cule. Electrostatic interaction between VIP, a strongly basic protein[17], and acidic groups of phospholipids may also be involved and could explain the differential effect of phospholipases C and D in altering VIP binding to membranes. In conclusion, the observed effect of phospholipases on VIP binding clearly indicate the important role of phospholipids in the process of VIP binding to its membrane receptors.

1 This work was supported by INSERM (CRL 827017) and the Fondation pour la Recherche Médicale Française.

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0014-4754/85/050631-03$1.50 + 0.20/0

Changes of the level of proteinase inhibitors in rat plasma during turpentine-induced inflammation[1]

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Summary. The levels of rat plasma α-macroglobulins, α-cysteine proteinase inhibitor, haptoglobin and antipapain activity were studied during the acute-phase reaction after an injection of α-pinen. An increase in concentration of all the compounds examined was observed.
Key words. α-Cysteine proteinase inhibitor; haptoglobin, α-macroglobulins; inflammation; rat plasma.

Three high mol.wt cysteine proteinase inhibitors have been described in serum: α_2-macroglobulin (α_2-M), haptoglobin (Hp) and α-cysteine proteinase inhibitor (α-CPI) which appears in different molecular forms[14,21]. Recently, cysteine proteinase inhibitors of low mol.wt have been identified in human, rat and bovine serum[4,5,7]. The biological function of the α-CPI and the low mol.wt inhibitor are still unknown. However, it has been suggested that these inhibitors might play a role in the control of cysteine proteinases participating in the inflammatory response[14,16], and these which are involved in myofibrillar protein degradation[19].

The aim of this study was to investigate changes in the rat plasma concentration of α-CPI in comparison with other known acute-phase reactants such as haptoglobin and α-macroglobulins during the course of turpentine inflammation.

Materials and methods. Experiments were carried out with 35 healthy male rats of the Buffalo strain. Inflammation was induced by c.s. injection of α-pinen (0.2 ml/100 g b.wt) in the scapular region. After one, two, three, five and seven days of the experiment the groups of five rats were anesthetized with ether and bled by intracardiac puncture into a syringe containing heparin. The control group (10 animals) received a similar volume of saline and were bled 24 h after injection. The amount of functional α-macroglobulin was determined by Ganrot's method[2]. The sum of the normally present α_1-macroglobulin and the acute-phase α_2-macroglobulin is determined by this method[3]. The concentration of haptoglobin was measured by the peroxidase method of Jayle[8]. Antipapain capacity (APC) of rat plasma was determined in the presence of 6 mmoles/l cysteine according to Sasaki et al.[17] using casein as a substrate. The inhibitory activity of α-CPI against papain was determined after methylamine inactivation of α-macroglobulins according to Minakata et al.[12]. One unit of α-CPI or antipapain capacity was expressed as the amount inhibiting 1 mg of papain (up to 50% inhibition).

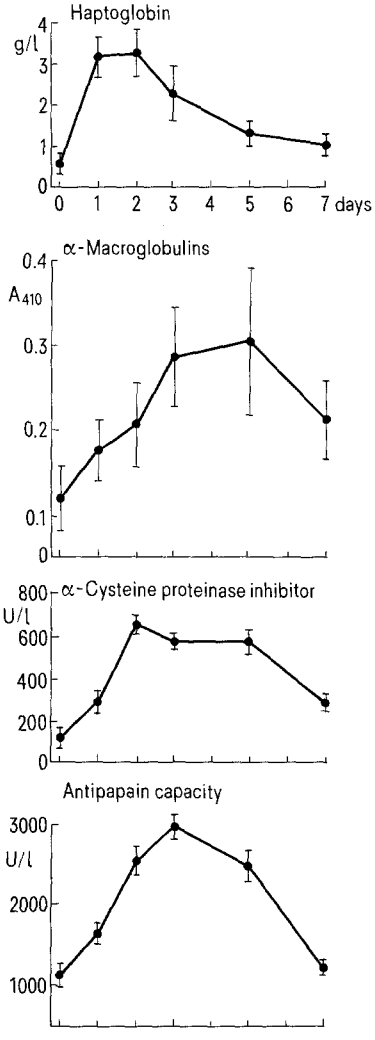
Results and discussion. As can be seen in the figure, during the inflammatory reaction increased levels of haptoglobin, α-macroglobulins, α-CPI and antipapain capacity were observed.

