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# Early post hatch nutrition on immune system development and function in broiler chickens

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**The onset of feeding broiler chicks after hatch is often delayed by 48-72 hours due to variations in hatching time and follow up hatchery practices, affecting their ultimate performance at marketable age. In the newly hatched chicks, residual yolk serves as a source of nutrients until access to exogenous sources of feed is established. However, despite the residual yolk being sufficient to maintain the chicks during the first three to four days of life, it does not provide the required level of nutrients to fully support the genetic expression of the bird's potential for growth, development of the gastro-intestinal tract or the immune system. Development of the avian immune system is triggered during embryogenesis, but is not evolved until a few weeks of age *post hatch*, and can be stunted due to the unavailability of nutrients if hatchlings are deprived of food immediately after hatch. Early feeding is not only associated with immune organ development, but also with the functioning of the immune system in broiler hatchlings. With the continued increase in the economic importance of broiler chickens, an understanding of the development and function of the immune system in these birds and their capacity to respond effectively to divergent antigens is necessary.**

**Keywords:** early post hatch nutrition; immune system development and function; broilers

## Introduction

Significant progress has been made during the past three decades on enhancing juvenile body weights in broiler chickens. Along with faster growth (2.2-2.4 kg at 40d), feed efficiency (1.6-1.7) and the proportion of breast meat have been improved in commercial broilers. However, the faster growth has a negative effect on the immune system; thereby increasing the incidence of early chick mortality, probably due to reduced resistance to infectious diseases (Yunis *et al.,* 2000; Juul-Madsen *et al.,* 2004). The immune system provides the body with the ability to mount a defence against foreign organisms and antigenic substances. Many reports have suggested that chicks in the first week of hatch

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lack adequate immune responses and remain highly susceptible to infections (Mast and Goddeeris, 1999; Juul-Madsen *et al.,* 2004). This has been attributed to the inadequacy of the immune systems development and its functional inability to effectively respond to the diversity of antigens required for protecting the chick during the early part of life (Panda *et al.,* 2010). Development of systemic immune competence in the chickens occurs during the *post hatch* period (Erf *et al.,* 1998; Mast and Goddeeris, 1999).

With the continued increase in the economic importance of broiler chickens, a better understanding of the development and functioning of the avian immune system in these birds is essential. The avian immune system begins in the embryo and continues for several weeks after hatch. Several factors influence the early development of immune system in newly hatched chick, and one of the important factors is initial feed intake (Dibner *et al.,* 1998). The transition from use of yolk sac derived nutrients to those present in exogenous feeds provides the correct platform for rapid functional and structural development of the gut (Geyra *et al.*, 2001). Feed provides nutrient substrates, which are essential for the growth and development of both primary and secondary lymphoid organs (Dibner *et al.*, 1998). The immune system of the hatchling, particularly the mucosal immune system, also requires oral feed intake for rapid development (Bar Shira *et al.,* 2005). However, in commercial poultry operations, poultry hatch is delayed by over 2-days and chicks are only moved from the hatchery after the majority of chicks have emerged from their shells. Following the release, other hatchery practices including sexing, vaccination and packaging are carried out before they are boxed for transportation. Thus, in practice, chickens from a hatchery may often spend considerable time (48-72 h) without any access to feed or water. It has been reported that early feed deprivation not only affects bird growth (Noy and Sklan, 1999; Bhanja *et al.,* 2009; Panda *et al.,* 2009) but also immune system development (Juul-Madsen *et al.,* 2004; Panda *et al.,* 2010). Early dietary restriction was reported to produce metabolic changes leading to impaired systemic immune competence (Nathan *et al.,* 1977; Hulsew *et al.,* 1999) possibly due to depleted lymphoid organ growth (Dibner *et al.,* 1998; Panda *et al.,* 2010). It has been reported that the primary lymphoid organ, the thymus, is very sensitive to the duration of feed deprivation, and causes a rapid decline in its cellularity and weight gain (Klasing, 2007). Feed deprivation beyond 48 hours in chicks impaired the activity of gut associated lymphoid tissue in the hind gut and delayed the development of lymphocyte populations in the bursa during the first two weeks of life (Bar Shira *et al.,* 2005; Panda *et al.,* 2008). Thus, the time from hatching to the onset of receiving food is the critical period for their development and subsequent performance of the hatchlings. Although the effect of early *post hatch* nutrition has been widely studied and reviewed, most of the information published remains confined to broiler performance in terms of body weight gain, feed conversion, GI tract development and meat yield, and not on the immune system or immune competence on long-term scale. The present review aims specifically on the development and functioning of immune system and the role of early *post hatch* nutrition on the maturation of the immune system in broiler chickens.

