Review

Early intestinal growth and development in poultry

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ABSTRACT While there are many accepted "facts" within the field of poultry science that are in truth still open for discussion, there is little debate with respect to the tremendous genetic progress that has been made with commercial broilers and turkeys (Havenstein et al., 2003, 2007). When one considers the changes in carcass development in poultry meat strains, these genetic "improvements" have not always been accompanied by correlated changes in other physiological systems and this can predispose some birds to developmental anomalies (i.e. ascites; Pavlidis et al., 2007; Wideman et al., 2013). Over the last decade, there has been increased interest in intestinal growth/health as poultry nutritionists have attempted to adopt new approaches to deal with the broader changes in the overall nutrition landscape. This landscape includes not only the aforementioned genetic changes but also a raft of governmental policies that have focused attention on the environment (phosphorus and nitrogen excretion), consumer pressure on the use of antibiotics, and renewable biofuels with its consequent effects on ingredient costs. Intestinal morphology has become a common research tool for assessing nutritional effects on the intestine but it is only one metric among many that can be used and histological results can often be interpreted in a variety of ways. This study will address the broader body of research on intestinal growth and development in commercial poultry and will attempt to integrate the topics of the intestinal: microbial interface and the role of the intestine as an immune tissue under the broad umbrella of intestinal physiology.

Key words: intestine, digestion, goblet cells, mucin, innate immunity

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INTRODUCTION

Overview

The significant changes in the growth of commercial poultry have focused interest on intestinal development from two related but different directions. Commercial broilers are largely grown to somewhat fixed market weights at ever decreasing ages, thus there is the recognition that the first week(s) posthatch represents an increasing proportion of the total growing period and thus an important period relative to optimizing digestive efficiency and performance (Willemsen et al., 2008). One objective of this report is to discuss intestinal growth and development with an emphasis on those factors effecting early physiological function. This portion of the paper was intentional in its inclusion of older literature which laid the foundation for the more basic research of the last 20 years. The term "intestinal health" has become an increasingly popular term, primarily as it refers to the increasing frequency of secondary bacterial challenges and the onset of such pathologies as necrotic enteritis (Forder et al., 2012). In this regard, this paper will integrate the synergistic role that microbial colonization plays in early intestinal development together with its role in facilitating innate immunity, particularly during the first week posthatch (Bar-Shira et al., 2003).

INTESTINAL GROWTH AND DEVELOPMENT

From a strictly developmental standpoint, Lilja (1983) put forth the concept that during the early posthatch growth period, there is a hierarchy for available nutrients that is divided among "supply" and "demand" tissues. The "demand" tissues are largely users of energy and protein (i.e. skeleton, muscle, adipose) while the "supply" tissues would include the respiratory and cardiovascular systems and the intestine. Two excellent reviews on the biological relationship between intestinal development and growth in altricial versus precocial avian species (poultry) can be

