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Nutrition and helminthiasis in chickens

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Lapage (1956) lists sixty-nine species of helminths that may be found in the domestic fowl, but of these, only the nematodes, *Ascaridia galli*, *Heterakis gallinarum* and *Capillaria* sp. are of economic importance and probably only *A. galli* and *C. obsignata* are of pathogenic significance. Occasionally cestodes, such as *Raillietina* and *Davainea* sp., are found and trematodes very rarely.

Wakelin (1963) has discussed the importance of *Capillaria* sp. in chickens and in a recent survey has found that its incidence can exceed that of *A. galli*. Heavy infections by either of these helminths produce retardation of growth and stunting in young stock, and a decrease in egg production in laying birds which often become severely emaciated. In contrast, *H. gallinarum* is itself not particularly harmful to the fowl although it is often present in large numbers. Its importance lies in the part it plays in the transmission of the protozoan disease histomoniasis.

The influence of nutrition on experimental infections in general, and on host–helminth relationships in particular, has been discussed by several groups of workers (Clark, McClung, Pinkerton, Price, Schneider & Trager, 1949; Chandler, 1953; Hunter, 1953; Scrimshaw, Taylor & Gordon, 1959). Any appraisal of the problem must take into account the following aspects: (1) the virulence of the parasite, the size of the infective dose and route of entry, (2) the host that becomes infected, and in this respect genetics, age and sex are important, (3) the nutritional status of the host, (4) the nature of the resistance produced. Consequently, the host–parasite interrelationship is never static but complex and dynamic with either organism capable of producing constant changes in the other. Thus, any particular combination of the host and parasite can produce effects which range from no observable interaction to a serious disease which may result in the death of the host. Between these two extremes lies a whole range of interactions which can be influenced by nutrition.

There is no doubt that faulty nutrition can be an important factor in the outbreak and spread of helminth infections in poultry but often there have been difficulties in accumulating acceptable evidence. Under natural conditions there are many unmeasurable variables which may be influencing an infection so that it is difficult to establish conclusively that nutrition alone is responsible. However, during the last two decades there has been an impressive increase in knowledge of the nutritional requirements of chickens and by applying the rigid techniques of nutritional investigation to this complex problem valid results are being obtained.

The effect of nutrition on the course of a parasitic infection falls broadly into two categories, (1) the effect on the parasite and (2) the effect on the defence mechanisms of the host. Under the former are included those nutrients which are required by the parasite, substances that may have a stimulating effect on the parasite and components of the diet that may produce changes in the bacterial flora and pH of the intestine. In the second category are those nutrients that may enhance or inhibit the development of antibodies and also the protective integrity of the epithelium.

At the same time it is necessary to define the terms 'resistance' and 'susceptibility' which are so frequently used in the literature on this subject. Both are relative terms which can be applied with equal force to the same overall phenomenon, namely the infection. Thus a host can be described as 'more resistant' or 'less susceptible', terms which are equivalent and interchangeable. But when an analysis is made of the fundamental causes of these differences in the host's physiology then it is necessary to enter the fields of immunology and allied subjects. With chickens, most workers have been content to assess resistance (or susceptibility) in terms of the mortality and growth rate of the host and numbers and lengths of worms recovered at the end of a specified period after infection. With *A. galli*, experimental investigations commonly employ an infective dose of from 50 to 500 eggs, but the number of worms recovered is often very low being of the order of 5–10% of the initial dose. Consequently, the interpretation of results may be difficult. With *Capillaria* it is possible to obtain a greater recovery (about 50%) for similar dose levels.

Schneider (1951) distinguishes between 'susceptibility factors' whose absence from the diet decreases the effect of an infection and 'resistance factors' whose absence leads to an increase. On the other hand, Scrimshaw *et al.* (1959) express this difference by the terms 'antagonism' when a nutritional deficiency results in a decrease in the severity of an infection and 'synergism' when a deficiency results in an increase. Thus 'antagonism' occurs when the main effect is on the infecting agent, whereas 'synergism' is the result primarily of a nutritional deficiency on the host.

Some confusion has also resulted from the failure to distinguish between undernutrition and malnutrition due to a specific deficiency. The former is the result of a proportional reduction in intake of all essential nutrients, whereas the latter results from a relative or absolute deficiency of one or more specified nutrients. In fact, Tugwell (1955) has shown that birds on a restricted diet tend to carry a heavier burden of helminths than those being fed *ad lib.* The induction of such a nutritional

deficiency often leads to a retardation in growth rate which is accompanied by a self-induced restriction in food consumption. In such an event experimental results must be interpreted with care since they may be due to the reduced food intake alone and not to the effect of a specific deficiency. Unfortunately, this fact has been little recognized.

