ABSTRACT

BARASCH, ILANA BETH. The Effects of Yeast-Derived Feed Additives and Their Potential as Antibiotic Alternative Feed Additives on the Growth Performance of Turkey Toms Raised to Market Age. (Under the direction of P. R. Ferket).

The purpose of this research was to examine the effects of supplementing feed additives, derived from *Saccharomyces cerevisiae* yeast, on the growth performance of male turkeys raised to market age. In the investigation for alternatives to the inclusion of sub-therapeutic levels of antibiotics in poultry feed, yeast-based products have been demonstrated as having potential. The goals of this research were to look at two different products, both derived from yeast, but different in design and mode of action. Two growth trials were conducted, each including one of the yeast-based products, to examine the influence of the dietary yeast additive on body weight and feed conversion ratio of male turkeys raised until market age of 20 weeks.

In one trial, turkey breeder hens (for the first two weeks of lay) as well as the male progeny produced, were fed an industry standard ration that was unsupplemented or supplemented with a yeast fermentation product (Original XPC™, Diamond V Mills, Inc., Cedar Rapids, IA, USA). The inclusion of Original XPC™ in the breeder hen diet during the first two weeks of lay did not have a carry over effect on the performance of the male progeny compared to the unsupplemented control breeder hens. The male progeny supplemented with Original XPC™ had higher body weights (BW) until 14 weeks of age compared to the unsupplemented progeny, as well as improved feed conversion ratio (FCR) at 3 and 14 weeks of age. Breast meat yield was also evaluated throughout the trial as well as meat quality at the conclusion of the trial (20 weeks). Supplementation of Original XPC™

improved early breast meat yield (2 weeks), but did not improve meat quality at 20 weeks of age, measured by drip loss and color value.

In the second trial, male turkeys were placed on previously-used litter and fed a control ration, or the control ration with the addition of a mannose-rich yeast cell wall fraction (Actigen™, Alltech, Inc., Nicholasville, KY, USA), from day of hatch until 12 weeks of age. During the starter period, birds from each of the two dietary treatments were euthanized and evaluated for differences in ileal morphology at 6, 10, and 14 days of age. Birds supplemented with Actigen™ had increased villous surface area (10 d) and decreased villous height:crypt depth ratio (14d) than the unsupplemented birds, indicating Actigen™ inclusion improved early intestinal health. All rations fed until 12 weeks included monensin, but there was no measurable interaction effect from the combination of monensin and Actigen™ during this period. From 13 to 20 weeks of age, birds were fed rations with no additive, dietary Actigen™, or an antibiotic (virginiamycin).   The birds fed Actigen™ throughout the whole study performed similarly to the birds only receiving Actigen™ from 13-20 weeks. Dietary supplementation of virginiamycin from 13-20 resulted in a marginally better growth performance response than those fed Actigen™-supplemented diets throughout the trial. However, a benefit of virginiamycin supplementation over Actigen™ supplementation during 13-20 weeks could not be detected.

In conclusion, either of these yeast-derived products could have the potential to be included in turkey diets as alternatives to antibiotics. However, more research should be done to further explore the benefits from these products.

The Effects of Yeast-Derived Feed Additives and Their Potential as Antibiotic Alternative Feed Additives on the Growth Performance of Turkey Toms Raised to Market Age

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BIOGRAPHY

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INTRODUCTION

Turkey consumption has increased 110 percent since 1970. There were over 244 million turkeys produced in the United States (U.S.) in 2010 (NTF, 2010). In 2010, U.S. consumption of turkey was 16.4 pounds per person, compared to 8.3 pounds per person in 1975 (NTF, 2010). Since the 1980's turkey consumption has shifted from a specialty item, mostly consumed around the holidays, to a protein that is consumed throughout the year in a variety of forms, from deli products to further processed food product to products in major fast food chains (NTF, 2010; EPA, 2009). There has been increasing attention to the low-fat, high-protein qualities of turkey meat and the versatility it allows in cooking and food preparation. The amount of exported turkey meat has also greatly increased, from about 1.2% in 1990 to about 10% in 2010. Currently the top export markets for the U. S. are Mexico, China, Hong Kong, and Canada (NTF, 2010).

The value of the U.S. turkey production increased 22 percent from 2009 to 2010 (U.S. Poultry & Egg Association, 2012). In 2010, the top turkey-producing states were Minnesota, North Carolina, and Arkansas (NTF, 2010). Turkey production companies in the United States are vertically integrated. This means each company controls the all phases of the bird production and processing, from breeding to marketing. By using a vertically integrated model, the industry is able to produce safe, high-quality products at the lowest possible cost to consumers.

Turkeys raised for meat production are raised separated into females (hens) and males (toms) because of the difference in their growth rates. Hens are typically raised in houses, or barns, of about 10,000 birds and are kept until 12-16 weeks of age. Toms are typically raised in houses of about 6-8,000 birds 18-20 weeks. Because of the large number of birds raised in close proximity and the long duration of production, it is common for producers to include an antibiotic in the feed during the growing and finishing periods (EPA, 2009). Antibiotics are included at sub-therapeutic levels to promote overall flock health. With the inclusion of antibiotics in animal diets, there are appropriate withdrawal times to ensure there are no antibiotic residues found in the meat. The Food and Drug Administration's (FDA) Center for Veterinary Medicine (CVM) is required by law to approve all antibiotic drugs for safety and efficacy. Specific regulations govern their safe use and proper withdrawal period. USDA's Food Safety and Inspection Service (FSIS) monitors residues of antibiotics or other medications (NTF, 2010). The inclusion of antibiotics in turkey feed has helped with maintaining good intestinal heath and growth of the birds.

LITERATURE REVIEW

A. Gastrointestinal Health

Enteric diseases are a significant concern to animal production because of lost productivity, increased mortality, and the associated contamination of products for human consumption (Patterson and Burkholder, 2003). Birds have been selected for growth rate and feed efficiency in order to provide a more economical and uniform meat product for consumers. As the growth rate of turkeys increases to shorten the time to market, the neonatal growth and development become an increasing proportion of the productive life of a turkey (Uni and Ferket, 2004). A shorter time to market also means that post-hatch enteric development is very important and a more rapid maturation could be an advantage to the health and productivity of the bird.

There are more enteric challenges in today's industry due to a shift in feed ingredients. As human population has increased, so has the demand for an affordable high quality animal protein, which drives demand for least-cost animal feed. This shift in consumption and usage patterns often leaves the animals with feed made using non-traditional ingredients and by-products that are less digestible than the traditional corn and soybean meal (Mathews and McConnell, 2009). For example, wheat and dried distillers grains (DDG) are two examples of ingredients that are high in non-starch polysaccharides, and have been used to replace, or partially replace, corn in some diets to reduce the cost of the diets (Mathews and McConnell, 2009). The nutritive value of a diet consumed by the bird can alter and effect intestinal morphology (Yamauchi, 2002). It has been shown that rations including ingredients high in non-starch polysaccharides (NSP) cause an increase in digesta viscosity resulting in decreased villous height and surface area, and consequently decreased nutrient absorption (Teirlynck *et al*., 2009). High levels of NSP in the diet can also induce enteric stress and lead to the development of undesirable microflora composition (Choct *et al*., 1996). This could affect the animals' resistance to infection as well as digestive efficiency. Poor digestion of feed and malabsorption increases the susceptibility of poults to opportunistic pathogens, resulting in impaired growth (de Oliveira *et al*., 2009). This could present a challenge especially as the amount of by-products included in rations increases in the diets because they are typically not as digestible (Barletta, 2010). This could lead to more problems with pathogens.

Intestinal pathogens are met with many levels of defense including low gastric pH, rapid transport through the intestinal tract, as well as intestinal microbiota, and mucosal immune systems (Patterson and Burkholder, 2003). When functioning correctly, each of these defense mechanisms plays an important role in protecting the gut. However, if the intestinal environment is changed and one or more of these defenses is not functioning properly, the gut can become overpopulated with pathogens or unfavorable bacteria. Gastric pH is low to maintain a caustic environment for foreign pathogens as well as provide a favorable environment for endogenous digestive enzymes (Wyatt *et al*., 2008). A change in pH could cause unfavorable bacterial growth, and decrease the efficiency of certain digestive enzymes (Wyatt *et al*., 2008). When passage rate of the digesta is slowed, due to increased viscosity or bulk of the digesta, microbial fermentation has an increased opportunity to flourish (Choct *et al*., 1996).

Enteric issues are especially important in the poultry industry because of the intensity and concentration of animals during production. Animals have also experienced enteric challenges because of intensive and confined housing and high animal population density, which lead to pathogen concerns (i.e. salmonella) that can be detrimental to animal health as well as a food safety concern. Modern rearing methods place animals in intensive rearing systems of high animal density, which increases the risk of microbial challenge. These enteric issues have been alleviated by the use of drugs such as antibiotics and coccidiostats, as well as other feed additives (Lohren *et al*., 2008).

The turkey is a good model to study for enteric health because turkeys are raised for a longer period of time before market than broilers, and they have time not only for their immune systems to completely develop, but there is enough time to harbor pathogens to the point where they cause problems. Genetic selection of turkeys has resulted in improved growth and feed efficiency. As a result of these improvements, turkeys have been left susceptible to enteric diseases, bacterial challenges, and protozoal issues, all of which cause a decrease in growth performance and feed efficiency (Plyler and Watkins, 2008)

Turkey production has a greater economic impact than broiler production because turkeys consume more feed for longer periods of time. In current poultry production, feed accounts for about 60-70% of the cost of bird production (Hybrid). Flocks of turkeys are raised for at least twice as long as flocks broiler chickens, requiring a longer investment of time in each flock. A typical male turkey raised to market age of 20 weeks will consume about 50 kilograms (kg) of feed to reach a body weight of 20 kg, compared to a broiler raised to 8 weeks of age and will consume 8 kg of feed to reach 4 kg body weight (Hybrid;

Aviagen). With these large investments of time and money it is important to make sure the birds have good enteric health so the feed can be utilized as efficiently as possible.

B. Antibiotics in Animal Feed

Antibiotics have been used in animal agriculture as growth promotants since the late 1940's (Dibner and Richards, 2005). Early on it was observed that inclusion of antibiotics in poultry and swine feed had a beneficial effect on production efficiency. When antibiotics are included in animal feed at sub-therapeutic levels feed conversion can be improved, mortality rates can be reduced, animals are more uniform in size, and the occurrence of disease outbreaks can be decreased (Patterson and Burkholder, 2003). Only antibiotics that are not absorbed in the digestive tract are authorized as growth promoters (Castanon, 2007); if antibiotics were absorbed by the animal, there would be concerns of antibiotic residues in the meat products (Donoghue, 2003). At sub-therapeutic levels, antibiotics have a prophylactic effect against bacterial microflora (Casewell *et al*., 2003). By decreasing the microbial burden in the intestinal tract, the susceptibility to disease is also decreased (Chowdhury *et al*., 2009). Decreases in disease should subsequently decrease the occurrence of immune system responses of the animal. A major immune response will partition nutrients to the immune system, taking them away from growth; this is why antibiotics act as "growth permitters" (Adil *et al*., 2010).

As early as the 1950's, there were reports of resistance in food animals after experimental feeding with antibiotics (Dibner and Richards, 2005; Starr and Reynolds, 1951). In the 1980's pathogen bacteria resistant to numerous antimicrobial agents emerged

(Dibner and Richards, 2005; Aarestrup *et al*., 2003). Reports by the Institute of Medicine, a Council for Agricultural Science and Technology, and a Committee on Drug and Use in Food Animals all recommended that a ban be put in place for the use of antimicrobials in food animals as a precautionary action. Data collected and reported did not prove that resistant microorganisms selected by the use of antibiotic growth promoters (AGP) in animal production cause antibiotic-resistant infections in humans (Dibner and Richards, 2005; Phillips *et al*., 2004). However, Castanon (2007) explains that the wide use of antibiotics as AGP in the long run could contribute to the development of resistant bacteria, and those bacteria with resistant genes could pose potential danger if transferred to humans. Prescott (2008) attributes most of the cause of antibiotic resistance to the extensive use of antimicrobial drugs in humans over the past 60-70 years. Although the use of antimicrobials in animals contributes to the total resistance of human pathogens, Prescott (2008) suggests it is probably relatively slight. Jones and Ricke (2003) pointed out the discrepancies even in estimations of use in antimicrobials in animals due to a lack of unbiased estimated reports.

In 1986, Sweden was the first nation to eliminate the use of antimicrobials for growth promotion. Over the next decade several other antimicrobials were banned in the European Union (EU), and by 1999 even more AGP were banned by the EU Commission because they belonged to classes of antimicrobials also used in humans (Dibner and Richards, 2005; Casewell *et al*., 2003; WHO 2003). By 2006 the EU Commission removed approval for any remaining AGP allowed in EU nations. It is believed by some that simply banning antibiotic growth promoters will stop the progression toward antibiotic resistance. The speculative concern for human public health is that the use of AGP will cause resistant microorganisms to be transferred from animals to humans, mostly through the food chain (Philips, 1999). If humans are exposed to these resistant microorganisms when they get sick, treatment with typical antibiotics will not work. Even if these organisms are transferred to humans by way of the food chain, the organism must survive the consumers' gastrointestinal tract, including stomach acid, the transient nature of digesta, and excretion in the feces. After this whole process, the organisms would still need to be alive to pass on any resistant genes to similar organisms found in humans (Patterson and Burkholder, 2003; Phillips, 1999). Lohren *et al.* (2008) addresses the conflicting results in correlations reported between antibiotic use in poultry and the presence of resistant bacteria.

After the ban of AGP in the EU, there has been an overall decrease of antibiotic use in animal production, but there has been an increase in the therapeutic use of antibiotics to treat sick animals. The antibiotics used therapeutically are the same as those used in human medicine, theoretically creating a hazard to human health related to these zoonotic strains (Casewell *et al*., 2003). Contrary to expectations, the ban of AGP may result in an increased risk to food safety. Animal weight uniformity decreases, without the use of AGP, which results in more ruptures of the gastrointestinal tract and fecal spillage at the processing plant, leading to increases in the potential contamination with salmonella and campylobacter (Casewell *et al*., 2003). The greatest concern of organisms that are harmful to people are *Salmonellae* and *Campylobacters*. *Salmonellae* are almost unaffected by growth promoters, but are affected by products used in the treatment of sick animals. *Campylobacters* that are common in man do not produce disease in food animals. *Escherichia coli* are also largely unaffected by growth promoters (Phillips, 1999).

In the US poultry industry, there has been a recent trend to remove antibiotics from broiler chicken diets. Currently, broiler chickens in the United States are only raised to about 6-8 weeks of age. However, removing sub-therapeutic antibiotics from turkey production presents more of a challenge. Turkeys are raised for a longer period of time (12-20 weeks) than broilers. Hence, they have more time to encounter pathogens, as well as allowing pathogens to accumulate in the digestive tract to the point of causing digestive disturbances or disease.

The trend of the market over the past several decades indicates the possibility of future antimicrobial inclusion restrictions in the United States similar to those in the EU. Many poultry companies in the United States are reducing antimicrobial use at the request of their customers or to meet requirements for export products (Lohren *et al*., 2008). Before any restrictions are put into action, many researchers have been proactive in investigating possible alternatives to antibiotic growth promoters.

C. Alternatives to Antibiotics

There are several possible alternatives to the use of antibiotics in animal feed, including: probiotics, prebiotics, organic acids, and plant extracts (Houshmand *et al*. 2011; Yang *et al*., 2009). The interest in intestinal microbiology and the use of dietary pre- and probiotics came about in the late 1800s and early 1900s, sparked by the isolation of *Escherichia coli* (Patterson and Burkholder, 2003).

Antibiotics have been used for over fifty years in animal production and give positive responses in growth promotion and overall animal health (Casewell *et al*., 2003; Patterson and Burkholder, 2003). Although there are several options for antibiotic alternatives, many give inconsistent responses. The response to each of these alternatives varies greatly, depending on management and environmental stress conditions during production. Some researchers have reported improvement in bird health and performance with the dietary inclusion of antibiotic alternatives (Shashidhara and Devegowda, 2003; Parks *et al*., 2001b). In contrast, Houshmand *et al*. (2011) saw no significant improvements in broiler growth performance or villi morphology with the inclusion of prebiotic (MOS), probiotic, or organic acid mixture. To be a suitable alternative, researchers are looking for something that will still give the benefits of AGP inclusion, such as consistent improvements in growth performance efficiency and control of disease outbreaks.

Probiotics are defined as "a live microbial feed supplement that beneficially affects the host animal by improving its intestinal microbial balance" (Fuller, 1989). In the United States the term "probiotic" cannot correctly be used for a product until its efficacy is well documented and accepted by government regulatory agencies to have curative properties. To avoid regulation requirements, the feed industry now uses the more generic term of "direct-fed microbials," or DFM (Denev *et al*., 2007).

When ruminants are fed DFM, beneficial microbes compete with potential pathogens to prevent their establishment (the same as the competitive exclusion principle). DFM could also produce end-products with antimicrobial properties that limit the growth of pathogens. Feeding DFM to ruminants has resulted in the metabolism of toxic compounds and the production of stimulatory substances, but most bacterial-based DFM are probably beneficial because they have effects in the gut and not in the rumen (Denev *et al.*, 2007). The proposed mechanism for DFM in non-ruminants is the DFM will help to maintain a population of beneficial bacteria by inhibiting the growth of pathogenic microorganisms, and will increase nutrient utilization through improved intestinal health (Angel *et al*., 2005). There are conflicting reports of response, or lack of response of improved performance and feed utilization with the inclusion of DFM in poultry diets (Lee *et al*., 2010; Angel *et al*., 2005).

A main concern with the use of DFM as a feed supplement has to do with its stability and viability after feed processing and manufacturing. The high temperature and pressure of the pelleting process kills most living organisms (Quigley, 2011). This presents a challenge for the poultry industry because nearly all broiler and turkey feed manufactured in the United States' poultry industry is pelleted. However, some researchers have demonstrated that there were still viable populations of lactobacilli from the DFM after the pelleting process (Grimes *et al*., 2008; Angel *et al*., 2005). Although generally, most *Lactobacillus*, *Bifidobacterium*, and *Streptococcus* are destroyed by the high temperatures during the pelleting process, bacilli form stable endospores when conditions are unfavorable for growth, making them very resistant to heat, pH, moisture, and disinfectants (Kung, 2001). The endospore protects the beneficial bacteria of these DFM during the pelleting process, as well as the acidic conditions of the animal's forgut.

