Benefits of Early Nutrition on Neonatal Poultry

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INTRODUCTION

In today's global business environment focusing on continuous improvement is a key to obtaining and keeping a competitive advantage. The poultry industry is an area where research and technology innovations have served to make poultry production one of the most technologically advanced protein production systems in the world. However, continued improvement requires a re-examination of current practice in order to develop the new tools needed to maintain this advantage. A fundamental principle of the practice of nutrition is a clear determination of the essential nutrient requirements for the most cost-effective feeding system. The nutrition of hatchling poultry has not been systematically studied but this area could represent the next important tool for the continued improvement in poultry production.

The current approach to the nutritional needs of hatchling poultry can be generally described in two ways. There are those who believe that hatchlings must receive feed and water within several hours of processing in the hatchery. The solution in this case is to create an infrastructure in which all grow out facilities are within a couple hours drive of the hatchery. In this setting the overriding concern is that birds be placed quickly despite other management challenges of brooding light-weight birds, getting all birds to eat and drink quickly and the potential for early disease challenges. The U.S. and European broiler industries are typically in this category, but there are other ways to approach this problem.

The placement of some poultry is delayed from 24 hours up to 72 hours. This would include those birds that require substantial transit time such as day of age parent breeding stock and to some extent commercial layers, ducks and turkeys. Often the reason for long delivery times is simply a matter of distance to the customers grow out facilities. In the case of U.S. turkeys, up to 80% of poults are held overnight in the hatchery[1]. The explanation for this practice is usually that it gives poults time to recover from processing or that poults will simply go to feed and water more quickly when treated in this manner. Whether any delay in hatchling placement is routinely practiced or not, the measure of a successful placement is based on maintaining a certain minimum level of mortality over the first several days after placement, not optimal growth.

In neither of these cases do the specific nutritional needs of the hatchling come into consideration. In the first case, getting them to dry feed and water is the key; however, one can certainly make the case that a starter diet is more reflective of the nutritional needs of the 14-21 day old bird than the day old bird. When placement is delayed any discussion of nutrition and feeding will automatically be answered with the assertion that the yolk sac provides all the nutrition the young bird requires for the first 48-72 hours of life so they don’t need to be fed. This paper will challenge the basic underlying assumptions of hatchling nutrition as they are generally considered today. The following statements will be discussed.

- The optimum use of residual yolk is not to deliver nutrients to the hatchling unless its survival is at stake.

- Carbohydrates, not lipids, are the hatchlings primary energy source.
- The nutritional needs of the hatchling are different than the rest of the starter period.
- Delaying balanced nutrition for even 24 hours can retard growth and development of the gastrointestinal and immune systems and impact growth long after birds are placed in grow out facilities.

Functions of Residual Yolk

It is true that the yolk provides the nutrients necessary for the development of the embryo while inside the egg. It is also true that the bird internalizes the residual yolk just prior to hatching. So it certainly makes sense that the contents of the yolk ought to serve as a ready reservoir of nutrients for the neonatal hatchling, especially if you consider that it contains 50% water, 25% protein and 25% lipid. However, a closer look at the actual protein and lipid components indicate that using the residual yolk protein as an amino acid source or its lipid as an energy source is a false economy. The protein and lipid remaining at hatching can play a much more specific role if not required for nutrition. Certainly neonatal poultry have the ability to search out food within hours of hatching. In addition, a long history of domestication has further increased the likelihood that residual yolk will not be needed in hatchling poultry to provide energy for survival. Over time, this has allowed the residual components of yolk to become an important means of providing the neonate not so much with nutrients as with macromolecules that it is unable to synthesize for itself. Data supporting this hypothesis are clear regarding residual yolk protein.

Although the protein fraction is partly constitutive (albumen), a large fraction of the egg protein consists of antibodies the hen was making during the immune responses it experienced at the time the egg was laid[2]. This is clearly selective because evolutionarily, the chick would hatch into the hen's environment and would presumably benefit by having a supply of antibodies specific for the current disease challenge. It is important to note that, during incubation, the developing embryo must be supplied with amino acids, but these do not come from maternal antibodies. Under normal circumstances, maternal antibody is not digested during the incubation process, leaving these immunoglobulins intact and fully functional at the time of hatch[3]. This argues strongly that these antibodies, i.e. the residual yolk protein, are not intended to be digested into their building blocks and just used as a source of amino acids, at least not unless the bird is under threat of starvation. In optimal or even normal conditions, these specific proteins would be best used for passive immunity until the neonate could mount an effective immune response.