Effect of nutritional deficiencies on helminth infections

It has been suggested (Chandler, 1953) that severe chronic intestinal nematode infections are unusual in an otherwise normal host on an adequate diet and that only under conditions of very heavy exposure to infection before a specific immunity develops is a severe infection likely to occur. On the other hand, several groups of workers have shown that diets deficient in one or more of several essential nutrients will lower the resistance of chickens to such infections (Table 1).

Table 1. *Effect of nutritional deficiencies on helminth infections in chickens*

Deficiency	Helminth	Action*	Reference
Vitamin A	*Ascaridia galli*	Synergistic	Ackert *et al.* (1927)
			Ackert *et al.* (1931)
			Pande & Krishnamurty (1959)
	Syngamus trachea	Synergistic	Clapham (1934)
	Heterakis gallinarum	No effect	Clapham (1933)
	Capillaria obsignata	Synergistic	Wakelin & Chubb (unpublished)
Vitamin D_3	*A. galli*	No effect	Ackert & Spindler (1929)
Pteroylglutamic acid	*A. galli*	Synergistic	Sadun *et al.* (1949)
			Sadun *et al.* (1950)
Pyridoxine	*A. galli*	Synergistic	Brody (1954)
Vitamin B_{12}	*A. galli*	Synergistic	Brody (1954)
			Hansen *et al.* (1953)
			Hansen *et al.* (1954)
Lysine	*A. galli*	No effect	Riedel (1950*a*)
Tryptophan	*A. galli*	No effect	Riedel (1954*a*)
Glycine	*A. galli*	No effect	Riedel (1954*b*)
Methionine	*A. galli*	No effect	Todd (1951)
			Riedel (1955)
Manganese	*Raillietina cesticillus*	Synergistic	Harwood & Luttermoser (1938)
	A. galli	No effect	Gaafer & Ackert (1953)
Calcium	*A. galli*	Antagonistic	Gaafer & Ackert (1953)
Phosphorus	*A. galli*	Antagonistic	Gaafer & Ackert (1953)

*Terminology of Scrimshaw *et al.* (1959).

Vitamin A. Ackert and his co-workers (Ackert, Fisher & Zimmerman, 1927; Ackert, McIlvaine & Crawford, 1931) were the first to show a relationship between vitamin A and the degree of resistance of chickens to *A. galli*. They found that deficient chickens harboured more and larger worms than the non-deficient controls and suggested that weakened peristalsis and the larger amount of faeces present in the intestines of the deficient birds may have provided a better food supply for the worms. More recently Pande & Krishnamurty (1959) have confirmed this close interrelationship between hypovitaminosis A and a predisposition to infestation with *A. galli* under practical farm conditions. Clapham (1934) has also presented some limited experimental evidence that chickens deficient in vitamin A are more susceptible to *Syngamus trachea* infection than are those receiving a well-supplemented

diet. On the other hand, she found that *H. gallinarum* infection was unaffected by the level of this vitamin in the diet (Clapham, 1933). Siefried (1933) observed a high frequency of *C. annulata* in vitamin A-deficient chickens and some recent experiments (Wakelin & Chubb, unpublished) have indicated that this vitamin may increase resistance to experimental infections with *C. obsignata.*

Vitamin D_3. Ackert & Spindler (1929) found with chickens that a deficiency of this vitamin was without effect on their resistance to *A. galli.*

Vitamin B complex. As long ago as 1926 it was observed that more but smaller ascarid worms occurred in chickens deprived of several members of the vitamin B complex (Zimmerman, Vincent & Ackert, 1926; Ackert & Nolf, 1931). More recently Sadun, Totter & Keith (1949) and Sadun, Keith, Pankey & Totter (1950) investigated the effect of certain of these vitamins on *A. galli* infection in birds fed on purified diets. These workers showed that a deficiency of pteroylglutamic acid (PGA) lowered the resistance of young chickens to this infection. Thymic atrophy, leucopenia (lymphocytes and basophils) and an eosinophilia were found in the deficient birds, and it was suggested that these factors may have been partly responsible for the reduced degree of resistance through their adverse effect on the mechanisms of antibody formation. On the other hand, birds given a PGA-deficient diet to which had been added a large amount of 'injectable' liver extract were found to harbour more and larger worms than those on the deficient diet alone. It was therefore suggested that the vitamin B_{12} in the liver extract may have been responsible for the additional growth of the worms. However, it should be pointed out that there was a considerable variation in the number of worms recovered in the different experiments. These studies were extended by Brody (1954), who found that a simultaneous deficiency of PGA and vitamin B_{12} resulted in increased worm numbers and worm lengths. Unlike Sadun *et al.* (1950), however, he found that the addition of vitamin B_{12} to the deficient diet inhibited the growth of the worms. The simultaneous administration of these two vitamins decreased both counts and mean lengths. He also found with chickens that vitamin C had a similar effect when added to a diet deficient in vitamin B_{12} and PGA but not when vitamin B_{12} was present. Leucovorin appeared to be equivalent to PGA and vitamin B_{12} in its effect on worm numbers and length. In addition, Brody showed that pyridoxine-deficient chickens harboured more and larger worms than those fed on a complete diet. Hansen and his co-workers (Hansen, Norris & Ackert, 1953; Hansen, Petri & Ackert, 1954) also showed that chlortetracycline alone or with vitamin B_{12} added to an all-vegetable diet increased resistance to *A. galli* infection.