Prebiotics are defined as " non-digestible food ingredients that beneficially affects the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon" (Gibson and Roberfroid, 1995). To be classified as a prebiotic, the ingredient can neither be hydrolyzed nor absorbed in the upper part of the gastrointestinal tact, be a selective substrate for one or a limited number of beneficial bacteria in the colon that are stimulated to grow and/or are metabolically activated, must be able to alter colonic flora in favor of a healthier composition, and must induce luminal or systemic effects that are beneficial to the host animal (Gibson and Roberfroid, 1995). An advantage of prebiotics over probiotics is that they can withstand the pelleting process because they are not live products.

Prebiotics are often non-digestible oligosaccharides, such as fructooligosaccharides (FOS) and mannanoligosaccharides (MOS). In poultry diets, research has shown MOS can be included at lower levels in the diets than FOS (Kim *et al*., 2011). FOS are found naturally in some cereal crops and onions, while MOS are derived from the cell walls of yeast that are indigestible to non-ruminant animals. Some oligosaccharides are thought to enhance the growth of beneficial organisms in the gut, whereas others are thought to function by competitive exclusion, or competing with the pathogenic bacteria for attachment sites in the gut lumen (Kim *et al*., 2011). By either of these modes of action they help provide favorable conditions for beneficial species of intestinal *Lactobacilli* (Solis de los Santos, 2007).

Another group of possible antibiotic alternative are organic acids, which are given to poultry to reduce unwanted bacteria during production. (Griggs and Jacob, 2005). Organic acid use has been reported to protect young chicks by competitive exclusion, and enhance nutrient utilization, growth and feed conversion efficiency (Adil *et al*., 2010). The acids can act against *C. perfringens*, salmonella, campylobacter, and listeria, all of which are challenges to the poultry industry (Hofacre *et al*., 2005). Common organic acids used are combinations of acetic, formic, and propionic acid, and can be added to either the feed or the drinking water. The intention of supplementing poultry with the acids to is to lower the pH of the gut to create an acidic intestinal environment to suppress pathogenic bacteria and promote beneficial bacteria growth (Adil *et al*., 2010; Hofacre *et al*., 2005). By reducing the colonization on the intestinal wall by the pathogenic bacteria, damage to the epithelial cells is prevented, thus improving nutrient digestibility. In the non-dissociated form, the organic acids can penetrate the bacterial cell well and disrupt the normal physiology of the bacteria (Adil *et al*., 2010). The concentration and pH of the acids varies from one to another and can alter the degree of antimicrobial effect. This variation can lead to inconsistent results in poultry with the supplementation of organic acids.

Hofacre *et al*. (2005) reported an improvement in feed conversion ratio and body weight gain in coccidia-challenged birds when supplemented with organic acids. This improvement was comparable to the response of the birds receiving an antibiotic (BMD), and both were better than the non-treated control. Adil *et al*. (2010) also observed significant improvements in body weight gains and feed conversion with dietary supplementation of organic acids compared to those not supplemented.

Researchers have also investigated the use of plant extracts as antibiotic alternatives. Many studies have shown essential oils from plant extracts inhibit the growth of bacteria, such as *Clostria*, *Salmonella*, and *E. coli*. However, most of this research has been demonstrated in culture media, not in live birds (Griggs and Jacob, 2005). There have also been documented effects, including growth promotion and modulation of the immune system (Reisinger *et al*., 2011). Responses to the inclusion of plant extracts or essential oils have been inconsistent. There is inconsistency with the strength of the oils themselves, based on where they were grown and if they have been exposed to heat damage (Deans and Ritchie, 1987). Reisinger *et al*. (2011) did not observe improved feed conversion or nutrient digestibility with the addition of an essential oil blend when supplemented to broilers with and without coccidia exposure and vaccination.

D. Yeast in animal feed

Saccharomyces cerevisiae, commonly known as "bakers yeast", is one of the most widely commercially used species of yeast. These uses include bakery products, brewing beer, the distillation of alcohol, fermenting wine, and dietary feed additives for animals (Stone, 1998). Yeast has been supplemented to ruminants since the late 1800's for various purposes, such as a protein source, an appetite stimulant, or a way to stabilize rumen microflora for dairy cows (Stone, 1998; Williams *et al*., 1991; Eckles, 1925). Eckles (1925) studied yeast supplementation and its use as a source of vitamin B in dairy cows and suggested yeast's possible effect on increasing digestive efficiency.

Yeast could be a good option as an alternative for sub-therapeutic inclusion levels of antibiotics in animal feed. Unlike antibiotics, there is no concern with developing resistance. Yeast is a natural product that is common to consumers and will not pose ethical issues or concerns if fed to animals (Owens and McCracken, 2007).

Yeast can be feed to animals as whole yeast, yeast cell wall, or a yeast fermentation product. When given to ruminants, dietary yeast (a respiring organism) can significantly increase the oxygen-consuming activity in the rumen fluid. Oxygen is toxic to anaerobic bacteria and can inhibit the growth of rumen bacteria (Newbold *et al*., 1996). The inclusion of yeast or yeast products in non-ruminant diets has been associated with earlier gut development, more favorable gut microflora population, and increase in immunity (Janardhana *et al*., 2009; Shen *et al*., 2009). Zhang *et al*. (2005) measured similar improvements in gut morphology and body weights in broiler chickens with the supplementation of either yeast cell wall or whole live yeast compared to an unsupplemented control. However, Gheisari and Kholeghipour (2006) did not see a benefit on growth performance or immune response with the addition of live yeast, powdered or granular for, to broiler chicken rations.

As with DFMs, a major concern with live yeast is its viability after the pelleting process. The heat from the steam during conditioning denatures the enzymes in the yeast, stopping its reproductive and metabolic processes (Stone, 1998). However, by processing the yeast through fermentation or a fractionation process, the yeast will no longer be a living, reproducing organism, but it will still retain its viability as a prebiotic (Stone, 1998). The benefit of brewer's dried yeast could be due to its composition, a source of both MOS and beta-glucans (Solis de los Santos *et al*., 2007). Yeast is unique in the fact that it can be fractionated into different parts, allowing for more consistent results because the proportion in a feed additive can be known and controlled. The cell wall of the yeast *Saccharomyces cerevisiae* can be separated from the rest of the yeast cell and fed as a prebiotic. The cell wall consists of an inner cell wall fraction (beta-glucan) and an outer cell wall fraction (mannanoligosaccharide). The cell walls purified from *Saccharomyces cerevisiae* consists of 80-90% (of their dry weight) of equal amounts of mannans (alpha-linked mannose) and glucans (beta-linked glucose)(Giaimis *et al*., 1993). Through processing of yeast cell wall product, the material can be attenuated to increase its ability to bind specific proteins or toxins (Dawson, 2011). The first commercially available yeast cell wall product, sold as an alternative growth promoter to antibiotics for turkeys, was introduced in 1993 by Alltech, Inc. (Alltech, Inc., Nicholasville, Kentucky USA). This product, Bio-Mos®, was derived from the outer cell wall of *Saccharomyces cerevisiae* (Hooge, 2004).

The work of Parks (2001a) compared growth performance and immune response of turkeys supplemented dietary Bio-Mos® (Alltech, Inc., Nicholasville, Kentucky USA) or industry standard antibiotics, bambermycin and virginiamycin. Parks (2001a) observed an improvement in early feed conversion (0-3 weeks) with the addition of Bio-Mos® bambermycins, virginamycin and Bio-Mos® and bambermycin used in combination. The addition of Bio-Mos®, as well as the treatments receiving virginiamycin, increased overall bodyweight as compared to unsupplemented dietary treatments. A similar study by Parks (2001a) conducted with hens showed an improvement in body weight gain with the addition of virginiamycin, but not with the inclusion of Bio-Mos®. Benites *et al*. (2008) reported an increased body weight in broiler chickens supplemented with Bio-Mos® as compared to the control treatment or those supplemented with another MOS product (SAF-Mannan®, Lasaffre et Compagnie, France). Benites *et al*. (2008) also reported that neither MOS product affected feed consumption and FCR in comparison to the control. Based on a meta-analysis of several trials, Hooge (2004) reported that dietary MOS can improve body weights and decrease mortality in turkeys as compared to negative control diets, but dietary MOS supplementation does not always improve feed conversion ratio. Based on the statistical similarity of bird performance response to either MOS-supplementation or antibiotic-supplementation, Hooge (2004) suggests that MOS could replace sub-therapeutic antibiotics in turkey feed.

The main component of MOS is the sugar mannose, which is a sugar that many enteric bacteria have receptors that bind to it, called Type 1 fimbriae (Griggs and Jacob, 2005; Oyofo *et al*., 1989). Binding to these receptors instead of the intestinal wall can result in the movement of undesirable bacteria though the gut without colonization; most bacteria must attach to the gut wall in order to colonize. In a meta-analysis of dietary MOS, Hooge (2004) summarizes three probable modes of action: 1) Adsorption of pathogenic bacteria containing Type 1 fimbrae with mannose sensitive lectins; 2) Improved intestinal function/gut health, such as increased villi height and uniformity; and 3) Immune modulation stimulates gut associated and systematic immunity by acting as a non-pathogenic microbial antigen. Although the commercial MOS, or yeast cell wall, products are thought to have a similar mode of action, they differ one from one another by the purity or concentration of the cell wall components, such as mannans, beta-glucans, chitin and protein.

The cell wall can be further fractionated to create a mannose-rich fraction. This concentrated MOS product can be fed at low levels in the diet, but is limited to very specific actions. The commercially available product Actigen™ (Alltech, Inc., Nicholasville, Kentucky USA) is a mannose-rich product derived from the outer cell wall of the yeast *Saccharomyces cerevisiae*. Actigen™ is a second-generation product, and is reported to be 2.5 times higher concentration of biological activity than its predecessor, Bio-MOS® (Alltech, Inc., Nicholasville, Kentucky USA). The proposed mode of action of Actigen™ is similar to that of Bio-Mos®, but was developed using nutrigenomic technology, allowing detection of changes in the gene expression in intestinal cells (Hooge and Connolly, 2011). When yeast is fractionated to remove the cell wall, the remaining fraction can still be used for other purposes. The inner cell wall of the *Saccharomyces cerevisiae* contains glucomannans, which have been demonstrated to have good binding affinity for toxins (Dawson *et al*., 2006). For this reason, several companies, including Alltech, Inc. (Mycosorb®, Alltech, Inc., Nicholasville, Kentucky USA), market yeast cell wall products for use as mycotoxin binders in animal feed.

Yeast can also be included in animal feed as a fermentation product, or yeast culture such as Original XPC™ (Diamond V Mills, Inc., Cedar Rapids, IA, USA). Yeast fermentation products are produced by distressing yeast, causing the cells to produce compounds and metabolites to help sustain their ecosystem (Francois and Parrou, 2001). The yeast culture is dried and fed as a prebiotic including the yeast, the media it was grown on, as well as any metabolites or by-products produced during fermentation. Metabolic products can include antioxidants, vitamin B complexes, organic acids (such as citric acid), polyphenols, and nucleotides, as well as yeast cell wall fragments (beta-glucans and MOS), and residual yeast cells (Yalcin, 2011; Stone, 1998). The fermentation products in the yeast cultures stimulate bacterial growth in the digestive tract (Stone, 1998). *Saccharomyces cerevisiae* produces the sugars, glycogen and trehalose, and under stressful conditions the yeast can increase production of these carbohydrates (Francois and Parrou, 2001). During fermentation, the yeast cells also produce citric acid (Yalcin *et al*., 2010). Citric acid inclusion in the rations of broiler chickens has been shown to improve phosphorus utilization (Boling-Frankenbach *et al*., 2001) and growth performance (Chowdhury *et al*., 2009).

Because of these metabolic products, a yeast fermentation product may give not only the beneficial effects of MOS, but also of organic acids and other bioactive compounds. Yeast cultures have been shown to be viable at high temperatures making them easier to handle during feed manufacturing (Stone, 1998).

Yeast fermentation product inclusion has been shown to accelerate gastrointestinal maturation in turkey poults by enhancing ileum villous height, surface area, lamina propria thickness, crypt depth and goblet cell density (Solis de los Santos *et al*., 2007). When pigs were supplemented with the yeast fermentation product in addition to the yeast cell wall product, there was no significant difference than with the addition on the yeast fermentation product alone (van der Peet-Schwering *et al*., 2007). Not measuring an additive effect of the yeast cell wall could be because the fermentation product already contains yeast cell walls (MOS).

E. Summary

Dietary feed additives derived from the yeast *Saccharomyces cerevisiae* could be a good option as alternatives to antibiotic growth promotants. There has been much success seen in poultry performance with the inclusion of yeast cell wall products, such as Bio-Mos® (Hooge, 2004). However, there is limited literature regarding mannose-rich products, specifically their use in turkeys. In this thesis research project, the objective was to study the growth performance response of male turkeys raised to market age supplemented with two yeast-based products that differed by their source, manufacturing process, and composition. The objective of this work was not to compare the efficacy of these two yeast products against one another in a single experiment, but to evaluate each yeast product in separate experiments for their growth promoting effects. The experiment evaluating the yeast fermentation product was not compared with a pharmaceutical antimicrobial, whereas the experiment with the mannose-rich cell wall product was compared to an antibiotic growth promoter (AGP). The two products used were from opposite sides of the spectrum: a yeast fermentation product and a concentrated, mannose-rich fraction of yeast cell wall. The yeast fermentation product, or yeast culture, used in this research was Original XPC™ (Diamond V Mills, Inc., Cedar Rapids, IA, USA). A yeast fermentation product is a conglomeration of various parts of the yeast cell, as well as metabolites from the fermentation process, resulting in a product with several modes of action, but not one specific action. On the other hand, the mannose-rich product used in this research was Actigen™ (Alltech, Inc., Nicholasville, Kentucky USA). Actigen™ is a specific, concentrated product of the mannose isolated from the outer cell wall from *Saccharomyces cerevisiae*. Our interest with these two very different products was to observe if there is an advantage of using a highly refined product compared to a product that is an unknown mixture of products.

F. References

Aarestrup, F. M., M. Lertworapreecha, M. C. Evans, A. Bangtrakulnonth, T. Chalermchaikit, R. S. Hendriksen, and H. C. Wegener. 2003. Antimicrocial susceptibility and occurrence of resistance genes among *Salmonella enterica* serovar Weltevreden from different countries. J. Antimicrob. Chemother. 52:715-718.

Adil, S. T. Banday, G. A. Bhat, M. S. Mir, and M. Rehman. 2010. Organic acids on performance, intestinal histomorphology, and serum biochemistry of broiler chicken. Vet. Med. Int. 2010: 1-7.

Angel, R., R. A. Dalloul, and J. Doerr. 2005. Performance of broiler chickens fed diets supplemented with a direct-fed microbial. Poult. Sci. 84:1222-1231.

Aviagen. Aviagen Group. <www.en.aviagen.com>

Barletta, A. 2010. Current Market and Expected Developments. Enzymes in Farm Animal Nutrition, 2nd Edition. (1):1-11.

Benites, V., R. Gilharry, A. G. Gernat, and J. G. Murillo. 2008. Effect of dietary mannan oligosaccharide from Bio-Mos or SAF-Mannan on live performance of broiler chickens. J. Appl. Poult. Res. 17:471-475.

Boling-Frankenbach, S. D., J. L. Snow, C. M. Parsons, and D. H. Baker. 2001. The effect of citric acid on the calcium and phosphorus requirements of chicks fed corn-soybean meal diets. Poult. Sci. 80:783-788.

Casewell M., C. Friis, E. Marco, P. McMullin, and I. Phillips. 2003. The European ban on growth-promoting antibiotics and emerging consequences for human and animal health. J. Antimicob. Chemother. 52:159-161.

Castanon, J. I. R. 2007. History of the use of antibiotic as growth promoters in European poultry feeds. Poult. Sci. 86:2466-2471.

Choct, M., R. J. Hughs, J. Wang, M. R. Bedford, A. J. Morgan, and G. Annison. 1996. Increased small intestinal fermentation is partly responsible for the anti-nutritive activity of non-starch polysaccharides in chickens. Brit. Poult. Sci. 37:609-621.

Chowdhury, R., K. M. S. Islam, M. J. Khan, M. R. Karim, M. N. Haque, M. Khatun, and G. M. Pesti. 2009. Effect of citric acid, avilamycin, and their combination on the performance, tibia ash, and immune status of broilers. Poult. Sci. 88:1616-1622.

Dawson, K. A., J. Evans, and M. Kudupoje. 2006. Understanding the adsorption characteristics of yeast cell wall preparations associated with mycotoxin binding. <www.en.engormix.com>

Dawson, K. A. 2001. The application of yeast and yeast derivatives in the poultry industry. Proc Aust Poult Sci Sym. 13:100-105.

de Oliveira, J. E., S. Druyan, Z. Uni, C. M. Ashwell, and P. R. Ferket. 2009. Prehatch intestinal maturation of turkey embryos demonstrated through gene expression patterns. Poult. Sci. 88:2600-2609.

Deans, S. G., and G. Ritchie. 1987. Antibacterial properties of plants essential oils. Int. J. Food Microbiol. 5:165-180.

Denev, S. A., Tz. Peeva, P. Radulova, N. Stancheva, G. Staykova, G. Beev, P. Todorova, and S. Tchobanova. 2007. Yeast cultures in ruminant nutrition. Bulg. J. Agric. Sci. 13:357-374.

Dibner, J. J., and J. D. Richards. 2005. Antibiotic growth promoters in agriculure: History and mode of action. Poult. Sci. 84:634-643.

Donoghue, D. J. 2003. Antibiotic residues in poultry tissues and eggs: Human health concerns? Poult. Sci. 82:618–621.

Eckles, C. H., and V. M. Williams. 1925. Yeast as a supplementary feed for lactating cows. J. Dairy. Sci. 8:89-93.

Environmental Protection Agency (EPA). 2009. Poultry production phases: Broilers, turkeys, ducks (meat-bird production). <www.epa.gov>

Francois, J., J. L. Parrou. 2001. Reserve carbohydrates metabolism in the yeast *Saccharomyces cerevisiae*. FEMS Microbiol. Rev. 25:125-145.

Fuller, R. 1989. Probiotics in man and animals. J. Appl. Bacteriol. 66:365-378.

