INTRAERYTHROCYTIC CHANGES OF METABOLIC REGULATOR, FRUCTOSE 1,6 DIPHOSPHATE (FDP) IN VITRO

BY

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ABSTRACT

Fructose 1,6 diphosphate (FDP), a metabolic regulator of the glycolytic pathway in the red blood cells is known to have the important roles in regulating cellular function and metabolism. Intraerythrocytic changes of FDP, subjected to various environmental conditions were followed in vitro, using a modified enzymatic method of Michal and Beutler (1974).

Both human and rat blood samples were taken into the heparinized plastic tubes, plasma was removed by centrifugation and red cells were washed with isotonic phosphate buffer before incubated at 10°C in different mediums for 1 and 24 hours. Packed red cells were deproteinized with perchloric acid, followed by K₂CO₃ neutralization before the FDP determinations.

At physiological pH (7.4), the presence of glucose significantly elevated intraerythrocytic levels of FDP after incubated for 24 hours. In the contrary, citrate which has been used as anticoagulant in clinical blood sampling and storage was found to lower the levels of FDP when incubated with the red cells. The suppressive effect of citrate on intraerythrocytic FDP was pH dependent; i.e. acidic citrate (ACD, pH 4.8) was more effective in lowering the level of FDP than the less acidic citrate. The change in pH per se, as varying the pH (from 7.4, 6.4, 5.5 to 4.8) in the phosphate buffer, used for red cell incubation caused no change to intraerythrocytic FDP under this in-
The levels of intraerythroycytic FDP in the Diabetic (DM) and Thalassemia (THAL) patients were variably changed presumably by the pathological states. These could be due to prior exposure of red cells to relatively high blood glucose in the diabetic patients thus the intraerythroycytic levels of FDP were significantly elevated before the in vitro incubation, and then followed the pattern of FDP levels in normal blood from healthy volunteers, after incubated with the assigned medium for both 1 and 24 hours. This indicates that the glycolytic regulation of the DM's red cell is not defective. Levels of intraerythroycytic FDP in thalassemia were similarly elevated at pre- and post-1 hour incubations, while the levels of intraerythroycytic FDP after 24 hour-incubation of the splenectomized (SP) and non-splenectomized (NS) were in contrast difference. The FDP level of the SP-THAL dropped down below but that of the NP-THAL continuously elevated beyond their respective pre-incubation levels suggesting some glycolytic regulatory defects in the thalassemic red cells, of these SP-Thal in particular. The elevated FDP levels in THAL; of both NS an SP patients at pre- and 1 hour after post-incubation may be resulted from the underlining hyperglycemia in these patients. The defects in the glycolytic regulation in the splenectomized thalassemic red cells is obviously different from the non-splenectomized patients. The distinction in such defects merits further studies.