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found in Konarzewski et al. (1989, 1990). Immediately posthatch, the proportional growth of the small intestine is greater than that of BW and peaks within the same approximate age range for both poults and chicks (6 to 10 d; Katanbaf et al., 1988; Sklan, 2001). Dror et al. (1977) compared the relative growth of the various components of the digestive system in light and heavy breed chickens. They reported that the relative weight of the duodenum peaked at 3 d posthatch and was highest among the 3 intestinal segments measured. This was followed by a subsequent decline in relative intestinal growth through 21 d in both breeds. In a follow-up study, forced-feeding doubled the weight of the duodenum in light breed chicks (1.71 vs 3.52 g) compared with an approximate 30% increase in heavy breed chicks (2.47 vs 3.33 g; Nir et al., 1978). The respective differences in the small intestine were + 56 and +21% for the light and heavy breeds, respectively. One interpretation of the data could be that the heavy breed chicks had already adapted to increased feed intake as a correlated response to selection for BW. Newcombe and Summers (1984) compared the length and relative weight of the small intestine in leghorn and broiler chicks fed diets with different energy densities. In both breed types, the relative weight of the small intestine was increased in the chicks fed the low versus high energy diets and almost twice as high in the leghorn versus broiler chicks. Mitchell and Smith (1991) compared the intestinal components in highly selected, relaxed, and unselected broilers. They reported that the relative length of the duodenum, jejunum and ileum were similar in all 3 strains but relative weights were decreased due to selection. Villus area, however, was significantly increased in the highly selected line and this was in line with a previous study by Smith et al. (1990) who reported that enterocyte migration rates within the villus were 40% greater in intestinal samples from the highly selected chicks when compared with the relaxed and unselected chicks. In later studies by Uni et al. (1995a,b), villus area was observed to be greater in broiler versus layer embryos beginning at 14 d incubation and continued through 7 d posthatch. These authors also reported that the number of enterocytes per villus increased with age and was greater in the broiler embryos/chicks. Dibner et al. (1996) and Iji et al. (2001a) confirmed the latter observations that the first week posthatch was a critical period for overall intestinal growth. In more recent studies, differences in cereal grains (corn, wheat, sorghum) and form of the grain (whole wheat, ground wheat) that resulted in significant performance results was not due to differences in intestinal morphology (Gabriel et al., 2008; Thomas and Ravindran, 2008). In turkey poults, Sell et al. (1991) reported that the relative weight of the small intestine increased 4-fold between hatch and 4 d posthatch and this was supported by Uni et al. (1998b). Applegate et al. (2005) compared commercial turkeys and Pekin ducks, species which have similar egg weights, incubation periods, and hatchling weights. By 3 d posthatch, however, the ducklings were 36% heavier and were almost twice as heavy by 7 d (290 vs 150 g). Of greatest interest relative to this discussion, however, were the significant differences in villus height and crypt depth already established at hatch and the continued increase in this differential at 1 and 3 d age. These differences, albeit in different species, emphasize the importance of intestinal development relative to supporting accelerated growth within the same or different species. It is also important to note that the changes in intestinal morphology and function during the first week posthatch as defined by histological measurements are accompanied by concomitant maturational changes. This maturational process involves the transition from enterocyte proliferation throughout the villus during the late embryonic/early posthatch period to localized proliferation within the crypt as observed in both chicks and poults (Uni et al., 1998a,b; Applegate et al., 1999; Geyra et al., 2001a). Within the last 20 years, there has been considerable research and widespread commercial acceptance of *in ovo* delivery of vaccines. This has generated research interest into the *in ovo* delivery of selected nutrients with the goal of accelerating the process of intestinal growth and development. The published results to date have shown increased posthatch intestinal carbohydrate absorption in poults (Foye et al., 2007), increased brush border carbohydrase activity at hatch and increased BW through 10 d posthatch in chicks (Tako et al., 2004), increased villus surface area at hatch through 3 d posthatch in chicks (Smirnov et al., 2006) and increased expression of selected brush border transporters (Tako et al., 2005).

INTESTINAL DEVELOPMENT–POSTHATCH ENVIRONMENT

The contents of the residual yolk are one source of nutrients for hatchlings and yolk nutrients can be transferred to the hatchling via both the blood and intestine up to 72 h posthatch (Noy et al., 1996). These authors also reported that residual yolk sac uptake was faster in fed versus fasted chicks which supported earlier observations in chicks (Heywang and Jull, 1930) and poults (Moran and Reinhart, 1980). Triglycerides and phospholipids represent a high proportion of residual yolk sac lipids in both chicks and turkeys but only amount to approximately 1 g actual triglyceride at hatch (Noble and Moore, 1964; Donaldson, 1967; Ding et al., 1995; Lilburn, 1998). Hurwitz et al. (1978) estimated that chicks had a maintenance energy requirement of 4.5 kcal/day. Noy and Sklan (1999) calculated that chicks fasted posthatch for 48 h lost 5.3 kcal/d in residual yolk lipid and protein while at the same time had a net increase in intestinal weight. The results from a later study by Noy and Sklan (2001) concluded that the immediate priority for residual yolk nutrients is for intestinal development rather than BW gain per se and

there is a selective hierarchy for fatty acid uptake (oleic acid) over glucose or methionine.

