THE RELATIONSHIP BETWEEN REGULATION OF PROSTAGLANDIN METABOLISM AND PLATELET AGGREGATION.

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Platelet aggregation is mediated by prostaglandin (PG) endoperoxide and cAMP. But exogenous PGE may modify platelet function and might play a major role in the PG metabolism in platelet as well as endogenous PGs do. We studied the relationship between quantitative and qualitative regulation of PGs and cAMP and platelet aggregation. By incubation of platelets with aspirin and cAMP, we found that thromboxane (TX) and cAMP levels were inversely correlated. The TX level was decreased by cAMP, and the cAMP level was increased by TX.

The correlation (r=0.896) was better than that of other clinically applied methods, although patho-physiological conditions such as individual inhibitor composition, extra-vascular enzyme distribution and inhibitor binding may in part be responsible for the observed variability of results with all methods.

FORMATION OF PROSTAGLANDIN CYCLOPEROXIDES AND PROSTAGLANDINS BY PHOSPHOLIPASE A,

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Phospholipase A has a biphasic action upon rat platelet aggregation, enhancing after shorter and inhibiting after longer incubation periods. The first effect is inhibited by indomethacin.

Cycloperoxides and thromboxane-like substances and prostaglandins are formed on incubation with the enzyme in the same amounts whether aggregation is induced or not. Formation of cycloperoxides and thromboxanes is not leading to aggregation. Phospholipase A enhances the aggregation of the platelets of essential fatty acid deficient rats. Only very small amounts of cycloperoxides and thromboxane-like substances are found. It is concluded that cycloperoxides and thromboxanes can enhance, but not induce aggregation in rat platelets.