LETTER TO THE EDITORS-IN-CHIEF

ON THE PROCOAGULANT ACTIVITY OF PLATELETS STIMULATED BY COLLAGEN AND THROMBIN.

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In a recent paper, Brox and Østerud describe the appearance of lysis independent procoagulant activity after activation of washed human platelets by thrombin (1). Bevers et al (2,3) demonstrated that platelet procoagulant activity was induced only after simultaneous activation by collagen and thrombin, and not after activation of the platelets by either one of these agonists separately. Brox and Østerud ascribe this discrepancy to a difference in platelet isolation procedure and a different procedure to lyse the platelets by sonication in order to make a calibration curve of platelet procoagulant activity vs platelet lysis. However, other differences between both studies may give an explanation for the discrepancy in the results. A reduction in clotting time as a result of platelet activation might be due to three different causes: 1. platelet lysis, which exposes the procoagulant cytoplasmic side of the platelet membrane, 2. release and activation of factor V from the α-granules during the platelet release reaction and 3. exposure of procoagulant phospholipids at the outer surface of the platelet plasma membrane. In order to measure the latter activity (termed platelet factor 3, PF3) the assay system should be in-

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dependent of the release of factor V. Although controversial data on the factor V content of platelets exist in literature (4-6) it can be calculated that the amount of factor V added in the PF3 assay as used by Brox and Østerud is less or almost equal to the amount of factor V that can be released from the platelets. Furthermore, even if an excess of exogenous factor V were added, it would be insufficient, because it is essential to add the activated form of factor V in this assay. Therefore, we feel that the major difference between the two studies is that we have added an excess of factor Va in our assay system for PF3, while Brox and Østerud use factor V instead of factor Va. Thus it is doubtful if their PF3 assay is indeed independent of the release of factor V/Va from the platelets, because an excess of factor Va is lacking. Considering the data from table I in their paper, the reduction in clotting time in the PF3 assay, observed upon activation of the platelets with thrombin could also be explained by release of factor V from the platelets, which is subsequently activated into factor Va. Also the lower PF3 activity seen with platelets activated by collagen can be explained by the fact that although release of factor V occurs, there is no subsequent activation of this factor.

REFERENCES