Gheisari, A and Kholeghipour, B. 2006. Effect of dietary inclusion of live yeast (*Saccharomyces cerevisiae*) on growth performance, immune responses and blood parameters of broiler chickens. Proceedings from XII European Poultry Conference, Verona, Italia, 6p.

Giaimis, J., Y. Lombard, P. Fonteneau, C. D. Muller, Rachel Levy, M. Makaya-Kumba, J. Lazdins, P. Poindron. 1993. Both mannose and beta-glucan receptors are involved in phagocytosis of unopsonized, heat-killed Saccharomyces cerevisiae by murine macrophages. J. Leukoc. Biol. 54: 564-571.

Gibson, G. R., and M. B. Roberfroid. 1995. Dietary modulation of the human colonic microbiota: introducing the concept of prebiotics. J. Nutr. 125:1401-1412.

Griggs, J. P., and J. P. Jacob. 2005. Alternatives to antibiotics for organic poultry production. J. Appl. Poult. Res. 14:750-756.

Grimes, J. L., S. Rahimi, E. Oviedo, B. W. Sheldon, and F. B. O. Santos. 2008. Effects of a direct-fed microbial (Primalac) on turkey poult performance and susceptibility to oral *Salmonella* challenge. Poult, Sci. 87:1464-1470.

Hofacre, C. L., G. F. Mathis, and M. A. Quiroz. 2005. Natural alternatives to prevent necrotic enteritis. Int. Poult. Prod. 13:7-9.

Hooge, D. M., and A. Connolly. 2011. Meta-analysis summary of broiler chicken trials with dietary Actigen® (2009-2011). Int. J. Poult. Sci. 10:819-824.

Hooge, D. M. 2004. Turkey pen trials with dietary mannan oligosaccharide: meta-analysis, 1993-2003. Int. J. Poult. Sci. 3(3):179-188.

Houshmand, M., K. Azhar, I. Zulkifli, M. H. Bejo, and A. Kamyab. 2011. Effects of non-antibiotic feed additives on performance, nutrient retention, gut pH, and intestinal morphology of broilers fed different levels of energy. J. Appl. Poult. Res. 20:121-128.

Hybrid Turkeys. A Hendix Genetic Company. <www.hybridturkeys.com>.

Janardhana, V., M. M. Broadway, M. P. Bruce, J. W. Lowenthal, M. S. Geier, R. J. Hughes, and A. G. D. Bean. 2009. Prebiotics modulate immune responses in the gut associated lymphoid tissue of chickens. J. Nutr. 139:1404-1409.

Jones, F. T., and S. C. Ricke. 2003. Observations on the history of the development of antimicrobials and their use in poultry feeds. Poult. Sci. 82:613-617.

Kim, G. B., Y. M. Seo, C. H. Kim, and I. K. Paik. 2011. Effect of dietary prebiotic supplementation on the performance, intestinal microflora, and immune response of broilers. Poult. Sci. 90:75-82.

Kung, Jr., L. 2001. Direct-fed microbials for dairy cows. University of Delaware Extension Research. Department of Animal and Food Sciences.

Lee, K. W., S. H. Lee, H. S. Lillehoj, G. X. Li, S. I. Jang, U. S. Babu, M. S. Park, D. K. Kim, E. P. Lillehoj, A. P. Neumann, T. G. Rehberger, and G. R. Siragusa. 2010. Effects of diect-fed microbials on growth performance, gut morphometry, and immune characteristics in broiler chickens. Poult. Sci. 89:203-216.

Lohren, U., A. Ricci, and T. S. Cummings. 2008. Guidelines for antimicrobial use in poultry. Guidelines to antimicrobial use in animals. 8:126-142.

Mathews, Jr., K. H., and M. J. McConnell. 2009. Ethanol co-product use in U.S. cattle feeding: lessons learned and considerations. United States Department of Agriculture (USDA). A report from the Economic Research Service.

National Turkey Federation (NTF). 2010. http://www.eatturkey.com/about/list.html.

Newbold, C. J., R. J. Wallace, and F. M. McIntosh. 1996. Mode of action of the yeast *Saccharomyces cerevisiae* as a feed additive for ruminants. Brit. J. Nutr. 76:249-261.

Owens, B., and K. J. McCracken. 2007. A comparison of the effects of different yeast products and antibiotic on broiler performance. Brit. Poult. Sci. 48:49-54.

Oyofo, B. A., R. E. Droleskey, J. O. Norman, H. H. Mollen- hauer, R. L. Ziprin, D. E. Corrier, and J. R. DeLoach. 1989. Inhibition by mannose of *in vitro* colonization of chicken small intestine by *Salmonella typhimurium*. Poult. Sci. 68:1351–1356.

Parks, C. W. 2001a. The effects of supplementing diets with virginiamycin or mannan-oligosaccharides on immune system properties and intestinal morphology. Ph.D. Dissertation. North Carolina State University, Raleigh, NC, USA.

Parks, C. W., J. L. Grimes, P. R. Ferket, and A. S. Fairchild. 2001b. The effect of mannanoligosaccharides, Bambermycins, and Virginianmycin on performance of large white male market turkeys. Poult. Sci. 80:718-723.

Patterson, J. A., and K. M. Burkholder. 2003. Application of prebiotics and probiotics in poultry production. Poult Sci. 82:627-631.

Phillips, I. 1999. Assessing the evidence that antibiotic growth promoters influence human infections. J. Hosp. Infect. 43:173-178.

Prescott, J. F. 2008. Antimicrobial use in food and companion animals. Animal Health Research Reviews. 9:127-133.

Quigley, J. 2011. A Bovine Alliance of Management and Nutrition (BAMN) publication: Direct-fed microbials (probiotics) in calf diets. AFIA publications.

Reisinger, N., T. Steiner, S. Nitsch, G. Schatzmayr, and T.J. Applegate. 2011. Effects of a blend of essential oils on broiler performance and intestinal morphology during coccidial vaccine exposure. J. Appl. Poult. Res. 20:272-283.

Shashidhara, R. G., and G. Devegowda. 2003. Effect of dietary mannan oligosaccharide on broiler breeder production traits and immunity. Poult. Sci. 82:1319-1325.

Shen, Y. B., X. S. Piao, S.W. Kim, L. Wang, P. Liu, I. Yoon, and Y.G. Zhen. 2009. Effects of yeast culture supplementation on growth performance, intestinal health, and immune response of nursery pigs. J. Anim. Sci. 87:2614-2624.

Solis de los Santos, F., A. M. Donoghue, M. B. Farnell, G. R. Huff, W. E. Huff, and D. J. Donoghue. 2007. Gastrointestinal Maturation is accelerated in turkey poults supplemented with a mannan-oligosaccharide yeast extract (Alphamune). Poult Sci. 86:921-930.

Starr, M. P., and D. M. Reynolds. 1951. Streptomycin resistance of coliform bacteria from turkeys fed streptomycin. Proceedings of the 51st General Meeting, Society of American Bacteriology, Chicago, IL. Page 15-34.

Stone, C. W. 1998. Yeast products in the feed industry: A practical guide for feed professionals. Diamond V Mills, Inc. Cedar Rapids, IA, USA. Page 1-16.

Teirlynck, E., L. Bjerrum, V. Eeckhaut, G. Huygebaert, F. Pasmans, F. haesebrouck, J. Dewulf, R. Ducatelle, and F. Van Immerseel. 2009. The cereal type in feed influences gut wall morphology and intestinal immune cell infiltration in broiler chickens. Br. J. Nutr. 102:1453-1461.

U.S. Poultry & Egg Association. 2012. <www.uspoultry.org>

van der Peet-Schwering, C. M. C., A. J. M. Jansman, H. Smidt, and I. Yoon. 2007. Effects of yeast culture on performance, gut integrity, and blood cell composition of weanling pigs. J. Anim. Sci. 85:3099–3109.

WHO (World Health Organization). 2003. Impacts of antimicrobial growth promoter termination in Denmark.

Williams, P. E., C. A. Tait, G. M. Innes, and C. J. Newbold. 1991. Effect of the inclusion of yeast culture (*Saccharomyces cerevisiae* plus growth medium) in the diet of dairy cows on milk yield and forage degradation and fermentation patterns in the rumen of steers. J. Anim. Sci. 69:3016-3026.

Wyatt, C. L., T. Parr, and M. Bedford. 2008. Mechanisms of action for supplemental NSP and phytase enzymes in poultry diets. Proceedings from Carolina Feed Industry Association 35th Poultry Nutrition Conference.

Yalcin, S., S. Yalcin, P. Can, A. O. Gurdal, C. Bagci, and O. Eltan. 2011. The nutritive value of live yeast culture (*Saccharomyces cerevisiae*) and its effect on milk yield, milk consumption and some blood parameter of dairy cows. Asian-Aust. J. Anim. Sci. 24:1377-1385.

Yalcin, S. K., M. T. Bozdemir, and Z. Y. Ozbas. 2010. Citric acid production by yeasts: Fermentation conditions, process optimization and strain improvement. Current Research, Technology and Education Topics in Applied Microbial Biotechnology. 2:1374-1382.

Yang, Y., P. A. Iji, and M. Choct. 2009. Dietary modulation of gut microflora in broiler chickens: a review of the role of six kinds of alternatives to in-feed antibiotics. World's Poultry Science Journal. 65:97-114.

Zhang, A. W., B. D. Lee, S. K. Lee, K. W. Lee, G. H. An, K. B. Song, and C. H. Lee. 2005. Effects of yeast (*Saccharomyces cerevisiae*) cell components on growth performance, meat quality, and ileal mucosa development of broiler chicks. Poult Sci. 84:1015-1021.

MANUSCRIPT I. The Effect of Feeding Original XPC^{TM} to Turkey Breeders During Early Lay and Their Progeny on Growth Performance

ABSTRACT: A study was conducted to determine the effect of a yeast fermentation product (Original XPC^{TM}, Diamond V Mills, Inc., Cedar Rapids, IA, USA) in turkey breeder hen diets and the diets of their progeny. Eggs were collected and incubated from young turkey breeder hens (33 weeks of age) that were fed a standard diet or a standard diet supplemented with Original XPC^{TM} during the first two weeks of lay. The male progeny of these two groups of hens were raised in a curtain-sided house in litter floor pens to 20 weeks and were provided feed and water *ad libitum*. The male poults were randomly placed in 40 pens, with 15 birds per pen, and assigned to one of two standard corn-soybean based dietary treatments: unsupplemented or supplemented with Original XPC^{TM} (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20). The male progeny were evaluated for growth performance from day of hatch until market age (20 weeks) and for meat quality characteristics at 20 weeks of age. Body weights (BW) and feed consumption were determined at 3, 6, 9, 12, 14, 16, 18, and 20 weeks and feed/gain (FCR) calculated. At 20 weeks, 20 birds per treatment were slaughtered and carcass parts yield was determined. There were no significant breeder effects or breeder X progeny interaction effects on BW or FCR throughout the experiment. BW of progeny fed Original XPC^{TM} was greater than controls at 3, 6, 9, 12, and 14 weeks ($P<0.05$). However, cumulative FCR of progeny fed Original XPC^{TM} was lower than unsupplemented birds at 3 and 14 weeks but this advantage was lost by 20 weeks. There were no significant treatment effects on carcass yield at 20 weeks. Supplementing male turkeys with Original XPC^{TM} in

standard corn-soy diet improved bird performance until 14 weeks of age, but there was no measureable difference in growth performance during the finishing period.

(*Keywords*: turkeys, *Saccharomyces cerevisiae*, fermentation product, Original XPC^{TM}, growth performance, carcass yield)

Description of Problem

Since the 1950's sub-therapeutic levels of antibiotics have been included in poultry feed to mitigate decreases in growth performance and feed efficiency caused by bacterial infections, disease, or other enteric challenges. However, there has been a growing concern about the development of antibiotic resistant bacteria with the continued use of antibiotics in animal production (Dibner and Richards, 2005). Restrictions have been put in place throughout the European Union since 2006 banning the use of antibiotics as growth promoters (AGP) in an attempt to slow the increasing numbers of resistant bacteria (Lohren *et al*., 2008; Dibner and Richards, 2005). For the past several years there have been legislative proposals made to ban or limit the use of antimicrobials in the United States (Lohren *et al*., 2008). Although these limitations have not yet become legal requirements, many poultry companies in the Unites States are voluntarily reducing antimicrobial use at the request of their customers or to meet requirements for export products (Lohren *et al*., 2008; Korver, 2006). The concern for human public health and legislative restriction has lead to the search for alternatives to antibiotic use in animal feed (Philips, 1999).

There is interest in the use of alternatives to antibiotic feed additives in the poultry industry, but it is of special concern in turkey industry. Although turkeys may not be considered a minor food animal species in the US, they are in other countries to which the US wishes to export meat. Producers of pharmaceuticals must submit separate data for each species for product registration approvals at a considerable cost (Lohren *et al*., 2008). Consequently, some pharmaceutical products never became available for minor species, such as turkeys, because the market potential is not big enough to recoup the costs of FDA approval and registration. Moreover, to export turkey meat to other countries, all restrictions in that country must be abided by, thus further limiting the options for dietary antimicrobial inclusion for the turkey industry compared to major poultry species, such as broiler chickens or laying hens.

There have been several proposed alternatives to AGP inclusion in animal feed including: probiotics, prebiotics, organic acids, and plant extracts (Griggs and Jacob, 2005; Shashidhara and Devegowda, 2003; Parks et al., 2001). Among these possible alternatives are a variety of yeast products, most of which are prepared from the yeast *Saccharomyces cerevisiae*. Yeast-based feed additive products are available in a variety of forms, such as whole yeast, fermented yeast products, fractionated yeast products, yeast cell wall, and yeast extract. Many of the yeast products are classified as prebiotics and can withstand the high temperatures of the pelleting process during feed manufacturing. The benefits of yeast products have been reviewed by several researchers (Hooge, 2004; Stone, 1998).

Yeast fermentation product, produced during the fermentation of *Saccharomyces cerevisiae* under various conditions, contains all fractions of the yeast, fermentation products, and residual fermentation media (Shen *et al*., 2011). During fermentation the yeast cells are put under distress, resulting in the generation of metabolic byproducts or bioactive compounds that have protective effects (Francois and Parrou, 2001). After the fermentation process, the whole product is dried so that it retains activity of the metabolic byproducts produced, and the product can be added to animal rations in a dry form (Yalcin *et al*., 2008).

Anaerobically fermented yeast products, or yeast culture, may be a possible alternative to the sub-therapeutic antibiotics included in poultry diets. The yeast culture, a good source of nutritional metabolites, mannanoligsaccharides, and beta-glucans, may be able to improve gut health and immunity (Bradley and Savage, 1995), which would lead to better growth performance (Price *et al*., 2010). Inclusion of yeast culture in poultry diets has been demonstrated to improve performance in laying hens (Wang *et al*., 2009), broiler chickens (Gao *et al*., 2009), and turkeys (Paiva *et al*., 2010).

In a study by Gao *et al*. (2009), yeast fermentation product was shown to improve growth performance and immune response when supplemented to broilers infected with coccidia. Firman *et al.* (2009) reported improved feed efficiency and increased *Pectoralis major* yield in turkey toms fed a diet containing yeast culture from 15 to 18 weeks. Bradley and Savage (1995) and Hayat *et al*. (1993) demonstrated that dietary supplementation of a yeast fermentation product improved the reproductive performance of some turkey breeder hens. They concluded that yeast culture inclusion in turkey breeder hen diets could improve reproductive efficiency, such as hatchability of fertile eggs.

The nutrients provided to the breeder hen will affect the nutrient composition of the egg and consequently what nutrients the embryo receives during incubation (Wang *et al*., 2009; Plyler *et al*., 2008; Surai, 2000). Poult quality is greatly influenced by the nutrients and antibodies the poult receives from the egg yolk (Plyler *et al*., 2008). It is possible that the transfer of nutrients from hen to progeny could have a carry-over effect on the progeny growth performance and health. Thus the study reported herein was designed to evaluate the possible carry-over effects the inclusion of yeast fermentation product in the diet of young turkey breeders on the early growth performance of their progeny.

It is well known that poults that are hatched from eggs laid by breeder hens early in their laying cycle tend to perform poorly. Young turkey hens in the beginning of lay produce smaller eggs than when they are older and more mature and their progeny exhibit inferior growth characteristics (Yalcin *et al*., 2005; Applegate, 2002). Yadgary *et al.* (2010) suggested that the absorption of yolk fat in early embryonic developments is lower in the progeny from younger hens than older hens, likely due to a lower initial yolk fat content in eggs produced by the younger hens. This creates a nutritional deficiency and higher nutritional demands on the embryo during the incubation process. They also suggest that the lipid transport in the yolk sac membrane is less efficient in younger hens than older ones. If performance of breeder hens can be improved by dietary yeast culture supplementation, then it could improve the efficiency of the younger hens to transfer more critical nutrients into their eggs, thus providing the embryo better *in ovo* nutrition to support more advanced development and subsequent growth performance and health post-hatch.

The yeast fermentation product, or yeast culture, used in this study was Original XPC^{TM} (Diamond V Mills, Inc., Cedar Rapids, IA, USA). This product is produced by fermenting selected liquid and cereal grain raw ingredients with bakers yeast (*Saccharomyces cerevisiae*) and drying the entire culture-media without destroying the yeast factors, B-vitamins and other fermentation products (www.diamondv.com).

Our first objective was to determine if there was a carry-over effect from the Original XPC^{TM} supplementation of breeder hens to the progeny. The second objective was to determine the effect of Original XPC^{TM} on early growth performance of the male progeny. Lastly, we hypothesized a possible interaction effect of feeding Original XPC^{TM} to the breeder hens and/ or the male progeny.

Materials and Methods

Birds and Housing

Hybrid Converter turkey hens were placed at a commercial breeder Farm (Cuddy Farms, Sandy Ridge Farm, Strathroy, Ontario, Canada) and photostimulated at 28 weeks of age. Two barns of 3,500 hens were fed an unsupplemented control diet and three barns of 3,500 hens were fed a similar diet supplemented with 0.075% Original XPC. When the breeder hens were 33 weeks of age, in the second week of lay and after being fed experimental treatments for two weeks, eggs were collected from a single collection time and an equal number of eggs from each treatment barn was pooled and transferred to the North Carolina State University Hatchery (Raleigh, NC), where they were set for single stage incubation and hatching in a Jamesway 252 incubator (Butler Manufacturing, Fort Atkinson, WI) according to breeder recommendations. Within six hours of hatching and sex determination, the male turkey poults were transported and placed in litter floor pen facility and randomly assigned to 40 pens containing 15 poults per pen. The toms were raised in 3.4 m^2 floor pens of a curtain-sided facility until market age at 20 weeks of age. Each replicate pen was equipped with one tube feeder and one Plasson drinker (Plasson, Ltd., Menashe, Israel) for *ad libitum* consumption of feed and water, respectively. Soft pine shavings were used as litter. All the birds were raised according to typical management practices and according to approval by the Institutional Animal Care and Use Commitee. The birds received 23 hours incandescent lights from 1-10 days and natural lighting subsequently until the experiment was terminated at 140 days of age.

