

anti-rat serum rabbit serum in SD rat. Drugs were orally administered one hour before antigen challenge. HY titer were determined the hemolysis of sheep red blood cell (sRBC) to spleen cells. Two weeks after sensitization of i.p. injection of sRBC, the mice were challenged with sRBC. On five day after the mice were rechallenged, spleen cells were isolated by cytosieve (100 mesh), the viability of spleen cells was determined by trypan blue exclusion test immediately before used. HY titer to sRBC were carried out to determine hemolysis in inactivated mice serum added guinea-pig complement, and exhibited as  $\log_2 X$  (X is the highest dilution). Drugs were orally administered one hour before the last challenge of antigen. It shows that Compound-A has dose-dependently inhibited the RCA as compared with control : Its inhibitory activity at a dose of 25 and 50 mg/kg were  $18.0 \pm 2.4$  and  $23.4 \pm 1.4$  %, respectively ( $p < 0.05$ ). Its activity at a dose of 50mg/kg was same as prednisolone acetate at a dose of 10 mg/kg and its activity at a dose of 25 mg/kg was same as disodium cromoglycate at a dose of 20 mg/kg. And Compound-A at a dose of 50 mg/kg inhibited significantly the HY titer as compared with control ( $34.1 \pm 3.0$  %,  $p < 0.05$ ), but its activity was more active than disodium cromoglycate (20 mg/kg) and less active than prednisolone acetate (10 mg/kg). Compound-A has the dose-dependently inhibitory action on RCA and HY titer. These results showed that Compound-A has the inhibitory activity of type II hypersensitivity such as RCA and CDH.

[PB2-6] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]

### **Compound-A inhibited the Asthmatic Responses in the Conscious Guinea Pigs**

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Effect of Compound-A, a phenylpropanoid isolated from *Arctium lappa* fruit, on the early- (EAR) and late-phase asthmatic responses (LAR) of guinea pigs were studied in vivo. Guinea pigs were sensitized by injection of 100 mg of ovalbumin (OA). Twenty-one days after sensitization, animals were challenged with exposure to aerosolized 1 % OA for five minutes in double-chambered plethysmograph box with jet nebulizer. Immediately and twenty-four hours after challenge, EAR and LAR asthmatic responses were determined the tidal volume (TV), respiration rate (RR) and specific airway resistance (sRaw), and then animals anesthetized and taken the bronchoalveolar lavage fluid (BALF) by lavage the lung with HEPES buffer through cannulation into trachea. BALF cytopspined and stained by Wright's stain, and the contents of leukocytes, histamine, phospholipase A<sub>2</sub> (PLA<sub>2</sub>), eosinophil peroxidase (EPO) and protein were measured in BALF. The TV at a dose of 25 mg/kg inhibited  $42.1 \pm 9.3$  % in EAR,  $21.1 \pm 6.3$  % in LAR as compared with control, respectively ( $p < 0.05$ ). sRaw at a dose of 25 mg/kg increased  $187.5 \pm 33.4$  % in EAR,  $97.1 \pm 15.5$  % in LAR as compared with control, respectively ( $p < 0.05$ ). but its activity was less than dexamethasone (5 mg/kg) and disodium cromoglycate (10 mg/kg). And Compound-A at a dose of 50 mg/kg inhibited significantly recruitment of total leukocytes and neutrophils ( $p < 0.05$ ), and inhibited significantly recruitment of eosinophils at a dose of 25 mg/kg, but its activity was less than dexamethasone (5 mg/kg) and disodium cromoglycate (10 mg/kg). Also Compound-A at a dose of 50 mg/kg inhibited significantly protein exudation and PLA<sub>2</sub> ( $p < 0.05$ ), its activity was same as dexamethasone (5 mg/kg) and disodium cromoglycate (10 mg/kg), respectively. Compound-A dose-dependently inhibited the histamine release ( $p < 0.05$ ), but its activity was less than dexamethasone (5 mg/kg). These results showed that Compound-A dose-dependently inhibited asthmatic responses.

[PB2-7] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]

### **Effect of Ascorbic Acid on the Activity and Gene Expression of Cytochrome P450 in Sepsis**

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Sepsis remains common surgical problems with high morbidity and mortality despite improvement in the management for septic patient. Although hepatocellular dysfunction occurs during sepsis, the mechanism responsible for this remains unclear. In sepsis, a state of severe oxidative stress is encountered, with host endogenous antioxidant defenses overcome. Therefore, the aim of this study was to determine whether specific