A similar argument can be made that residual yolk lipid and the hydrophobic material associated with it are best used for growth and not simply as an energy source for maintenance. Recently, a number of studies have shown a regulatory or second messenger role for particular fatty acids and compounds derived from them. Since the fatty acid composition of yolk lipid is in part dependent on the hen's fatty acid profile at the time of lay, these fatty acids, if spared, may well influence the partitioning of nutrients or the pace of development of a particular organ system. Indeed, recent literature has documented elevated levels of fat-soluble vitamins and ω-3-fatty acids in chicks from hens fed diets rich in the corresponding compound[4]. This could, for example, influence the hatchlings early inflammatory response. The role of fatty acids as second messengers is not fully understood but immunoregulatory compounds such as eicosanoids derived from yolk fatty acids could influence the development and character of the early immune response.

In addition to this potential regulatory role, yolk lipids may also serve a structural purpose. Many of the residual yolk lipids, specifically the cholesterol and phospholipids, are important components of cell membranes. One of the fastest growing tissues in the neonate is the central nervous system. This growth requires very high amounts of structural lipid for the myelin sheath that surrounds the axons of large neurons. It would not be selective to use such structural lipids for energy unless survival is at issue.

Energy Metabolism after Hatching

A dominant feature of energy metabolism in the neonatal bird is the abrupt change from a blood-borne, lipid-based metabolism to an oral, carbohydrate-based system upon hatch[5]. The digestive enzyme and gut transport systems for carbohydrate utilization are more developed at the time of hatch than those for lipid utilization and neonatal birds readily digest complex starch[6]. In contrast, lipid digestibility is significantly poorer in chicks and poults especially when fed saturated fats[7]. This indicates that the bird is primed at hatching to provide for its energy needs through oral consumption of carbohydrates.

If oral carbohydrates were not available at hatching, using the entire contents of residual yolk for energy would not meet the hatchlings minimum needs. The maintenance energy requirement for a hatchling broiler chick for the first 24 hrs has been estimated at approximately 11 kcal (112 kcal $W^{.75}$)[8]. Assuming the amount of residual yolk released in the first 24 hours were used solely for energy with 100% efficiency (i.e. gross energy = metabolizable energy), the total potential is only 9.4 kcal. Thus, without additional nutritional supplies the chick is clearly in energy deficit and will invariably lose weight. Research has shown that provision of oral nutrients actually increases the rate of residual yolk utilization, perhaps by initiating growth or causing increases in gut motility and yolk emptying through the yolk stalk[9]. While oral supplementation of residual yolk to deutectomized chicks provided some benefit compared to deutectomized controls, no additional benefit was observed when intact chicks were orally supplemented with residual yolk[10]. We postulate that the value of residual yolk lipid for cell regulation and for structural components is greater than its value as an energy source and that providing supplemental nutrition after hatching can help preserve it for that use.

Nutrient Requirement of Hatchlings

The nutrient requirements of neonatal hatchling broiler chicks were examined by feeding a variety of nutrient formulations as the sole source of feed and water over the first two days immediately following hatching. Formulations consisted of a 25% dry matter semi-solid containing the following nutrient ranges: fat 0-10%, protein 0-15% and carbohydrate 0-20% of the total formulation. Once the nutrient ranges had been determined, ingredients were chosen from which to assemble the hydrated mixture. For this experiment, the carbohydrate was cornstarch, the fat was soybean oil and the protein source was porcine plasma.