Dietary treatments

The experimental diets (Table 1) were corn-soy based diets formulated to meet or exceed the nutrient level recommendations by Hybrid Turkeys at the time of the trial (http://www.hybridturkeys.com/en/Hybrid%20Resources/Nutrition.aspx). The diets were not supplemented with any pharmaceutical antimicrobial feed additives. Each pen was assigned to one of four dietary treatments. This study was designed as a split-plot design with the two main plots as 1) the breeder hens fed control diets or 2) diets supplemented with 0.075% Original XPC^{TM}, and the subplots as the male progeny of these hens fed either 1) a control diet or 2) a diet supplemented with Original XPC^{TM} (0.125% XPC weeks 1 to 6; 0.0625% XPC weeks 7 to 20). Each dietary treatment was represented by 10 replicate pens, with 15 poults each, with a total of 40 pens used in the study.

Measurements

Individual body weights and pen feed consumption data were obtained at 3, 6, 9, 12, 14, 16, 18, and 20 weeks of age. Mortalities were recorded and the weight was used to adjust pen feed conversion.

At 2, 6, 12, 14, 16, 18, and 20 weeks of age, four birds per treatment were sampled for carcass parts yield determination after 4 hours without access to feed, although drinking water was provided. Live body weights were determined and then the birds were euthanized by electrocution, scalded in 65 C water for 2 minutes and feathers removed by a rotary picker. The carcasses were then air dried and weighed again to determine feather weight. The *pectoralis major*, *pectoralis minor*, and viscera content were dissected and weighed to determine yield relative to live body weight. At 20 weeks, 20 birds per treatment were euthanized by electo-stunning and exsanguination. Weights of the carcass without the head, blood, viscera, feet, and feathers were determined and then carcasses were chilled over-night (12 hours) in chill-tanks filled with ice water. The chilled carcasses were removed from the tanks, weighed, and yield of the following parts was determined: *pectoralis major*, *pectoralis minor*, neck, fat pad, thigh, drum, breast skin, wings, and rack (back, ribs, and keel bone).

From the *pectoralis major* of each sampled bird, a sliced breast filet was dissected (approximately 1 inch wide) from the middle section of the left lobe of the muscle. After weighing each sample, individual breast filets were placed in half-gallon zip-lock bag and refrigerated at 4 C for subsequent color determination. After 2 and 7 days of refrigerated storage a color value for L* (lightness), a* (redness), and b* (yellowness) was obtained from each filet, using a portable reflectance colorimeter (Minolta Chroma Meter CR 300, Minolta Corp., Ramsey, NJ.). At the time of meat color evaluation, 7 days after storage, filets were also removed from the plastic bags, the excess fluids wicked into a paper towel, and then the filets were reweighed. Purge fluid (drip) loss was calculated by weight differenced of the meat at the beginning and end of the 7 day refrigerated storage period.

Statistical Analysis

This study was analyzed as a 2x2 factorial, with the factors as breeder hens fed control diets or diets supplemented with 0.075% Original XPC^{TM}, and the male progeny of these hens fed either a control diet or a diet supplemented with Original XPC^{TM} (0.125% XPC weeks 1 to 6; 0.0625% XPC weeks 7 to 20). Growth parameter data were analyzed as a completely randomized block design using the GLM procedure of JMP 8.0 (SAS Institute, 2008) for ANOVA. Means were separated using the LSMeans procedure of JMP at $P<0.05$. The pen was used as the experimental unit for growth parameters and individual birds were used as the experimental unit for carcass analysis. Data reported in percentages were transformed before analysis using arc sine √percent, but values are reported in tables as percentage values.

Animal Ethics

This study was conducted according to the guidelines of the Institutional Animal Care and Use Committee at North Carolina State University. All husbandry practices and euthanasia were performed with full consideration of animal welfare.

Results and Discussion

Up to the point of egg collection from the breeder hens, there were no treatment differences on reproductive performance, such as hen-day egg production rate, feed conversion, mortality rate, hatchability rate, fertility rate, hatchability of fertile eggs, egg weight, and eggshell weight. There was no carry-over effect of breeder treatment on progeny performance.

In this study there was no significant breeder x progeny treatment interaction effects or dietary Original XPC™, or yeast fermentation product, supplementation of breeders on progeny growth performance. Dietary supplementation of the yeast fermentation product for breeders had no effect on progeny growth performance. Therefore, there was not an added benefit in performance passed on by the breeder hens fed the yeast fermentation product, with the study design utilized. This observation agrees with the response seen by Brake (1991) in broiler breeders and their progeny. Brake (1991) evaluated the effect of yeast culture on the performance of broiler breeders and their progeny. Eggs from breeders, both yeast-supplemented and non-supplemented, were collected, incubated, and raised to 17 days of age in battery cages. With the dietary addition of yeast culture over the control diets, Brake (1991) did not observe significant affects in egg production, feed conversion, mortality, hatchability of fertile eggs, egg weight, eggshell weight, or percentage shell. There was also no effect on the progeny weight, feed conversion, and mortality in the treatments that the hen had been supplemented with yeast culture. Bradley and Savage (1995) also saw no differences in post-hatch livability (1 to 10 days) for the progeny of turkey breeder hens with and without yeast culture supplementation. These authors suggested that the inconsistent responses observed among researchers could be due to the differences in genetic strain of the hens used and how they respond to the feed additive, as well as not always identifying the yeast products being included, leaving it difficult to make comparisons between experiments. Response to the yeast culture, like any feed additive, depends on inclusion levels, duration of additive inclusion, management conditions, other stresses on the animal, and sufficiency of nutrient levels in the diet. The lack of breeder x progeny interaction response could be due to adequate management and environmental conditions at both the breeder and progeny, level, or the short duration of supplementation of only two weeks at the breeder hen level. Huff *et al*. (2007) suggested the age of the breeder hen can influence the response to feed additives and should be considered when designing studies to evaluate such products.

A likely explanation related to the present study is the short time period that the hens were supplemented. In the present study, the breeder hens were only supplemented during the first two weeks of lay. In most studies evaluating nutrient transfer from hen to egg, the hens were supplemented with dietary treatments several weeks prior to the onset of lay (Yalcin *et al*., 2008; Surai, 2000; Latour *et al*., 1998). Galobart *et al*. (2002) concluded it takes at least two to four weeks of dietary supplementation to the hen before stable levels of a supplement will be deposited in the eggs laid, as was demonstrated with α-Tocopherol supplementation. It is not likely that there was adequate time to pass a nutritive effect on to progeny because there was not time for the eggs to be enriched. Since we did not observe a carry-over effect of the breeder treatment to the progeny, the focus for the remainder of this discussion will be primarily on the performance of the progeny.

In the present study, an improvement was seen in the performance of the male progeny with the addition of the yeast fermentation product as seen in Tables 2 to 4. The toms receiving the yeast fermentation product had higher body weights (BW) up to 14 weeks of age than the birds fed the non-supplemented diet. This difference in BW was not seen for the remainder of the study (14-20 weeks).

The higher BW observed in the yeast fermentation product-fed birds corresponded with higher feed intake measured in these same birds up to 14 weeks of age. Feed intake is highly linked to growth; the more a bird eats, the more it will grow. Dietary inclusion of the yeast fermentation product could act as a feed stimulant, causing increased daily feed intake, and as a result increased BW gain (Shen *et al*., 2009; van Heugten *et al*., 2003). Stress, due to enteric issues or environmental heat, can cause a reduction in feed intake (Ferket and Gernat, 2006). The present study took place from December to April. The toms were 14 weeks of age in the middle to end of March when the average daily temperatures are starting to increase. After 14 weeks, there was no longer a difference in feed intake observed. As a response to the increasing temperatures, the birds could have reduced their feed intake in an effort to reduce their metabolic heat production. The average daily gain (ADG) of turkey toms peaks at about 100 days of age (Hybrid). This relative reduction, or slowing, in daily feed intake could be a result of the growth rate having peaked and then decreased.

In this study, there was an improvement in feed conversion ratio (FCR) at 3 and 14 weeks of age with the dietary inclusion of the yeast fermentation product. The improvement in FCR during the first 3 weeks could be a result of improved digestive capacity and nutrient absorption. Enteric stress can interfere with nutrient digestion and absorption (Hoerr, 1998), especially among young birds that have an immature digestive system relative to older birds (Bedford, 1996). Better digestibility could be an indication of improved gut health. Similar to the early treatment response on FCR, the improvement of FCR observed about 14 weeks of age could indicate less enteric distress experienced by the toms fed the yeast fermentation product. Around this age, tom turkeys often have a poorer FCR due to their slowing rate of ADG. The toms in the current study showed an improvement in FCR with the yeast fermentation product supplementation, which could be indicative of improved nutrient digestibility. This could be due to less enteric stress, or an advantage from the yeast fermentation product, which helped the birds tolerate stress better. Improved nutrient digestibility efficiency results in better FCR because the bird has to consume less feed to gain the same amount of BW.

No effect for dietary inclusion of the yeast fermentation product was observed on progeny performance from 16-20 weeks. A similar response was seen in a study by Paiva *et al*. (2010), who conducted a study with turkey hens raised to 9 weeks of age with several inclusion levels of the yeast fermentation product. The birds receiving the yeast fermentation product had higher BW gain at four and six weeks of age as compared to control birds. By 9 weeks of age, a significant difference was no longer observed (Paiva *et al.*, 2010). The peak of ADG for turkey hens is about 75 days of age, about 25 days earlier than in toms. Thus, Paiva *et al*. (2010) observed a disappearance of response to dietary yeast fermentation product supplementation about 25 days earlier in production than observed in the present study, indicating a similar response relative to the allometric growth of turkeys. With the inclusion of the yeast fermentation product in earlier diets an improvement in performance was observed, but later in the study a difference from the control treatments was not seen. The lack of performance difference between treatments in the finishing period could be due to the control birds catching up to the level of the yeast fermentation product-fed birds, mainly because the yeast fermentation product birds were physiologically more advanced and their growth rate began to plateau sooner. Moreover, the yeast fermentation product may help in stimulating early feed consumption leading to an improvement in early growth performance and advanced physiological development.

There is a strong relationship between BW and breast meat yield (Bagley, 2010). In the poultry industry, BW gain is a priority for most producers because body weight is often a good indicator of breast meat yield. Based on the analysis of the carcass characteristics, there was a response early on to the yeast fermentation product supplementation, but this difference disappeared by 6 weeks (Table 5). At 2 weeks of age, those toms receiving the yeast fermentation product had a higher breast meat yield than the controls (higher *Pectoralis major* and *minor*). During this starter period, the yeast fermentation product-fed birds also had a superior FCR. The higher percentage breast meat yield and improved FCR seen during this starter period could mean that more nutrient resources were available for breast meat development. During times of stress, a decrease in breast meat yield can be observed (Klasing *et al*., 1987). During periods of negative energy balance, a common problem associated with disease is catabolism of muscle proteins to supply amino acids for the synthesis of hepatic acute phase proteins (APP) and serve as a source of energy (Korver, 2006; Gruys *et al*., 2005). Acute phase reactions, related to inflammatory response, are known to be counter-productive to muscle anabolism (Gruys *et al*., 2005). Muscle anabolism is a concern in poultry because of meat production, specifically breast meat yield. Higher breast meat yield could be an indicator of less inflammatory response and thus better gut health. Although the benefit of the yeast fermentation product supplementation on early breast muscle development was not observed to persist from 6 until 20 weeks of age, this could be due to an insufficient sample size and increased variability of the breast tissue weight.

There was no effect on carcass analysis observed at 6 weeks of age (Tables 5 to 7). At 12 weeks of age, those toms receiving the yeast fermentation product had lower percentage of viscera weight (7.92 versus 8.57, P=0.0439) but higher blood and featherweight (5.01 versus 4.65, P=0.0462). No other effect on carcass analysis was seen at 12 weeks or for the remainder of the study.

In turkey production, breast meat generates 60 to 70% of the income of the bird (Bagley, 2010), making meat quality important to the industry. Pale, soft, exudative (PSE) breast meat is harder to process and more prone to drip loss, which can affect shelf life of the meat (Aslam *et al*., 2011; Woelfel *et al*., 2002). PSE is caused by a rapid drop in pH of the meat soon after death, before the meat is chilled, causing denaturation of the muscle proteins responsible for the color of the muscle and its ability to hold water during storage and cooking (Owens *et al*., 2009; Barbut *et al*., 2008). It has been proposed that *Saccharomyces cerevisiae* could have antioxidant properties (Lingnert *et al*., 1989). Barbut *et al.* (2008) suggested that oxidation in the meat after slaughter could lead to the rapid drop in pH and protein degradation leading to PSE. Zhang *et al*. (2005) showed improved oxidative stability in breast and thigh meat of broiler chickens that had been fed diets supplemented with *Saccharomyces cerevisiae* as compared to those fed a standard corn-soy diet. In a study by Milewski and Zaleska (2011), the meat quality was evaluated in lambs fed concentrate supplemented or not supplemented with a *Saccharomyces cerevisiae* product. The meat of the lambs supplemented with the dried yeast product had lower L* value, higher a* value and a higher water-holding capacity. In this study there was no difference in drip loss at 7 days of storage for breeder level effect or breeder*commercial interaction (Table 9). However, the toms receiving the yeast fermentation product lost more fluid than the control birds after 7 days of storage (2.44% loss vs. 3.39%, p=0.0079). The greater amount of fluid loss by the toms receiving the yeast fermentation product could be related to the faster growth rate in these birds, or higher moisture content of the meat to start with. There is a positive correlation with percent drip loss and body weight traits in turkeys at later ages, which could be due to the increase in glycogen contents of the breast muscle (Aslam *et al*., 2011). In an analysis of the genetic and phenotypic correlations related to meat quality in turkeys, rapid growth has been associated with greater incidences of PSE and subsequently increased drip loss (Aslam *et al*., 2011; Owens *et al*., 2009). The toms receiving the yeast fermentation product had higher feed consumption and higher body weights than the non-supplemented birds through 14 weeks, indicating faster growth. The progeny of breeders who received the yeast fermentation product had higher a* (redness) and b* (yellowness) values at 2 days of storage; however, there were no treatment differences in meat color values at 7 days of storage. Although the treatments receiving the yeast fermentation product showed greater fluid (drip) loss, the color of the meat did not indicate PSE. The toms supplemented with the yeast fermentation product had higher body weights through the growing period but this was not translated into greater meat production or improvement in overall meat quality.

Our objective in this study was to see if the feeding of yeast fermentation products to the breeder hens could have a lasting effect on their progeny. A carry-over effect from the early supplementation of Original XPC™ to breeder hens on the growth performance of their male progeny was not observed. However, we did observe an advantage in growth performance with the supplementation of Original XPC™ to the progeny. Although, no direct measurements were taken to determine enteric health of the male progeny, higher BW, higher breast meat yield, and lower FCR during the starter period indicate improved nutrient absorption with the supplementation of Original XPC™ to young poults.

Conclusion and Applications

1. Dietary supplementation of 0.075% Original XPC to turkey breeders during the first two weeks of lay does not significantly affect progeny growth performance.
2. Dietary supplementation of 0.125% Original XPC significantly improves growth performance (increased feed intake, body weight gain, and FCR) and breast muscle development during the brooding phase (1-21 days of age).
3. Dietary supplementation of Original XPC (0.125% XPC weeks 1 to 6; 0.0625% XPC weeks 7 to 20) significantly improves growth performance of growing turkeys through at least 14 weeks of age, but persistence of this positive response is dependent upon management conditions.

References

Applegate, T. J. 2002. Reproductive maturity of turkey hens: egg composition, embryonic growth and hatchling transition. Avian Poult Biol Rev. 13:31-41.

Aslam, M. L, J. W. M. Bastiaansen, R. P. M. A. Crooijmans, B. J. Ducro, A. Vereijken, and M. A. M. Groenen. 2011. Genetic variances, heritabilities and maternal effects on body weight, breast meat yield, meat quality traits and the shape of the growth curve in turkey birds. BMC Genetics. 12:14.

Bagley, L. 2010. Breast meat yield in turkeys. Nutrition Line. Diamond V.

Barbut, S., A. A. Sosnicki, S. M. Lonergan, T. Knapp, D. C. Ciobanu, L. J. Gatcliffe, E. Huff-Lonergan, and E. W. Wilson. 2008. Progress in reducing the pale, soft and exudatative (PSE) problem in pork and poultry meat. Meat Sci. 79:46-63.

Bedford, M. R. 1996. Interaction between ingested feed and the digestive system in poultry. J. Appl. Poult. Res. 5:86-95.

Bradley, G. L., and T. F. Savage. 1995. The influences of pre-incubation storage duration and genotype on the hatchability of Medium White turkey eggs from hens fed a diet containing a yeast culture of *Saccharomyces cerevisiae*. Anim Feed Sci and Tech. 51:141-152.

Brake, J. 1991. Research note: Lack of effect of a live yeast culture on broiler breeder and progeny performance. Poult Sci. 70:1037-1039.

Casewell M., C. Friis, E. Marco, P. McMullin, and I. Phillips. 2003. The European ban on growth-promoting antibiotics and emerging consequences for human and animal health. J. Antimicob. Chemother. 52:159-161.

Dibner, J. J., and J. D. Richards. 2005. Antibiotic growth promoters in agriculture: History and mode of action. Poult. Sci. 84:634-643.

Ferket, P. R., and A. G. Gernat. 2006. Factors that affect feed intake of meat birds: A review. Int. J. Poult. Sci. 5:905-911.

Firman, J. D., and D. Moore. 2009. Effects of dietary inclusion of a *Saccharomyces cerevisiae* fermentation product on performance and gut characteristics of male turkeys to market weight. Poult. Sci. 88 (Suppl.1): 96.

