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III. Vitamin A Deficiency on Concentration of Sugar, Alkaline Reserve, and Glycogen Content of the Liver.*

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We have employed 24 animals, 29 to 55 days of age, that were transferred from our stock diet No. 2[1] to vitamin A deficient rations. The experimental basal ration used in this work had the following composition: Casein (hot-alcohol extracted) 20; Northwestern yeast 10; salts No. 185,[2] 4; lard 2; dextrin 64, irradiated for 30 minutes to insure an adequacy of vitamin D. In a number of experiments the lard in the ration was replaced by 1 to 2% of butter fat, in order to prolong the experimental period, so that the animals would be suffering from an insufficiency of vitamin A rather than from complete depletion of this dietary factor. This was done to prevent sudden death from pneumonia which occurs when all traces of vitamin A are removed from purified diets. The period of experimentation ranged from 80 to 150 days.

The methods used for the determination of blood constituents have been reported in previous publications.[3, 4] The same technique was employed in our studies of vitamins D and G deficiencies, the results of which follow in the subsequent articles. In this study, as well as in those that follow, daily records were kept of food and water intake in the case of all animals, and the blood sugars were determined twice weekly. In addition, determinations of specific gravity were made at each bleeding, in order to obtain information on blood concentration.

Our results show that in various stages of vitamin A deficiency characterized by the severity of eye lesions, there are no significant changes in the concentration of true blood sugar. The figures approximate those found in animals on satisfactory diets.[3] In a good many instances the concentration of apparent sugar is considerably higher in the pathological animals than in the controls. The results of work in progress will reveal whether such high values for the

* Research paper No. 194, Journal series, University of Arkansas.

1 Sure, B., *J. Biol. Chem.*, 1928, **76**, 728.

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

3 Sure, B., and Smith, M. E., *J. Biol. Chem.*, 1929, **84**, 727.

4 Sure, B., and Smith, M. E., *J. Biol. Chem.*, 1929, **82**, 307.

latter constituent may be due to non-protein nitrogen contained in the non-reducing sugars.

We have not encountered the acidosis in vitamin A deficiency as we have in vitamin B deficiency.[3] Only 2 animals showed considerable reduction in the carbon dioxide volume capacity, one (28%) with mild ophthalmia, and the other (27.5%) with advanced eye lesions.

Expressed as milligrams of glucose per 100 gm. of liver the vitamin A deficient animals were found to contain 145.5 mg. glycogen which shows no noteworthy deviation from the figure of 138.3 mg. for our control adult animals.

Autopsy examinations revealed gross pathological changes in the respiratory tract of all the animals, either pus in the bronchi, hemorrhages in the lungs, or pneumonia, bronchial pneumonia being the most common. The following fact has, however, become clear on the careful study of the records of the 24 animals studied that, although there is a reduction in the food intake in vitamin A deficiency in most of the animals, it is not as pronounced as in vitamin B deficiency, complete anorexia being rather infrequent. It became also apparent that there was no specific relation between the water and food intake in advanced stages of vitamin A deficiency. It was observed, however, that in a good many instances excessive volumes of water, as much as 15 to 25 cc. daily, were consumed when the daily food intake was not more than 1 to 4 gm.

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IV. Vitamin D Deficiency on Concentration of Sugar, Alkaline Reserve, and Glycogen Content of the Liver.*

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In this study we have employed 25 animals transferred from our stock diet No. 2[1] to Steenbock and Black ricketic ration No. 2965.[2] Thirteen of these rats, which served as controls, received the same diets supplemented with vitamin D. The latter was supplied either

* Research paper No. 195, Journal series, University of Arkansas.

1 Sure, B., *J. Biol. Chem.*, 1928, **76**, 728.

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