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.

by irradiation of that ration or by the addition of 6 drops of cod liver oil daily per animal. At the end of the experiment a line test[3] was made on each animal.

Five out of the 13 animals showed absolutely normal calcification. The rest of the 8 controls showed a slight deviation from normal calcification, and yet could not be considered as cases of even mild rickets, judging from the line tests. Exposing some of these animals to light did not improve their calcification, because we encountered better calcification on some animals that received the irradiated ration and cod liver oil in the dark. We found only 2 cases out of 12 with severe rickets, the other 10 showing a narrow line of calcification which places them in a group of moderate rickets, as evidenced by the line tests.

An analysis of our data shows that there are no demonstrable changes in true blood sugar, alkaline reserve, or glycogen content of the liver in moderate or severe rickets, after a comparison is made between ricketic and control animals. The liver glycogen is higher than in our animals depleted of vitamin A, the average figure for the control being over 200 mg. expressed as glucose per 100 gm. of liver. This higher figure is undoubtedly due to the fact that the animals were younger, corresponding to our control weaned rats which show a figure of 164.8 mg.

We encountered high figures for apparent sugar both in control and pathological animals, the maximum average figure for non-sugar reducing substances in the control group being 48 mg. %, and 54 mg. % in the pathological group. When it is considered, however, that the Steenbock-Black ration No. 2965 is not an entirely satisfactory diet for optimum physiological function, it is not surprising to find high values for apparent sugar (166 to 178 mg. %) on that diet even if it is supplemented with vitamin D. The high figure of such blood constituent in pathological animals, therefore, cannot be attributed to a deficiency of vitamin D.

A detailed examination of the records of 25 animals leads us to conclude that vitamin D deficiency has no influence on food and water intake.

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