Francois, J. and, J. L. Parrou. 2001. Reserve carbohydtares membolism in the yeast Saccharomyces cerevisiae. FEMS Microbiol. Rev. 25: 125-145.

Fritts, C.A., and P. W. Waldroup. 2003. Evaluation of Bio-Mos® mannan oligosaccharide as a replacement for growth promoting antibiotics in diets for turkeys. Int. J. Poult. Sci. 2:19-22.

Galobart, J., A. C. Barroeta, L. Cortinas, M. D. Baucells, and R. Codony. 2002. Accumulation of α-Tocopherol in eggs enriched with ω-3 and ω-6 polyunsaturated fatty acids. Poult. Sci. 81:1873-1876.

Gao, J., H. J. Zhang, S. G. Wu, S. H. Yu, I. Yoon, D. Moore, Y. P. Gao, H. J. Yan, and G. H. Qi. 2009. Effect of *Saccharomyces cerevisiae* fermentation product on immune functions of broilers challenged with *Eimeria tenella*. Poult Sci. 88: 2141-2151.

Griggs, J. P., and J. P. Jacob. 2005. Alternatives to antibiotics for organic poultry production. J. Appl. Poult. Res. 14:750-756.

Gruys, E., M. J. M. Toussaint, N. Upragarin, A. M. Van Ederen, A. A. Adewuyi, D. Candiani, T. K. A. Nguyen, J. Sabeckiene. 2005. Acute phase reactants, challenge in the near future of animal production and veterinary medicine. J. Zhejiang Univ Sci. 6B:941-947.

Hayat, J., T. F. Savage, L. W. Mirosh. 1993. The reproductive performance of two genetically distinct lines of medium white turkey hens when fed breeder diets with and without a yeast culture containing *Saccharomyces cerevisiae*. Anim Feed Sci Tech. 43:291-301.

Hoerr, F. J. 1998. Pathogenesis of enteric diseases. Poult. Sci. 77:1150-1155.

Huff, G.R., W. E. Huff, N. C. Rath, F. Solis de los Santos, M. B. Farnell, and A. M. Donoghue. 2007. Influence of hen age on the response of turkey poults to a cold stress, *Escherichia coli* challenge, and treatment with a yeast extract antibiotic alternative. Poult. Sci. 86: 636-642.

Hybrid Turkeys. A Hendix Genetic Company. <www.hybridturkeys.com>.

Klasing, K. C., D. E. Laurin, R. K. Peng, and D. M. Fry. 1987. Immunologically mediated growth depression in chicks: Influence of feed intake, corticosterone and interleukin-1. J. Nutr. 117:1629-1637.

Korver, D. R. 2006. Overview of the immune dynamics of the digestive system. J. Appl. Poult. Res. 15:123-135.

Latour, M. A., E. D. Peebles, S. M. Doyle, T. Pansky, T. W. Smith, and C. R. Boyle. 1998. Broiler breeder age and dietary fat influence the yolk fatty acid profiles of fresh eggs and newly hatched chicks. Poult. Sci. 77:47-53.

Lingnert, H., G. Akesson, and C. E. Eriksson. 1989. Antioxidative effect of superoxide dismutase from *Saccharomyces cerevisiae* in model systems. J. Agric. Food Chem. 37:32-28.

Lohren, U., A. Ricci, and T. S. Cummings. 2008. Guidelines for antimicrobial use in poultry. Guidelines to antimicrobial use in animals. 8:126-142.

Milewski, S., and B. Zaleska. 2011. The effect of dietary supplementation with *Saccharomyces cerevisiae* dried yeast on lambs meat quality. J Anim Feed Sci. 20:537-545.

Owens, C. M., C. Z. Alvarado, and A. R. Sams. 2009. Research developments in pale, soft, exudative turkey meat in North America. Poult. Sci. 88:1513-1517.

Paiva, D.M., C. L. Walk, R. Lehman, J. R. Sottosanti, C. F. Honaker, D. T. Moore, and A. P. McElroy. 2010. Turkey response to the inclusion of a *Saccharomyces cerevisiae* fermentation product, Original XPC™, in antibiotic free diets following a coccidia vaccination. Poult. Sci. 89 (E-Suppl. 1): 283-284.

Parks, C. W., J. L. Grimes, P. R. Ferket, and A. S. Fairchild. 2001. The effect of mannanoligosaccharides, Bambermycins, and Virginianmycin on performance of large white male market turkeys. Poult. Sci. 80:718-723.

Phillips, I. 1999. Assessing the evidence that antibiotic growth promoters influence human infections. J. Hosp. Infect. 43:173-178.

Plyler, J., and S. Watkins. 2008. Gut Health: Is anything more important in turkey production? University of Arkansas Avian Advice. 10 (1):4-7.

Price, K. L., H. R. Totty, H. B. Lee, M. D. Utt, G. E. Fitzner, I. Yoon, M. A. Ponder, and J. Escobar. 2010. Use of *Saccharomyces cerevisiae* fermentation product on growth performance and microbiota of weaned pigs during *Salmonella* infection. J. Anim. Sci. 88:3896-3908.

Shen, Y. B., J. A. Carroll, I. Yoon, R. D. Mateo, and S. W. Kim. 2011. Effects of supplementing *Saccharomyces cerevisiae* fermentation product in sow diets on performance of sows and nursing piglets. J. Anim. Sci. 89(8):2462-2471.

Shen, Y. B., X. S. Piao, S.W. Kim, L. Wang, P. Liu, I. Yoon, and Y.G. Zhen. 2009. Effects of yeast culture supplementation on growth performance, intestinal health, and immune response of nursery pigs. J. Anim. Sci. 87:2614-2624.

Sims, M. D., K. A. Dawson, K. E. Newman, P. Spring, and D. M. Hooge. 2004. Effects of dietary mannan oligosaccharide, bacitracin methylene disalicylate, or both on the live performance and intestinal microbiology of turkeys. Poult. Sci. 83:1148-1154.

Stone, C. W. 1998. Yeast products in the feed industry: A practical guide for feed professionals. Diamond V Mills, Inc. Cedar Rapids, IA, USA. Page 1-16.

Surai, P. F. 2000. Effect of selenium and vitamin E content of the maternal diet on the antioxidant system of the yolk and the developing chick. Brit Poult Sci. 41:235-243.

van Heugten, E., D. W. Funderburke, and K. L. Dorton. 2003. Growth performance, nutrient digestibility, and fecal microflora in weanling pigs fed live yeast. J. Anim. Sci. 81:1004-1012.

Wang, Z. G., X. J. Pan, Z. Q. Peng, R. Q. Zhao, and G. H. Zhou. 2009. Methionine and selenium yeast supplementation of the maternal diets affects color, water-holding capacity, and oxidative stability of their male offspring meat at the early stage. Poult Sci. 88:1096-1101.

WHO (World Health Organization). 2003. Impacts of antimicrobial growth promoter termination in Denmark.

Woelfel, R. L., C. M. Owens, E. M. Hirschler, R. Martinez-Dawson, and A. R. Sams. 2002. The characterization and incidence of pale, soft, and exudative broiler meat in a commercial processing plant. Poult. Sci. 81: 579-584.

Yadgary, L., A. Cahaner, O. Kedar, and Z. Uni. 2010. Yolk sac nutrient composition and fat uptake in late-term embryos in eggs from young and old breeder hens. Poult. Sci. 89:2441-2452.

Yalcin, S., B. Ozsoy, H. Erol, and S. Yalcin. 2008. Yeast culture supplementation to laying hen diets containing soybean meal or sunflower seed meal and its effect on performance, egg quality traits, and blood chemistry. J. Appl. Poult. Res. 17:229-236.

Yalcin, S., S. Ozkan, M. Cabuk, J. Buyse, E. Decuypere, and P. B. Siegel. 2005. Pre- and postnatal conditioning induced thermotolerance on body weight, physiological responses and relative asymmetry of broilers originating from young and old breeder flocks. Poult. Sci. 84:967-976.

Zhang, A. W., B. D. Lee, S. K. Lee, K. W. Lee, G. H. An, K. B. Song, and C. H. Lee. 2005. Effects of yeast (*Saccharomyces cerevisiae*) cell components on growth performance, meat quality, and ileal mucosa development of broiler chicks. Poult Sci. 84:1015-1021.

Table 1: Composition and nutrient content of dietary feeding phases of experimental diet in place of corn fed to turkey toms placement to 20 weeks of age.

Dietary phase	**Prestarter**	**Starter**	**Developer 1**	**Developer 2**	**Grower 1**	**Grower 2**	**Finisher 1**	**Finisher 2**
Weeks of age	0-3	3-6	6-9	9-12	12-14	14-16	16-18	18-20
Ingredients				---------- *% of Total diet* ----------				
Corn	37.77	47.98	50.76	53.47	61.44	66.51	66.71	68.38
Soybean Mean 48	48.46	38.63	35.14	32.02	23.80	18.25	16.87	14.79
Poultry Meal	5.00	5.00	5.00	5.00	5.00	5.00	5.00	5.00
Poultry Fat	3.14	2.59	3.82	4.97	5.56	6.46	8.02	8.64
Dicalcium Phosphate 18.5	3.00	2.83	2.60	2.09	1.90	1.69	1.60	1.46
Limestone	0.94	1.07	0.96	1.00	0.91	0.92	0.75	0.75
Alimet	0.37	0.39	0.32	0.25	0.24	0.14	0.17	0.20
Salt (NaCl)	0.33	0.32	0.33	0.33	0.33	0.33	0.33	0.33
Trace mineral premix[1]	0.20	0.20	0.20	0.20	0.15	0.15	0.13	0.08
Choline Chloride 60%	0.20	0.23	0.22	0.17	0.16	0.09	0.05	0.05
L-Lysine	0.16	0.32	0.32	0.18	0.22	0.16	0.10	0.05
Vitamin premix[2]	0.15	0.10	0.10	0.08	0.08	0.08	0.05	0.05
Sodium Selenite premix[3]	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Orginal XPC™ [4]	0.125	0.125	0.0625	0.0625	0.0625	0.0625	0.0625	0.0625
L-Threonine	0.00	0.06	0.02	0.02	0.00	0.00	0.00	0.00
Nutrient analysis								
ME Poultry, Kcal/kg	2950	3000	3100	3200	3300	3400	3500	3550
Crude protein, %	29.50	26.00	24.44	23.00	19.60	17.29	16.50	15.50
Crude fat, %	5.54	5.20	6.46	7.63	8.35	9.34	10.84	11.51
Calcium, %	1.45	1.40	1.30	1.20	1.10	1.00	0.95	0.90
Available Phosphorus, %	0.80	0.75	0.70	0.60	0.55	0.50	0.48	0.45
Sodium, %	0.18	0.18	0.18	0.18	0.18	0.18	0.18	0.18
Total Lysine, %	1.85	1.70	1.60	1.4	1.20	1.00	0.90	0.80
Total Met +Cys, %	1.15	1.15	1.05	0.95	0.85	0.70	0.70	0.70
Threonine, %	1.15	1.05	0.95	0.90	0.74	0.65	0.62	0.58
Choline, mg/kg	2720	2700	2570	2570	2025	1565	1285	1285

[1] Each kilogram of mineral premix (0.1% inclusion) supplied the following per kg of complete feed: 60 mg Zn as $ZnSO_4H_2O$; 60 mg Mn as $MnSO_4H_2O$; 40 mg Fe as $FeSO_4H_2O$; 5 mg Cu as $CuSO_4$; 1.25 mg I as $Ca(IO_3)2$; 1 mg Co as $CoSO_4$.

[2] Each kilogram of vitamin premix (0.1% inclusion) supplied the following per kg of complete feed: 13,200 IU vitamin A; 4,000 IU cholecalciferol; 66 IU alpha-tocopherol; 110 mg niacin; 22mg pantothenic acid; 13.2 mg riboflavin; 8 mg pyridoxine; 4 mg menadione; 2.2 mg folic acid; 4mg thiamin; 0.253 mg biotin; 0.04mg vitamin B_{12}; 100 mg ethoxyquin.

[3] $NaSeO_3$ premix provided 0.3mg Se/kg of complete feed.

[4] In dietary treatments not receiving Original XPC™, corn was used as a replacement.

Table 2: Effect of dietary supplementation of Original XPC™ to breeders and commercial level progeny on male progeny Body Weight until 20 weeks of age.[1]

		Weeks of Age							
% Dietary XPC		3	6	9	12	14	16	18	20
Breeder	Progeny	(kg)							
0	0	0.565	2.49	5.58	10.15	13.18	16.69	19.77	20.82
0	0.125/0.0625[3]	0.626	2.65	6.16	10.53	13.54	16.95	19.84	20.91
0.075	0	0.562	2.46	5.55	10.15	13.18	16.60	19.60	20.84
0.075	0.125/0.0625	0.639	2.65	6.07	10.51	13.45	16.66	19.55	20.77
Breeder Main Effect									
0% XPC		0.596	2.57	5.87	10.34	13.36	16.82	19.80	20.86
0.075% XPC		0.601	2.56	5.81	10.33	13.31	16.63	19.57	20.80
Progeny Main Effect									
0% XPC		0.564	2.47	5.57	10.15	13.18	16.64	19.68	20.83
0.125/0.0625% XPC		0.633	2.65	6.11	10.52	13.50	16.81	19.69	20.84
Source of Variation		*P values*							
Breeder		0.5537	0.5957	0.3281	0.8823	0.6136	0.1110	0.0717	0.6926
Progeny		<0.0001	<0.0001	<0.0001	0.0002	0.0041	0.1680	0.9361	0.9496
Breeder x Progeny		0.3430	0.6527	0.6383	0.9364	0.6481	0.4065	0.6354	0.5977
SEM (35)[2]		0.008	0.029	0.065	0.087	0.099	0.113	0.121	0.149

[1] Values are means of 10 replicate pens of *ca.* 15 toms per pen.
[2] SEM (35)= Standard error of the mean with 35 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).

Table 3: Effect of dietary supplementation of Original XPC™ to breeders and commercial level progeny on male progeny average cumulative Feed Intake until 20 weeks of age.[1]

		Weeks of Age							
% Dietary XPC		**3**	**6**	**9**	**12**	**14**	**16**	**18**	**20**
Breeder	**Progeny**	---- *(kg)* ----							
0	0	0.871	2.81	9.23	19.52	28.46	37.67	45.99	53.12
0	0.125/0.0625[3]	0.907	3.05	10.40	20.87	29.02	38.17	46.50	53.53
0.075	0	0.859	2.82	9.53	19.79	27.79	37.89	45.83	53.30
0.075	0.125/0.0625	0.920	3.05	10.64	21.62	29.69	38.32	47.09	53.72
Breeder Main Effect									
0% XPC		0.889	2.93	9.81	20.19	28.74	37.92	46.25	53.32
0.075% XPC		0.890	2.93	10.08	20.70	28.74	38.11	46.46	53.51
Progeny Main Effect									
0% XPC		0.865	2.82	9.38	19.65	28.13	37.78	45.91	53.21
0.125/0.0625% XPC		0.914	3.05	10.52	21.24	29.35	38.24	46.80	53.62
Source of Variation		----*P values*----							
Breeder		0.9729	0.9362	0.1770	0.2455	0.9994	0.7847	0.7575	0.8153
Progeny		<0.0001	<0.0001	<0.0001	0.0007	0.0254	0.5036	0.2027	0.6076
Breeder x Progeny		0.1789	0.8681	0.8775	0.5851	0.2092	0.9573	0.5862	0.9926
SEM (35)[2]		0.009	0.042	0.062	0.004	0.517	0.659	0.647	0.776

[1] Values are means of 10 replicate pens of *ca.* 15 toms per pen.
[2] SEM (35)= Standard error of the mean with 35 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).
[a,b] Means within a column with no common superscript are significantly different (P≤0.05).

Table 4: Effect of dietary supplementation of Original XPC™ to breeders and commercial level progeny on male progeny cumulative Feed Conversion Ratio adjusted for mortality until 20 weeks of age.[1]

		Weeks of Age							
% Dietary XPC		**3**	**6**	**9**	**12**	**14**	**16**	**18**	**20**
Breeder	**Progeny**	*(kg Feed Intake : kg Body Weight Gain)*							
0	0	1.72	1.53	1.66	2.00	2.23	2.35	2.49	2.78
0	0.125/0.0625[3]	1.63	1.52	1.71	1.99	2.15	2.36	2.59	2.87
0.075	0	1.70	1.53	1.68	1.92	2.18	2.35	2.50	2.88
0.075	0.125/0.0625	1.60	1.53	1.70	2.01	2.10	2.32	2.52	2.91
Breeder Main Effect									
0% XPC		1.67	1.53	1.68	1.99	2.19	2.36	2.54	2.82
0.075% XPC		1.65	1.52	1.69	1.97	2.14	2.34	2.51	2.89
Progeny Main Effect									
0% XPC		1.71	1.53	1.67	1.96	2.21 [b]	2.35	2.49	2.83
0.125/0.0625% XPC		1.62	1.53	1.70	2.00	2.12 [a]	2.34	2.55	2.89
Source of Variation		*P values*							
Breeder		0.2771	0.2255	0.8523	0.6896	0.1049	0.6443	0.6304	0.2904
Progeny		0.0001	0.6041	0.2306	0.5341	0.0130	0.7913	0.3365	0.3549
Breeder x Progeny		0.6160	0.4566	0.6792	0.3993	0.9942	0.7394	0.5424	0.6356
SEM (35)[2]		0.021	0.008	0.025	0.061	0.030	0.043	0.060	0.062

[1] Values are means of 10 replicate pens of *ca.* 15 toms per pen.
[2] SEM (35)= Standard error of the mean with 35 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).
[a,b] Means within a column with no common superscript are significantly different ($P \leq 0.05$).