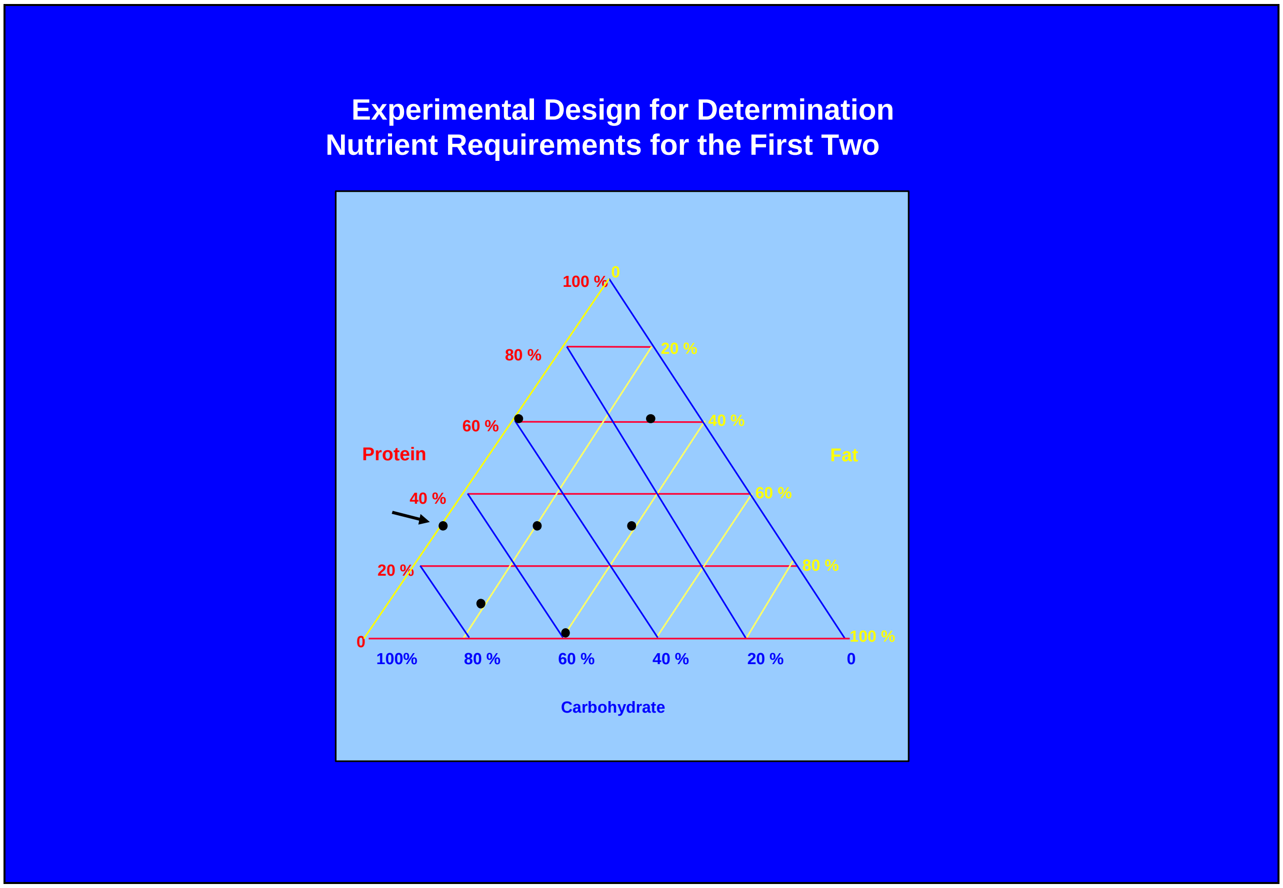

Figure 1

Figure 1 shows the nutrient composition of the experimental treatments. Each black dot represents a different nutrient formulation (dry matter basis) whose composition can be determined by following the appropriate line to the nutrient of interest. For example, at the arrow is a dot representing a formulation whose dry matter consists of 30% protein, 0% fat, and 70% carbohydrate. Mixture design methodology was used to determine the appropriate nutrient combinations required to best define the response surface[11].

Following 2 days of consumption of the various mixtures all birds were given the same corn and soybean meal starter and grower diets from day 2-21 and day 22-41, respectively. Birds and feed were weighed on days 7, 21 and 41. Response surface methodology was used to create a response model[12] to determine the optimal combination of the nutritional components using bodyweight corrected feed conversion as the response measure (Figure 2).

The red area represents the best feed conversion with each layer of color representing .01 units poorer in body weight corrected feed to gain as shown in the legend. Results from this study show that the optimum nutritional mixture for a broiler chick the first 48 hours after hatch consists of dry matter with a nutrient balance of about 50% protein and 50% carbohydrate with no added fat. All mixtures with additional fat resulted in a substantially negative impact on performance. The white dot present in Figure 2 indicates the nutrient makeup of a common starter diet and demonstrates the degree of difference between the optimum nutrient mixture for the neonate and what is commonly supplied in the diet. It is apparent that providing the optimum nutrition in the first 48 hours can have a substantial impact on final bird performance.