## Avian immune system

Among the avian species, the immune system of the chicken has been the most extensively studied. Because of its widespread function, the immune system is a complex system, both structurally (*Figure 1*) and functionally (*Table 1*). The immune system is widely dispersed throughout the body and it is composed of organs, cellular elements, and soluble (humoral) elements. Like in mammals, immunity in birds is



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developed through the lymphoid system. The organs of the immune system can be classified into primary (central) and secondary lymphoid organs (*Figure 1*).

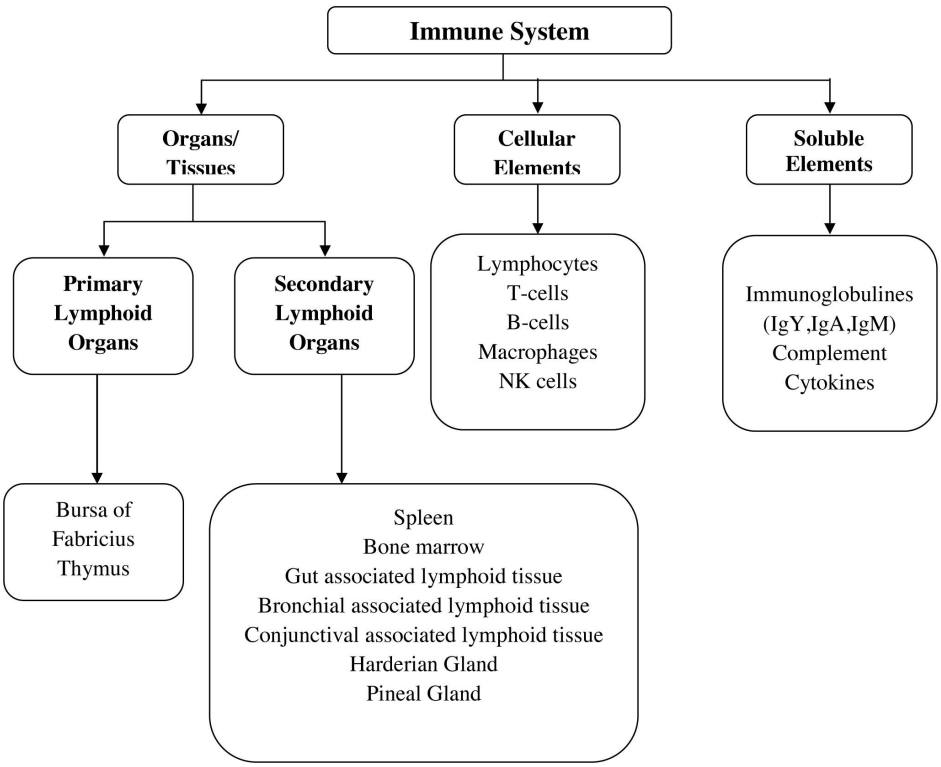



**Figure 1 Avian immune system.**

The bursa of Fabricius and thymus are the primary lymphoid organs in which the lymphocyte precursors develop into immune competent native lymphocytes. Secondary lymphoid tissues are the spleen, bone marrow, harderian gland, pineal gland and organised lymphoid tissues associated with mucosal surfaces (MALT), including bronchial-associated lymphoid tissues (BALT), gut-associated lymphoid tissues (GALT), conjunctival associated lymphoid tissues (CALT) and other less-prominently organised clusters of lymphoid cells in various organs. These lymphoid tissues are located at strategic sites where foreign antigens entering the body from either the skin or a mucosal surface can be trapped and concentrated. Lymphocytes, antigen transporting and presenting cells and other regulatory cells are located in these anatomically defined tissues and are thought to be organised into structures that optimise cellular interactions to support the efficient removal of unwanted pathogens (McCorkle and Glick, 1980; Klasing, 2007).



