ULTRASTRUCTURAL AND PARTIAL BIOCHEMICAL CHARACTERIZATION OF PLATELETS FROM CHEDIAK-HIGASHI CATTLE. K.M. Meyers, C.I. Seacord., G. Hopkins, and H. Holmsen. Washington State College of Veterinary Medicine, Pullman, Washington, U.S.A. and Thrombosis Research Center, Temple University, Philadelphia, Pennsylvania, U.S.A.

To provide additional information on the platelet defect which is associated with the Chediak-Higashi syndrome (CHS), platelet rich plasma from normal and CHS cattle was incubated with ^{14}C-adenine. Platelets were then isolated by gel filtration and treated with thrombin. Both the resting amount and extent of secretion of ATP, ADP, several acid hydrolasis, serotonin, calcium and magnesium was determined. Nucleotide profiles and electron micrographs of resting and thrombin treated platelets were also obtained. The markedly reduced secretion of nucleotides, serotonin, and metals demonstrate that CHS cattle have a storage pool defect. Furthermore, there appears to be significant differences in both the resting amount and extent of secretion of several of these measured substances between normal cattle and human platelets.

INCORPORATION OF ^{3}H-GLYCEROL INTO PHOSPHOLIPIDS OF NORMAL AND ABNORMAL PLATELETS. R.A.Hutton and R.M.Hardisty. Institute of Child Health, London, England and D.Deykin, V.A. Hospital, Boston, Ma., U.S.A.

Glycerol-2-^{3}H uptake into phospholipids separated by unidirectional chromatography on silica gel H plates, was measured in normal platelets and those from patients with Glanzman's thrombasthenia (GT), Bernard-Soulier syndrome (B-S), Storage pool deficiency (SPD) or aspirin-like release defect . The results were compared with the release of adenine nucleotides (AN) and with changes in platelet factor 3 availability (PF3a). In normal platelets, secondary aggregation induced by adenosine diphosphate (ADP), Epinephrine (EPI), Collagen (COL) or Ristocetin (RI) led to a marked stimulation of glycerol uptake over a 60 minute period of incubation, the most striking increase being in the phosphatidyl inositol (PI) fraction.

No quantitative differences in phospholipid distribution were noted in any of the patients tested. In all cases, the uptake of glycerol was proportional to the degree of platelet aggregation, being minimal where no aggregation was observed (GT platelets mixed with ADP or EPI), of intermediate level where primary aggregation alone occurred (SPD platelets with all agents or with GT platelets with RI), and similar to normal where secondary aggregation was induced (B-S platelets stirred with ADP or EPI). The results suggest that the defective ^{3}H-glycerol uptake by abnormal platelets is a consequence rather than a cause of the decrease in platelet aggregation. Enhanced PI turnover most probably reflects the cells response to membrane damage or distortion incurred during aggregation and the release reaction.