out to determine hemagglutination of sRBC and exhibited as $\log_2 X$ (X is the highest dilution). PFC was calculated with microscope and exhibited as the number of PFC. It shows that Compound-A at a dose of 50 mg/kg inhibited significantly the Arthus reaction as compared with control (37.7±4.14 %, $p<0.05$). Its activity was more than prednisolone acetate (10 mg/kg) and disodium cromoglycate (20 mg/kg). And Compound-A has dose-dependently inhibited formation of PFC. Its inhibitory activity at a dose of 25 and 50 mg/kg inhibited 37.8±3.0 and 36.8±3.0 %, respectively ($p<0.05$). These results can be showed that Compound-A inhibited reaction of Type Ⅲ Hypersensitivity.

[PB2-2] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

LPS-induced Imbalanced Expression of Hepatic Vascular Stress in Hepatic Ischemia and Reperfusion

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Hepatic ischemia and reperfusion predisposes the liver to secondary stresses such as endotoxemia possibly via dysregulation of the hepatic microcirculation secondary to imbalanced regulation of vascular stress gene. In this study, we determined the effect of endotoxin on hepatic vasoregulatory gene expression in response to hepatic ischemia and reperfusion (I/R). Rats were subjected to 90 min of hepatic ischemia and 6 h of reperfusion. Lipopolysaccharide (LPS, 1 mg/kg) was injected intraperitoneally after reperfusion. Liver samples were obtained 6 h after reperfusion for RT-PCR analysis of mRNA for genes of interest: endothelin (ET-1), its receptors ET_A and ET_B, endothelial nitric oxide synthase (eNOS), inducible nitric oxide synthase (iNOS), heme oxygenase-1 (HO-1), and tumor necrosis factor-a (TNF-a). The activities of serum alanine aminotransferase and aspartate aminotransferase significantly increased in I/R group. This increase was markedly potentiated by LPS treatment. Although there were no changes in HO-1 and COX-2 mRNA levels in LPS alone, the levels of ET-1, ET_B, iNOS and TNF-a mRNA significantly increased. The expression of ET-1, ET_B, HO-1 and COX-2 mRNA significantly increased in I/R alone, which was not affected by LPS treatment. The levels of iNOS and TNF-a mRNA significantly increased in I/R alone. This increase was significantly potentiated by LPS treatment. There were no significant differences in ET_A and eNOS mRNA levels among any of the experimental groups. Our findings suggest that I/R challenged with a secondary insult of endotoxemia aggravate the imbalanced vasoregulatory gene expression.

[PB2-3] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Effects of Compound-A on the Early-Phase Anaphylactic Type Hypersensitivity

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