TWO HUMAN IMMUNOCHEMICALLY DISTINCT LOW MOLECULAR FIBRINOLYTIC INHIBITORS. U. Hedner and M. Gallimore. Coagulation Laboratory, Allmänna Sjukhuset, Malmö, Sweden and Institute for Thrombosis Research, Oslo, Norway.

Recently a fast reacting $\alpha_2$-antiplasmin (Müllertz 1972, Collen et al 1975) not identical with the inhibitor of the plasminogen activation (Hedner and Collen 1976) was described. Using the antiserum of Dr Aoki his inhibitor (Moroi and Aoki 1976) was found to be identical with the $\alpha_2$-antiplasmin but distinct from the inhibitor of the plasminogen activation. Furthermore the inter-$\alpha$-antiplasmin described by Gallimore (1975) was found to contain both material reacting with the antiserum of Collen and with the antiserum against the inhibitor of the plasminogen activation. By means of specific immunoadsorption of the fraction eluted close to the plasminogen activation inhibitor by the method described 1973 (Hedner 1973) these two inhibitors have been separated into two fractions, which do not cross react immunochemically. In a caseinolytic system plasma from which the $\alpha_2$-antiplasmin was removed still has got an inhibitory activity against plasminogen activation. Also the $\alpha_2$-antiplasmin, plasminogen, activation inhibitors as well as $\alpha_2$-macroglobulin did not show any covariation in patients with different diagnoses. The inhibitor of the plasminogen activation did not interact with purified urokinase or plasmin but the $\alpha_2$-antiplasmin forms complexes both with human and porcine plasmin.

Thus there seem to exist at least two different low molecular fibrinolytic inhibitors migrating as $\alpha_2$-globulins in human blood.

PLASMA AND PLATELET ANTIPLASMINS: RADIAL DIFFUSION IN FIBRIN(OGEN). W. A. Andes. Oxford Haemophilia Centre, Churchill Hospital, Headington, Oxford, United Kingdom.

An antiplasmin assay has been developed utilizing radial diffusion of plasma from wells cut in plasmin-enriched, fibrinogen-agarose plates. After diffusion the fibrinogen is clotted. Zones of fibrin protected from background fibrinolysis develop as the result of plasma antiplasmin activity. The sizes of the fibrin antiplasmin zones are proportional to the concentration of plasma placed in the wells. A pooled plasma standard was taken to contain 100% antiplasmin activity. Antiplasmin activity of 52 normal subjects varied from 64 to 132%. Washed platelets (bovine albumin gradient) contained 1-5% antiplasmin activity. Using antisera to precipitate individual inhibitors, physical methods of separation, and electrophoresis of plasma in agarose, several different proteins were found to have antiplasmin activity. Thus, $\alpha_2$-macroglobulin contributed 56%, $\alpha_1$-antitrypsin 20%, antithrombin III 2%, and other proteins 22% of the total antiplasmin activity. One millilitre of whole plasma neutralized 7.0 CTA units of plasmin. Using this assay, patients with haemophilia treated with fibrinolytic inhibitors had significantly increased antiplasmin levels.

DETERMINATION OF A FAST ACTING PLASMIN INHIBITOR IN PLASMA FROM PATIENTS WITH TENDENCY TO THROMBOSIS AND INCREASED FIBRINOLYSIS. A-C. Teger-Nilsson, E Gyzander, H Myrwold, H Noppa, R OLsson and L. Wallmo Departments of Clinical Chemistry, Internal Medicine II, Surgery I, and Obstetrics and Gynecology, University of Gothenburg, Sahlgrens Hospital, Gothenburg, Sweden.

Antiplasmin activity has been determined by means of the plasmin specific tripeptide substrate H-D-Val-L-Leu-L-Lys-p-nitroanilide (S-2251) It is concluded that the method mainly measures the antiplasmin described by Collen and coworkers (Thromb. Res. 7,245,1975 ), although $\alpha_2$-macroglobulin may interfere to a small extent. Different categories of patients with tendency to thrombosis and increased fibrinolysis have been studied During normal middlesized operations antiplasmin decreased slightly, but increased postoperatively. Antiplasmin likewise decreased during deliveries, and increased in th puerperium Patients with acute deep vein thrombosis had normal antiplasmin activity at the time for diagnosis, but antiplasmin increased later during the course of events Streptokinase treated patients showed a pronounced and rapid drop in antiplasmin activity, which was normalized as soon as the streptokinase administration was interrupted Patients with liver cirrhosis had lower antiplasmin activity than normals , and so had patients with disseminated intravascular coagulation.