ENHANCED ASSOCIATION OF FIBRINOGEN WITH ITS PLATELET RECEPTOR DUE TO SODIUM CITRATE INDICATION FOR ONE FIBRINOGEN RECEPTOR ONLY. S.K. Bowry, Clinical Research Unit for Blood Coagulation and Thrombosis of the Max-Planck-Gesellschaft, Goffenstr. 11, D-6000 Giessen, West-Germany.

Washed platelet suspensions are almost always prepared from blood anticoagulated with sodium citrate. As citrate has been implicated to affect platelet function and as estimates of fibrinogen to washed platelets from citrated blood of binding sites, a high-affinity site (\(K_d 10^{-5}\) M) and a low-affinity site (\(K_d 10^{-6}\) M) were determined for PCl. However, Fbg bound to two glycoproteins with molecular weights of 130,000 and 65,000, which are known to be involved in the fibrinogen binding to platelets. The results indicate that the major reason for the disparities in the fibrinogen binding data is due to the effects of citrate on the platelets.

ABOLITION OF PLATELET GROWTH FACTOR SUMMARY AND THE LEUKOCYTE peripheral blood mononuclear cells (PBMC) and the human platelet membrane glycoprotein IIb-IIIa complex and a family of functional leukocyte cell membrane antigens, LFA-1 (L), Mac-1 (M) and p150,95 (X), possess known structural analogs in platelets. Our results suggest that the two protein bands responsible for this binding have been visualized by ligand blotting. Both types of ligand specifically bind to two glycoproteins with molecular weights of 130,000 and 65,000. Binding of both classes of plasma lipoproteins, though competitive, has been shown by several groups to facilitate platelet activation. Isolated washed platelets were treated with high concentrations of LDL even in the absence of known platelet activators. The results strongly support the assumption, that the two protein bands associated with lipoprotein binding are constituents of the GP-IIb/IIIa complex. This document may have great implications for our understanding of the mechanism by which lipoproteins facilitate platelet stimulation.

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Platelets possess specific binding sites for low density lipoproteins (LDL) and high density lipoproteins (HDL) (1). Binding of both classes of plasma lipoproteins and lipoprotein structure, though competitive, has been shown by several groups to facilitate platelet activation. Isolated washed platelets were treated with high concentrations of LDL even in the absence of known platelet activators. The proteins responsible for these binding sites are known to be involved in the fibrinogen binding to platelets. The results indicate that the major reason for the disparities in the fibrinogen binding data is due to the effects of citrate on the platelets.

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