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Dermatitis in Pyridoxine-Deficient Rats.

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Pyridoxine deficiency in rats has always been associated with a "specific dermatitis" which has been named acrodynia.[1] Dermatitis can be readily produced when thiamine and riboflavine are the only members of the Vitamin B complex fed.[2, 3] While such a supplemented diet has often been referred to as a pyridoxine-deficient diet, it is deficient also in factor 2 or the filtrate factors. Factor 2 concentrates are now known to carry pantothenic acid plus other as yet unidentified filtrate factors. Addition of factor 2 will sometimes promote the development of dermatitis[4] and sometimes will give erratic results.[5]

In this laboratory for a long time no dermatitis appeared on large numbers of pyridoxine-deficient rats, though the rats were obviously deficient since they either did not grow at all or grew slowly and responded spectacularly to pyridoxine administration. Because a good deal of dermatitis had been observed on pyridoxine-deficient rats in the past, the records were searched for changes in technic which might account for the absence of dermatitis on pyridoxine-deficient rats. The absence of dermatitis was found to be coincident with a change in technic involving feeding of supplements to the basal diet. In previous work, 21-day-old rats were put on the synthetic diet[4] without any addition of water-soluble vitamins for 10 to 12 days. Thiamine and riboflavine were then added for 14 days after which factor 2 concentrates[4] were added. A large proportion of these rats developed dermatitis. The change in technic consisted in feeding thiamine, riboflavine and factor 2 concentrates to the rats when they were put on the synthetic diet at 21 days of age without any depletion period. Such rats, though they developed pyridoxine deficiency, seldom developed dermatitis.

Forty rats were accordingly put on the basal diet[4] for a depletion period of 12 days after which, for a period of 2 weeks, only thiamine

1 Birch, T. W., György, P., and Harris, L. J., *Biochem. J.*, 1935, **29**, 2830.

2 György, P., *Biochem. J.*, 1935, **29**, 741.

3 Antopol, W., and Unna, K., PROC. SOC. EXP. BIOL. AND MED., 1939, **42**, 126.

4 Lepkovsky, S., Jukes, T. H., and Krause, M. E., *J. Biol. Chem.*, 1936, **115**, 557.

5 Dann, W. J., *J. Biol. Chem.*, 1939, **128**, XVIII.

and riboflavine were fed. At this time symptoms of dermatitis began to appear. Factor 2 concentrates were added, and definite dermatitis appeared in a week. In 2 weeks, 16 of the rats developed severe dermatitis, 15 mild, and 12 rats showed no dermatitis. After 5 weeks of factor 2 feeding, only 4 rats remained without dermatitis. Three rats with slight dermatitis had cured spontaneously.

It thus appears, at least from these experiments, that dermatitis develops as a result of a factor 2 deficiency superimposed upon a pyridoxine deficiency. If rats are early exposed to such an extra deficiency, the effects, insofar as they affect dermatitis, seem to carry over after the addition of factor 2 concentrates, and the rats become deficient only in pyridoxine. The question may well be raised whether dermatitis or acrodynia is a reliable expression of uncomplicated pyridoxine deficiency.

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Experimental Necrotizing Arteritis. II. Mercuric Chloride as Effective as Uranium Nitrate in its Production.*

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In a recent publication[1] the senior author described well marked necrotizing arterial lesions affecting principally the large elastic arteries (aorta, sinuses of Valsalva, endocardium of the left auricle, coronary and pulmonary arteries) which appeared unexpectedly during the course of the experiments designed to determine whether heavy metal poisoning is influenced by altering the plasma protein level.

Normal adult dogs maintained on a standard low protein diet were made hyperproteinemic by repeated intravenous injections of plasma obtained from healthy donor dogs. Each dog then received a single subcutaneous injection of 5.0 mg of uranium nitrate per kilo. In 4 of the 5 dogs in which this procedure was carried out, acute necrotizing arterial lesions were found at necropsy 8-17 days after the injection of the heavy metal; and healed lesions were found when the 5th dog

* This investigation was aided by a grant from the John and Mary R. Markle Foundation.

1 Holman, Russell L., *Am. J. Path.*, 1941, **17**, 359.