The logistics of commercial incubation is such that there is often a lag time between hatch and initial access to feed. This lag time is the result of normal variability in the hatch window, removal of chicks/poults from the hatchers followed by hatchery processing and delivery to growing facilities. The hatch window can be influenced by many factors including variability in incubation temperature and age of hen. The delivery of poults is of greatest potential concern in the United States because independent turkey hatcheries are often one or more states removed from rearing facilities. The potential effect of this lag period on early intestinal growth is similar in scope to what is observed in swine immediately postweaning (Wijtten et al., 2012). Delayed access to feed posthatch has been used by numerous authors as an experimental model to study the effects of early nutrition (or lack thereof) during this critical neonatal period (Geyra et al., 2001b; Noy et al., 2001). Various studies have addressed the effects of delayed placement on specific aspects of intestinal development (i.e., goblet cells; Uni et al., 2003) and strategies for addressing this issue (Noy and Pinchasov, 1993; Noy and Sklan, 1997; Batal and Parsons, 2002; Kornasio et al., 2011).

The day a chick or poult arrives at the growing farm is Day 1 from a production standpoint and studies comparing immediate or delayed access to feed in chicks or poults have often not included a third treatment corresponding to days postfeeding (**DPF**). For example, in Geyra et al. (2001b), fasted chicks (48 h) weighed significantly less than fed chicks at 4 d posthatch (59 vs 85 g) but not 4 d postfeeding (85 vs 85 g). Their intestinal crypt data supports the BW data in that by 5 to 6 d age, the effects due to fasting are diminished. In a study with poults (Potturi et al., 2005), there were no differences in villus height, width, or crypt depth in poults with immediate or delayed access to feed (48 h) at 1 and 2 DPF but there was, however, a small but significant decline in both these variables at 5 DPF. There were also no treatment effects on proliferating cells per crypt or goblet cell numbers per 100 μm villus. From a practical perspective, Turner et al. (1999a) had used a similar experimental approach as Potturi et al. (2005) and found no differences at 13 DPF in each of two experiments. In a similar study with ducklings that had either immediate or delayed access to feed and water posthatch, there were no differences between treatments in BW or selected aspects of intestinal development or intestinal immunity at 6 d postfeeding (Loundon et al., 2011).

INTESTINAL DEVELOPMENT–MICROBIAL INTERACTIONS

One of the interesting observations by Potturi et al. (2005) was the increased presence of aerobic bacteria within the ileum in poults with delayed access to feed. It has been recognized for many years that the intestinal–bacterial interface is established shortly after hatch or feeding (Shapiro and Sarles, 1949; Naqui et al., 1970) and this is largely the basis for the use of probiotics and the concept of competitive exclusion (Rantalaa and Nurmi, 1973; Nurmi et al., 1992). The establishment of an intestinal microflora population is also essential for early intestinal development (Furuse and Okumura, 1994; Lan et al., 2005). Cook and Bird (1973) reported that duodenal villus area was significantly increased in conventional versus germ-free chicks at 5 d posthatch with large numerical differences also being observed at 1, 2 and 4 d age. These authors studied enterocyte proliferation via radiolabelled thymidine and observed that at 7 d posthatch, conventional chicks had twice the number of radiolabelled nuclei. Rolls et al. (1978) subsequently showed that the rates of enterocyte migration were greater in conventional chicks, was reduced in the upper versus lower intestine, and was not influenced by the inclusion of dietary fiber (wheat bran).

While the literature supports the positive effects of the intestinal microflora on intestinal development, this did not necessarily result in increased growth as germ-free chicks and poults were heavier than conventional birds (Forbes et al., 1958; Forbes and Park, 1959; Coates et al., 1963). There were only small differences in feed passage rate in germ-free versus conventional chicks and no differences in starch digestibility or glucose absorption (Coates, 1973; Coates et al., 1981). Salter and Fulford (1974) reported no effects of the gut microflora on protein digestibility but Kussaibaiti et al. (1982) and Yokota and Coates (1982) reported improved protein digestibility and methionine absorption, respectively, in germ-free chicks. Muramatsu et al. (1987) reported that overall protein synthesis was enhanced in the intestine and liver of conventional chicks and hypothesized that the increased energetic cost of protein synthesis may have decreased the available energy needed for overall BW gain.

