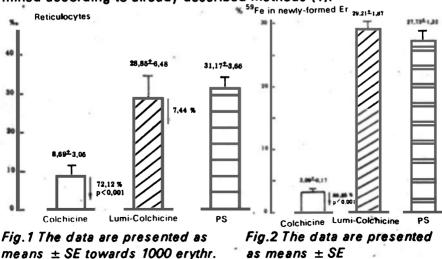
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EFFECTS OF COLCHICINE ON ERYTHROPOIESIS AND ON SOME PROPERTIES OF THE ERYTHROCYTES IN RATS T. Ganchev

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It is known that assembling of cell tubulin molecules in microtubules is blocked by colchicine (CCH) (2,4). That disturbs mitotic processes and functions in many cell systems (2-4). Blood system at treatment with high doses of CCH reacts with leukocytosis, thrombocytosis and bone marrow aplasia. CCH depresses thrombocyte formation under "in vitro" (7), and "in vivo" conditions (5). There are no complex studies of CCH influence on erythropoiesis and on some properties of erythrocytes. Our aim was to investigate CCH influence on erythropoiesis and on some functions of mature erythrocytes. Experiments were carried out on 207 white female Wistar rats with 170-180 g body mass divided in three groups: 1st group (86) treated with CCH once daily - 0,5 mg/kg b.m. Colchicine (Merck, Germanv): 2nd group (26) treated with gamalumi-colchicine (Sigma, USA) once daily - 0.5mg/kg b.m. and 31 group (95) treated with the solvent for CCH and lumi-CCH (0,9 % NaCl) for control. Duration of the experiment was 3 days. Serum iron, ⁵⁵Fe incorporation in newly formed erythrocytes, erythrocytes' deformability and G-6-PD activity were determined according to already described methods (1).



Determination of erythrocytes peroxide haemolysis was performed according to the method of Jager (8). Results are presented as means \pm SE. Considerable decrease of reticulocytes with 72,12% (p<0,001) and of ⁵⁹Fe incorporation in newly formed erythrocytes with 88,85% (p<0,001) (fig.1 and 2) in CCH treated rats was observed. Serum iron and coefficient of transferin saturation were increased (fig.3). Activity of G-6-PD in erythrocytes showed

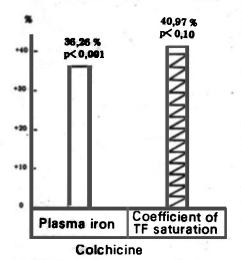


Fig.3. Data are presented as percent deviation to control levels accepted as 0

tendency for increase in comparison with the control group. Deformability of erythrocytes and peroxide haemolysis after treatment with CCH did not show significant changes versus control group. The probable reason for the depression of erythropoiesis is antitubulin effect of CCH possibly on early erythroid cells and may be also reduced biogenesis of erythropoietin described by George and Rodgers (6). The reason for the increased serum iron according to us is its diminished utilization by ervthroid bone marrow, indicator for which is the considerable decrease of ⁵⁹Fe incorporation by newly formed erythrocytes. It is obvious that the underlined de-

pression of erythropoiesis by CCH does not influence substantially on the erythrocytic functions: deformability, peroxide resistance and activity of G-6-PD.

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