The plasma concentration of Hp increased significantly 24 h after turpentine injection and reached a maximum level of seven times the normal value after 48 h. It was observed that α-CPI activity increased twice after 24 h and five times after 48 h. α-macroglobulins and antipapain capacity did not increase so markedly, and reached the maximum value of about 2.5 times normal after 72 h. Calculated correlation coefficients 'r' between α-CPI and Hp, α-M, and antipapain capacity are: 0.52, 0.72 and 0.94 respectively.

Inflammation induced by various agents is reflected in the production of modulatory proteins by the liver. These proteins, referred to as 'acute phase reactants'[9], circulate in the blood. The precise functions of these proteins have not been elucidated yet. The observed changes in Hp and α-macroglobulin concentrations during the course of inflammation are in good agreement with the previous findings[3,6,10,11,18]. The correlation between α-CPI and antipapain capacity is rather low. In the human serum the inhibitor activity of α-CPI against ficin was found to be twice higher then that of α_2-macroglobulin. In our experiments we used papain as a cysteine proteinase and we found that the inhibitory activity of α-CPI in the rat plasma comprised ony about 10% of the antipapain capacity. From the work of Sasaki et al.[17] it is known that the inhibitory power of α-CPI towards the hydrolytic activity decreases in the following order: ficin, papain, cathepsin B and bromelain. We choose papain as a cysteine enzyme because of its general resemblance to cathepsins B and H[20]. Furthermore, Valeri et al.[22] demonstrated that plasma antithrombin III is also capable of

reaction with papain. In addition, the low mol.wt proteinase inhibitor found in rat serum inhibited papain, but the concentration of the inhibitor was very low[7].

The observed increase of α-CPI in the acute-phase rat plasma allows us to consider it as one of the markers of inflammation, more precisely as one of the acute-phase reactants with a positive response. Esnard and Gauthier[1] found that α-CPI isolated from acute-phase rat serum was very similar to α_1-acute phase globulin. Minakata et al.[13] established that pregnancy significantly enhanced the α-CPI level in human serum, while such pathological conditions as myoma of the uterus, endometritis, cervical cancer, ovarian cyst and ovarian cancer did not. Such results indicate that α-CPI is not an acute-phase reactant in humans. We also did not observe α-CPI level in sera of patients during polychemotherapy of ovarian cancer (not published).



Time-course changes in rat plasma levels of haptoglobin, α-macroglobulins, α-cysteine proteinase inhibitor and antipapain capacity following turpentine inflammation.

Acknowledgment. This work was supported by the grant No. R-I-9 from the Ministry of Science and Education.

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0014-4754/85/050633-02$1.50 + 0.20/0

Normetanephrine and metanephrine oxidized by both types of monoamine oxidase

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Summary. Both normetanephrine and metanephrine were found to be oxidized by both types of monoamine oxidase in mouse liver mitochondria. Both K_m and V_{max} values of type B MAO for both substrates were higher than those of type A MAO, which caused the shift of inhibition curves with clorgyline and deprenyl according to the increase in substrate concentration.
Key words. Mouse liver mitochondria; normetanephrine; metanephrine; monoamine oxidase.

Normetanephrine and metanephrine are important O-methylated metabolites of norepinephrine and epinephrine, respectively, in mammalian tissues[1]. These compounds are further metabolized by monoamine oxidase (MAO) to yield finally 3-methoxy-4-hydroxymandelic acid. The enzyme responsible for the oxidation of normetanephrine and metanephrine has been believed to be type A MAO[2]. In the present communication, however, we demonstrate that these compounds are also oxidized by type B MAO in mouse liver mitochondria.

Materials and methods. Mitochondrial fraction was prepared