Proteins and amino acids. Ackert & Beach (1933) found that chickens given an all-cereal diet supplemented with certain animal proteins contained fewer and shorter worms than those given a similar diet supplemented with groundnut meal. Branson (1944) found that soya-bean meal was as effective as certain animal proteins in increasing resistance to ascarid infections. Riedel & Ackert (1950, 1951) suggested that this effect was due to the high content of certain essential amino-acids in these protein sources. However, subsequent work (Riedel, 1950*a*, 1954*a*,*b*, 1955; Todd,

1951) failed to show that the individual amino acids, lysine, tryptophan, glycine and methionine, had any part in resistance to ascarid infections.

There is some evidence that a mild hyperthyroid condition induced by orally administered thyroactive iodocasein may improve resistance to *A. galli* and *H. gallinarum* infections (Todd, 1948), but such treatment did not maintain growth at the same rate as in the non-infected chickens.

Todd, Hansen, Kelley, Wyant & Culton (1950) gave breeding hens either an all-vegetable diet or this diet with added fish meal or vitamin B_{12}. The resistance of their progeny (all fed on the same commercial starter diet) to *A. galli* was then studied. Although no worm counts or measurements were made, some improvement in resistance (based on body-weight gains) resulted from the addition of either the fish meal or vitamin B_{12} to the breeder diet.

Minerals. Little work has been carried out with any of these nutrients. Harwood & Luttermoser (1938) found that a manganese deficiency lowered the resistance of chickens to *Raillietina cesticillus*, but Gaafer & Ackert (1953) found that it was without effect on *A. galli*. The latter workers showed, however, that calcium and phosphorus deficiencies could increase resistance to *A. galli*. Riedel (1950*b*) found no evidence that the giving of grit *per se* produced any significant effect upon an *A. galli* infection in young chickens.

Effect of helminths on the nutrition of the host

In most species of animal it has been found that a helminth infection causes a loss in body-weight. It is probably the result of loss of appetite, decreased digestion of protein, poorer utilization of certain minerals, and the selective utilization by the parasite of some of the vitamins in the host's diet (Hunter, 1953). Surprisingly, however, Todd & Hansen (1951) found that, contrary to this view, progressively greater weight gains in young chickens could be correlated with increasing numbers of *A. galli*. They suggested that in those birds harbouring fewer parasites the energy employed in resisting infection had prevented their making maximal weight gains. On the other hand, Reid, Pate & Kleckner (1958) and Reid & Carmon (1958) found a highly significant decrease in weight gain as the worm population increased. A similar effect has also been noted with *Capillaria* infections (Levine, 1938; Wakelin, unpublished).

Effect of nutrition on acquired resistance

The relationship of nutrition to actively acquired immunity, either natural or artificially induced, has been discussed by Axelrod & Pruzansky (1955). Their experiments have clearly shown that antibody response in certain mammals at least can be markedly inhibited in various vitamin deficiency states. Obviously this phenomenon is significant only with those infections in which the importance of antibodies has been clearly established. In chickens this interrelationship with helminth infections has not been studied, because the nature of the immune response is at present unknown.

Conclusion

There is considerable evidence that the nutritional state of the host can have a profound effect on the course of many different helminth infections. In chickens on a well-balanced diet the effect of such an infection is generally a retardation of growth of the host and poorer utilization of the food. On the other hand, some dietary deficiencies have been shown to lead to an increase in the number and size of the helminths. The nature of these interrelationships has not yet been clarified since it is only by the in vitro culturing of the helminths with media deficient in specific nutrients that such information can be obtained. In addition, until more is known of the nature of the immunity produced it will not be possible to study the effect of nutrition on this phenomenon.

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