of platelets and suggest that both types of cells may have similar receptors sensitive to thrombin.

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M. J. Stuart, H. Holmsen and F. A. Oski (Upstate Medical Center, Dept. of Pediatrics, Syracuse, N.Y. and Specialized Center for Thrombosis Research, Temple University, Philadelphia, U.S.A.): **Hydrogen Peroxide (H_2O_2) an Inhibitor of the Platelet Release Reaction.** **(7)**

The effect of H_2O_2 on platelet metabolism, aggregation and release was studied by the in vitro exposure of ^{3}H-adenine labelled platelet rich plasma (PRP) to H_2O_2. On incubation of PRP with H_2O_2 (100 to 500 μM final conc.) over a 30 minute period, there was a mean drop of 38% in the baseline steady state levels of radioactive metabolic ATP, the fall occurring in the first 3 minutes of incubation, with a corresponding increase in the levels of radioactive inosinemonophosphate and hypoxanthine. This was *not* a nonspecific lytic effect on the platelet since no extracellular leakage of platelet nucleotides occurred during the incubation. Further, mean decreases of 8 to 38% in steady state levels of platelet metabolic ATP were observed during incubations of PRP with 5 to 500 μM H_2O_2 respectively. Finally, the action of H_2O_2 on adenosine diphosphate (ADP) induced biphasic aggregation and release was studied in PRP preincubated for 3 minutes with H_2O_2 (100 to 500 μM). Partial inhibition of the primary wave, and complete inhibition of the second wave of ADP induced aggregation was observed in the H_2O_2 pretreated platelets, concommitant with inhibition of release of platelet non-metabolic ATP and ADP, when compared to the control saline preincubated platelets. Since H_2O_2 is generated in vivo by bacteria and leucocytes during phagocytosis, the in vivo effects of the described inhibition of H_2O_2 and its possible role in platelet-leucocyte-bacterial interaction requires further elucidation.

J. F. Mustard, R. L. Kinlough-Rathbone, H. J. Reimers, D. W. Perry and M. A. Packham (Department of Pathology, McMaster University, Hamilton, Ontario, Canada): **Inhibition of Collagen- or Thrombin-Induced Shape Change of Rabbit Platelets.** **(8)**

With suspensions of washed rabbit platelets, EGTA inhibits collagen- or thrombin-induced aggregation but does not inhibit shape change caused by these agents. Addition of apyrase or creatine phosphate/creatine phosphokinase (CP/CPK) to a suspension of washed platelets reduces the extent of collagen-induced aggregation but does not inhibit shape change. Combinations of EGTA with apyrase, CP/CPK, acetyl salicylic acid (ASA) (500 μM) or sulfinpyrazone (300 μM) inhibit collagen-induced shape change. Apyrase and ASA together also inhibit collagen-induced shape change. None of these combinations of inhibitors inhibits thrombin-induced shape change completely, although apyrase or CP/CPK cause partial inhibition. With thrombin-degranulated platelets, collagen induces shape change and a small increase in light transmission. This effect of collagen is inhibited by ASA but not by CP/CPK. The addition of collagen to ASA-treated platelets causes platelet shape change although aggregation does not occur. Addition of collagen to a mixture of thrombin-degranulated and ASA-treated platelets results in shape change and aggregation. It seems likely that endoperoxides formed by the thrombin-degranulated platelets in response to collagen cause shape change and aggregation of the ASA-treated platelets which are unable to form endoperoxides. These observations demonstrate that collagen-induced shape change of rabbit platelets involves two different mechanisms, one of which is inhibited by CP/CPK (and hence is probably caused by released ADP) and one which is inhibited by ASA (and may involve endoperoxide formation). Although thrombin can induce shape change by releasing ADP, it can also induce shape change through a mechanism which is independent of adenine nucleotides release and is not inhibited by ASA.