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**Table 1 Avian immune system, its related organs and their structure and functions.**

| Organ | Location | Structure | Function |
|---|---|---|---|
| Bursa of Fabricius | Dorsal to cloaca | Round/oval sac with lymphoid follicles | Production and development of B lymphocytes |
| Thymus | Along two sides of the neck | Multi lobular with follicles | Production and development of T lymphocytes |
| Spleen | Right side of proventiculus- gizzard junction | Variable shape | Systemic immunity |
| Meckle's diverticulum | Attached to mid-intestine | Loosely follicular | Local immunity |
| Caceal tonsils | Proximal end of caeca | ---------- | Local immunity |
| Peyer's patches | Ileocecal junction | ---------- | Local immunity |

Source: Panda and Reddy (2007)

Chickens basically have two types of defence systems to counter infectious agents; innate (non-specific) immunity and adaptive immunity (specific immunity). Non-specific immunity comprises of physical and chemical barriers (to prevent invasion) and cellular components (macrophages, neutrophils, natural killer cells, etc.). Heterophils are the avian equivalent to mammalian neutrophils. Specific immunity is made up of two parts; humoral immunity (carried out by antibodies) and cell-mediated immunity (carried out by T cell activity). The cellular components of specific immunity are lymphocytes. Chicken lymphocytes consist of B cells, which develop in the bursa of Fabricius and, the T cells in the thymus. Both, the B and T cells express antigen receptors on their surface. It has been reported that each B cell and T cell has a homogenous set of antigen receptors specific for a given antigen (Dibner *et al.*, 1998).

## Ontogeny of the avian immune system

An understanding of immune system development in neonate broiler chicks and its capacity to respond effectively to the diversity of antigens during the early part of their life is a priority. The development of the immune system in the embryonic and neonatal stages is a dynamic process involving the migration of lymphocyte stem cells to primary immune glands, the rapid and extensive proliferation of lymphocytes in the absence of any antigen within the bursa and thymus, the concurrent differentiation of bursal and thymic lymphocytes, and the eventual seeding of differentiated lymphocytes to the peripheral lymphoid organs such as the spleen and caecal tonsils (Bucy *et al.,* 1988). The immune system in poultry is partially developed at hatch. The primary immune organs such as the bursa and thymus are present and are populated by lymphoid tissue. However, the cell mediated immune system has a fairly high degree of development during embryogenesis as compared to the humoral immune system (Perey and Bienenstock, 1973; McCorkle *et al.,* 1983).

The stem cell migration to chick thymus occurs in three waves during embryogenesis at 6.5, 12 and 18 days of incubation, and these cells pass through thymus to populate peripheral tissues (Bucy *et al.,* 1988). The thymocytes are CD3+ (avian homologue) and develop to CD4 or CD8 antigens during embryogenesis (Lillehoj and Trout, 1996). The seeding of bursa by lymphocytes occurs between 7.5 and 14 days of embryogenesis (Peault *et al.,* 1987). At this stage, these cells are committed B cells but are capable of expressing IgM at hatch only (Leslie and Martin, 1973). Dibner *et al.* (1998) reported that



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the dominant immunoglobulin in the bursa of the hatchling is IgM. The IgM-bearing lymphocytes are the precursor cells for the expression of IgG or IgA. The hatchlings do not have the capacity to produce IgG at this stage, and are entirely dependent upon maternal antibodies for humoral immune protection (Martin and Leslie, 1973; Shawky *et al.,* 1993).

The secondary immune organs such as the spleen, caecal tonsils and Meckel's diverticulum are incomplete at hatch (Schat and Myers, 1991). IgM-secreting B-cells are found in the spleen at three days of age whereas IgG-secreting B-cells are not detected until six days of age (Lawrence *et al.,* 1981). Although B cells are detected in caecal tonsils at hatch, they only express IgM. There are T cells in the lamina propria and epithelium of the gut and other secondary immune organs, but these do not develop cytotoxic ability until some period after hatch. The ability to mount a secondary response, as indicated by the presence of germinal centres or circulating IgG and IgA, begins to appear between the first and fourth week *post hatch* in broiler chicks (Leslie, 1975). Bar Shira *et al.* (2003) reported that gut associated lymphoid tissue contained functionally immature T and B lymphocytes at hatch and their functional maturation occurred during the first two weeks of life.

