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Interaction of Nutrition and Infection in Poultry - A Review

A.B. Mandal*, A.V. Elangovan and S.V.S. Verma
Division of Avian Nutrition and Feed Technology
Central Avian Research Institute
Izatnagar - 243 122, India

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ABSTRACT

Nutrition is one of the major areas of importance amongst the acquired characteristics in relation to disease resistance. Nutrition of the host may affect pathogenesis either synergistically or antagonistically. In synergistic cases the host's impaired nutritional status tends to decrease the resistance for a pathogen. In antagonistic interaction impaired nutrition protests the host against the virulence of the pathogen, or infection may improve the nutritional status. Not a single nutrient acts prophylactically against infection. However, impaired nutrition is associated with reduced capacity of host to form specific antibodies, decrease in the phagocytic activity, altered tissue integrity, diminished inflammatory response, collagen formation and wound healing, and decreased antibody affinity and complement system. Again nutrition does not influence all infections equally and all nutrients do not have similar influence to a particular infection. Feeding management, different feed additives and toxins may also influence the immunocompetence of the birds. The present review highlights the mainstream research and speculates on areas, which need consideration.

Key words : Disease, Feed toxins, Immunity, Interaction, Nutrients, Probiotics.

INTRODUCTION

The poultry production activity in India has picked up the momentum during the last three decades or so and has now assumed the shape of an well organized industry. The high yielding poultry stocks are vulnerable to a wide variety of infections, some of which respond readily to treatment, whereas others persist and often resist control measures causing considerable production losses. The situation has become more serious due to increased stress arising from changed breeding, feeding and managemental practices that are becoming highly

*Reprint request : Dr. A.B. Mandal, Tel : +91-581-447223; Fax : +91-581-447321, E-mail : elangocari@rediffmail.com

specialised in wake of economic prospects of poultry farming in the modern times.

The importance of proper nutrition in economic poultry production and health promotion can not be over emphasized. The objective of poultry feeding so far has been to achieve optimum live weight gain and feed conversion efficiency on the basis of minimum nutrients supplied at the least cost. With gradual unfolding of the scientific knowledge to control poultry diseases and parasitic infestations, the influence of proper nutrition on various infections and diseases become all the more apparent. Nutritional status of the host can influence the occurrence of various infectious diseases (Scrimshaw *et al.* 1959; Chandra, 1999). Nutritional deficiency apparently increases the severity of infection by viral, bacterial, fungal or parasitic pathogens. Studies indicate that host's nutrition has an important impact on the cell mediated immune reactions and complement (Latshaw, 1991; Praharaj *et al.,* 1997). Impaired nutrition tends to decrease resistance to infection or its infection in consequent turn. Wakelin (1978) ranked nutrition as one of the major area of importance amongst the acquired characteristics in relation to disease resistance.

Nutrition of the host may affect pathogenesis either synergistically or antagonistically. In synergistic cases, the impaired nutritional status of the host tends to decrease the resistance to a pathogen. Some of the nutritionally induced determinants of synergism as summarized by Schrimshaw *et al.* (1968) include the followings: (a) a reduced capacity of the host to form specific antibodies, (b) decreased phagocytic activity of both, the microphages and macrophages, (c) an interference with production of non-specific protective substances, (d) reduced non-specific resistance to the bacterial toxins, (e) an alteration in tissue integrity, (f) the diminished inflammatory response and alterations in collagen formation and wound healing, (g) effects originating in intestinal flora and (h) variations in endocrine activity of the host. In antagonistic interactions, impaired nutrition protects the host against the virulence caused by pathogens, or the infection may improve nutritional status of the host. One classical example of antagonistic interaction has been that the lean birds resisted better than those of well nourished ones to *Rous sarcoma* virus. Moreover, research work and experience have demonstrated that certain feedstuffs or feed additives also have health

promotion qualities. An effort has been made to review the available reports on the role proper nutrition and feeding play in poultry disease conditions and their prevention.

