

Pyridoxine Deficiency in Turkeys.

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The effects of pyridoxine deficiency have been described for the rat,¹ dog,² pig,³ and chick.⁴⁻⁷ In order to find whether the turkey would respond to pyridoxine deficiency in a similar manner to the chicken and the rat, poults were fed deficient and standard diets.

Deficient diet. The pyridoxine-deficient diet was similar to that reported in a previous publication.⁶ However, it differed in that extracted liver and alfalfa were included each at a level of 5%.

The alfalfa meal was prepared in the same manner as the wheat bran.⁶

Beef liver was passed through a meat grinder, suspended in water, and allowed to autolyze 2 days under benzene at 37° C. The

suspension was then treated with live steam until all protein was coagulated. The solids were separated in a wine press, suspended in hot water, and repressed. This was repeated twice. The solids were then dried. The combined filtrates were treated 5 times with fuller's earth, concentrated at reduced pressure, and added back to the dried residue.

Methods. Bronze turkey poults were weighed at hatching, placed in electrically heated wire battery brooders with raised wire floors, and fed a regular turkey starting mash for different periods of time, as shown in Table I. At the start of each test the birds were reweighed and segregated into groups of similar weights and gains. Experimental diets and water were given *ad libitum*. In 2 of these tests the regular all-mash ration fed by the Division of Poultry Husbandry to turkeys during the first few weeks after they are hatched was used as the only positive control. In the third test, 3 mg of pyridoxine per kg of basal diet and the regular turkey starting mash were both used as positive controls.

Results. Poults receiving the deficient diet appeared weak and apathetic, although they were hyperexcitable when disturbed. Some of them would dash around the cage bumping into any object in their path. Jerky uncon-

¹ Chick, H., El Sadr, M. M., and Worden, A. N., *J. Biochem.*, 1940, **34**, 594.

² Fouts, P. J., Helmer, O. M., Lepkovsky, S., and Jukes, T. H., *J. Nutrition*, 1938, **16**, 197.

³ Chick, H., Macrae, T. F., Martin, A. J. P., and Martin, C. J., *J. Biochem.*, 1938, **32**, 2207.

⁴ Carter, C. W., and O'Brien, J. R., Proc. 7th World's Poultry Congress, Cleveland, 1939, p. 126.

⁵ Jukes, T. H., Proc. Soc. Exp. Biol. Med., 1939, **42**, 180.

⁶ Lepkovsky, S., and Kratzer, F. H., *J. Nutrition*, 1942, **24**, 515.

⁷ Mills, R. C., Hegsted, D. M., Elvehjem, C. A., and Hart, E. B., *Poultry Sci.*, 1942, **21**, 379.

TABLE I.

Test	Diet	Days on normal* diet, No.	Poults started, No.	Poults finished, No.	Convulsions observed in poults, No.	Gain in weight	
						Grams	% Normal
I	Basal	4	11	2	3	32.4	12.5
	Normal*	25	10	9	0	256.7	100.0
II	Basal	11	11	7	3	43.9	11.9
	Normal*	32	10	9	0	368.8	100.0
III	Basal	6	10	3	3	20.0	6.3
	Basal + 3.0 mg pyridoxine per kg	6	10	10	0	270.5	85.2
	Normal*	28	10	9	0	317.5	100.0

*Normal = regular all-mash ration.

trollable movements of the head and neck were observed. Convulsions were observed in 9 out of 32 poults and largely resembled those already reported for the chick.⁶

Two convulsions in each of 3 poults followed a somewhat different pattern prior to death. These resembled the convulsions seen in acute strychnine poisoning. At the start the poults were in a comatose state lying on their sides. A fine tremor of the feet was noticed which increased in severity. Suddenly the neck would arch until the head touched the back, the legs assumed extreme extension, and the whole body became rigid and in opisthotonos. Respiratory movements ceased during such an attack. After an interval of approximately one-half minute, the poult would relax and respiratory movements would begin. After these seizures the poults were completely exhausted. In each case the poult died during the second observed convulsion. Several poults on the

deficient diet were found dead with this characteristic curve of the neck with the head arched toward the back. They may have died in convulsions but such were not observed. None of these convulsions could be elicited but appeared spontaneously. These observations indicate that the effects of pyridoxine deficiency on poults are similar to those reported for chicks.⁶ In turkeys, convulsions are followed by death much more quickly than in chicks.

Poults receiving the basal diet plus 3.0 mg pyridoxine per kilo of diet were normal in appearance and grew at a rate which approximated 85% of the growth of poults on the turkey starting mash.

Summary. Pyridoxine deficiency in turkeys is characterized by loss of appetite, poor growth, apathy, hyperexcitability when disturbed, convulsions, and death. Pyridoxine prevented the deficiency symptoms.

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Further Studies on Properties of the Gastric Secretory Depressant in Gastric Juice.*

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A gastric secretory depressant has been observed in acid gastric juice of both humans and dogs. In patients with achlorhydria and gastric carcinoma or pernicious anemia, this factor is present in relatively high concentration.¹ This factor is contained in the flocculent precipitate obtained by addition of 4 volumes of absolute alcohol to neutralized gastric juice. About 1 g of precipitate obtains from a liter of juice. Three hundred to 500 mg of the precipitate, suspended in saline and injected intravenously into gastric pouch dogs fed meat, affords consistently an

achlorhydria of the pouch lasting at least 2½ hr.

Alkalinization of a suspension of the precipitate results in solution of the latter, which however reprecipitates with acidification (pH 5 to 6). This second precipitate is inactive as far as the gastric secretory depressant is concerned, as is also the supernatant fluid, hence alkalinization destroys the depressant factor. Five hundred mg of active precipitate were suspended in 20 cc of N/10 N HCl and U.S.P. pepsin added to .2% concentration. After incubation for 24 hr at 37°C no decrease in the potency of the gastric secretory depressant factor was noted. Longer periods of incubation resulted in decreased potency of the factor.

The question is raised if the gastric secre-

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¹ Brunschwig, A., Schmitz, R. L., Rasmussen, R., *J. Nat. Cancer Inst.*, 1941, **1**, 481.