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POSTER SYMPOSIUM VI

Platelets: Interaction With Collagen.

ADHESION OF PLATELETS TO COLLAGEN. F.A. Meyer and Z. Weisman, The Weizmann Institute of Science, Rehovot, Israel.

Platelets adhere to collagen fibers, undergo the release reaction and aggregate, soluble collagen, however, does not cause release or aggregation. Platelets, however, recognize soluble collagen since adhesion (but not aggregation) occurs when a soluble collagen coated surface is exposed to washed rabbit platelets in Tyrode and in competitive experiments preincubation of platelets with soluble collagen reduces the number adhering to a collagen surface. The latter effect is concentration dependent; preincubation with 100 µg/ml of soluble collagen reduces platelet adhesion by 70%.

Similar competitive experiments were performed to examine the features on collagen responsible for platelet adhesion. Preincubation of platelets with a variety of soluble collagens, denatured soluble collagen, pepsin- and periodate-treated soluble collagen and cyanogen bromide peptides all decrease the ability of platelets to bind to a collagen surface. Similar effects arise moreover with the synthetic copolypeptides $(Gly\text{-}Pro\text{-}Ala\text{-}Gly\text{-}Pro\text{-}Pro)_n$, $(Gly\text{-}Pro\text{-}Pro)_n$ and $(Pro_2Gly)_n$. In fact, the homopolymers, polyproline and polyhydroxyproline (but not proline or hydroxyproline) as well give effects similar to those of the collagen materials. The effect is rather specific since preincubation with plasma proteins and synthetic polypeptides based on other major amino acids present in collagen, viz. polyalanine, polyglutamic acid, polyaspartic acid, polyarginine and polylysine do not inhibit the binding of platelets. It would therefore appear that proline and hydroxyproline sequences incorporated in a macromolecular chain are the important, if perhaps not the only determinants involved in platelet recognition of collagen.

Soluble collagen coated surfaces were used in this study, however, similar effects of the test material on platelet adhesion to collagen fibers could be demonstrated.

PLATELET-COLLAGEN INTERACTION : ADHESION TO ALPHA 1 (I) CB6 AND ALPHA 1 (III) CB4 PEPTIDES FROM CALF-SKIN COLLAGEN. F. Fauvel, Y.J. Legrand and J.P. Caen. I.N.S.E.R.M. U. 150, Hôpital Lariboisière, Paris, France

The interaction of blood platelets with collagen is the trigger of haemostasis and, at least partly, of thrombosis. Two types of fibrillar collagens (type I and type III), present in the vessel wall, can induce the adhesion of blood platelets. This study was designed to determine which part of their molecule is involved in that phenomenon. We then devised a quantitative test based upon differential filtration of non adhesive and adhesive ^{14}C serotonin labeled platelets through a sepharose column for the evaluation of platelet adhesion to collagen or collagen derivatives. No differences were observed in the adhesive properties of calf-skin type I and type III collagens when the degree of multimerisation was identical. Isolated alpha 1 (I) and alpha 2 chains were not active ; but platelets adhered to reassociated $(\text{alpha 1 (I)})_3$ trimers, not to $(\text{alpha 2})_3$ trimers ; in type I collagen, alpha 1 chains seems to have the most essential role for platelet adhesion. Various cyanogen bromide peptides were tested : only the 217 C-terminal amino acids alpha 1 CB6 from type I and the 149 central amino acids alpha 1 CB4 from type III were active.

The adhesive properties of collagen seems then associated with chemical groupings localized in these peptides : a random folding of the entire isolated chains could make these "sites" unavailable to the platelets, whereas in the triple helix characteristic of fibrillar trimers, they would be more accessible. This could explain why fibrils and small peptides are active, while isolated chains are not.