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Nutrition and reproduction in the domestic fowl

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In the domestic fowl (*Gallus domesticus*) the fertilized egg in the infundibulum of the oviduct consists of an enormous mass of yolk about 40 mm in diameter on which rests the blastodisc about 3 mm in length. The whole is enclosed by a vitelline membrane, but as the egg passes down the oviduct (a process requiring about 24 h) albumen is deposited around it, shell membranes are laid down enclosing the albumen and, finally, calcium salts are deposited on them. This macrolecithal egg provides a readily assimilable food supply for the extremely rapid extragenital development of the embryo. If certain nutrients are lacking or are present in insufficient amounts, death or morphological abnormalities may occur.

Several excellent reviews have been published on both the qualitative and quantitative nutritional requirements of breeding fowls for the maintenance of a high reproductive performance (Cruickshank, 1940–1; Taylor, 1949; Landauer, 1951; Bolton, 1958) and I therefore propose to discuss some of the effects of individual nutritional deficiencies on embryonic development, with special emphasis on the age at death of the embryo and the occurrence of specific abnormalities.

Nutrition and fertility in the male

Knowledge of the effect of specific nutrients on fertility in the male is extremely limited. Vitamin A deficiency results in a reduction in comb size, underdeveloped testes, a decrease in sperm concentration and motility, and an increase in abnormal sperms (Craft, M'Elroy & Penquite, 1926; Burrows & Titus, 1938; Garcia & Paredes, 1957; Lowe, Morton, Cunningham & Vernon, 1957).

Adamstone & Card (1934) investigated the effect of a practical-type diet in which vitamin E had been destroyed by ethereal ferric chloride and found that by the end of the 2nd year some birds had become sterile and showed degenerative changes in the testes. Recently, Lorentz, Abbott, Asmundson, Adler, Kratzer, Ogasawara & Carson (1959) reported that male chickens maintained for 2 months on a purified diet deficient solely in vitamin E showed reduced testis size and degenerative changes in the seminiferous tubules.

There is no conclusive evidence available that nutritional deficiencies in the diet of the male influence the embryonic development and hatchability of eggs fertilized by him. The paucity of available information in this field suggests that further research is required.

Nutrition and reproduction in the female

Vitamin A. The necessity of vitamins for reproduction in the female has received considerable attention, although much of the work has been concerned with determining the quantitative requirements by the maternal organism to support optimum hatchability and viability in the young chicken. Such experiments have yielded little information on the role of these substances in the metabolism of the chick embryo.

The effect of various quantities of vitamin A or its precursors on egg production and hatchability has been studied by a large number of workers (see Landauer, 1951). There is evidence to suggest that the vitamin A requirement for maintaining egg production is higher than that for hatchability. Temperton & Dudley (1946) found that the increase in embryonic deaths in deficient eggs occurred between the 18th and 21st days of incubation, although the more severe the depletion of maternal body-stores the earlier did the embryo die. The only abnormality found in such embryos was the occlusion of the ureters with urates (Temperton & Dudley, 1948).

Vitamin D. Vitamin D_3 is intimately concerned with the assimilation and utilization of calcium and phosphorus by the fowl. A deficiency of this vitamin in the diet of the breeding female results in a fall in egg production and the appearance of thin-shelled eggs. Insko & Lyons (1936) found that embryos from deficient eggs contained less calcium and phosphorus than those receiving an adequate amount of vitamin D. The maximum difference occurred on the 18th and 19th days of incubation when there was also an increase in embryo mortality in the deficient eggs.

Vitamin E. There is some evidence that vitamin E is necessary for early embryonic development. Adamstone (1931) observed a marked retardation in both the growth rate and tissue differentiation of deficient embryos after 24 h incubation. A lethal ring of mesodermal proliferation was found to have developed in the vascular area outside the embryo which completely encircled it by the 4th day of incubation. More recently, however, Singsen, Matterson, Bunnell, Kozeff & Jungherr (1954) found that both egg production and hatchability remained normal in fowls that had been maintained on a purified diet low in vitamin E for 9 months, although the blood content of tocopherols was nil.

B vitamins. The role of thiamine in enbryonic development has not been investigated, although Ellis, Miller, Titus & Byerly (1933) did show that chickens from parents maintained on a thiamine-deficient diet exhibited polyneuritis soon after hatching. A deficiency of riboflavin in the diet of the breeding female leads to a retardation of embryonic growth with two characteristic peaks of mortality in the deficient eggs (Davis, Norris & Heuser, 1938; Lepkovsky, Taylor, Jukes & Almquist, 1938). The first occurs between the 9th and 14th days of incubation, when the embryos are found to be oedematous, and the second between the 17th and 21st days when a condition known as 'clubbed down' (failure of the feather follicles to erupt) and degeneration of the embryonic Wolffian duct are the most characteristic lesions. Embryonic micromelia and degeneration of the myelin sheath has also been observed in riboflavin-deficient embryos (Engel, Phillips & Halpin, 1940; Romanoff & Bauernfeind, 1942).

Dietary pantothenic acid is essential for the production of hatchable eggs (Gillis, Heuser & Norris, 1942, 1947). Embryonic mortality in the deficient eggs rises sharply after the 14th day of incubation, and the chickens that succeed in hatching exhibit a general debility, muscular incoordination, swollen hocks and poor-quality down.