Nutrition and bacterial infections

The role of dietary protein and bacterial infections in chickens has received the attention of different workers from time to time. Increasing the dietary protein level from 10 to 20 or 30% by employing soybean and casein, increased the fatality rate of chickens following inoculation with *Salmonella gallinarum* (Hill and Garren, 1961). However, chicks fed 15% protein in diet suffered higher fatality rate than those fed 30% protein on inoculation with *E. coli* (Boyd and Edwards, 1963). Severity of infection was high in chicks fed diets of high nutrient density (Boa-Amponsem *et al.,* 1991). Similarly, fast growing chicks receiving 24% CP and 3146 kcal ME/kg diet had higher lesion score and mortality than those fed on 20% CP and 2685 kcal ME/kg diet (Praharaj *et al.,* 1997). Deleterious effects of *E. coli* were also more on high plane of nutrition. In another experiment (Nitsan *et al.,* 1989), chickens receiving extruded soybean had lower mortality rate than those fed non-extruded soybean meal in diet. Survival rate of *Salmonella gallinarum* infected chicks increased when the birds received excess iron either through injection or iron supplemented diet (Hill and Smith, 1974). Whereas, the chicks receiving iron deficient diet were more susceptible to *S. gallinarum.* Similar reports indicating the critical role of iron in inducing infection to chicks are also available (Smith *et al.,* 1977a,b; Hary, 1979). Iron is an essential element for growth of pathogenic enteric bacteria including *Clostridium botulinum.* Elevated iron concentrations in drinking water or the feed had been attributed to intestinal proliferation of *C. botulinum* and subsequent by the occurrence of botulism (Recelunas *et al.,* 1999).

Stress originating due to over crowding of birds increased mortality rate in chicks inoculated with *E. coli,* the effect being more severe in terms of mortality when ethylenediamine tetraacetic acid (EDTA) was added in diet (Tufft and Nockels, 1991). Elemental, Cu, Zn and Fe changes namely of in serum and other tissues resulting from EDTA might have predisposed the chicks to a higher mortality rate from *E. Coli* infection as compared to those of the control birds. Taurine has been known to play important roles in cardiovascular and nervous system. However, its addition in diet

at 0 to 0.2% level did not alter resistance or susceptibility of chicks to *E. coli* infection (Dunnington *et al.,* 1996). Addition of vitamin A in diet of chickens suffering from *Mycobacterium tuberculosis* improved survival rate by 26% (Solotorovsky *et al.,* 1961). Dietary supplementation with vitamin A or vitamin E reduced *E.coli*-induced mortality in chickens (Cheville, 1979) and the effect was attributed to increased antibody production and phagocytosis (Tengerdy and Brown, 1977). Supplementation of 150-300 mg vitamin E/kg diet in form of D-a tocopheryl acetate gave increased protection against a relatively moderate mortality in *E. coli* infection in birds (Heinzerling *et al.,* 1974).

Several feed restriction programmes have been *in vogue* to control feed intake and thereby improve reproduction in broiler breeders, forced molting and slaughter traits of birds. The chicks shifted from alternate-day to the *ad lib* item feeding were found to be more susceptible to the *E. coli* infection (Katanbaf *et al.* 1988). Feed withdrawal i.e. starvation before processing of broiler chickens has also been associated with increased colonization by bacterial enteropathogens. The crop serves as a reservoir for salmonellae in chicks (Hargis *et al.,* 1995) and the feed withdrawal has been linked to an increase in the number of colonized salmonellae in the crop of broilers (Ramirez *et al.,* 1997).

Osteomyelitis outbreaks by *Staphylococcus aureus* have been found associated with severe feed restriction, poor nutrition, over crowding in turkeys (Mutalib *et al.,* 1983, Zhu and Hester, 2000). Flocks of chicks harbouring S. aureus in their yolk sacs at one-day of age may develop staphylococcosis when they are stressed by restricted feeding programmes (Zhu and Hester, 2000). Similarly, feed restriction is a common programme in raising of broiler breeders and may play an important role in the development of staphylococcal osteomyelitis and synovitis (Page, 1982).