As discussed above, the physiological importance of intestinal–microbial interactions has been recognized and accepted for over 50 years and is currently an active area of research given increased consumer perceptions relative to food safety and dietary subtherapeutic antibiotics. Much of the research has focused on understanding the mechanisms of bacterial attachment and modulation of the intestine. The interface between intestinal bacteria and the intestine is a mucous layer that is largely an aqueous layer containing a biological mixture of electrolytes, enzymes, some sloughed cells and glycoproteins called mucins (Satchithanandam et al., 1990). The mucus layer varies in thickness and composition throughout the gut and protects the underlying enterocytes lining the villus from mechanical, enzymatic, and chemical challenges in addition to being a source of intestinal lubrication (Sharma and Schumacher, 1995). Mucin proteins are the backbone of mucus and they are a heterogeneous family of proteins coded by the *MUC* gene family. Individual mucins contain a central glycosylated region composed of tandem peptide repeats rich in serine and threonine with O-linked oligosaccharide side chains that may contribute up to 80% of

the mass of the mucin–glycoprotein complex (Kim and Gum, 1995; Perez-Vilar and Hill, 1999). Both the number of tandem repeats in the central region (major domain) and the amino acid profile is a function of unique mucin genes. At the C- and N-terminal regions of the protein are smaller or minor domains that are non-glycosylated and rich in cysteine (Desseyn et al., 2000; Forder et al., 2007). The oligosaccharide side chains vary in both length and composition and this can be influenced by diet (Sharma et al., 1997; Deplancke and Gaskins, 2001). Differences in histochemical staining led to the classification of mucins as either neutral or acidic with the latter category being further delineated into sulfated or sialylated mucin types (Forder et al., 2007). The carbohydrate side chains are hypothesized to be important regulators of bacterial attachment and mucin degradation (Tsai et al., 1991; Macfarlane and Gibson, 1991; Hoskins, 1993; Mack et al., 1999).

Mucins are further characterized as either membrane bound or secretory (gel-forming, non gel-forming; Desseyn et al., 2000) and the predominant gel-forming mucin in the small and large intestine is *MUC2*. Mucin gene expression, packaging of the carbohydrate side chains, and secretion of mucus occurs via goblet cells, a form of enterocyte that primarily originates at the base of the crypt (Cheng and Leblond, 1974). Goblet cells are found throughout the intestinal tract but their proportional contribution to the entire population of enterocytes within a segment of the intestine is variable (Specian and Oliver, 1991). Within the Golgi apparatus of goblet cells, membrane-bound glycosyltransferases facilitate the synthesis of the oligosaccharide chains via the addition of individual monosaccharides whereas sulfate is transferred via a Golgi sulfotransferase (Paulson and Colley, 1989; Brockhausen et al., 1998). Goblet cell mucin secretion occurs by 2 distinct processes, simple exocytosis (baseline secretion) and compound exocytosis. In simple exocytosis, mucin glycoproteins are assembled and stored in granules before they are secreted from the apical surface. Compound exocytosis or the accelerated release of stored granules can occur in response to an inflammatory challenge or endocrine stimulation (Deplancke and Gaskins, 2001). The expression of different mucins as defined by their unique protein backbones and different glycosylation patterns will vary within different segments of the digestive tract and as mentioned previously, *MUC2* is the primary mucin in the small intestine and colon of poultry whereas *MUC5AC* is more largely expressed in the proventriculus (Smirnov et al., 2004).

There are a number of factors which can influence mucin biosynthesis and secretion including de novo bacterial interactions, supplemental probiotics, diet, and poultry management including immediate or delayed access to feed and feed withdrawal. Smirnov et al. (2005) observed a considerable increase in goblet cell area and staining intensity in chicks supplemented with a probiotic when compared with Control chicks or those fed an antibiotic. Forder et al. (2007) compared chicks reared in a low bacterial load environment with conventional chicks and reported no differences in the morphology or number of goblet cells at 4 d posthatch but the conventionally reared chicks had an increased proportion of goblet cells expressing acidic mucin that were sialylated versus sulfated. They hypothesized that the extended period of sulphated mucin expression was an indication of immature goblet cells as proposed by Turck et al. (1993). In a recent publication, Cheled-Shoval et al. (2014) reported that in germ free chicks at 7 d posthatch, the number of goblet cells with both neutral and acidic mucins was reduced with sulfated goblet cells being predominant among the acidic cells. Direct dietary supplementation with probiotics has been reported to positively affect goblet cell numbers and morphology and overall mucosal thickness in poults and broiler chicks (Rahimi et al., 2009; Tsirtsikos et al., 2012).

