

same blood picture was encountered on all the 9 control animals that showed normal calcification. We, therefore conclude that vitamin D deficiency has no influence on hematopoietic function.

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Hematopoietic Function in Avitaminosis. VI. Vitamin G Deficiency.*

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That pellagra is an avitaminosis, produced by a deficiency of a dietary factor, associated with the vitamin B complex, is now generally recognized. Recently, however, Bliss¹ has advanced the theory that pellagra is an iron deficiency disease and claims that the curative agent of the vitamin-containing foods employed by Goldberger and other investigators is not the "so-called vitamin G" but iron. Although it has not yet been conclusively established whether the pellagra-like disease in the rat is the analogue of human pellagra or not, we trust that the character of the results of our studies on the hematopoietic function in vitamin G deficiency in the rat may be of interest to investigators in this field.

The dietary regime for the production of vitamin G deficiency has been described elsewhere.^{2, 3} The optimum ration for the production of dermatitis was found to be one deficient in the vitamin B complex, supplemented by a daily allowance of 500 mg. of rice polishings, and irradiated for 10 hours, in order to insure the destruction of the greater portion of vitamin G.⁴ We have found no specific relation between failure in growth and the incidence of pellagra-like symptoms in the rat, the dermatitis being prevalent in some animals that made normal growth and absent in others that entirely failed in growth and finally collapsed following great losses of weight. The latter phenomenon we encountered most frequently when our basal diet was supplemented with alcoholic extracts from whole wheat, as a source of vitamin B. We, therefore, suggested

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¹ Bliss, S., *Science*, 1930, **72**, 578.

² Thatcher, H. S., Sure, B., and Walker, D. J., *So. Med. J.*, 1930, **23**, 143.

³ Thatcher, H. S., and Sure, B., *Arch. Path.*, 1931, in press.

⁴ Hogan, A. G., and Hunter, J. E., *J. Biol. Chem.*, 1928, **78**, 433.

that the antidermatitis factor is a syndrome distinct from the growth-promoting substance, both of which are associated with "so-called vitamin G."⁵

A total of 44 animals was employed for this study. Bleedings were performed once to twice weekly and determinations were made of hemoglobin and specific gravity, and erythrocyte counts. Records were also kept of food and water intake. The experimental period lasted from 76 to 147 days.

The results on animals that showed loss of weight, unaccompanied by dermatitis, are as follows: Twenty-two animals were used in this investigation, 16 pathological, and 6 positive controls. We found no noteworthy changes in the concentration of hemoglobin or erythrocytes in 10 animals. The anemias observed in 3 animals may be attributable to inanition, since anorexia became quite pronounced in these rats. Three animals, however, which showed a normal food intake and which were apparently in excellent state of nutrition, showed a reduction of 25 to 50% in the concentration of hemoglobin.

Our results on 14 animals, showing dermatitis, compared with 8 positive controls, may be summarized as follows: Ten out of 14 animals showed marked anemia. In 2 cases the reduction of erythrocytes ran parallel to the decrease of hemoglobin, both of which occurred in animals that developed marked anorexia. In the rest of the cases the anemia was due mainly to the reduction of hemoglobin to the extent of 30 to 50%. The latter animals were consuming 7 to 8 gm. of the ration daily, which is considered normal for the rat.

We found no significant changes in the total and differential leucocyte count in this avitaminosis.[†]

While our results are somewhat irregular, it would seem that an anemia occurs in vitamin G deficiency when accompanied by skin lesions comparable to that found in human pellagra. Since our diets had an abundance of ferric citrate, and since in such anemia we found no response to administration of the ash from yeast, we conclude that the anemia we encountered is not produced by a mineral deficiency.

⁵ Sure, B., Smith, M. E., and Kik, M. C., *Science*, 1931, in press.

[†] Credit is due Miss Dorothy J. Walker for making the total and differential leucocyte counts.