Nutrition and viral infections

The low protein diets (8% CP) or those with excess protein (41% CP) increased severity of Newcastle disease as indicated clinically and also by the RNA / DNA ratio observed in liver cells (Squibb, 1964). However, growth depression was less with lysine deficient than on control diet, and fatality rate increased in chicks receiving borderline vit. A deficient diet (Squibb, 1961). Similarly, Sijtsma *et al.* (1989) observed

that pre-existing marginal vitamin A status increased the severity (mortality and morbidity) of the disease. Following Newcastle Disease Virus (NDV) infection, the plasma vitamin A level reduced to a level that could be regarded as deficient. Birds receiving 3600 or 10,000 IU of vitamin A/lb of diet experienced greater live weight during active disease condition following infectious bronchitis infection (Panda *et al.,* 1962) but the course of the disease was not altered even when vitamin A concentration was 5 times than the requirement (Gratzl *et al.,* 1963). Pardue (1987) reported that supplementation of vitamin C in diet of chicks increased resistance against infectious bursal disease. The effect of vitamin E deficiency on chick mortality as induced by avian encephalomyelitis (AE) virus was examined (Cheville and Monlux, 1966), the deficient chicks appeared slightly less susceptible than the control chicks.

Nutrition and gut protozoal infections

Although no nutrients are known to act prophylactically against coccidia (Fayer and Reid, 1982), the nutritional status of the host is known to increase as well as decrease the severity of infection. Supplemental vitamin A in diet over normal level did not reduce mortality or morbidity but helped the chicks to recover fast (Reid, 1972). Mortality due to *E. tenella* and *E. necatrix* infections was reduced on supplementation of diet with vitamin K but no beneficial effect against non-haemorrhagic species of coccidia e.g., *E. acervulina, E. brunetti* and *E. maxima* (Ryley and Hardman, 1978) were observed. Vitamin K @ 0.53 mg/kg diet reduced clotting time and prevented haemorrhage. Colnago *et al.* (1984) concluded that supplementation of selenium @ 0.25 mg/kg or vitamin E @ 100 mg/kg diet reduced mortality and increased body weight gain of non-immunized chicks infected with *E. tennella.* The coccidia require many vitamins and metabolites for normal development viz., thiamine, riboflavin, nicotinic acid, biotin, folic acid, panthothenic acid, pyridoxine, retinal, calciferol, ascorbic acid, menadione, choline, inositol, cyanocobalamin, para-amino benzoic acid and glutamic etc. Deficiency of these vitamins or the presence of antimetabolites or vitamin antagonistic elements would interfere with their normal development. Several anticoccidial drugs act by antagonizing vitamins; for example sulphonamides antagonize para-amino-benzoic acid, amprolium is an antimetabolite of thiamin, and pyrimethamine

antagonizes folic acid (Fayer and Reid, 1982). Coccidiosis and ascorbic acid @ 150 mg/kg diet interacted to increase feed intake, lower plasma concentration and heterophil; lymphocyte ratios (Mckee and Harrison, 1995).

In groups of chickens fed diets containing 0, 5, 10, 15, 20 or 30% protein before inoculation with oocysts, fatalities increased progressively upto 15% dietary protein (Britton *et al.,* 1964). *Eimeria* oocysts require trypsin for excystation and releasing of infective sporozoites. A protein deficiency or as little as 48 hours of fasting reduces available trypsin to inconsequential levels and thereby protects the birds. On addition of trypsin to the oocyst inoculum, the antagonism was avoided (Britton *et al.,* 1963; 1964).

