PROCEEDINGS OF THE NUTRITION SOCIETY

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SYMPOSIUM ON 'RECENT ADVANCES IN POULTRY NUTRITION'

Nutrition and embryonic development in the domestic fowl

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Nutritional deficiencies in the diet of the hen are directly reflected in an abnormal development of the avian embryo (Taylor & Grau, 1968; Couch & Ferguson, 1972). Excesses of some nutrients will also have an adverse effect on embryonic development. Three stages in the embryonic development of the chick which can be related to nutritional deficiences are 2–4, 9–14 and 18–21 d of incubation. A chick that is unable to pip the shell or one that pips the shell and dies may be the victim of a maternal nutritional deficiency. Two vulnerable stages in the embryonic development of the turkey are 2–4 and 24–28 d of incubation.

Riboflavin. Embryonic abnormalities in the chicken produced by giving riboflavin-deficient breeder diets occur from 9–14 d. These consist of the following: anaemia, 'clubbed down', curled toes, prognathism, micromelia, shortened leg bones, degeneration of embryonic Wolffian bodies, degeneration of the myelin sheath of the peripheral nerves, oedema, parrot beak, chondrodystrophy and a dwarfing of body size (Lepkovsky, Taylor, Jukes & Almquist, 1938; Engel, Phillips & Halpin, 1940; Romanoff & Bauernfeind, 1942; McClymont & Hart, 1947).

Biotin. A deficiency of biotin in the breeder diet produces peaks of embryonic mortality in chickens at 3 and 18–21 d of incubation (Cravens, McGibbon & Sebesta, 1944; Couch, Cravens, Elvehjem & Halpin, 1948*a*, *b*). The anomalies recorded at 18–21 d of incubation consist of perosis, chondrodystrophy, syndactyly, parrot beak, and skeletal deformities which appear to result from a shortening, thickening and bending of the humerus, radius, ulna, capula, tibiotarsus, tarsometatarsus, cranium and mandibula. Ataxia and spasticity occur in chicks that emerge from the shell. This cannot be relieved by biotin injection.

Pantothenic acid. Turkey embryos from eggs laid by turkey hens fed on a diet deficient in pantothenic acid live to the end of the 28th day, but do not pip the shell (Kratzer, Davis, Marshall & Williams, 1955; Dawson, Ferguson, Deyoe & Couch, 1962). These embryos are decreased in size, have a wiry type of stiff down, exhibit

abnormalities of the legs, are haemorrhaged, have fatty livers, pale dilated hearts and an opacity in the eyes.

Folic acid. Chicks in eggs from breeders fed on diets deficient in folic acid pip the shell and expire (Sunde, Cravens, Elvehjem & Halpin, 1950). Abnormalities in these embryos are: syndactyly, deformed mandibles, parrot beak and shortening and bending of the tibiotarsus. Folic acid injection into the deficient egg on the 17th day produces normal chicks.

Embryos from turkey hens fed on a diet deficient in folic acid are reduced in size and die at 26–28 d (Kratzer, Davis & Abbot, 1956). There is an enlargement of the hock joint, and twisting of the legs, micromelia, a crossed beak, haemorrhages and oedema.

Cyanocobalamin. The peak of embryonic mortality in vitamin B_{12}-deficient eggs is at 16–18 d of incubation (Olcese, Couch, Quisenberry & Pearson, 1950; Ferguson & Couch, 1954; Ferguson, Rigdon & Couch, 1955). A gross microscopic examination of the embryos reveals that there is a high incidence of the malposition 'head between the thighs', a decrease in size, perosis, myoatrophy of the leg muscles, shortening of the legs, oedema and a general body haemorrhaging under the skin, in the muscles and in the internal organs. There are focal areas of necrosis in the liver, brain and spinal cord and a marked increase in fat in the parenchymatous tissues. The thyroid gland is enlarged and has a reduced capacity for ^{131}I uptake (Ferguson, Rigdon & Couch, 1957; Ferguson, Trunnell, Dennis, Wade & Couch, 1957). The hearts of the deficient embryos are pale, enlarged and irregularly shaped. The presence of fat in the parenchymatous tissues suggests a basic disturbance in liver metabolism. The oedema and haemorrhages suggest a possible disturbance in protein metabolism (Ferguson *et al.* 1955).

Vitamin E. The peak of embryonic mortality in vitamin E-deficient eggs occurs at 84–96 h. Growth of the embryo is retarded and there is haemorrhaging and a failure of development of the circulatory system (Adamstone, 1931).

There is a bulging of the cornea and a protruding eye in vitamin E-deficient turkey embryos, with a yellowish-white spot between the lens and the cornea at 24–28 d of incubation. Cataracts and haemorrhages occur in the eyes, with a liquefaction of the lens protein (Ferguson, Atkinson & Couch, 1954; Atkinson, Ferguson, Quisenberry & Couch, 1955; Ferguson, Rigdon & Couch, 1956).

Manganese. The Mn-deficient chick embryo dies at 20–21 d, showing retarded growth, and is chondrodystrophic with shortened and thickened leg and wing bones and a parrot beak, with an anterior bulge of the skull (Lyons & Insko, 1937; Gallup & Norris, 1939; Wilgus, Norris & Heuser, 1939). Chicks that hatch are ataxic and micromelic (Caskey, Norris & Heuser, 1944; Caskey & Norris, 1950).

Iodine. Growth is retarded, hatching time is delayed and there is an increase in the size of the thyroid, which exhibits hypertrophy of epithelial cells with a block of colloid follicles in I-deficient chicken embryos (Rogler, Parker, Andrews & Carrick, 1959).

Zinc. In Zn-deficient embryos there are abnormalities of the trunk (anouria, ectrosomia and lordosis) and limbs (micromelia, ectrodactyly and symmelia),

oedematous hatching muscle, abnormal brains and visceral arches, microphthalmia and celosomia (Blamberg, Blackwood, Supplee & Combs, 1960; Kienholz, Turk, Sunde & Hoekstra, 1961).

Phosphorus. Embryonic mortality is increased at 12–14 d by giving a low-P maternal diet. Gross specific abnormalities were not recorded (O'Rourke, Bird, Phillips & Cravens, 1954).

Linoleic acid. The abnormal position (head over right wing) occurs when a linoleic acid-deficient diet is given, with peaks of embryonic mortality at 2–4 and 20–22 d (Menge, Calvert & Denton, 1965*a*, *b*).

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