w. from 2,000 to 30,000). As a consequence, poly-L-lysine looses its ability to aggregate platelets. Serotonin reacts with D-glucose to form a stable and strongly reducing Amadori type compound. No change in the platelet aggregating ability of this compound has been observed, but other biological properties of the amine are highly affected.

R. Castillo, S. Maragall, J. A. Guisasola, F. Casals, C. Ruiz and A. Ordinas (Hospital Clínico y Provincial, Barcelona 11, Spain): **Inhibition of Human Platelet Aggregation by the Proteolytic Effect of Streptokinase (SK). Role of the Factor VIII Related Antigen. (234)**

Defective ADP-induced platelet aggregation has been observed in patients treated with streptokinase. This same effect appears "in vitro" when adding SK to platelet rich plasma (PRP). Classic hemophilia and normal platelet poor plasmas (PPP) treated with SK inhibit the aggregation of washed platelets; plasmin-treated normal human serum also shows an inhibitory effect on platelet aggregation. However, von Willebrand SK-treated plasmas do not inhibit the aggregation of washed platelets. The same results appear when plasmas are previously treated with a rabbit antibody to human factor VIII.

This confirms that the antiaggregating effect is mainly linked to the digested factor VIII related antigen.

The inhibition of ADP-induced platelet aggregation has been proved in gel filtration-isolated and washed platelets from SK-treated PRP.

Defective ristocetin-induced platelet aggregation has also been observed. This action does not appear in washed platelets from SK-treated PRP in presence of normal PPP, but it does in presence of SK-treated PPP, which suggests that the inhibition of the ristocetin-induced aggregation is due to the lack of factor VIII and not to the factor VIII-related products.

Heparin, either "in vivo" or "in vitro", has corrected the antiaggregating effect of SK.

J. L. Gordon, D. E. MacIntyre, A. H. Drummond (University Department of Pathology, Cambridge CB2 1QP, England): **Experimental Modification of the Platelet Release Reaction Induced by Collagen. (235)**

Collagen-induced release of platelet constituents can be divided into two kinetically-distinct phases, only one of which is associated with platelet aggregation, and the aggregation-independent release is less susceptible to inhibition by pharmacological agents (Drummond and Gordon, 1974). Minor variations in experimental conditions alter the release reaction profile. Collagen was incubated with platelet rich plasma (PRP) for one minute at 37°, under conditions in which no aggregation occurred (+3nM EDTA or in absence of stirring). The reaction was 'terminated' by addition of ice-cold EDTA-saline and samples were then centrifuged (14,800 g) under the conditions described in the table.

Time of Centrifugation	Centrifugation Temperature	Storage Time	Storage Temperature	Release of 5HT
30 s	4°	2 hours	4°	—
120 s	4°	2 hours	4°	—
30 s	20°	2 hours	4°	—
120 s	20°	2 hours	4°	+
30 s	4°	1 Min	20°	—
30 s	4°	5 Min	20°	+

No significant release was detected under any of the above conditions if saline were substituted for collagen suspension.

These results indicate that the storage temperature and time after centrifugation are as important as the centrifugation conditions themselves, and suggest reasons for discrepancies reported in previous studies of collagen induced release kinetics.

Drummond, A. H. and Gordon, J. L. (1974). Brit. J. Pharmacol. *52*, 130 P.