Chicks artificially infected with *E. tenella* and fed 24% protein in diet suffered high mortality than those fed 16 or 20% crude protein. However, in *E. acervulina* infection higher protein was beneficial to protect weight loss (Sharma *et al.,* 1973). After a natural outbreak of intestinal coccidiosis, a high protein diet seemed more beneficial (Harms *et al.,* 1967). Diet supplemented with 4% fish oil attenuated the growth depressing effect of *E. tenella* (Korver *et al.,* 1997). Chicks suffering from *E. acervulina* or mixed coccidiosis responded remarkably with increased gain and improved feed conversion efficiency when excess zinc added to the high calcium (1.82% or 2.00%) diet (Bafundo *et al.,* 1984; Khanagwal *et al.,* 2000a,b). Supplementation of diet with extra calcium and zinc on the face of coccidial outbreak was beneficial in improving feed conversion efficiency (Singh *et al.,* 1997a, b). Dietary level of calcium and or zinc did not alter the course of coccidiosis as oocysts output remained similar. However, mortality was lower at high calcium (1.98%) and high zinc (93 or 123 ppm) containing diet (Khanagwal, 1995; Singh *et al.,* 1997a,b). Supplementation of phosphorus in the form of disodium phosphate did not prove beneficial and rather decreased feed intake due to unpalatability of feed. Aflatoxin in diet of birds increased the severity of *E. tenella* (Wyatt *et al.,* 1975) and *E. acervulina* infections (Ruff and Wyatt, 1978). The addition of sodium carbonate in diet at 0.2 to 0.4% level yielded benefits in lesion score and body weight (Hooge *et al.,* 1999). However, the responses of supplemental nutrients are variable depending upon the type of coccidial infection and pathogenesis involved.

Nutrition and immunocompetence

The ability of poultry to withstand infectious disease caused by bacteria, virus or protozoa depends upon the integrity of immune system. The immune system consists of diverse cell populations. Avian cellular immune system consists of rapidly dividing cells (Dietert *et al.,* 1990). The commercial poultry environments harbour different microorganisms, which are challenging the immune system continuously. Proper functioning of the immune system depends upon the availability of nutrients, the precursors for cell growth and activity. Immunity can be impaired by nutrient deficiency and biological amines. Immune stimulation decreases appetite and muscle protein accretion, increases metabolic rate, body temperature, and oxidative damage to cells (Swick, 1995).

Both low and high vitamin A intakes resulted in impaired immune responses (Friedman and Sklan, 1997). Low dietary vitamin A level caused reduced antibody production, defective T-cell responses and reduced phagocytosis, and decreased resistance to infection by bacterial pathogen (*E. coli;* Friedman *et al.,* 1991), Mycoplasma (Boyd and Edwards, 1962); viral pathogen viz. Newcastle disease virus (Davis and Sell, 1989) and turkey pox (Sklan *et al.,* 1995) and protozoan enteropathogens e.g. coccidia (Singh and Donovan, 1973). Optimum immune responses in growing chicks and poults were observed at 3-10 fold higher dietary vitamin A levels than specified by NRC(Friedman and Sklan, 1997). The retinoids direct differentiation, development and morphogenesis by pre-activated gene amplification in immune response cells (Wolf, 1991; Friedman *et al.,* 1991). Nutritional deficiencies of vitamin E and selenium impaired immune function as measured in terms of humoral response to sheep erythrocytes in young chicks (Marsh *et al.,* 1981). The deficiency of vitamin E and Se in chicks depressed bursa weight, reduced the overall number of lymphocytes found in the primary lymphoid organs and spleen, and resulted in destructive histological changes within these tissues (Marsh *et al.,* 1982). Vitamin E supplementation at higher levels (150-300 IU/kg) increased antibody production against NDV vaccination (Raza *et al.,* 1997). It has been established that immunostimulation effect by vitamin E and Se were related to their antioxidant properties, and that the stimulation of Se was independent of vitamin E nutrition of the host (Colnago *et al.,* 1984).

