90 days might be due to reduced Hb production. Lead inhibits ferrochelatase, the enzyme that catalyzes the incorporation of iron into the porphyrin ring leading to reduced iron incorporation in Hb [10,17], and might result in reduced oxygen transfer by RBCs. This was compensated for by increased number of these cells with smaller volume [18], and was confirmed by the significant reduction of MCH and MCHC. This is in agreement with observations in this study regarding RBC and reticulocyte count and MCV, suggesting that bone marrow could overcome lead toxicity because of sub-chronic exposure which was not at high dose, but suppressed the production of Hb [19] unless it would indicate impaired marrow function or lack of erythropoietin stimulus [20,21].

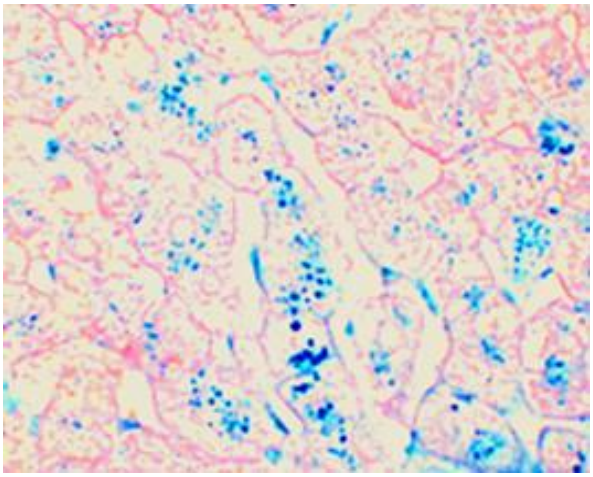

Fig. 2. Liver section from rabbits administered lead was stained with Prussian blue, showing heavy deposition of iron in periportal hepatocelluler, kupffer cells and portal tracts macrophages in middle and periportal zone (400×)

On the other hand, results of this study demonstrated that vitamin E and methionine played an important role in improvement of Hb biosynthesis, RBCs production, which was confirmed by the significant elevation of total RBCs count, Hb, and MCHC. This protective mechanism of vitamin E and methionine against lead adverse effects could be explained by the direct effect of vitamin E on improving the morphology of RBCs by its ability to stabilize their membrane [22], and the free radical scavenger activities of methionine residues as powerful sulfur-containing endogenous antioxidant V [17]. In addition, methionine is a very important nutrient during the Hb biosynthesis and RBC production in bone marrow.

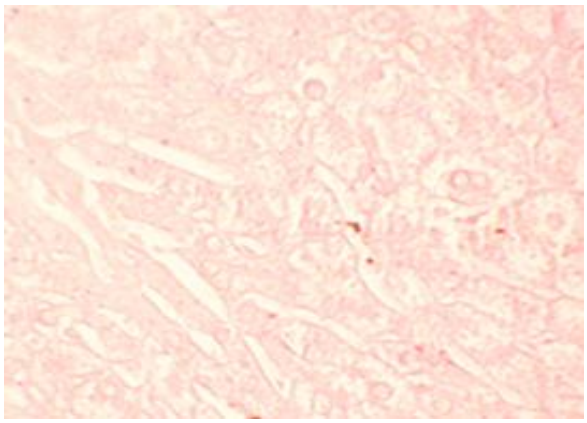

Fig. 3. Liver section form rabbits received lead+methionine stained with Prussian blue, showing a heavy deposition of iron in periportal hepato-celluler, kupffer cells and portal tracts macrophages (400×).

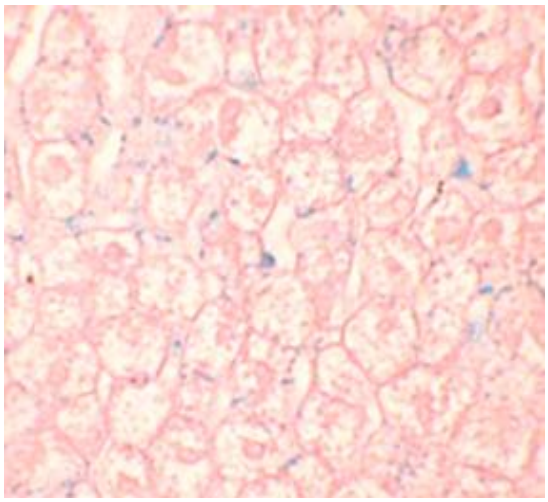

Fig. 4. Liver section form rabbits received lead+vit.E stained with Prussian blue, showing mild panlobuler hepatocyte and kupffer cell iron deposition, iron present in portal tract macrophage (400×).

The increased platelet count found in the present study (Table 2), refer to other