The role of goblet cells and mucous secretion as a first line of enterocyte protection is well established (Lievin-Le Moal and Servin, 2006). Intestinal goblet cells also secrete a family of small protease resistant peptides called trefoil factors (Wong and Poulsom, 1999; Taupin and Podolsky, 2003). In mammals, Trefoil Factor 3 is often referred to as intestinal trefoil factor and it works synergistically with mucin to enhance the intestinal barrier response to luminal challenges and facilitates epithelial repair (Kjellev, 2009). Jiang et al. (2011) reported that Trefoil Factor 2, which is largely absent in the intestine of humans and rodents, is found in the small intestine of chickens so there may be species and location differences in the expression of specific trefoil factor isoforms. In the latter report, embryonic expression levels were high in the duodenum, jejunem, and ileum followed by a significant decline at hatch. In the jejunem and ileum, however, the significant decline at hatch was followed by a progressive increase through 5 d posthatch.

The regulation of mucin synthesis is in concert with microbial colonization of the gut in mammals and they are important components of the innate immune system (Dharmani et al., 2009; Kim and Ho, 2010; McGuckin et al., 2011). In poultry, the developing microbiome and diet are the 2 primary sources of antigens encountered by gut associated lymphoid tissue (**GALT**) in the first d posthatch (Friedman et al., 2003). Caecal tonsils, Meckel's diverticulum, and the bursa fabricius are well-recognized components of the intestinal GALT in poultry (Muir, 1998). Bar-Shira et al. (2003) reported that the tissues comprising the GALT contained T and B cells that were developmentally immature until approximately 2 wks posthatch. In a subsequent report by Bar-Shira and Friedman (2006), it was speculated that the initial innate immune response consisted of a heterophil response in combination with maternal antibodies followed by a secondary mucosal response after feeding and the presence of microflora in the gut. This supported the previous report by Honjo et al. (1993) that the establishment of a microflora population played a role in IgG and IgA synthesis within the gut as germ-free chicks had diminished

lymphoid follicle development within the cecal tonsils when compared with conventional chicks.

With respect to maternal immunoglobulins, IgY is considered to be the equilvalent of IgG in mammals and is found primarily in the egg yolk and there is little IgA found in the yolks of fresh eggs (Leslie and Chem, 1969; Rose et al., 1974). IgA is deposited primarily in the albumin and Kaspers et al. (1996) showed that there can be a 36 to 44% transfer of IgA to the yolk during the course of incubation although it is not subsequently transferred into the embryonic circulation (Rose and Orlans, 1981). Bar-Shira et al. (2014) suggested that maternal antibodies, particularly IgA (**mIgA**), are a source of intestinal mucosal protection until approximately 7 d posthatch when the mucosal immune system is mature. These authors presented evidence showing that mIgA levels were consistently high from hatch through 3 d posthatch followed by a significant decline through 6 d. There was a subsequent reappearance at 8 d to levels similar to those at hatch. Immunohistochemical staining of goblet cells confirmed the presence of presumed mIgA and not IgY which led to the hypothesis that there needed to be a mechanism for prolonging the half-life of mIgA until the onset of mucosal production. These authors concluded that goblet cells were the reservoir for mIgA that was secreted along with mucin and supports a role of goblet cells in immunity, similar to what has been described in mammals by McDole et al. (2012).

Feed management can also influence mucin/mucous homeostasis, both immediately posthatch and at older ages. Uni et al. (2003) reported that delayed access to feed immediately posthatch (48 h) reduced the total number of enterocytes, particularly in the jejunum, but increased the density of goblet cell staining in both the ileum and jejunum. Smirnov et al. (2004) further reported that fasted chicks (72 h) had decreased BW and villus area but there were no effects on the proportion of goblet cells relative to total cells but the size of goblet cells was significantly greater than in fed chicks. This was accompanied, however, by a decrease in the mucus adherent layer and the authors speculated that this could have been the result of increased mucolysis. Bar Shira et al. (2005) showed that similar to what has been reported for delayed feeding responses on overall intestinal development, GALT development was also delayed in chicks that had feed and water withheld for 72 h posthatch. These authors reported a prolonged reduction in antibody response to rectal immunization (hindgut) with hemocyanin compared with a much shorter term reduction following oral immunization of the foregut with bovine serum albumin.