## Residual yolk-sac and immunity in neonates

Towards the end of the incubation, residual yolk is internalised into the abdominal cavity and is the only nutrient source to the newly hatched chick until exogenous feeds are supplied (Romanoff, 1960). At hatch, the yolk constitutes 20% of the chick's weight and provides immediate *post hatch* energy and protein for growth (Sklan and Noy, 2000). Although the protein fraction is partly from albumen, a large content of the egg protein in hatchlings is made up of maternal antibodies (Dibner *et al.,* 1998). In general, the maternal antibodies are not digested during the incubation process, leaving those immunoglobulins intact and fully functional at the time of hatch, indicating that the protein component of the yolk sac is used for passive immunity of the chicks. Thus, the chick should utilise the residual yolk sac faster to provide early immunity to the newly hatched chick. It is reported that residual yolk is used up more quickly by chicks that have early access to feed compared to those that are fasted for 48 hours (Noy *et al.,* 1996, Bhanja *et al.,* 2009). The reason being that anti-peristaltic movement of yolk from yolk-stalk to the duodenum is stimulated by the presence of feed in the gut (Noy and Sklan, 1999; Panda *et al.,* 2006). However, hatchery operations in commercial poultry practices delay the access of feed and water and under practical conditions newly hatched chicks generally receive their first food and water 48-72 hours after hatching. This delay in access to nutrients may limit the utilisation of the yolk sac and result in poor immunity in the newly hatched chick (Bhanja, 2008).

Furthermore, despite the residual yolk being sufficient to keep the birds alive for the first three to four days, it does not support the expression of the bird's genetic potential for body weight gain, development of the gastro-intestinal tract or immune system (Dibner *et al.,* 1998). The same researchers studied the effect of early feeding on lymphocyte proliferation in bursa compared to fasting for 48 hours. It was observed that early feeding led to extensive proliferation of lymphocytes compared to fasted chicks, and that the nutrients of residual yolk may not be a substitute for oral intake in maintaining lymphocyte proliferation, as seen in chicks receiving oral nutrition. This could be attributed to the priority of use of the nutrients by the gut (Ricklefs, 1987) and may be related to the presence of nutrient substrates and antigen exposure (Rao *et al.,*



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1978). Consequently, prolonged feed restriction not only reduced yolk sac utilisation but also affected the development of the immune system.

## Early nutrition and the development of immune organs and function

Early feeding has a significant effect on bursa and spleen development of chicks. Dibner *et al.* (1998) reported significantly higher bursa weight on the 3rd day of broiler chicks fed immediately after hatch compared to those fasted for 48 hours, and the effect remained up to 21days of age. In addition, the lymphocyte proliferation in bursa was faster in the immediately fed chicks. In another study, Juul-Madsen *et al.* (2004) observed that feed deprivation for 24 hours was acceptable for normal immunological response, but further delay in feeding up to 48 hours was unfavourable for immune response and growth in broilers, suggesting that newly hatched broilers can cope with a short duration of starvation. However, the results of Bhanja *et al.* (2009) and Panda *et al.* (2010) indicated that even the shorter duration of 24 hours feed deprivation *post hatch* depressed development of bursa and spleen, while feed deprivation up to 48 hours *post hatch* affected broiler growth. Later studies inferred that the immune system was more sensitive and susceptible to nutrient deprivation than growth and development. In a recent study, Rammouz *et al.* (2011) reported that even 12-hour feed deprivation *post hatch* was harmful for the development of the immune system in poultry. Therefore the conflicting information reported by various researchers on immune system development due to delayed feed access requires further investigation, and the exact duration of *post hatch* deprivation that would not adversely affect the immune system maturation and its functioning needs to be determined.

Approximately 70% of the body's immune system is found in the digestive tract of chickens. One of the key stimulators of intestinal development in the chick is physical exposure to feed, while feed deprivation delays the onset of gut development (Geyra *et al.,* 2001; Panda *et al.,* 2008). Further, intestinal development occurs in continuation with that of gut associated lymphoid tissue (GALT), which is a component of the mucosal immune system (Thompson *et al.,* 1996; Bar Shira *et al.,* 2003). GALT is made up of several types of lymphoid tissue, the caecal tonsils, Peyer's patches, Meckel's diverticulum and the bursa of Fabricius that produce and store immune cells to attack and defend against pathogens. GALT in chickens is responsible for the initiation of immune responses against bacterial, viral and parasitic challenges. The development of GALT in the hindgut of broiler chicks was delayed due to early feed deprivation, which persisted for two weeks of life (Bar Shira *et al.,* 2005). Delay in the onset of feeding results in delay of the maturation and expression of the functional activity of T lymphocytes in the hindgut and also delays lymphocyte proliferation in the bursa of fabricius. It should be emphasised here that the initial first week of life is very critical for the chick's survival and subsequent performance, as delayed access to feed takes nearly two weeks to achieve normalcy. Therefore, chicks with no access to feed immediately after hatch may be more vulnerable to environmental pathogens than those fed immediately after hatch. This vulnerability needs attention particularly in situations where chicks are in transit from hatchery to farms for extended periods.