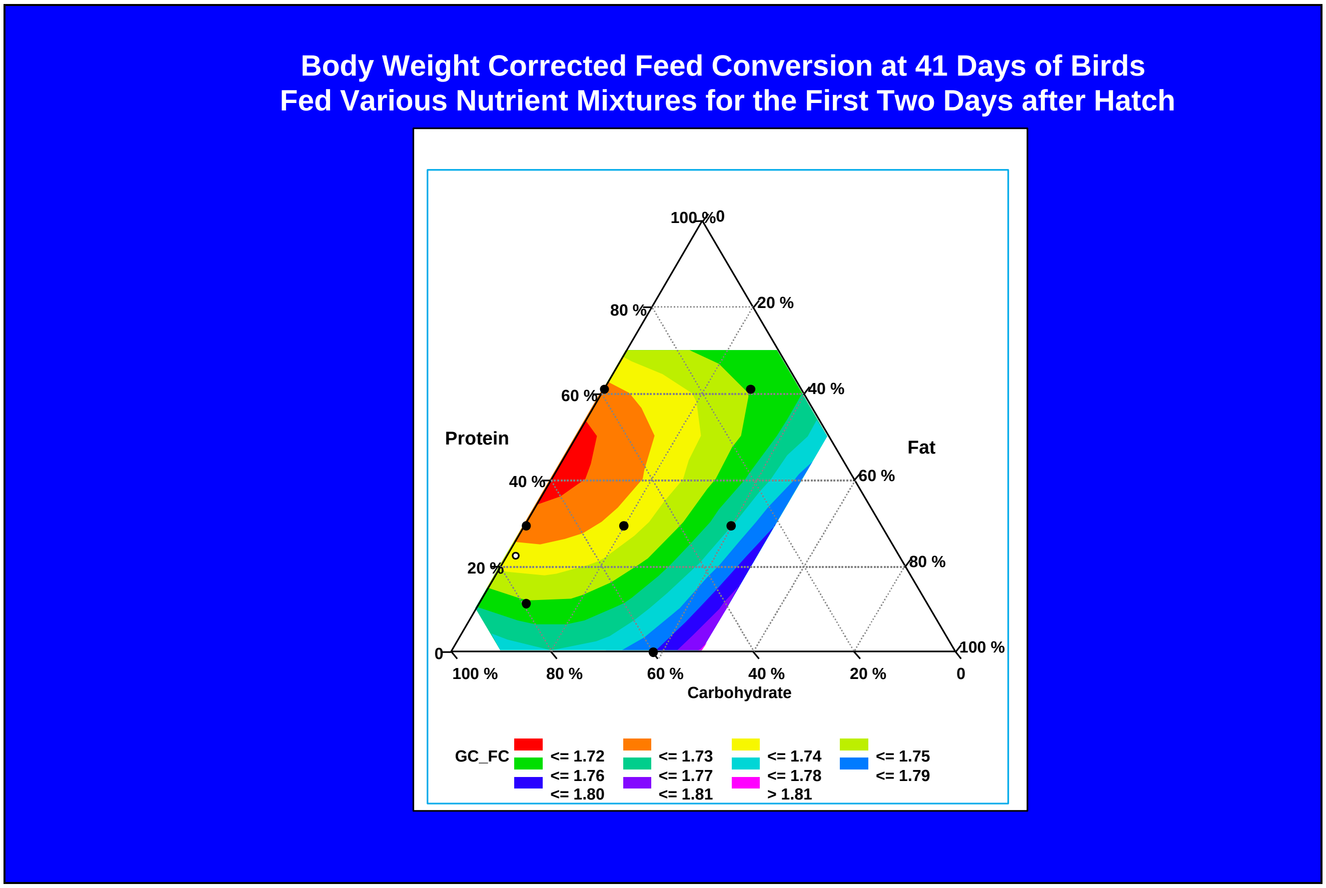

Figure 2

Systems Requiring Oral Intake for Optimum Early Development

All post hatch development requires nutrients, but some systems are uniquely and specifically dependent on oral intake. Oral intake is essential for the provision of amino acids and carbohydrates in order to spare the macromolecules present in residual yolk as discussed above. There are, however, other critical systems whose ontogenetic development depends on oral intake. First, gut development, not only villus growth but also enterocyte differentiation depends in part on oral intake[13]. Intestinal motility, nutrient transport systems, pancreatic enzyme secretion and bile salt synthesis are all examples of systems which are partly developed at hatch but whose development to adult levels requires oral intake[14]. A particularly critical component of early gut development that is dependent on oral intake is the establishment of a desirable microflora before the birds arrive at the production house. Early exposure to a defined microbial population is already widely practiced.

Rapid growth of a healthy bird is the ultimate goal. Unless the gastrointestinal supply system can quickly mature and provide the necessary substrates, demand organs such as muscle never do catch up. Nutrients must be available quickly and at levels well beyond those required for maintenance. Thus, early provision of nutrients affects not only immediate survival and disease resistance but also the ultimate attainment of genetic potential[15].