The role of nicotinic acid in the nutrition of breeding fowls is still obscure. The developing embryo is able to synthesize this vitamin (Dann & Handler, 1941; Snell & Quarles, 1941). Briggs, Groschke & Lillie (1946) showed that a diet low in nicotinic acid and containing high levels of protein low in tryptophan, leads to complete failure to hatch; nicotinic acid was found to be effective in recovery experiments.

A deficiency of dietary pyridoxine leads to anorexia and a rapid decline in egg production. Consequently the effect of such a deficiency on embryonic development has not been satisfactorily investigated.

Folic acid is indispensable for embryonic growth (Taylor, 1947). Deficient embryos die towards the end of the incubation period without showing any gross defects, although a few may exhibit syndactylism and beak abnormalities.

A biotin deficiency has a pronounced effect on embryonic development. The most characteristic feature is the occurrence of skeletal abnormalities, which include a parrot-like beak often associated with chondrodystrophy and syndactyly (Cravens, McGibbon & Sebesta, 1944). Ataxia has also been observed in chickens that hatch from deficient eggs (Couch & Cravens, 1947). This ataxia could not be relieved by oral or parenteral administration of biotin, but embryonic development could be restored to normal by injecting biotin into the albumen of the egg before incubation.

Vitamin B_{12} is indispensable for normal embryonic development (Lillie, Olsen & Bird, 1949). Maximum embryonic mortality in the deficient eggs occurs between the 8th and 18th days of incubation, the principal macroscopic lesions being oedema about the eyes, shortening of the beak, curled toes, myoatrophy of the legs and haemorrhages (Hill, McConachie, Gartley & Branion, 1950; Olcese, Couch, Quisenberry & Pearson, 1950). Ferguson and his co-workers (Ferguson & Couch, 1954; Ferguson, Rigdon & Couch, 1955, 1957; Ferguson, Alexander & Couch, 1956) have also found that the vitamin B_{12}-deficient embryo has an enlarged thyroid gland, focal areas of necrosis in the liver, brain and spinal cord, with demyelination of the last.

Minerals. The interrelationship between calcium and vitamin D has been discussed. A diet deficient in this mineral soon leads to thin-shelled eggs and reduced hatchability. Embryonic mortality also increases when the calcium level of the maternal diet is high, the peak rising sharply during the last 3 days of incubation (Titus, Byerly, Ellis & Nestler, 1937). Though a diet low in phosphorus will not support normal egg production and embryonic development, the requirement of breeding females for this element has been found to be considerably lower than hitherto supposed (O'Rourke, Bird, Phillips & Cravens, 1954).

The presence of manganese in the egg is essential for the developing embryo (Lyons & Insko, 1937). Fertile eggs from fowls that had received a diet low in this element fail to hatch. The peak of embryonic mortality occurs just before the end of

the incubation period and the affected embryos may exhibit micromelia, chondrodystrophy and parrot beak. Addition of manganese to the diet or its injection into the albumen before incubation prevents the occurrence of these abnormal embryos. Chicks hatched from manganese-deficient eggs may also exhibit ataxia (Caskey, Norris & Heuser, 1944).

Until recently iodine was not thought to be necessary for embryonic growth, although excessive amounts have been found to reduce hatchability (Wilgus, Gassner, Patton & Harshfield, 1948). However, Rogler, Parker, Andrews & Carrick (1959) have shown that iodine deficiency of the dam's diet leads to a reduction in the iodine content of the egg, decreased hatchability and prolonged hatching time. Injection of potassium iodide into the air cell of deficient eggs as late as the 20th day of incubation produces a marked improvement in hatchability.

No studies have been carried out on the role of iron, copper, magnesium, zinc, cobalt and potassium in egg production or embryonic growth and development.

Proteins. Many experiments have been conducted on the effect of different proteins on egg production and hatchability, but their evaluation has been difficult because the different protein concentrates investigated contained varying amounts of the essential amino acids and B-complex vitamins.

Until recently it had not proved possible to formulate a diet containing individual amino acids that would maintain egg production (Grau & Taylor, 1948) but Johnson & Fisher (1956*a*,*b*; 1958) have now overcome this difficulty and determined the minimal levels of essential amino acids necessary to support egg production.

Unidentified factors. In recent years a number of unidentified nutritional factors essential to maintain optimal growth of young chickens have been found in distillers dried solubles, dried whey, fish solubles, liver fractions and grass juice (Scott, 1957). Some of these factors also appear to be essential for normal embryonic growth and hatchability (Arscott & Combs, 1953; Kurnick, Svacha, Reid & Couch, 1956; Jensen & McGinnis, 1957).

Conclusions

Adequate nutrition of the breeding fowl is essential for normal embryonic development and the hatching of viable chickens. Though the effect of many nutritional deficiencies on embryonic development has been widely investigated, especially in relation to the age of death of the embryo and gross pathological lesions exhibited, there is still a paucity of histopathological information about the embryos that die. In addition, more research is needed on the metabolic function of some of these nutritional factors in the developing embryo.

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Influence of nutrition on reproduction in laboratory rodents

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It is almost a platitude that diet is closely related to reproduction. Even with good nutrition and management 20–25% of both farm and laboratory livestock matings