[Biochem. J., 275, 121-126 (1991)]

[Lab. of Biochemistny]

## Inhibition of Dimeric Dihydrodiol Dehydrogenases of Rabbit and Pig Lens by Ascorbic Acid.

AKIRA HARA\*, MICHIO SHINODA, TAKUSHI KANAZU,

Toshihiro Nakayama, Yoshihiro Deyashiki, Hideo Sawada

Dimeric dihydrodiol dehydrogenases purified from pig and rabbit lenses were reversibly inhibited by either L-ascorbate or isoascorbate, at pH 7.5. Isoascorbate was a more potent inhibitor than ascorbate, but 1 mM-dehydroascorbate gave low inhibition. The inhibition patterns by the two compounds were competitive with respect to dihydrodiols and uncompetitive to NADP, but those in the reverse reaction were uncompetitive with respect to both carbonyl substrate and NADPH. The results indicate that ascorbate and its epimer directly bind to and enzyme: NADP complex as dead-end inhibitors. Ascorbate may be an important modulator of DD in the lens.

(Inflammation, 15, 317-330 (1991))

[Lab. of Pharmacology]

## Participation of Collagenase and Elastase in LPS-Induced Airway Hyperresponsiveness in Guinea Pigs.

H. Nagai, F. Tsuji, T. Shimazawa, S. Goto,

K. Yoshitake, A. Koda\*

Bacterial lipopolysaccharide(LPS) caused airway hyperreactivity in guinea pigs pretreated with metopirone. Increases of pulmonary capillary permeability and leukocytes in bronchoalveolar lavage fluid were observed after the inhalation of LPS. Collagenase and elastase activities were elevated 2 h after the inhalation of LPS. The inhalation of collagenase and elastase caused bronchial hyperreactivity and increased pulmonary permeability. Present results indicate the participation of collagenase and elastase in the onset of LPS-induced airway hyperreactivity in guinea pigs.

[Japan. J. Pharmacol., 56, 13-21 (1991)]

[Lab. of Pharmacology]

## Effect of Three Novel K<sup>+</sup> Channel Openers, Cromakalim, Pinacidil and Nicorandil on Allergic reaction and Experimental Asthma.

H. NAGAI, K. KITAGAKI, S. GOTO, H. SUDA, A. KODA\*

Anti-allergic and anti-asthmatic activities of cromakalim, pinacidil and nicorandil were investigated. PCA in mice, antigen-induced histamine release from guinea pig lung tissue, and mediator-induced vasculitis in rats were not affected by these drugs. Antigen-induced and high potassium-, histamine-, LTC<sub>4</sub>-, and U-46619-caused constrictions of isolated guinea pig tracheal muscle were relaxed by these drugs. Increased airway resistance caused by histamine, LTD<sub>4</sub> and U-46619, and experimental asthma in guinea pigs were inhibited by these drugs.