Another system requiring oral intake for full and rapid development is the immune system, particularly the mucosal immune system[16]. Antigens, both directly from the intestine and secondarily from the cloaca, reach the bursa and are engulfed by the bursal epithelium[17]. (In fact, the bursa can be considered as much a part of the gastrointestinal system as of the immune system: For example, the microscopic structure of the bursa strongly resembles an isolated, large cluster of elongated villi whose lamina propria is occupied by very large lymphoid follicles.) In the hatchling, the humoral immune system consists of only IgM bearing lymphocytes, primarily found in the bursa itself[18]. Preventing contact between the bursal lymphocytes and environmental antigens can seriously retard the development of other isotypes and of antibody diversity[19]. Indeed, in the absence of oral intake and in gnotobiotic (germ-free) birds, lymphocytes fail to colonize mucosal sites such as the cecal tonsils[20]. These important secondary immune organs remain undeveloped.

Evolutionarily, most environmental antigens were introduced to the system by inhalation and ingestion but in the modern hatchling additional antigen is introduced by deliberate administration with vaccines to stimulate an immune response. An effective vaccine response, i.e. one that generates immune memory, requires mature B and T lymphocytes[21]. Timely maturation of the immune system requires a large amount of the cell proliferation (and cell death) for which glucose metabolism is the energy source. Intake of carbohydrates is necessary to supply substrates for lymphocyte energy metabolism and also for the next round of division.

Figure 3 shows the effects of early fasting on bursa weight. In this study broiler

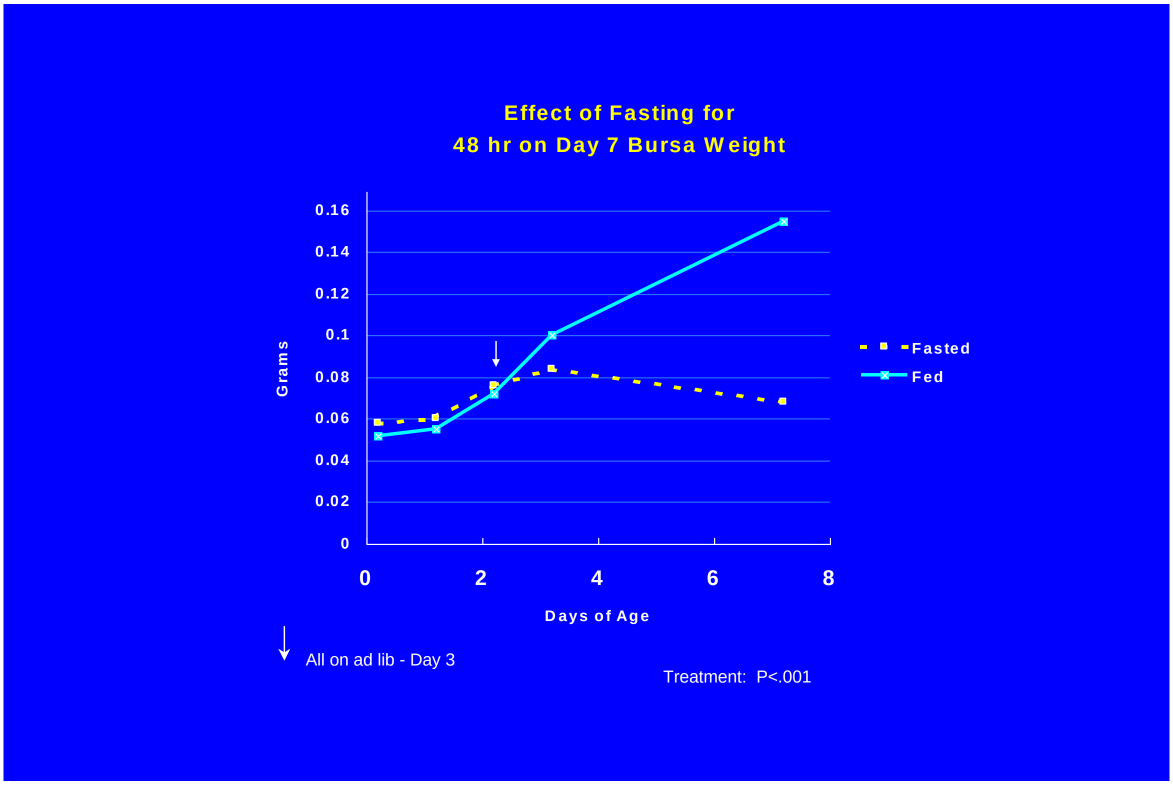

Figure 3

chicks were fasted or fed a high moisture semi-solid for the first 3 days of life. Thereafter, all birds were placed on the same corn soy starter diet ad lib. Figure 3 shows that bursa weight in fasted birds was very slow to respond to feeding and remained significantly lighter than in the fed controls through day 21 (data not shown). This affects not just the bursa itself but the secondary immune tissue populated by bursal lymphocytes during the first weeks of the hatchling's life.

