

Dr. J. R. Hobbs, of the Harvard University Medical School, by personal communication of May 16, 1931, states that one of his experimental rabbits, naturally recovered from an infection of the South American variety of myxomatosis, has resisted subsequent inoculation with the California strain of virus. Dr. Hobbs' and our results thus indicate that recovery from either South American or Californian types of myxomatosis will render immunity to subsequent inoculations with both strains of virus.

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Effect of Vitamin C Diet on Blood Formation in Experimental Scurvy of Guinea Pigs.*

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We recorded¹ that a variable degree of anemia occurred in a large percentage of individuals suffering from vitamin C deficiency. That a reticulocyte response could be induced in such patients by the administration of a diet rich in vitamin C. That the evidence suggested an altered function of the bone marrow, in great measure directly dependent upon the chronic lack of vitamin C. Since anemia, reported by Meyer and McCormick² and others is of regular incidence in experimental scurvy of guinea pigs, it seemed desirable to determine the effect of vitamin C-containing foods on blood formation. Accordingly, experiments were made (1) to ascertain the effect of a diet deficient in vitamin C on the bone marrow of the guinea pig; (2) to ascertain the effect of a diet containing vitamin C on the bone marrow administered to animals with manifest scurvy.

Adult guinea pigs weighing between 300 to 550 gm. were used throughout the experiment. The males were segregated from the females and all animals were observed over a preliminary period of one to 3 weeks.

* Read before the Section on Medicine of the American Association for the Advancement of Science, Pasadena, California, June 17, 1931.

¹ Mettier, Stacy R., Minot, George R., Townsend, Wilmot C., *J. Am. Med. Assn.*, 1930, **95**, 1089.

² Meyer, A. W., and McCormick, L. M., *Stanford University Publications*, 1928, **2**, 199.

The diet to produce scurvy was calculated to be adequate in all food factors with the exception of vitamin C-containing substances.

In a group comprising 10 animals, signs of scurvy began to appear 10 to 15 days after being given the scurvy-producing diet, and death usually occurred in from about 21 to 30 days. Coincident with the appearance of signs of scurvy, there developed an anemia which became progressively more severe. The red blood cells decreased from an average normal of about 5,000,000 per cu. mm. to as low as 2,500,000 per cu. mm. The hemoglobin decreased from an average normal of 12 gm. to as low as 5.5 gm. In all animals the reticulocytes, which had been less than 1% during the control period, appeared in moderately increasing numbers as the anemia became more marked. There was a terminal rise in the reticulocytes of from 5 to 10%.

The bone marrow removed from these animals after death and examined under the microscope showed a marked increased cellularity. There was an almost complete disappearance of fat cells. There was a marked decrease in the number of mature erythrocytes but an increased number of normoblasts. Only an occasional mitotic figure was noted.

In another group of 3 animals with manifest scurvy and definite anemia, 3 cc. of orange juice was fed daily. There followed soon after an augmented rise in the reticulocytes in the peripheral blood in all animals of from 12 to 20% and prompt alleviation of the anemia and disappearance of signs of scurvy.

Sections of bone marrow removed from these animals at the height of the reticulocyte response also showed marked cellularity. In contrast to the bone marrow removed from animals with scurvy before treatment was started, there was a marked increase in the number of mitotic figures and a more active appearance of the bone marrow.

It is concluded from these experiments that the lack of vitamin C from the diet of the guinea pig results in a profound anemia that is accompanied by definite cytological changes in the bone marrow indicative of retarded production of red blood cells.