DIGESTIVE ENZYMES

The first 2 wks posthatch are also critical for both chicks and poults in terms of increased digestive enzyme secretion with the magnitude of the increase varying for individual enzymes (Marchaim and Kulka, 1967). Nitsan et al. (1991) reported that in broiler chicks, the relative growth of the pancreas and intestine both plateaued at approximately 8 d age and the linear decline thereafter through 21 d was much greater for the intestine. In a subsequent study, Nir et al. (1993) reported a similar age associated pattern of enzyme activity (amylase, trypsin, lipase, chymotrypsin; units/kg BW) was observed through the first 15 d post hatch although amylase activity peaked at 10 d. Noy and Sklan (1995) observed greater than 85% starch digestibility by 4 d posthatch and Reisenfeld et al. (1980) reported greater than 95% digestibility by 12 d so it appears that at very young ages, there is sufficient amylase to facilitate near maximal starch utilization. When enzyme activity was expressed per unit of pancreas weight, however, there was a lag phase through 5 to 8 d posthatch for trypsin, lipase, and chymotrypsin activity followed by a sharp linear increase. In turkey poults, the lag phase was slightly longer (14 d) followed by the same linear increase in digestive enzyme activity although lipase activity was somewhat influenced by diet (Krogdahl and Sell, 1989). With respect to actual nutrient absorption, there is a far greater substrate by age interaction in very young versus older chicks or poults and dietary fat is a good example of this. It is well documented in the literature that lipid digestion/absorption improves with age and the magnitude of the improvement is greater for lipid sources with high levels of polyunsaturated versus saturated fatty acids (i.e. vegetable oils, lard versus tallow; Whitehead and Fisher, 1975; Krogdahl, 1985; Sell et al., 1986). This is often interpreted to mean that only low levels of supplemental fat should be incorporated into the starter or prestarter diets when in fact, selected fat sources are still highly digestible and excellent energy sources. Carew et al. (1972) reported that apparent fat absorption from a semipurified diet containing 20% corn oil was 80% from 2 to 7 d age which is similar to the 82 to 89% apparent digestibility range reported by Noy and Sklan (1995) from 4 to 21 d for a practical-type diet (corn–soybean meal) with 6% added soy oil. From a growth response and nutrient utilization standpoint, Sell et al. (1986) reported that 8 and 12% supplemental tallow or A-V fat fed from 0 to 8 wks improved BW when compared to diets containing 0 or 4% of the same fat sources. In the latter report, there was also a significant linear decrease in feed conversion with both sources as supplemental fat was incrementally increased from 4 to 12%. Turner et al. (1999a) also observed a significant improvement in BW at 12 d when poults were fed a high fat (10.8% A-V) versus high carbohydrate diet. In a subsequent study, Turner et al. (1999b) reported that the high fat diet significantly increased feed intake (3 to 5 d, +16.6%; 6 to 8 d, +19.8%; 9 to 11 d, +24.2%) coupled with a concomitant 47 to 59% age associated increase in lipid intake and 7 to 17% increase in determined ME. This is a reminder that while percentage digestibility of key nutrients has a significant age component, it is the actual absorption of key nutrients that is critical to early growth. Iji et al. (2001b) reported that the specific activity of selected brush border enzymes (maltase,

sucrase, amino peptidase) was maximal at 1 day age and subsequent increases with age were simply a function of villus growth together with increased enterocyte number and area. This latter observation suggests that the enterocyte is not a primary limiting factor in nutrient absorption if macro-substrate digestion can be more effectively accomplished.

CONCLUSION

The literature which has contributed to our knowledge base on early intestinal development in poultry has been developed over the last 50 years. In the last 20 years, however, the application of more sophisticated techniques to understanding intestinal physiology and novel approaches to potentially modulating early intestinal development (*in ovo* feeding) has accelerated our interest in the multiple contributions the intestine makes to the establishment of whole animal posthatch homeostasis. This heightened interest has included inputs from the realm of poultry management (immediate versus delayed access to feed), microbiome development (endogenous versus probiotics), and innate immunity (maternal antibodies). The broiler and turkey industries are already encountering constraints on the use of antibiotics (possibly including ionophores), increased reliance on anticoccidial vaccines, shortened and longer production cycles (product dependent), and heightened concerns with food safety. These challenges underscore the continuing need for novel research ideas combined with the integration of data that is already in the literature to facilitating a healthy and robust intestine.

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