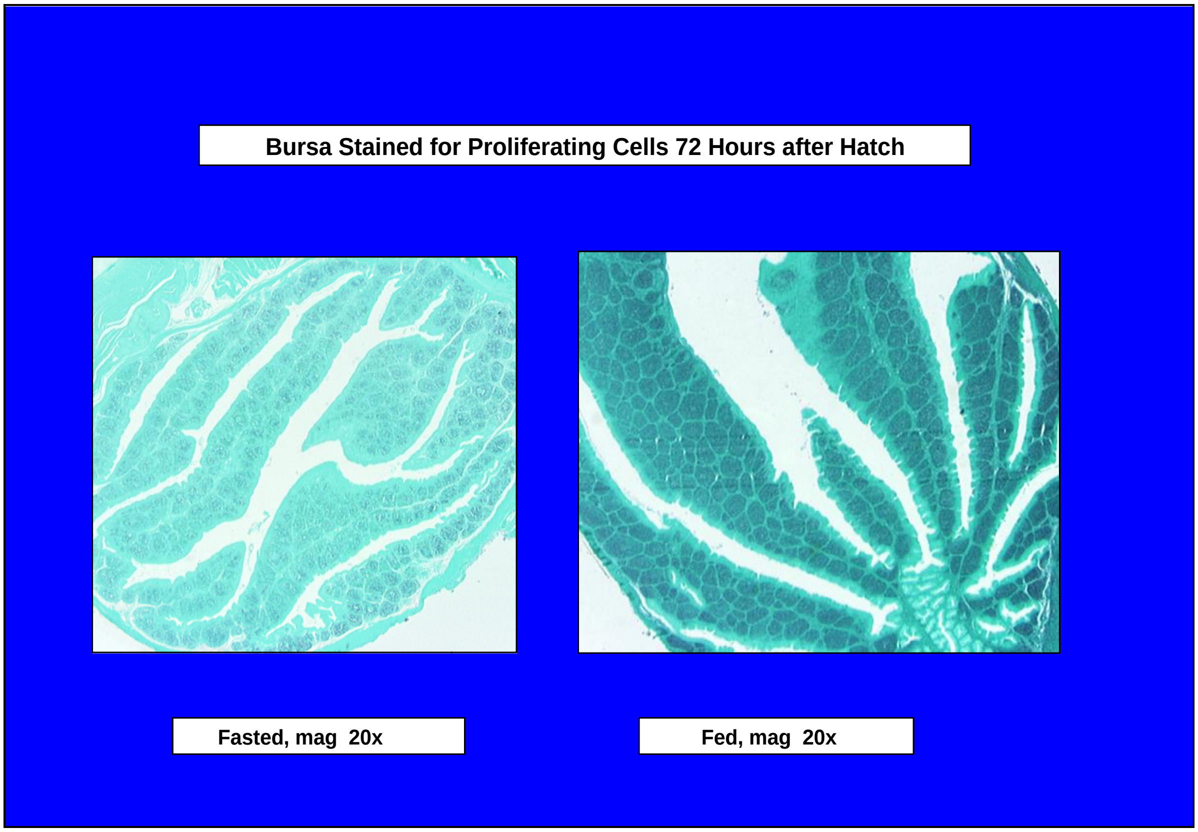

Figure 4

Figure 4 is a tissue slide of bursa from this study stained with bromodeoxyuridine, which highlights proliferating cells by binding to nucleic acids[22] during DNA synthesis. Figure 4 shows evidence that the lighter bursal weight is due to differences in lymphocyte proliferation in the fasted birds compared to those that have been fed. Other researchers have observed a lower antibody titer in fasted birds vaccinated for infectious bursal disease[23]

While it is obvious that provision of substrates is essential for the expansion of the response repertoire, immune development is also suppressed for another reason when the hatchling is not fed. Fasting stimulates secretion of corticosteroids which are powerful inhibitors of immune cell proliferation, including that required for the hatchling to response to a vaccine[24]. It makes no more sense to vaccinate a fasted animal than to hyperimmunize a hen and then use the resulting maternal antibodies as a source of amino acids instead of passive immunity.

