C. S. P. Jenkins, D. R. Phillips, K. J. Clemetson, Dominique Meyer and Marie-José Larrieu (Institut de Pathologie Cellulaire, Hôpital de Bicêtre, 94270 Le Kremlin Bicêtre, France and Theodor Kocher Institute, Bern 9, Switzerland): **Platelet Membrane Glycoproteins Implicated in Ristocetin-Induced Aggregation. (453)**

Ristocetin induces aggregation of platelets in the presence of Willebrand Factor. Certain preparations of ristocetin when preincubated with washed normal platelets render the platelets less responsive to the addition of Willebrand Factor. It has been demonstrated that such preparations are contaminated with a proteolytic enzyme(s). Platelets treated in this way with different ristocetin preparations were analysed using disc gel electrophoresis. Membrane glycoproteins I and II were depleted only when using contaminated ristocetin preparations. Platelets labelled with ^{3}H-adenine when treated with this ristocetin did not demonstrate leakage of ^{3}H-labelled material, thus showing that the platelet membrane remained intact.

Platelets were treated with proteolytic enzymes of known specificities and following further washing were tested in an aggregating system consisting of ristocetin and Willebrand Factor. The aggregation response was markedly reduced compared to control platelets. These results point to the glycoproteins of the platelet membrane being part of the receptor for the ristocetin-Willebrand Factor complex.

G. Gasic, T. Gasic, B. Hsu, P. Koch and S. Niewiarowski (Dept. Path., Univ. Penna Sch. Med. and Spec. Ctre Thrombos Res and Dept. of Med., Temple Univ. Hlth. Sci Ctre, Philadelphia, Pa.): **Platelet Interaction with Tumor Cells. (454)**

Previous investigations demonstrated that mouse tumors cause platelet aggregation (PA) and increase platelet turnover. Depletion of platelets by neuraminidase and inhibition of PA by aspirin reduced the number of metastases (Gasic et al., *Int. J. Cancer 11*, 704, 1973). The purpose of this investigation was to study further interaction of cells from various mouse and human tumors with platelets. Cells of 7 mouse tumors (1 mammary adenocarcinomas, 5 sarcomas, 1 melanoma) and 14 human tumors (8 breast, 3 colonic adenocarcinomas, 1 cancer of the ureter, 1 Wilms tumor, and 1 neuroblastoma) aggregated homologous platelets suspended in heparinized plasma. Three mouse tumors (2 mammary and 1 sarcoma) and 5 human tumors (2 breast, 1 sarcoma, 1 Wilms, and 1 neuroblastoma) did not. PA was accompanied by the release of radio-activity from ^{14}C-serotonin labeled platelets (range 15–90%). PA activity was not correlated with fibrinolytic or procoagulant activity. The contribution of plasminogen activators, thrombin, and tumor immune complexes has been excluded. However, gamma globulin of tumor bearing mice contained a "blocking factor" which delayed PA. Since enzymatic removal of ADP reduced PA it is possible that the ADP release either by tumors or by platelets played a contributory role. The pattern of PA by tumor cells ressembled that induced by collagen. Indeed preliminary evidence suggests that collagen-like material associated with tumor cells might be involved in platelet adherence to these cells and subsequent aggregation.

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M. Steinbuch, A. Friess, J. Drouet and P. Amouch (Centre National de Transfusion Sanguine, 6, rue Alexandre-Cabanel, Paris XVè): **Absence of Interaction between Streptokinase/Plasmin(ogen) Complexes and α_2-Macroglobulin (α_2M). (455)**

Streptokinase, plasmin(ogen), the activator complex and α_2M have different, well defined electrophoretic mobilities in agarose/acrylamide in function of their electric charge and molecular size. Highly purified I^{131}-labeled streptokinase has been used to study whether an interaction with α_2M can be seen. No radioactivity associated with α_2M could be found. Thus, with respect to α_2 M, streptokinase does not behave like urokinase. Furthermore, activator complexes formed in the cold (4° C) to avoid any dissociation did not react with α_2M either as shown by the same technique whereas I^{131}-labeled plasmin became normally fixed to α_2M under the same conditions. Several anodic radioactive