The possible mechanisms for Se action may have been due to a greater glutathione peroxidase activity which helps to protect membranes of lymphocytes from prooxidants. Selenium may also be involved by modifying the metabolism of arachidonic acid to prostaglandin precursors or related compounds, thus enhancing immune response by reducing endogenous production of prostaglandins because the prostaglandins and related compounds are involved in immune response (Stenson and Parker,1980; Goodwin and Webb, 1980, Colnago *et al.,* 1984; El Boushy,1988). Supplemental vitamin C in diet of healthy chicks did not augment antibody production but did so in the immunosuppressed chicks (McCorkle et al., 1980; Pardue et al., 1985). Fasting as practiced in meat-type breeders, increased resistance to deleterious effects of *E.coli* (O' Sullivan *et al.,* 1991) and E. tenella (Zulkifli *et al.,*1993) infections. Feed restriction enhanced development of endocrine glands and also humoral immunoresponsiveness (Klassing, 1988). Feeding alterations such as imbalance of protein, low energy and dietary fibre impaired immune competence and disease resistance (Scott *et al.,* 1982; Boa-Amponsem *et al .,*1991). Though, the level of dietary energy alone or energy and protein did not alter antibody titre to sheep RBC or weight of lymphoid organs (bursa and spleen) in commercial broilers (Reddy *et al.,* 1998; Praharaj *et al.,* 1999a), but an interaction existed between genotype and dietary energy level in another experiment (Praharaj *et al.,* 1999b). Broiler chicks fed higher protein in diet (23% CP) revealed better persistency in antibody production to sheep RBC at 10 or 15 days post-injection (Praharaj *et al.,* 1998). However, long-term obesity but not short term weight gain had deleterious consequences for reproductive characteristics, response to sheep RBC antigen, resistance to *E.coli* and livability (O' Sullivan *et al.,*1991). Evidences (Tsiagbe *et al.,* 1987; Hall *et al.,* 1986) also existed that methionine is also an essential input for proper functioning of the immune system. Methionine deficiency depressed immune responsiveness. Lymphocytes have also specific methionine requirements. However, important benefits arise from increasing the dietary level beyond a level required for optimum growth.

Certain trace elements like zinc, iron, copper, manganese and selenium are important for normal immune function and disease resistance (Fletcher *et al.,* 1988; Reddy and Frey, 1990). Presence of adequate zinc in the biological system is crucial to normal development,

maintenance and functioning of the immune system (Dardenne and Bach, 1993). Zinc is important for proper functioning of heterophils, mononuclear phagocytes and T-lymphocytes (Kidd *et al.,* 1996). As the avian immune system consists of rapidly dividing cells (Dietert *et al.,* 1990), an adequate cellular zinc is crucial for enzyme functions that facilitate their proliferation (Vruwink *et al.,* 1993).

Role of feed additives in disease prevention

Probiotics

Probiotics are the live microbial feed supplements which beneficially affect host animal by improving its microbial balance, nutrition and health. Continuous feeding of probiotics helps in maintaining health by two ways viz. competitive exclusion of pathogens and antagonistic activity towards pathogenic bacteria. Lactic acid bacteria are able to inhibit the growth of *Salmonella* strains and *E. coli* (Chateau *et al.,* 1993; Oyarzabal and Conner, 1995; Jin *et al.,* 1996). The administration of *Enterococcus faecium* significantly decreased clostridial counts adhering to the crop wall as well as in the caecal content (Kmet *et al.,* 1993). An interaction (competitive exclusion) between *Bifidobacteria* and *Salmonella* had also been reported (Farnworth *et al.,* 1995). *Salmonella* colonization was reduced ($P < 0.05$) but *Campylobacter* colonization was not affected by yeast treatment (Line *et al.,* 1998). In a field trial, there were significant decrease in the number and detection rate of *Campylobacter*, detection rate of *Salmonella*, number of *Clostridium perfringens* and *Enterobacteriaceae* and increases in the number of lactobacilli after *B. subtilis* administration (Maruta *et al.,*1996). Chicks inoculated with caecal culture had lesser number of adherent microbes (*Salmonella*) in the caecal mucosa (Yu *et al.,* 1999). The possible mechanism for antagonistic effect of probiotics has been reviewed by Jin *et al.* (1997). The antagonistic effect of lactobacilli has been attributed to low pH, low redox potential and the production of inhibitory substances like bacteriocins, organic acids, hydrogen peroxides, etc. The proposed mechanisms for competitive exclusion include aggregation between lactic acid bacteria with pathogens, competition for adhesion sites, competition for nutrients and production of bacteriocidal substances (Jin *et al.,* 1997). Probiotics specially the lactobacilli and *Bacillus cereus* are also important in the development of immunocompetence against