SUMMARY

Research is underway to further understand the many ramifications and potential benefits associated with supplying neonatal poultry with optimum nutrition prior to placement in the growing facilities. The ideal situation for the hatchling may well be a period of relative isolation before placement for recovery from processing, replenishment of glycogen stores, distribution of maternal immunoglobulins to mucosal sites, establishment of a defined microflora, and initiation of vaccine responses. Relying solely on residual yolk as a nutrient source in the first 24-72 hours would appear to waste valuable resources and produce less than optimum production results. Satisfaction of nutrient requirements specific to the postnatal period have been shown to stimulate development of the gastrointestinal and immune systems and has been shown to result in performance benefits well after the neonatal feeding period. Thus, pProviding optimum nutrition in this important neonatal period could be the next tool for continued efficiency enhancements in poultry production.

[1] Grimes, J.L. and S. L. Pardue. 1966. A Survey of commercial turkey hatcheries in the United States. J. Appl. Poultry Res. 5:231-238.

[2] Losch, U., I. Schranner, R. Wanke and L. Jurgens. 1986. The chicken egg, an antibody source. J. Vet. Med. 33:609-619.; Wiedemann, V. , R. Kuhlmann, P. Schmidt, W. Erhardt and U. Losch, 1990. Chicken egg antibodies for prophylaxis and therapy of infectious intestinal diseases. J. Vet. Med. 37:163-172.

[3] Brierley, J. and W.A. Hemmings, 1956. The selective transport of antibodies from the yolk sac to the circulation of the chick. J. Embryol. Exp. Morph. 4:34-41.

[4]Cherian, G. and J. S. Sim, 1997. Egg yolk polyunsaturated fatty acids and vitamin E content alters the tocopherol status of hatched chicks. Poultry Sci. 76:1753-1759.

[5] Latour, M. A., E. D. Peebles, C. R. Boyle, J. D. Brake, and T. F. Kellogg. 1995. Changes in serum lipid, lipoprotein and corticosterone concentrations during neonatal chick development. Biol Neonate 67:381-386.

[6]Best, E. E. 1966. The changes of some blood constituents during the initial post-hatching period in chickens. Brit. Poult. Sci. 7:23; Moran, Jr., E. T. 1985. Digestion and absorption of carbohydrates in fowl through perinatal development. J. Nutr. 115:665-674; Ikeno, T., H. Sakamoto, K. Ikeno, and K. Niwa. 1992. The embryo pancreas is a source of increased yolk amylase in the fertilized eggs of domestic fowls. Exp. Anim. 41:19-23; Noy, Y. and D. Sklan, 1995. Digestion and absorption In the young chick. Poultry Sci. 74:366-373.

[7]Polin, D., T.L. Wing, P. Ki, and K.E. Pell. 1980. The effect of bile acids and lipase on absorption of tallow in young chicks. Poultry Sci. 59:2738-2743; Wiseman, J., and F. Salvador, 1991. The influence of free fatty acid content and degree of saturation on the apparent metabolizable energy value of fats fed to broilers. Poultry Sci. 70:573-582; Blanch, A., A. C. Barroeta, M. D. Baucells, and F. Puchal. 1995. The nutritive value of dietary fats in relation to their chemical composition. Apparent fat availability and metabolizable energy in two-week-old chicks. Poultry Sci. 74:1335-1340;;

[8] Akiba, Y. and H. Murakami. 1995. Partitioning energy and protein during early growth of broiler chicks and contribution of vitteline residue. World's Poultry Science Association Proceedings: 10th Eurpoean Symposium On Poultry Nutrition. October 15-19 1995.

[9] Chamblee, T. N., J. D. Brake, C. D. Schultz, and J. P. Thaxton. 1992. Yolk sac absorption and initiation of growth in broilers. Poultry Sci. 71:1811-1816; Noy, Y., Z. Uni and D. Sklan. 1996. Routes of yolk utilization in the newly hatched chick. Poultry Sci. 75S:13. (Abstr.); Sulaiman, A., E. D. Peebles, T. Pansky, T. F. Kellogg, W. R. Maslin and R. W. Keirs. 1996. Histological evidence for a role of the yolk stalk in gut absorption of yolk in the post-hatch broiler chick. Poultry Sci. 75S:48. (Abstr.)

[10] Nitsan, Z. 1995. The development of the digestive tract in posthatched chicks. World's Poultry Science Association Proceedings: 10th Eurpoean Symposium On Poultry Nutrition. October 15-19 1995.

