

this diet, so that one to 2% of lard was replaced by equivalent amounts of butterfat, in order to prevent sudden deaths from pneumonia frequently associated with this avitaminosis. In addition, we have studied the effect of vitamin A deficiency on the differential leucocyte count in 9 females and 9 males. In the females the disease was followed with daily examinations of the vaginal smears according to the technique of Evans and Bishop.³ Daily records were kept of food consumption, and in the majority of animals records were also kept of the daily water intake. Bleedings were performed on each animal twice weekly and determinations were made of hemoglobin and erythrocyte counts. Specific gravity determinations were also made, in order to secure information on blood concentration. The experimental period ranged from 68 to 173 days.

To summarize our results, we found no noteworthy disturbance in hematopoietic function during various stages of this avitaminosis, as characterized by the severity of ophthalmia and loss of body weight, nor any changes in the differential leucocyte count during the onset, and remission produced by vitamin therapy. Our results, therefore, do not substantiate the findings of Koessler and co-workers.⁴ Although secondary anemias have been encountered in keratomalacia in man,⁵ the investigators admit that other complications or infections associated with this disease may be the influencing factors rather than this avitaminosis.

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Hematopoietic Function in Avitaminosis. V. Vitamin D Deficiency.*

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In a study of the secondary anemia of childhood, Happ and Evans¹ pointed out that 9 of their 10 patients had rickets, although

² McCollum, E. V., and Simmonds, N., *J. Biol. Chem.*, 1918, **33**, 63.

³ Evans, H. M., and Bishop, K. S., *J. Metabol. Res.*, 1922, **1**, 10.

⁴ Koessler, K. K., Maurer, S., and Loughlin, R., *J. Am. Med. Assn.*, 1926, **87**, 476. Koessler, K. K., and Maurer, S., *J. Am. Med. Assn.*, 1927, **89**, 768.

⁵ Keefer, C. S., and Yang, C. S., *Nat. Med. J., China*, 1929, **15**, 419.

* Research Paper No. 199, Journal Series, University of Arkansas.

¹ Happ, W. M., and Evans, F. A., *Johns Hopkins Hosp. Bull.*, 1922, **33**, 7.

the anemia could not be attributed to a lack of vitamin D, since it did not improve following the addition of cod liver oil. Since in human cases we seldom encounter uncomplicated avitaminosis, for the reason that diets that are deficient in one or 2 vitamins are generally deficient in other respects, particularly in minerals, we have found it of interest to determine if an anemia could be produced on the Steenbock-Black ration No. 2965² which is now extensively used by nutritional workers in vitamin research for the production of uncomplicated rickets in the rat.

For this study we have employed 39 animals, 30 on the Steenbock and Black ricketic diet No. 2965 and 9 controls receiving the same diet supplemented with vitamin D by irradiating the ration 30 minutes with a mercury quartz vapor lamp.

TABLE I.
Representative Case of Hematopoietic Function on Steenbock and Black Ration No. 2965, Irradiated for 30 Minutes with a Quartz Mercury Vapor Lamp (♀ 6579).

Date (1929)	Age (days)	Weight (gm.)	Hemoglobin Erythrocytes.		Specific Gravity
			Index Gm. per 100 cc.	Count R. B. C. per mm. ³	
Jan. 29	36	52	11.2	6.1	1.0545
Feb. 5	43	58	9.6	6.2	1.0470
12	50	65	9.5	7.1	1.0493
19	57	71	7.7	6.6	1.0458
27	65	75	8.2	6.7	1.0448
Mar. 5	71	77	7.8	7.0	1.0461
12	78	88	8.4	7.3	1.0395
19	85	93	9.2	7.2	1.0427
26	92	96	8.0	6.2	1.0387

Bleedings of most of the rats were performed twice weekly and of the others once weekly. Daily records were kept of body weight and food intake. At each bleeding hemoglobin and specific gravity determinations and erythrocyte counts were made. In Table I we present the hematopoietic function of a representative animal receiving the Steenbock and Black ration 2965 supplemented with vitamin D, showing anemia, as evidenced by the concentration of hemoglobin and erythrocyte counts. This is the typical picture that was obtained on the ricketic animals as judged by the line tests,³ and the

² Steenbock, H., and Black, A., *J. Biol. Chem.*, 1925, **64**, 263.

³ Shipley, P. G., Park, E. A., McCollum, E. V., Simmonds, N., and Parsons, H. T., *J. Biol. Chem.*, 1921, **45**, 343.

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

* Research Paper No. 200, Journal Series, University of Arkansas.

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.