The mechanism involving the effect of feed deprivation on the recession of lymphoid organ weights is not known, but one possible explanation for the negative effects on weight could be due to the rise in glucocorticoids associated with fasting (Dibner *et al.,* 1998). Gulcocorticoids have been reported to hamper the involution of primary lymphoid organs, even in young birds (Nir *et al.,* 1996). Bigot *et al.* (2001) reported that fasting induced stress leading to the secretion of corticosterone hormones which inhibited the



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proliferation of lymphocytes and affected the immune system of birds. Further, he suggested that chicks fasted for more than 24 hours *post hatch*; degraded the immunoglobulins present in the residual yolk sac to produce proteins for their survival. The development of secondary immune organs, *e.g.* spleen, depends on the primary immune organs, *e.g.* bursa and thymus (Glick, 1997). If the development of the bursa is affected, spleen growth will be reduced. Another important factor on the development of immune organs appears to be due to antigen exposure (Rao *et al.,* 1978). Feeding immediately after hatch provides an early antigen stimulus, facilitating rapid differentiation of the immune organs (Ekino *et al.,* 1979; Bar Shira *et al.,* 2005). Additionally feed provides nutrient substrates that are essential for the growth and development of secondary lymphoid organs (spleen) as they are not matured at hatch (Dibner *et al.,* 1998).

The newly hatched chick can only express IgM, but not IgG and IgA. Though, IgG was detected in bursa of newly hatched chicks, this represented the maternal IgG deposited by the residual yolk sac (passive immunity) (Bhanja, 2008). Thus, the humoral immune system of hatchlings consists of IgM and maternal IgG only. IgA is a part of the mucosal immune system and is the last of the major isotypes to appear. Thus, the appearance and levels of biliary IgA is an indicator of humoral immune system maturation and functioning, as the hatchlings on their own have no ability to synthesize IgA. It was reported that early feeding resulted in more rapid appearance of biliary IgA and higher levels of Igs than those fasted for 48 hours (Dibner *et al.,* 1998). Germinal centres are the local concentrations of lymphocytes in which T cells, B cells and antigens presenting cells form an organised structure associated with the development of immune memory to antigen, as required for a vaccine response. Thus, early appearance of germinal centres is an indication of immune maturation (Schat and Myers, 1991). Dibner *et al.* (1998) observed that early feeding resulted in significantly higher numbers of germinal centres in the caecal tonsils of neonates compared to those fasted for 48 hours and the carryover effect was observed up to 21 days of age. Juul-Madsen *et al.* (2004) reported that prolonged feed restriction up to 48 hours was harmful for the development of the immune system. These researchers observed lower proportions of CD4 and CD8 cells on the seventh day *post hatch* in chicks deprived of feed for 48 hours compared to those with access to feed immediately after hatch. Bar Shira *et al.* (2005) also found impaired development of the functionality of GALT in the hind gut due to early feed deprivation in hatchlings. They reported that the colonisation of the hind gut (caecum and colon) by B and T lymphocytes was delayed and expression of IL-2 m RNA (indicates functional maturation of T lymphocytes) in hindgut T lymphocytes was delayed relatively to that expressed by T lymphocytes in the early fed chicks. It has been observed that thymus is very sensitive to the duration of feed deprivation, energy or amino acid deficiencies, which cause a rapid decline in cellularity and weight (Klasing, 2007). Longer duration of nutrient deprivation may result in fewer CD4+ T cells lowering IgG production and fewer CD8+ cells results in diminished delayed-type of hypersensitivity (Glick *et al.,* 1981).