enteric infections (Perdigon *et al.,* 1995). However, the responses obtained depended upon several factors viz. microbial strain, number of viable cells per unit feed, type and nature of diet, age of birds and sanitary conditions of poultry house.

Prebiotics

Prebiotics are non-digestible feed ingredients that beneficially affect the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon, that can improve the host health. Galacto-oligosaccharides, fructo-oligosaccharides and lactose derivatives have been used in poultry and other non-ruminants (Peeters *et al.,* 1992; Morisse *et al.,* 1992; Chambers *et al.,* 1997). The oligosaccharides may directly inhibit the growth of certain intestinal pathogenic species by increasing the concentration of lactic acid thereby decreasing pH in the lower gut (Choi *et al.,* 1994; Okumura *et al.,* 1994). Microbes are able to attach themselves to the mucosa through recognition of oligosaccharide binding sites on the wall (Morgan *et al.,* 1992). Dietary oligosaccharides attract microbes away from the intestinal binding sites and, therefore, reduce, colonization by pathogens. Chickens treated with fructo-oligosaccharides had a four-fold, reduction in the level of *Salmonella* present in caeca (Bailey *et al.,* 1991) and the reduction was attributed to a shift in the intestinal gut microflora. Moreover, certain oligosaccharides like 1,2-gluco-oligosaccharides substrate for beneficial *Bifidobacterium* spp., at lower tract which favoured their colonization but prevented those of the *Clostridium, Enterobacter, E. coli, Enterococcus,* etc. (Monsan and Paul, 1995). The effect of dietary lactose and *Lactobacillus acidophilus* on the colonization of *Salmonella enteritidis* in newly hatched Leghorn chicks concurrently infected with *Eimeria tenella* has been studied (Qin *et al.,* 1995). The caecal population of *S. enteritidis* was increased significnatly by *E. tenella* infection. Supplementation of lactose alone or the combination of lactose and *L. acdiphillus* reduced the population of *S. enteritidis* in the caeca of birds infected with *E. tenella* but the combination was more effective than lactose alone. Similarly, culture of indigenous caecal bacteria together with dietary lactose controlled *S. typhimurium* caecal colonization effectively in newly hatched broiler chicks (Corrier *et al.,* 1993, Kogut *et al.,* 1994). However, neither the lactose nor *Lactobacillus* (or organic acid, egg powder) prevented colonization of *S. enteritidis* or organ

invasion of chicks in another experiment (Opitz *et al* 1993).

The information provided herein is the clear indicative of close relation between nutrition and the health of poultry and that many of the nutrients have been found to influence the growth, production and reproductive capacity of the birds significantly. The effects have been more clear in the high-producing stocks. Therefore, the managemental practices involving host's nutrition can be a potential component in an integrated approach towards ensuring a sound health and thereby controlling infection, an area which has so far poorly been exploited. Nutritional studies in future should also incorporate immunological aspects along with the traditional growth and production criteria. Because the nutrition also has a direct bearing on outcome of disease in the infected animals, the interaction of infectious diseases with different nutrients along with the optimum level of interacting nutrients need to be established. As the immunoresponsive effects of various nutrients are observed at much higher levels than those actually recommended for optimum production, it is therefore necessary to give due considerations to the economic aspects of the whole exercise. There is also a need to consider the nutrition, disease control and flock management together to ensure maximum profitability from poultry production.

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