[11] Cornell, J. 1990. Experiments with mixtures. John Wiley & Sons, NY, NY.

[12] Box, G. & J. Drater. 1987. Empirical model building and response surface. John Wiley & Sons, NY, NY.

[13] . Baranyiova, E. 1972b. Influence of deutectomy, food intake and fasting on the digestive tract dimensions in chickens after hatching. Acta Vet. Brno 41:373-384; Cook, R.H., and F.H. Bird, 1973. Duodenal villus area and epithelial cellular migration in conventional and germ-free chicks. Poultry Sci. 52:2276-2280; Baranyiova, E., and J. Holman. 1976. Morphological changes in the intestinal wall in fed and fasted chickens in the first week after hatching. Acta Vet. Brno 45:151-158.

[14] Buddington, R. K., and J. M. Diamond. 1989. Ontogenetic development of intestinal nutrient transporters. Annu. Rev. Physiol. 51:601-619. Buddington, R. K. 1992. Intestinal nutrient transport during ontogeny of vertebrates. Am. J. Physiol. 263(Regulatory Integrative Comp. Physiol. 32): R503-509.

[15] Fredde, M. R., P. E. Waible, and R.E. Burger, 1960. Factors affecting the absorbability of certain dietary fats in the chick. J. Nutr. 70:447-452; Gomez, M. X., and D. Polin. 1976. The use of bile salts to improve absorption of tallow in chicks, one to three weeks of age. Poultry Sci. 55:2189-2195; Nir, I., Z. Nitsan, and B. Ben Avraham. 1988. Development of the intestine, digestive enzymes and internal organs of the newly hatched chick. Pp 861-864 *in*: Proceedings XVIII World's Poultry Congress, Nagoya, Japan; Noy, Y. and D. Sklan. 1995. Digestion and absorption in the young chick. Poultry Sci. 74:366-373; Palo, P.E., J. L. Sell, F. J. Piquer, L. Vilaseca, and M. F. Soto-Salanova. 1995. Effect of early nutrient restriction on broiler chickens. 2. Performance and digestive enzyme activities. Poultry Sci. 74:1470-1483. Uni, Z., S. Ganot, and D. Sklan, 1998. Posthatch development of mucosal function in the broiler small intestine. Poultry Sci. 77:75-82.

[16] Schaffner, T., J. Mueller, M. W. Hess, H. Cottier, B. Sordat, and C. Ropke, 1974. The bursa of Fabricius: A central organ providing for contact between the lymphoid system and intestinal content. Cellular Immunology 13:304-312.

[17] Bockman, D.E. and M.D. Cooper. 1973. Pinocytosis by epithelium associated with lymphoid follicles in the bursa of Fabricius, appendix and Peyer's patches. An electron microscopic study. Am J. Anat. 136:455-478.

[18] Lydyard, P.M., C.E. Grossi and M.D. Cooper. 1976. Ontogeny of B cells in the chicken. I. Sequential development of clonal diversity in the fursa. J. Exp. Med. 144:79-97.

[19] Honjo, K., T. Hagiwara, K. Itoh, E. Takahashi, and Y. Hirota. 1993. Immunohistochemcial analysis of tissue distribution of B and T cells in germfree and conventional chickens. J. Vet. Med. Sci. 55:1031-1034.

[20] Hegde, S. N., B. A. Rolls, A. Turvey and M.E. Coates. 1982. Influence of gut microflora on the lymphoid tissue of the chicken (Gallus domesticus) and Japanese quail (Coturnix coturnix japonica). Comp. Biochem. Physiol. 72a:205-209.

[21] Perey, D.Y., and J. Bienenstock. 1973. Effects of bursectomy and thymectomy on ontogeny of fowl IgA, IgG, and IgM. J. Immun. 111: 633-637.

[22] Kitchell, M.L. and J.J. Dibner (1989) Rapid detection of proliferating cells using microwave fixation and a monoclonal antibody to bromodeoxyuridine. J. Histotechnology 12:101-103.

[23] Casteel, E.T., J. L. Wilson, R. J. Buhr, and J. E. Sander. 1994. The influence of extended posthatch holding time and placement density on broiler performance. Poultry Sci. 73:1679-1684.

[24] Constantin, N., J. Raszyk, A. Holub, and V. Kotrbacek. 1977. Effect of adrenocorticotrophic hormone and starvation on adrenocortical function in chickens during the early posthatching period. Acta Vet. Brno 46:87-93; Siegel, H. S. 1980. Physiological stress in birds. Bioscience 30:529-534.