## Early *post hatch* nutrition and immune response

Nutrients are the major decisive factors that determine the expression of genetic potential of birds in terms of growth and immunity (Katanbaf *et al.,* 1988; Klasing and Barnes, 1988). Nutrients influence the magnitude of antibody responses and nature of responses (Latshaw, 1991). Early feed restriction in poultry has been reported to influence cellular and humoral response to sheep red blood cells (Klasing, 1988). Bhanja (2008) reported



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that feed deprivation beyond 24 hours has a negative effect on both cellular and humoral immune response of broiler chickens. In another study, Kadam *et al.* (2009) showed that delay in *post hatch* feeding by 48 hours resulted in poor haemagglutination titre (HI) (a measure of humoral immune response) to Newcastle disease (ND) vaccine at 14$^{th}$ and 21$^{st}$ day of age. A study was conducted on the effect of early *post hatch* feed privation on humoral immune response, as measured by antibody titre to sheep red blood cell (SRBC) inoculation and ND vaccine during the third week of age in broilers (Panda *et al.,* 2010). Antibody titre (both SRBC and ND) was significantly higher in the chicks with access to feed immediately or after 12 h *post hatch*, compared to those deprived of feed for either 24 or 48 hours. Similarly, the lymphocyte proliferation ratio (LPR) was decreased significantly in the birds deprived of feed for 24 and 48 hours compared to those with access to feed immediately or placement within 12 hours period.

Generally, antibody responses are generated in two sites in the gut, namely the cloacal bursa and the hindgut. Bar Shira *et al.* (2005) reported impaired antibody response (both systemically and locally in the gut) to a rectally administered antigen in the hatchlings that were deprived of feed for 72 hours after hatching. The decrease in antibody production by intestinal cells was not only an indication of impaired local response, but demonstrated decreased migration of plasma cells generated elsewhere (Von Andrian and Mempel, 2003). Nnadi *et al.* (2010) reported that early feeding significantly improved the antibody titre (HI titre) to ND vaccine (at 21 and 27 days of age) compared to cockerel chicks fasted for 72 hours. Similarly, Rammouz *et al.* (2011) observed significantly lower antibody response against ND and Infectious Bronchitis virus in the chicks that received feed early, compared to 12 hour delays, at 11 days of age. This higher immunity in the immediately fed chicks could be due to early availability of immunoglobulin due to faster utilisation of yolk sac and early developmental and functional maturation of the gut immune system (Nathan *et al.,* 1977; Dibner *et al.,* 1998; Panda *et al.,* 2008).

## Mechanisms of immune development

Three mechanisms have been proposed by Dibner *et al.* (1998) for the effect of oral nutrition on the immune system of hatchlings. Firstly, early nutrition provides substrates that may otherwise be limited. Secondly, feeding may influence endogenous levels of hormones or other immunomodulators and thirdly, the presence of antigens (feed) in the gastrointestinal tract is necessary to trigger full differentiation of the primary immune cells, particularly the B lymphocytes. The complete differentiation of B lymphocytes is essential for the eventual development of secondary immune structures such as germinal centres or caecal tonsils, along with the associated ability to respond to a vaccine with the development of immune memory. Another proposed mechanism of action is that early nutrition results in faster development of mucosal immune system (hind gut GALT) in chickens (Bar Shira *et al.,* 2005). Gut development occurs in concert with the development of GALT, and early feeding results in rapid functional and structural development of the gut along with GALT (Thompson *et al.,* 1996; Geyra *et al.,* 2001).

## Challenge models

Early feeding is reported to improve bird performance in disease challenged conditions (Dibner *et al.,* 1998). Using commercial coccidiosis vaccines (challenged orally on the 14 day) to evaluate disease resistance, the above researchers reported poor weight gain and



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FCR at 18 days of age in the broiler chicks deprived of feed for initial 24 hours of hatch compared to those fed immediately after hatch. The above findings suggest that if chicks are given the optimum nutrient formulation immediately after hatch, they respond to the physiological and environmental challenges in a better way. A similar observation in turkey poults exposed to a challenge model for poultry enteritis and mortality syndrome was observed (Edens *et al.,* 1998).

## Conclusions

The development of the avian immune system, though initiated very early during embryogenesis, is incomplete at hatch. The residual yolk-sac does not provide the required level of nutrients to fully support immune system maturation during the early hatchling period. Early *post hatch* feed deprivation not only affected the growth of immune organs like the bursa and spleen, but also reduced lymphocyte proliferation, thereby restricting the function of the immune system. The mucosal immune system in particular is more affected due to early feed deprivation, and needed almost a two week period for the chick to recover. This vulnerability should be considered seriously in situations where hatchlings are in transit for extended periods during transportation from hatchery to farm. Narrowing down the holding period in hatchlings could alleviate most of the problems associated with the early *post hatch* nutrient deprivation and allows the bird to perform optimally.

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