Table 5: Effect of dietary supplementation of Original XPC™ to breeders and commercial level progeny on male progeny Breast Meat Yield until 20 weeks of age.[1]

		Weeks of Age						
% Dietary XPC		**2**	**6**	**12**	**14**	**16**	**18**	**20**
Breeder	**Progeny**	---------------------------------- *(% Breast Meat Yield[4])* ----------------------------------						
0	0	12.29	19.23	23.35	23.85	22.56	24.78	25.24
0	0.125/0.0625[3]	15.17	18.39	23.77	22.26	24.26	24.52	25.85
0.075	0	12.34	19.71	24.02	23.11	23.95	25.71	25.90
0.075	0.125/0.0625	15.28	18.83	23.90	22.78	23.58	25.08	27.26
Breeder Main Effect								
0% XPC		13.73	18.81	23.56	23.06	23.41	24.65	25.54
0.075% XPC		13.81	19.27	23.96	22.95	23.76	25.39	26.58
Progeny Main Effect								
0% XPC		12.31	19.46	23.68	23.48	23.25	25.24	25.57
0.125/0.0625% XPC		15.22	18.61	23.83	22.52	23.92	24.80	26.56
Source of Variation		---*P values*--						
Breeder		0.841	0.487	0.486	0.848	0.485	0.336	0.174
Progeny		<0.0001	0.206	0.791	0.118	0.199	0.564	0.192
Breeder x Progeny		0.940	0.976	0.633	0.289	0.056	0.804	0.612
SEM (12)[2]		0.204	0.321	0.279	0.285	0.244	0.370	0.358

[1]Values are means of 4 toms per treatment; a total of 16 toms.
[2] SEM (12)= Standard error of the mean with 12 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).
[4] % Breast meat yield=weight of breast meat (includes both *Pectoralis major* and *Pectoralis minor*) as a percentage of carcass weight without blood and feathers. The carcass still had viscera, head, and feet.
[a,b] Means within a column with no common superscript are significantly different ($P \leq 0.05$).

Table 6: Effect of dietary supplementation of Original XPC™ to breeders and commercial level progeny on male progeny percent viscera until 20 weeks of age.[1]

% Dietary XPC		Weeks of Age					
		6	12	14	16	18	20
Breeder	Progeny	(% Viscera[4])					
0	0	10.30	8.72	8.12	6.31	6.77	5.83
0	0.125/0.0625[3]	9.90	8.16	8.41	6.26	6.74	5.69
0.075	0	11.17	8.42	8.21	6.91	6.32	5.90
0.075	0.125/0.0625	10.64	7.68	7.10	6.49	6.80	5.74
Breeder Main Effect							
0% XPC		10.10	8.44	8.27	6.28^{b}	6.75	5.76
0.075% XPC		10.90	8.04	7.65	6.70^{a}	6.56	5.82
Progeny Main Effect							
0% XPC		10.73	8.57^{a}	8.17	6.61	6.55	5.86
0.125/0.0625% XPC		10.27	7.92^{b}	7.75	6.37	6.77	5.71
Source of Variation		*P values*					
Breeder		0.055	0.194	0.060	0.036	0.670	0.819
Progeny		0.241	0.044	0.188	0.209	0.626	0.589
Breeder x Progeny		0.859	0.757	0.034	0.322	0.570	0.975
SEM (12)[2]		0.189	0.144	0.148	0.089	0.220	0.136

[1]Values are means of 4 toms per treatment; a total of 16 toms.
[2] SEM (12)= Standard error of the mean with 12 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).
[4] % Viscera=weight of visceral content as a percentage of carcass weight without blood and feathers. The carcass still had head and feet.
[a,b] Means within a column with no common superscript are significantly different (P≤0.05).

Table 7: Effect of dietary supplementation of Original XPC™ to breeders and commercial level progeny on male progeny percent Blood and Feathers until 20 weeks of age.[1]

% Dietary XPC		Weeks of Age					
		6	12	14	16	18	20
Breeder	**Progeny**	------------------------------- *(% Blood and Feathers[4])* -------------------------------					
0	0	7.31	4.43	3.69	2.82	3.65	2.86
0	0.125/0.0625[3]	7.82	5.05	4.09	3.63	2.87	2.41
0.075	0	7.65	4.89	4.61	3.51	4.29	3.06
0.075	0.125/0.0625	7.79	4.97	4.44	3.25	6.93	2.99
Breeder Main Effect							
0% XPC		7.56	4.74	3.89[b]	3.23	3.26	2.63[b]
0.075% XPC		7.72	4.93	4.52[a]	3.38	5.61	3.02[a]
Progeny Main Effect							
0% XPC		7.48	4.65[b]	4.14	3.16	3.97	2.96
0.125/0.0625% XPC		7.80	5.01[a]	4.26	3.44	4.90	2.70
Source of Variation		--*P values*--					
Breeder		0.790	0.260	0.005	0.566	0.247	0.032
Progeny		0.587	0.046	0.544	0.300	0.640	0.130
Breeder x Progeny		0.761	0.109	0.154	0.067	0.393	0.264
SEM (12)[2]		0.289	0.079	0.092	0.129	0.967	0.079

[1]Values are means of 4 toms per treatment; a total of 16 toms.
[2] SEM (12)= Standard error of the mean with 12 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).
[4] % Blood and Feathers= 1 –(weight of exsanguinated and plucked bird in kg / weight of live bird in kg) X 100.

Table 8: Effect of breeder and commercial turkey dietary supplementation of Original XPC on toms carcass parts yield at 20 weeks of age.[1]

% Dietary XPC		Cold Carcass Wt.[4]	Neck	Fat Pad	Thigh	Drum	Breast Skin	Wings	*Pectoralis major*	*Pectoralis minor*	Rack
		(kg)	------ % of Cold Carcass weight ------								
Breeder	**Progeny**										
0	0	18.43	2.68	0.48	15.18	11.57	4.30	9.07	27.76	4.63	23.72
0	0.125/0.0625[3]	18.84	2.70	0.61	15.00	12.02	4.67	8.78	27.38	4.41	24.03
0.075	0	19.11	2.62	0.54	15.10	11.43	4.50	8.87	28.00	4.57	23.44
0.075	0.125/0.0625	18.66	2.64	0.49	15.13	11.46	4.40	8.84	27.76	4.61	23.73
Breeder											
0%		18.63	2.69	0.53	15.09	11.79	4.48	8.92	27.57	4.52	23.87
0.075%		18.88	2.63	0.51	15.12	11.44	4.45	8.86	27.88	4.59	23.59
Progeny											
0%		18.77	2.65	0.50	15.14	11.50	4.40	8.97	27.88	4.59	23.58
0.125/0.0625%		18.75	2.67	0.54	15.07	11.73	4.53	8.81	27.57	4.52	23.87
Source of Variation		------ ***P-values*** ------									
Breeder		0.3200	0.6239	0.6726	0.8900	0.0117	0.8487	0.5579	0.3055	0.3788	0.1919
Progeny		0.9418	0.8615	0.5346	0.7197	0.0831	0.4661	0.1757	0.3025	0.3196	0.1782
Breeder x Progeny		0.0931	0.9813	0.1664	0.5942	0.1200	0.2230	0.2516	0.8060	0.1171	0.9749
SEM (68)[2]		0.126	0.062	0.032	0.096	0.067	0.095	0.057	0.149	0.041	0.111

[1]Values are means of *ca*. 17 toms per treatment, in a total of 72 toms.
[2] SEM (68)= Standard error of the mean with 68 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).
[4]Cold Carcass weight defined as bird weight after processing (exsanguination, plucking, evisceration, removal of head and feet) and chilling in a chill tank for 12 hours.

Table 9: Drip loss and breast meat filet color evaluation at 20 weeks of turkey toms with and without supplementation of Original XPC.

		Drip Loss	Color					
		7 days	2 days			7 days		
% Dietary XPC		%	*L**	*a**	*b**	*L**	*a**	*b**
Breeder	**Progeny**							
0	0	2.27	54.05	3.27	5.40	55.06	3.22	6.11
0	0.125/0.0625[3]	3.13	54.72	3.28	5.13	56.16	2.99	5.83
0.075	0	2.62	54.45	3.79	5.75	55.72	3.57	6.08
0.075	0.125/0.0625	3.65	55.15	3.68	5.95	55.75	3.22	6.04
Breeder Main Effect								
0%		2.70	54.38	3.28	5.26	55.73	3.10	5.97
0.075%		3.14	54.80	3.74	5.85	55.61	3.40	6.06
Progeny Main Effect								
0 %		2.44	54.25	3.53	5.58	55.39	3.40	6.09
0.125/0.0625%		3.39	54.94	3.48	5.54	55.96	3.11	5.94
Source of Variation		-- ***P-values*** --						
Breeder		0.1468	0.3673	0.0124	0.0383	0.8166	0.2236	0.8117
Progeny		0.0049	0.1407	0.7896	0.8931	0.2807	0.2259	0.6609
Breeder x Progeny		0.9911	0.9719	0.7251	0.3932	0.3055	0.7808	0.7379
SEM (68)[2]		0.16	0.45	0.17	0.27	0.51	0.23	0.36

[1]Values are means of *ca*. 17 toms per treatment, in a total of 72 toms.
[2] SEM (68) = Standard error of the mean with 68 degrees of freedom.
[3] Supplemented with Original XPC™ (0.125% weeks 1 to 6; 0.0625% weeks 7 to 20).

MANUSCRIPT II. The Effect of Dietary Inclusion of Actigen^{TM} and Virginiamycin on the Growth Performance and Ileal Morphology of Turkey Toms

ABSTRACT: Dietary supplementation of *Saccharomyces cerevisiae* mannan oligosaccharide (MOS) is commonly used to manage the enteric microbial ecosystem of animals, including turkeys. MOS is thought to inhibit the colonization of pathogens and stimulate innate immunity and mucosal barrier function. Actigen^{TM} (Alltech, Inc.), a concentrated mannose-rich oligosaccharide fraction derived from the cell wall of a specific strain of *Saccharomyces cerevisiae*, was evaluated for its effect on ileal villi morphology and the growth performance of turkey toms. Eight-hundred sixty-four male turkey poults (Hybrid Converter) were randomly distributed on day of hatch among 48 floor pens and raised in a curtain-sided house. Pens were randomly assigned to one of four dietary treatments and both feed and water were provided *ad libitum* to all birds until 20 weeks of age. All the poults were naturally challenged by placing them on used litter from a previous flock, top-dressed with clean soft pine shavings. From bird placement on day of hatch to 12 weeks of age poults were fed one of two dietary treatments: a control diet (CON) or a diet containing 400 ppm Actigen^{TM} (ACT); all diets contained monensin for the first 12 weeks. Eight birds per treatment were sampled on 6, 10, and 14 d and ileal sections dissected for histomorphometric analysis. From 13-20 weeks, birds were fed one of three dietary treatments: no additive, Actigen^{TM}, or an industry standard antibiotic (virginiamycin). Thus four dietary treatments were ultimately evaluated during the study: CC [0-20 weeks diets contain no additional additives]; AA [0-20 weeks diets contain Actigen^{TM}]; CA [0-12 weeks no additional additives, 13-20 weeks diets with Actigen^{TM}]; CV [1-12 weeks no additional

additives, 13-20 weeks diets with virginiamycin]. Body weights (BW) and feed consumption were determined at 3, 6, 9, 12, 14, 16, 18, and 20 weeks, and feed/gain (FCR) calculated. There was no statistically significant treatment effect during the first 12 weeks on BW, feed consumption, or FCR. The birds receiving virginiamycin had significantly higher BW than CC at 14 and 16 weeks and, and AA at 18 and 20 weeks ($P<0.05$). Cumulative (1-20 week) FCR among the AA treatment group was significantly higher than the CV (2.33 vs 2.26, $P=0.0168$), with CA (2.30) and CC (2.30) having intermediate values. Dietary supplementation with virginiamycin resulted in the highest 20 week BW. In contrast, Actigen^{TM} supplementation from 1 to 20 weeks resulted in inferior FCR only in comparison the CV treatment. At 10 d, ACT increased villous surface area by 18% over CON (306 vs 257 μ^2, $P<0.005$). At 14 d, ACT reduced apical width 15%, (78 vs 92 µ, $P<0.02$), base width by 15%, (173 vs 200 µ, $P<0.02$), and crypt depth by 20% (183 vs 229 µ, $P<0.005$), without effect on muscularis thickness or surface area. Therefore, Actigen^{TM} may enhance ileal mucosa health of turkey poults during the starter period and Actigen^{TM} could have the potential to be an alternative to antibiotic inclusion in turkey feed during the grower-finisher period.

(Keywords: turkey, antibiotics, Actigen^{TM}, growth, yeast, villi)

Description of Problem

Over the past several years there has been an increasing consumer demand for poultry meat produced from birds that are raised on antibiotic-free diets (Castanon, 2007; Casewell *et al*., 2003). This has been difficult for turkey producers due to the length of time it takes to raise the birds to market age, and the amount of disease and immune challenges that a turkey tom will be exposed to in 5 months (20 weeks) of production (Tabler, 2004). Most turkey producers include an antibiotic feed additive during the growing and finishing phases of production to reduce the adverse effects of enteric pathogens and intestinal microbial challenge. This enteric stress can leave the bird more susceptible to bacterial infections. Inflammation or other enteric stress can cause a reduction in feed intake and a decrease in nutrient absorption efficiency, resulting in a lack of potential growth, or a depression in growth rate (Patterson and Burkholder, 2003).

The intestinal microflora populations of adult birds are complex ecosystems, but young poultry are hatched without a stable microbial population (Yang *et al*., 2009). Since the chicks or poults in today's poultry industry are incubated and hatched in clean environments, without contact with adult birds, the young bird will pick up microbes from its environment to establish its intestinal population (Fuller, 2001). There are usually both beneficial and pathogenic bacteria within the microbial population. Some beneficial bacteria, such as *lactobacilli* and *bifidobacterial* species, seem to be sensitive to stress, which could lead to a decrease in these bacterial populations in the bird's intestines (Patterson and Burkholder, 2003), and allow pathogens to attach to the intestinal wall and proliferate. The intestinal microflora and the mucosal tissue act as barriers against invasive colonization by pathogens (Gibson *et al*., 2005; Fuller, 2001). If the intestinal lining is damaged due to enteric stress, such as necrotic enteritis from coccidiosis infection, pathogens are able to get through the protective barrier of the mucosal tissue and infect the bird, eliciting an immune response (Korver, 2006). Immune responses can cost a great deal of energy and metabolic resources that will be partitioned away from growth (Sandberg *et al*., 2007). Using a prebiotic to help these beneficial bacteria flourish can reduce the amount of enteric stress experienced and, as a result, decrease immune and inflammatory responses (Spring *et al*., 2000).

Dietary yeast products have been demonstrated as a possible antibiotic alternative in turkey feed. The dietary supplementation of *Saccharomyces cerevisiae* cell wall mannanoligosaccharide (MOS) is commonly used to manage the enteric microbial ecosystem of animals, including turkeys (Hooge, 2004). Dietary supplementation of MOS has been demonstrated to inhibit the colonization of pathogens, stimulate innate immunity, and enhance enteric mucosal barrier function in poultry (Santin *et al*., 2001; Spring *et al*., 2000; Ofoyo *et al*., 1988). Sims *et al*. (2004) reported an improvement in turkey performance with antibiotic addition (BMD) or MOS addition, and a synergistic benefit was observed when these two BMD and MOS were combined. Zhang *et al*. (2005) also saw improvement in FCR among broilers when fed diets supplemented with yeast cell wall from hatch to 3 weeks of age as compared to controls, but body weight gain was not affected. Zhang *et al*. (2005) also observed higher body weight gain (BWG) from 4 to 5 weeks of age among birds fed yeast cell wall as compared to the control birds. Mannan-oligosaccharides are not absorbed in the gastrointestinal tract of non-ruminants, but are thought to be fermented or complexed by beneficial bacteria in the gut, thus stimulating their growth and reducing the colonization of pathogenic bacteria (Stone, 1998). In addition to improving growth performance, dietary supplementation of MOS products have been shown to promote gut health (Parks *et al*., 2001b; Fritts and Waldroup, 2003; Sims *et al.*, 2004).

Good gut health and integrity is necessary for proper nutrient uptake and immune function (Janardhana *et al*., 2009; Ferket, 2003). The villi in the small intestine significantly increase the surface area of the gut wall and are critical in proper nutrient absorption. An increase in small intestine villous surface area facilitates greater nutrient absorption (Adil *et al*., 2010; Yamauchi, 2002). The intestinal villi develop mostly during the first 3 weeks (Uni *et al*., 1995) of the bird's life, so it is very important that the bird receive proper nutrition from the start to encourage proper gut development. A yeast extract product containing mannan-oligosaccharides and β-glucans (Alphamune®, Alpharma Animal Health, Antwerp, Belgium) has been shown to accelerate gastrointestinal maturation in turkey poults when included in the diet (Solis de los Santos *et al*., 2007). Solis de los Santos *et al*. (2007) measured an enhancement in ileum villous height, surface area, lamina propria thickness, crypt depth and goblet cell density in turkeys at 7 and 21 days with the inclusion of a yeast fermentation product in a standard corn-soy diet. These results were found to be more pronounced in the ileum compared to other portions of the small intestine. Other researchers (Santin *et al*., 2001; Parks *et al.*, 2001a) found similar responses in enteric development with the addition of yeast cell wall products, such as Pronady500® (Prodesa Ltda., Sao Paulo, Brazil) and Bio-Mos® (Alltech, Inc., Nicholasville, KY).

Actigen^{TM} (Alltech, Inc., Nicholasville, KY) is a concentrated form of mannan-oligosaccharide derived from yeast cell wall, which has been shown to enhance enteric barrier function, improve gut-associated immunity, and increase colonization resistance of enteric pathogens (Hooge and Connolly, 2011; Xiao *et al*., 2011). Actigen^{TM} has also been associated with the upregulation of genes involved in cellular immune response, inflammatory response, and antimicrobial responses in broiler chickens supplemented with Actigen^{TM} (Xiao *et al*., 2011). Although Actigen^{TM} has a much different mode of action than antibiotics, its beneficial response on growth performance of turkeys may position it in the market as an alternative or complementary to antibiotic growth promoters for turkeys. Actigen^{TM} is derived from the outer cell wall of the yeast, *Saccharomyces cerevisiae*, as Bio-Mos$^{®}$ (Alltech, Inc., Nicholasville, KY), but is claimed to be 2.5 times more concentrated with biological activity (Hooge and Connolly, 2011). With a mannose-rich fraction product, such as Actigen^{TM}, there should be more active compound to positively influence the colonization of beneficial bacteria and discourage the colonization of undesirable mircroflora that adversely affects enteric health and increases the risk of food safety. Perhaps with more active compound present, the positive effect on the microflora could happen more rapidly, decreasing the time it takes for the population of beneficial bacteria to increase and thus having a more stable gut microfloral population earlier in the bird's life.

