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ABSTRACT

Vitamin E deficiency can affect many species of animals in various distinct ways . One of the first consequences of this deficiency in man and related primates is anemia . In monkeys, depletion of plasma vitamin E level is accompanied by a concomit ant increase in erythrocyte susceptibility to hemolysis by hydrogen peroxide . Anemia is manifested by moderate decrease in hemoglobin concentration, hematocrit, red cell number and life time of red blood cells . During the anemic phase, plasma bilirubin is normal and no splenomegaly is observed. Erythrocytes of deficient animals are denser than those of control and the fragiligram profile indicates a heterogeneous cell population of decreased osmotic fragility . Normal peripheral blood picture and blood indices as well as an absence of reticulocytosis are observed . Upon one dimensional polyacrylamide gel electrophoresis, there is no difference in erythrocyte membrane proteins and glycoproteins between deficient and control animals under both sulfhydryl reducing and non reducing conditions . The bone marrow picture shows an accumulation of orthochromatic normoblasts and multinucleation of this late stage of erythroid precursors .

One hypothesis to account for the above phenomena is that vitamin E is involved in the differentiation of rapidly dividing cells, in particular in those stages during which segregation (translocation) or nuclei are necessary. Absence of vitamin E leads to a block in the process of enucleation of orthochromatic normoblasts,, thereby reducing the number of red cells exported into the peripheral system. Anemia is thus due to a bone marrow failure (ineffective erythropoiesis) and is not associated with detectable intra- or extravascular hemolysis.