lobe extract was controlled by injecting 2 cc. of the same extract intravenously into animal 4 and 1 cc. into animal 2 four days after the failure of the injection into the fetuses to produce ovulation. Following each of these, subsequent injections of the extract directly into the mother, ovulation occurred.

Apparently the tissues of the fetus or the placental barrier withheld the passage from fetus to mother of an amount of the substance sufficient to produce ovulation. These results indicate that anterior lobe extract is probably not transmitted from fetus to mother. It is possible, however, that with even greater dosage, or by direct introduction of the extract into the fetal blood stream, the substance might be induced to pass the placental barrier in sufficient amount to cause ovulation. Nevertheless the failure of anterior lobe extract to pass the placenta in the present experiments and to exhibit a biological effect in the mother is in keeping with observations on the failure of transmission of active principles of other glands (adrenalin, insulin, pituitrin, and parathyrin) (Snyder and Hoskins,² Hoskins and Snyder.³)

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A New Symptom Complex in Vitamin-G Deficiency in Rats.

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The diversity of symptoms resulting from vitamin-G deficiency and the lack of uniformity of results even in the same laboratory lead to confusion in evaluating the real significance of any one symptom. The present report deals with a new symptom which thus far has been found to occur regularly.

Some of the symptoms of rats on vitamin G deficient diets described most frequently by other investigators are dermatitis occurring at various sites on the body,¹ a peculiar oedematous dermatitis of the digits,³ alopecia,² blood stains on wrist and forepaws,⁸ and

² Snyder, F. F., and Hoskins, F. Meredith, Anat. Rec., 1927, 35, 23.

³ Hoskins, F. M., and Snyder, F. F., PROC. Soc. EXP. BIOL. AND MED., 1927, 25, 264.

¹ Goldberger, J., and Lillie, R. D., Pub. Health Rep., 1926, 41, 1025.

² Chick, H., and Roscoe, M. H., Biochem. J., 1927, 21, 698.

³ Chick, H., and Roscoe, M. H., Biochem. J., 1928, 22, 790.

dark reddish brown stains,⁴ which fail to give the tests for blood, on the lower abdomen, and about the urethra, chromogenic urine and constipation. In the present study all of these symptoms occurred at some time during the course of the experiment in different individuals. The most common one was the blood stained wrists and forepaws but this was later shown to be a dehydration phenomenon, occurring regularly in rats deprived of water.

Eighteen white albino rats of the Wistar Institute stock were placed on Sherman's vitamin G deficient diet No. 554^5 at the age of 45 days, for a period of 226 days. The 17 rats, surviving longer than 70 days, all developed a characteristic dermatitis of the tail which manifested itself grossly by a coating of brownish yellow waxy material which grew progressively worse until the animal died or had its diet supplemented with autoclaved yeast. Upon the addition of yeast, autoclaved for $2\frac{1}{2}$ hours at 15 pounds pressure, this condition gradually cleared up.

The material coated on the tails failed repeatedly to give a positive benzidine or guaiac test for blood. It did, however, give a positive test for lipoid material when dissolved in ether and stained with Sudan IV.

Microscopic sections* made from the tails of all rats having this dermatitis showed very characteristic histological changes in the skin (Fig. 1). Although the epithelium remained intact it showed thinning and disorganization. There was almost complete atrophy and disintegration of the sebaceous glands. In addition there was atrophy and fragmentation of the fibrillar material in the corium.⁶ There was no cellular infiltration. These histological changes preceded the gross changes by an appreciable period of time. The one rat dying at the end of 70 days on the diet showed no gross lesion, but microscopic sections of the tail showed characteristic histological changes described above.

If after 226 days on the deficient diet, a supplement of autoclaved yeast, at a level of 10%, is added for 36 days and the tails then biopsied and sectioned the histological picture is entirely changed. The epithelium is completely regenerated and the sebaceous glands and corium have returned to normal.

⁴ Leader, K. R., Biochem. J., 1930, 24, 1172.

⁵ Sherman, H. C., and Bourquin, A., J. Am. Chem. Soc., 1931, 53, 3501.

^{*} I am indebted to Dr. D. H. Sprunt of the Department of Pathology for examining the microscopic preparations and confirming statements made about them and to Miss Eleanor Milnor for technical assistance.

⁶ Denton, Jas., Am. J. Path., 1928, 4, 341.



Tail of Rat No. 36 after subsisting on G-deficient diet No. 554 for 226 days. Tail at this time exhibited the gross lesion described in text.

The same symptom complex was observed in 17 rats on a diet similar to one on which human beings frequently develop pellagra. This diet consisted of white, water-ground, corn meal 50%; pork fat 20%; cane syrup 15%; white flour 10%; and cane sugar 5%.

In order to rule out the effects of dehydration or inanition, 24 rats were subjected to 2 starvation regimes. In 10 rats deprived of food but given water there occurred merely loss of weight and general weakness but no characteristic lesion. Twelve rats deprived of water but given food developed the blood stained wrists often described as a symptom in vitamin G deficiency. The mouth, nose, and eyes were also stained. However, not a single individual in these 2 groups developed the tail lesions described above, either grossly or microscopically.

Sections made from the tails of 16 control rats were all entirely normal.

These experiments are being repeated and tests made to determine whether the microscopic changes might be used in developing a new assay method for vitamin G.