In this study, poults were placed on previously used litter to present a natural microbial challenge to the young birds. As an additional challenge, this trial took place during the summer months (May-September) to also present a natural heat stress. Because of the size of the adult turkeys and the metabolic heat they produce, the turkey industry is challenged during the summer months by a decrease in growth performance (Noll, 2004; Yahav *et al*., 1998). We hypothesize that Actigen^{TM} can improve performance of turkeys fed an ionophore anticoccidial (i.e. monensin) feed additive under commercial conditions and function as an effective alternative to an antibiotic feed additive to maintain gut health and promote growth of turkeys. Dietary supplementation of Actigen^{TM} may reduce enteric health challenge and reduce the associated performance depression, particularly in response to heat stress. In the study reported herein, the growth performance during the growing and finishing phases was compared among turkeys receiving diets that contained no feed additives, an industry standard antibiotic (virginiamycin), or dietary Actigen^{TM}. In order to test our hypothesis, the objectives of this experiment were as follows: 1) to determine the effect of supplementing Actigen^{TM} with monensin on ileal mucosa morphology during the starter phase (1 to 14 days of age) and growth performance of turkeys from 1 to 12 weeks of age and subsequent carry-over effects; and 2) to compare the effects of dietary inclusion of Actigen^{TM} and the antibiotic virginiamycin on the growth performance of turkey toms from 12 to 20 weeks of age.

Materials and Methods

Birds and Housing

Eight-hundred-sixty-four male Hybrid Converter turkey poults (Hendrix Genetics, Ontario, Canada) were obtained from a commercial hatchery (Sleepy Creek Hatchery, Goldsboro, NC) on the day of hatch (May 4, 2010) and randomly assigned to 48 pens containing 18 poults per pen. At six weeks of age, the number of birds was reduced to 15 per pen to comply with appropriate stocking density according to the animal care guidelines. The toms were raised in 3.4 m^2 floor pens of a curtain-sided facility until market age at 20 weeks of age. Each replicate pen was equipped with one tube feeder and one Plasson drinker (Plasson, Ltd., Menashe, Israel) for *ad libitum* consumption of feed and water, respectively. The birds were placed on litter used by a previous flock that was top-dressed with two inches of fresh soft pine shavings. Exposure to previously used litter was intended to present a practical microbial challenge to the birds. All the birds were raised according to typical management practices. The birds received 23 hours incandescent lights from 1 to 10 days and natural lighting subsequently until the experiment was terminated at 140 days of age.

Dietary treatments

Table 10 illustrates the corn-soy based diets that were formulated to meet or exceed the nutrient recommendations for Hybrid turkey commercial 8-phase feeding program (Hybrid). All diets, including those that contained Actigen™ (Alltech, Inc., Nicholasville, KY, USA), contained the ionophore coccidiostat, sodium monensin, as Coban® 90 (Elanco, Greenfield, IN, USA) during the first 12 weeks of the study, as typically used in the commercial turkey industry. Each pen was assigned to one of four dietary treatments, with 2 starter-developer phase treatments (with or without Actigen™) from 0 to 12 weeks of age, and 3 grower-finisher phase treatments (No additive, Actigen™, or virginiamycin) from 13 to 20 weeks of age. Actigen™ (400 g/tonne) and virginiamycin (20 g/ton, as Stafac® 20, Phibro Animal Health, NJ, USA) were included in the respective diets at the supplier's recommended levels at the expense of washed builder's sand so as not to change the nutrient density of the diet. The experimental diets were provided as crumbled pellets from 0 to 6 weeks of age, and whole pellets from 6 to 20 weeks of age. Each dietary treatment was randomly assigned to 12 replicates pens within 4 blocks of 12 pens of 18 turkeys each.

Measurements

Individual body weights within each pen and feed consumption data were measured at 3, 6, 9, 12, 14, 16, 18, and 20 weeks of age. Mortalities were recorded as they occurred and their body weights were included in the calculation of adjusted pen feed conversion ratio (FCR, feed:gain).

At 6, 10, and 14 days of age, one bird was randomly selected from eight different pens from each of the two starter treatments on each sampling day and was sampled for histomorphometric analysis. The birds sampled were euthanized by cervical dislocation and the intestinal tract from Meckel's diverticulum to the ileal-cecal junction was removed from the bird. The digesta contents were removed by using a syringe to gently flush the contents from the intestine with 0.9% saline solution. A 6 to 7 centimeter section from the distal end of the ileum was cut and placed in a tube with 10% formalin for 48 hrs. The intestinal sections were then dehydrated with an alcohol-xylene sequence and embedded in paraffin. Three pieces of 5 μm thick slices were prepared from each intestinal sample and stained with hematoxylineosin. Measurements were taken in micrometers (μm) of villi height, apical width, base width, crypt depth, and muscularis thickness of 20 villi per bird, using Image Tool Software (Version 3.0, University of Texas Health Science Center, San Antonio, TX, USA), and surface area (SA) was calculated[1]. Histomorphometric measurements were done to observe the differences in early gut morphology of poults fed diets containing the Actigen™ or the unsupplemented control during the starter period.

Statistical Analysis

Growth parameter data were analyzed as a completely randomized block design, using the GLM procedure of JMP 8.0 (SAS Institute, 2008) for ANOVA. Data collected during the starter and developer phase (0 to 12 weeks), were statistically analyzed to determine differences between the Actigen™ and control treatment at $P<0.05$. Data collected during the grower and finisher phase (13 to 20 weeks of age) were analyzed as four treatments: CC [0 to 20 weeks diets contain no additional additives]; AA [0 to 20 weeks diets contained Actigen™]; CA [0 to 12 weeks no additional additives, 13 to 20 weeks diets with Actigen™]; CV [0 to 12 weeks no additional additives, 13 to 20 weeks diets with virginiamycin]. The pen was used as the experimental unit for growth parameters and individual birds were used for the histomorphometric measurements.

Animal Ethics

This study was conducted according to the guidelines of the Institutional Animal Care and Use Committee at North Carolina State University. All husbandry practices and euthanasia were performed with full consideration of animal welfare.

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[1] Surface area=(2π)*((Apical width+ Base width)/4)*Villous height

Results and Discussion

Starter-Developer Feed Phase (0 to 12 weeks of age)

The growth performance observed during the starter-developer phase (0 to 12 weeks) is summarized in Table 11. There was no clear advantage of dietary Actigen^{TM} supplementation on growth performance from 1-12 weeks. In this study birds were placed on litter from a previous flock to provide a microbial challenge. All birds were also treated with monensin, an antimicrobial ionophore that could have masked the potential effect of Actigen^{TM}. The recommended inclusion level of Actigen^{TM} is 200-800 ppm, in the current study we used a level of 400 ppm. Perhaps our level of inclusion was not high enough to give an additive response to the monensin. Nollet *et al*. (2007) saw an improvement in FCR when a coccidiostat was added with MOS as compared to the coccidiostat alone. In contrast, McCann *et al*. (2006) treated coccidia-challenged broiler chickens with MOS and a coccidiostat they did not observe an improvement in performance over those birds only treated with a coccidiostat, and the MOS alone did not effect coccidiosis infection. Based on the results of the present study, we conclude that Actigen^{TM} does not enhance the effect of monensin in the diets of turkeys. However, if there was not enough of a microbial challenge provided with the used litter to elicit a response from the monensin alone, we would not be able to observe an enhancing effect with the addition of Actigen^{TM}.

Increases in small intestine villous surface area (SA) facilitates greater nutrient absorption (Adil *et al*., 2010). There is an increase in villous volume and crypt depth during the first 21 days post-hatch (Uni *et al*., 1995). With the addition of Actigen^{TM} we observed a more advanced rate of development compared to the control birds (Table 12). At 10 days of age, the Actigen™-fed birds had greater ileum villi SA than the control-fed birds. However, there was no statistical difference in the villous SA at 14 days, indicating that both treatment groups reached a similar level of development by this time. Poor digestion, or malabsorption of feed, increases the poults' susceptibility for pathogens, which will subsequently impair the bird's growth (Oliveira *et al*., 2009). A greater villous surface area, and increased nutrient uptake, earlier in life allows for more nutrients available for the bird to utilize in development of immune system (Forder *et al*., 2007). Earlier development could prepare the intestine to handle pathogens earlier and be less affected by enteric stress. Greater absorptive capacity also reduces the nutrients available for competitive pathogens in the intestines to use as colonization media. There is a school of thought that is most commonly attributed to breeding stock, called minimum cumulative nutrition (Brake, 2007). The thought is that a bird's development is based on the total nutrients it consumes, rather than the age of the bird or the total amount of feed consumed. A greater villous surface area could have allowed the bird to uptake more nutrients necessary for mucosal development, giving it an advantage for dealing with pathogens. In this study we did not analyze mircofloral population, so we do not know if the Actigen™ supplementation allowed for a healthier microfloral population. These results suggest that Actigen™ either stimulated early mucosal development or the birds in this treatment were less challenged by microflora.

The Actigen™-fed poults exhibited increased villous height:crypt depth at 14 days of age. Collett *et al*. (2011) also observed similar increases in villous height, villous height:crypt depth, and goblet cell count among broiler chickens fed diets supplemented with Actigen™ or BMD as compared to those fed an unsupplemented control diet. An improved ileal mucosal development response in 21-day-old broilers to a yeast cell wall feed additive in comparison to controls was also reported by Zhang *et al*. (2005). This improvement in ileal morphology was correlated with observed improvements in growth. Birds receiving yeast cell wall had greater villous height and villous:crypt depth, which indicates slower epithelial turnover rate and lower mucosal distress due to healthier gastrointestinal tract than controls. In contrast, a faster rate of epithelial replacement may indicate the presence of enteric stress or harmful pathogens (Van Kruiningen, 1988). Enteric stress, due to toxins or disease, has been associated with shorter villi and deeper crypts, thus decreasing the surface area for nutrient assimilation (Yason *et al*., 1987). Crypt depth is closely related to the proliferation rate of new epithelial cells, and these cells have less absorptive capacity than more mature cells near the apex of the villus. Blunted villi with deep crypts indicate fewer epithelial cells with absorptive function (Geyra *et al*., 2001; Simon and Gordon, 1995). Moreover, epithelial cell turnover has great impact on the protein and energy requirements of the small intestinal mucosa (Zhang *et al*., 2005). A slower epithelial turnover rate may decrease the amount nutrients required by the gut, thus allowing for the partitioning of more resources toward production and growth.

Grower-Finisher Feed Phase (13 to 20 weeks of age)

The growth performance observed during the grower-finisher phase is summarized in Table 4. During the grower-finisher phase, the treatments receiving Actigen™ were not statistically different from each other. Therefore, there was not an added advantage to growth performance of Actigen™ inclusion throughout the whole study (AA) as compared to dietary Actigen™ inclusion only during the grower-finisher phase. The birds receiving the antibiotic growth promoter in the grower-finisher phase (CV) had significantly higher BW than the CC birds at 14 and16 weeks of age, CA and AA birds at 18 weeks of age, and AA at 20 weeks of age. The birds receiving virginiamycin during the grower-finisher phase only had significantly better cumulative (0 to 20 wk) FCR than those receiving Actigen™ throughout the entire study. In contrast, dietary supplementation of virginiamycin during the grower-finisher phase had no significant advantage in FCR over those that received either the control or Actigen™-supplemented diets during this period. The CV treatment toms had significantly greater cumulative feed intake from 0 to 16 and 0 to 18 weeks of age than the CC group. Although virginiamycin supplementation of grower-finisher diets resulted in a marginally better response on the growth performance of toms than those fed Actigen™-supplemented diets throughout the trial, a benefit over Actigen™ supplementation of grower-finisher diets could not be detected.

A similar result was reported by Fritts and Waldroup (2003), who observed no significant effect of the dietary addition of yeast cell wall (Bio-Mos®) or an antibiotic (BMD) as compared to a non-medicated control diet during any period on body weight or feed conversion of turkeys raised until 20 weeks of age. They suggest that this lack of dietary treatment response could be due to fact that the birds were already achieving their maximum genetic potential for growth, and that the dietary antibiotic supplementation was not necessary to achieve any additional growth response. However, throughout the entire 20-week feeding period, these researchers did observe those receiving Bio-Mos® or BMD had significantly better FCR than those birds receiving the negative control diet. A similar response could have been the case in the present study. Our intention of placing the birds on used litter was to provide them with a sufficient microbial challenge to impede growth performance, and thus simulate industry conditions. However, the litter was not analyzed for pathogens or microbial load before the poults were placed post-hatch. If the litter did not contain detrimental microbial (pathogen) agents, it is possible that the used litter served more as a "probiotic" for the poults to establish a stable symbiotic gut microflora rather than a gut health challenge, as is often seen in industry (Lee *et al*., 2011).

Based on the results from this study, we did not have a clear growth performance improvement during the hot summer months with Actigen™ supplementation. A measurable response to the feed additive could have been constrained by high environmental temperatures. The thermoneutral zone for birds is 15.5-24 degrees Celsius (C); at about 29 C birds will start to make behavioral adjustments to try to keep their core body temperature from increasing too much. These behavioral changes due to heat stress result in decreases in feed intake and production (Noll, 2004). In this study the temperature in the turkey house, recorded on data loggers kept at bird height throughout the entire study, did not drop below 25 C. If birds decreased their feed intake to decrease their production of metabolic heat, they may not have consumed enough feed to yield a measureable growth response. The feed intake measured in this study was lower than expected for this strain of bird. When feed intake is lower than expected to permit maximal growth, due to environmental factors such as excessive heat, animals will partition protein stores to maintenance and immunity before growth (Sandberg *et al.*, 2007). This typically results in decreased body weight gain and poorer FCR.

The lack of growth response could be due to a less than optimal dietary inclusion level of Actigen™. The recommended levels of dietary inclusion used in the study were based on previous studies with chickens (Hooge and Connolly, 2011; Xiao *et al*., 2011). In the present study, we used a constant dietary inclusion level of Actigen™. It was demonstrated with chickens that a higher level of Bio-Mos® inclusion in starter feed is necessary to elicit a positive response, and a gradual decrease in Bio-Mos® supplementation was more effective than a constant dietary inclusion rate throughout the production period (Nollet *et al*., 2007). A similar optimum dosage schedule for Actigen™ was reported by Collett *et al*. (2011). Collett *et al*. (2011) raised broiler chickens on used litter with a control diet, Actigen™ (800 g/tonne 0 to 7 days, 400 g/tonne 7 to 21 days, and 200 g/tonne 21 to 42 days), or an antibiotic (BMD). They observed improved 42 day BW and FCR in the birds treated with Actigen™ or BMD. A feeding program similar to this, with gradually decreasing levels of Actigen™ inclusion, may be more effective than one constant level of inclusion for turkeys.

We hypothesized that Actigen™ could improve turkey performance when fed in a diet with an ionophore anticoccidial (i.e. monensin) feed additive. Based on the results of this study, dietary Actigen™ did not enhance the beneficial effect of monensin. We did see an enhancement in intestinal morphology with the inclusion of Actigen™ during starter period. We also hypothesized that the Actigen™ could function as an effective alternative to an antibiotic feed additive to maintain gut health and promote growth of turkeys. Based on the results of this study, we cannot conclude that Actigen™ is an effective alternative to virginiamycin. We did not see clear or consistent differences in growth performance during the grower-finisher period between any treatments. The birds receiving Actigen^{TM} gave responses intermediate of the treatment receiving virginiamycin and no additive, indicating that the Actigen^{TM} was not better than the control nor worse than the antibiotic inclusion. Further research is necessary to determine optimum dietary dose schedule or optimal conditions for Actigen^{TM} supplementation for turkeys. However we can conclude that Actigen^{TM} is a potential alternative to an antimicrobial pharmaceutical agent based on ileal mucosa morphology and its implications on gut health.

Conclusions and Applications

1. Dietary inclusion of 400 g Actigen^{TM}/tonne may enhance gut mucosa health of turkey poults at early age (1 to 14 days of age) when added with monensin.
2. There was no clear advantage of dietary Actigen^{TM} supplementation on growth performance from 1 to 12 weeks when fed to male turkeys.
3. There was no apparent carry-over effect of Actigen^{TM} inclusion (1-12 weeks) on the growth performance during the grower-finisher phase.
4. Based on our results, Actigen^{TM} and virginiamycin resulted in statistically similar results.

References

Adil, S. T. Banday, G. A. Bhat, M. S. Mir, and M. Rehman. 2010. Organic acids on performance, intestinal histomorphology, and serum biochemistry of broiler chicken. Vet. Med. Int. 2010: 1-7.

Brake, J. T. 2007. Breeder nutrition fundamentals in any poultry operation: recent developments in North Carolina. Proceedings of Alltech's 23rd Annual Symposium. p. 117-121

Casewell M., C. Friis, E. Marco, P. McMullin, and I. Phillips. 2003. The European ban on growth-promoting antibiotics and emerging consequences for human and animal health. J. Antimicob. Chemother. 52:159-161.

Castanon, J. I. R. 2007. History of the use of antibiotic as growth promoters in European poultry feeds. Poult. Sci. 86:2466-2471.

Collett, S. R., G. F. Mathis, B. Lumpkins, D. M. Hooge, K. M. Brennan, and J. L. Pierce. 2011. Live performance and intestinal morphology of broiler chickens fed diets supplemented with BMD, Actigen™ or neither in two pen trials on built-up litter. Poult. Sci. 90 (E-Suppl. 1):145.

Ferket, P. R. 2003. Controlling gut health without the use of antibiotics.

Forder, R. E. A., G. S. Howarth, D. R. Tivey, and R. J. Hughes. 2007. Bacterial modulation of small intestinal goblet cells and mucin composition during early posthatch development of poultry. Poult. Sci. 86:2396-2403.

Fritts, C.A., and P. W. Waldroup. 2003. Evaluation of Bio-Mos® mannan oligosaccharide as a replacement for growth promoting antibiotics in diets for turkeys. Int. J. Poult. Sci. 2:19-22.

Fuller, R. 2001. The chicken gut microflora and probiotic supplements. Poult. Sci. 38:189-196.

Geyra, A., Z. Uni, and D. Sklan. 2001. Enterocyte dynamics and mucosal development in the posthatch chick. Poult. Sci. 80:776-782.

Gibson, G. R., A. L. McCartney and R. A. Rastall. 2005. Prebiotics and resistance to gastrointestinal infections. Brit. J. Nutr. 93 (Suppl. 1): S31-S34.

Hooge, D. M., and A. Connolly. 2011. Meta-analysis summary of broiler chicken trials with dietary Actigen® (2009-2011). Int. J. Poult. Sci. 10:819-824.

Hooge, D. M. 2004. Turkey pen trials with dietary mannan oligosaccharide: Meta-analysis, 1993-2003. Int. J. Poult. Sci. 3:179-188.

Hybrid Turkeys. A Hendix Genetic Company. <www.hybridturkeys.com>

Janardhana, V., M. M. Broadway, M. P. Bruce, J. W. Lowenthal, M. S. Geier, R. J. Hughes, and A. G. D. Bean. 2009. Prebiotics modulate immune responses in the gut-associated lymphoid tissue of chickens. J. Nutr. 139:1404-1409.

Lee, K. W., H. S. Lillehoj, S. H. Lee, S. I. Jang, G. D. Ritter, D. A. Bautista, and E. P. Lillehoj. 2011. Impact of fresh or used litter on the posthatch immune system of commercial broilers. Avian Dis. 55:539-544.

McCann, M. E. E., E. Newell, C. Preston, and K. Forbes. 2006. The use of mannan-oligosaccharides and/or tannin in broiler diets. Int. J. Poult. Sci. 5:873-879.

Noll, S. 2004. Avoiding heat stress mortality in poultry. University of Minnesota Extension Service.

Nollet. L., G. Huyghebaert, and P. Spring. 2007. Effect of dietary mannan oligosaccharide (Bio-Mos) on live performance of broiler chickens given an anticoccidial vaccine (Paracox) followed by a mild coccidial challenge. J. Appl. Poult. Res. 16:397-403.

Ofoyo, B. A., R. E. Droleskey, J. O. Norman, H. H. Mollenhauer, R. L. Ziprin, D. E. Corrier, and J. R. DeLoach. 1988. Inhibition by mannose of *in vitro* colonization of chicken small intestine by *Salmonella typhimurium*. Poult. Sci. 68:1351-1356.

Parks, C. W. 2001a. The effects of supplementing diets with virginiamycin or mannan-oligosaccharides on immune system properties and intestinal morphology. Ph.D. Dissertation. North Carolina State University, Raleigh, NC, USA.

Parks, C. W., J. L. Grimes, P. R. Ferket, and A. S. Fairchild. 2001b. The effect of mannanoligosaccharides, Bambermycins, and virginianmycin on performance of large white male market turkeys. Poult. Sci. 80:718-723.

Patterson, J. A., and K. M. Burkholder. 2003. Application of prebiotics and probiotics in poultry production. Poult Sci. 82:627-631.

Sandberg, F. B., G. C. Emmans, and I. Kyriazakis. 2007. The effects of pathogen challenges on the performance of naïve and immune animals: the problem of prediction. Animal. 1:67-86.

Santin, E., A. Maiorka, and M. Macari. 2001. Performance and intestinal mucosa development of broiler chickens fed diets containing *Saccharomyces cerevisiae* cell wall. J. Appl. Poult. Res. 10:236-244.

Simon T. C., and J. I. Gordon, 1995. Intestinal epithelial cell differentiation: new insights from mice, flies and nematodes. Curr. Opin. Genet. Dev. 5:577–586.

Sims, M. D., K. A. Dawson, K. E. Newman, P. Spring, and D. M. Hooge. 2004. Effects of dietary mannan oligosaccharide, bacitracin methylene disalicylate, or both on the live performance and intestinal microbiology of turkeys. Poult. Sci. 83:1148-1154.

Solis de los Santos, F., A. M. Donoghue, M. B. Farnell, G. R. Huff, W. E. Huff, and D. J. Donoghue. 2007. Gastrointestinal Maturation is accelerated in turkey poults supplemented with a mannan-oligosaccharide yeast extract (Alphamune). Poult Sci. 86:921-930.

Spring, P., C. Wenk, K. A. Dawson, and K. E. Newman. 2000. The effects of dietary mannanoligosaccharides on cecal parameters and the concentrations of enteric bacteria in the ceca of salmonella-challenged broiler chicks. Poult. Sci. 79:205-211.

Stone, C. W. 1998. Yeast products in the feed industry: A practical guide for feed professionals. Diamond V Mills, Inc. Cedar Rapids, IA, USA. Page 1-16.

Tabler, G. T. 2004. The challenges facing turkey growers. Avian Advice. 6:9-11.

Uni, Z., Y. Noy, and D. Sklan. 1995. Posthatch changes in morphology and function of the small intestines in heavy- and light-strain chicks. Poult. Sci. 74:1622-1629.

Van Kruiningen, H. J. 1988. Morphologic Considerations in the Interpretation of Gastrointestinal Disorders. Toxicol Pathol 16: 110-117.

Xiao, R., R. F. Power, D. Mallonee, L. Spangler, K. Routt, K. M. Brennan, J. L. Pierce, and K. A. Dawson. 2011. Gene expression study reveals the association of dietary supplementation of Actigen^{TM} and the regulation of pathogen-influenced signaling pathways in broiler chickens. Poult. Sci. 90 (E-Suppl. 1):142.

Yahav, S., I. Plavnik, M. Rusal, and S. Hurwitz. 1998. Response of turkeys to relative humidity at high ambient temperature. Brit. Poult. Sci. 39:340-345.

Yamauchi, K. 2002. Review on chicken intestinal villus histological alterations related with intestinal function. J. Poult. Sci. 39: 229-242.

Yang, Y., P. A. Iji, and M. Choct. 2009. Dietary modulation of gut microflora in broiler chickens: a review of the role of six kinds of alternatives to in-feed antibiotics. World Poult. Sci. 65:97-114.

Yason, C. V., B. A. Summers, and K. A. Schat. 1987. Pathogenesis of rotavirus infection in various age groups of chickens and turkeys: pathology. Am J Vet Res. 48:927-938.

Zhang, A. W., B. D. Lee, S. K. Lee, K. W. Lee, G. H. An, K. B. Song, and C. H. Lee. 2005. Effects of yeast (*Saccharomyces cerevisiae*) cell components on growth performance, meat quality, and ileal mucosa development of broiler chicks. Poult Sci. 84:1015-1021.

Table 10: Composition and nutrient content of dietary feeding phases fed to turkey toms from placement to 20 weeks of age.

Dietary Phase	Prestarter	Starter	Developer		Grower		Finisher	
			1	2	1	2	1	2
Weeks of age	**0-3**	**3-6**	**6-9**	**9-12**	**12-14**	**14-16**	**16-18**	**18-20**
Ingredients	----------	----------	----------	----------	%	----------	----------	----------
Corn	40.99	47.33	50.76	53.32	61.50	66.26	66.33	68.27
Soybean Mean 48	45.23	39.05	34.96	32.01	23.58	18.44	17.06	14.79
Poultry Meal	5.00	5.00	5.00	5.00	5.00	5.00	5.00	5.00
Poultry Fat	2.84	2.85	3.95	5.11	5.65	6.60	8.17	8.70
Dicalcium Phosphate 18.5	3.08	2.86	2.63	2.11	1.91	1.69	1.60	1.45
Limestone	1.11	1.15	1.06	1.16	1.09	0.99	0.96	0.92
Alimet ®[1]	0.41	0.37	0.32	0.24	0.24	0.13	0.16	0.19
Salt (NaCl)	0.34	0.34	0.34	0.34	0.34	0.34	0.34	0.34
Trace mineral premix[2]	0.20	0.20	0.20	0.13	0.13	0.13	0.10	0.10
Choline Chloride 60%	0.21	0.23	0.22	0.17	0.16	0.09	0.05	0.05
L-Lysine-HCl, 78.8%	0.27	0.30	0.32	0.17	0.22	0.15	0.08	0.04
Vitamin premix[3]	0.15	0.15	0.10	0.1	0.08	0.08	0.05	0.05
Sodium Selenite premix%[4]	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05
Actigen™ [5]	0.04	0.04	0.04	0.04	0.04*	0.04*	0.04*	0.04*
Sand	0.00*	0.00*	0.00*	0.00*	0.01*	0.01*	0.01*	0.01*
Coban® 90[6]	0.03	0.03	0.03	0.03	--	--	--	--
L-Threonine	0.05	0.05	0.02	0.02	--	--	--	--
Calculated nutrient analysis								
ME Poultry, Kcal/kg	2950	3000	3100	3200	3300	3400	3500	3550
Crude protein, % Est.	28.50	26.00	24.27	22.89	19.44	17.23	16.50	15.50
Crude Protein, % Det.	27.60	26.93	24.22	22.32	20.35	18.10	17.50	16.82
Crude fat, %	5.33	5.45	6.59	7.79	8.47	9.49	11.03	11.58
Crude fiber, %	2.40	2.29	2.21	2.14	1.98	1.88	1.83	1.78
Calcium, %	1.45	1.40	1.30	1.20	1.10	1.00	0.95	0.90
Total Phosphorus, %	1.07	1.01	0.95	0.84	0.77	0.70	0.68	0.64
Available Phosphorus, %	0.80	0.75	0.70	0.60	0.55	0.50	0.48	0.45
Sodium, %	0.18	0.18	0.18	0.18	0.18	0.18	0.18	0.18
Potassium, %	1.33	1.19	1.10	1.03	0.84	0.72	0.68	0.63
Chloride, %	0.32	0.33	0.33	0.30	0.31	0.29	0.28	0.27
Na +K-CL, Meq/kg	329	291	266	257	205	180	176	165
Arginine, %	1.98	1.79	1.66	1.57	1.30	1.14	1.09	1.02
Lysine, %	1.85	1.70	1.60	1.40	1.20	1.00	0.90	0.80
Methionine, %	0.82	0.75	0.67	0.59	0.54	0.41	0.42	0.44
Met +Cys, %	1.25	1.15	1.05	0.95	0.85	0.70	0.70	0.70
Threonine, %	1.15	1.05	0.95	0.90	0.74	0.65	0.63	0.59
Tryptophan, %	0.33	0.30	0.27	0.26	0.21	0.18	0.17	0.16
Choline, mg/kg	2720	2700	2570	2265	2025	1565	1330	1285

[1]Alimet ® (Novus International, Inc., St. Charles, MO, USA) is 2-hydroxy-4-(methylthio)-butyric acid (88% active methionine analogue).
[2] Each kilogram of mineral premix (0.1% inclusion) supplied the following per kg of complete feed: 60 mg Zn as $ZnSO_4H_2O$; 60 mg Mn as $MnSO_4H_2O$; 40 mg Fe as $FeSO_4H_2O$; 5 mg Cu as $CuSO_4$; 1.25 mg I as $Ca(IO_3)2$; 1 mg Co as $CoSO_4$.
[3] Each kilogram of vitamin premix (0.1% inclusion) supplied the following per kg of complete feed: 13,200 IU vitamin A; 4,000 IU cholecalciferol; 66 IU alpha-tocopherol; 110 mg niacin; 22mg pantothenic acid; 13.2 mg riboflavin; 8 mg pyridoxine; 4 mg menadione; 2.2 mg folic acid; 4mg thiamin; 0.253 mg biotin; 0.04mg vitamin B_{12}; 100 mg ethoxyquin.
[4] $NaSeO_3$ premix provided 0.3mg Se/kg of complete feed.
[5] In dietary treatments not receiving Actigen™, washed builder's sand was used as a filler.
[6]Coban © 90 (Elanco, Greenfield, IN, USA) is Monensin sodium salt (90grams/pound or 198 grams/kg active), used for the prevention of coccidiosis.
* Con=0.05% sand; AA=0.04% Actigen™ + 0.01% sand; CA=0.04% Actigen™ + 0.01% sand; CV=0.05% Stafac® 20 (Phibro Animal Health, NJ, USA) virginiamycin (20 g/ton).

Table 11: Growth performance of tom turkeys fed diets supplemented with or without Actigen™ during the starter-developer phase (day of hatch until 12 weeks of age).[1]

	Age (weeks)			
Treatment	**0-3**	**0-6**	**0-9**	**0-12**
	-------- *(kg Body Weight/bird)* --------			
Control	0.47	1.95	4.73	8.05
Actigen™	0.47	1.95	4.75	8.06
P-value	0.9157	0.8924	0.7551	0.8773
SEM(43)[1]	0.007	0.019	0.040	0.052
	-------- *(kg Feed Intake/bird)* --------			
Control	0.80	3.22	9.25	14.91
Actigen™	0.82	3.23	9.10	15.00
P-value	0.2088	0.8930	0.1686	0.5892
SEM(43)[1]	0.012	0.031	0.072	0.113
	-------- *(kg Feed:kg Gain)* --------			
Control	1.915	1.703	1.725	1.866
Actigen™	1.913	1.696	1.725	1.875
P-value	0.9487	0.6620	0.9756	0.5195
SEM(43)[2]	0.018	0.012	0.008	0.0076

[1] Values for Control are means of 36 replicate pens of ca. 15 toms per pen; Values for Actigen™ are means of 12 replicate pens of *ca.* 15 toms per pen.
[2] SEM (43) = Standard error with 43 degrees of freedom.

Table 12: Histomorphometric measurements on ileal villi in male turkey poults fed diets supplemented with and without Actigen™.[1]

Age (days)	Diet[2]	Apical Width	Villi Height	Base Width	Crypt Depth	Muscularis Thickness	Villi Surface Area (μ^2)
		Measurements (microns)					
6	Control	72.8	381.3	127.9	107.6	88.8	121,450
	Actigen	64.9	406.7	118.1	118.8	84.6	116,530
	P-value	0.0335	0.4861	0.1573	0.1968	0.5629	0.7278
	SEM(14)[3]	2.38	25.15	4.62	5.87	4.95	9,793
10	Control	88.7	678.1	180.0	160.9	173.0	256,630
	Actigen	87.4	709.4	191.0	161.9	159.7	305,810
	P-value	0.7976	0.4698	0.2955	0.8940	0.5011	0.0028
	SEM(14)[3]	3.46	29.82	7.14	5.52	13.60	9,249
14	Control	92.2	690.7	199.8	229.2	153.6	336,765
	Actigen	78.1	808.2	172.8	183.2	146.8	290,265
	P-value	0.0192	0.0135	0.0324	0.0022	0.6642	0.2324
	SEM(14)[3]	3.77	29.06	7.98	8.72	10.890	26341

[1]Values are means of 20 villi from 8 sampled birds per treatment.
[2]Both control and Actigen diets contained the anticoccidial ionophore, Monensin.
[3]SEM (14)= Standard error with 14 degrees of freedom.

Table 13: Growth performance of tom turkeys fed diets supplemented with Actigen™ or virginiamycin until during the grower-finisher phase (13 to 20 weeks of age).[1]

Dietary Supplement 0-12 wk	13-20 wk	Trt	Age (weeks) 0-14	0-16	0-18	0-20
			(kg Body Weight/bird)			
Control	Control	CC	10.52 ^{b}	12.53 ^{b}	15.37 ^{ab}	17.86 ^{ab}
Actigen™	Actigen™	AA	10.59 ^{ab}	12.66 ^{ab}	15.18 ^{b}	17.69 ^{b}
Control	Actigen™	CA	10.74 ^{ab}	12.64 ^{ab}	15.23 ^{b}	17.81 ^{ab}
Control	Virginiamycin	CV	10.83 ^{a}	13.06 ^{a}	15.79 ^{a}	18.35 ^{a}
Source of Variation			**(P-Value)**			
P-value			0.0402	0.0166	0.0212	0.0167
SEM(43)[1]			0.082	0.117	0.145	0.150
			(kg Feed Intake /bird)			
Control	Control	CC	20.63	26.87 ^{b}	33.91 ^{b}	44.08 ^{ab}
Actigen™	Actigen™	AA	21.16	27.49 ^{ab}	34.62 ^{ab}	45.34 ^{ab}
Control	Actigen™	CA	20.81	27.25 ^{ab}	34.47 ^{ab}	43.86 ^{b}
Control	Virginiamycin	CV	21.29	28.07 ^{a}	35.42 ^{a}	45.51 ^{a}
P-value			0.1408	0.0237	0.0252	0.0158
SEM(43)[1]			0.220	0.260	0.329	0.425
			(kg Feed:kg Gain)			
Control	Control	CC	1.973 ^{ab}	2.153 ^{ab}	2.231 ^{ab}	2.303 ^{ab}
Actigen™	Actigen™	AA	2.005 ^{a}	2.203 ^{a}	2.270 ^{a}	2.332 ^{a}
Control	Actigen™	CA	1.966 ^{ab}	2.155 ^{ab}	2.238 ^{ab}	2.299 ^{ab}
Control	Virginiamycin	CV	1.957 ^{b}	2.120 ^{b}	2.193 ^{b}	2.263 ^{b}
P-value			0.0312	0.0073	0.0299	0.0485
SEM(41)[2]			0.0116	0.0156	0.0175	0.0168

[1] Values are means of 12 replicate pens of *ca.* 15 toms per pen.
[2] SEM (41)= Standard error with 41 degrees of freedom.
[a,b] Means within a column with no common superscript are significantly different (P≤0.05).

Table 14: Percent mortality of turkey toms fed diets supplemented with Actigen™ or virginiamycin until 20 weeks of age, summarized by starter-developer and grower-finisher phase.[1]

Dietary Supplement 0-12 wk	13-20 wk	Trt	Starter-Developer 0-12 wk	Grower-Finisher 13-20 wk
			------------ *% Mortalitiy*[3] ------------	
Control	Control	CC	2.32	1.52
Actigen™	Actigen™	AA	1.85	2.02
Control	Actigen™	CA	1.51	0.56
Control	Virginiamycin	CV	1.11	1.01
P-value			0.7484	0.5374
SEM (41)[2]			0.794	0.732

[1] Values are means of 12 replicate pens of *ca.* 15 toms per pen.
[2] SEM (41)= Standard error with 41 degrees of freedom.
[3] % Mortality= (number of birds found dead/ number of birds started)*100

SUMMARY

The overall objective of this research was to study the effects of two different commercially-available, yeast-based products on the performance of commercial turkeys, and determine if they could potentially be used to promote growth or used as alternatives to dietary antibiotic growth promotants. The composition and likely mode of action of the two products are different, one being a very refined cell wall MOS product (Actigen™, Alltech, Inc.) and the other being a relatively undefined yeast fermentation product mix (Original XPC™, Diamond V Mills).

Supplementing male turkey diets with Original XPC™ was shown to improve growth performance through 14 weeks of age as compared to an unsupplemented control diet. Inclusion of Original XPC™ stimulated increased feed intake during the starter and developer periods. Although gut health was not measured directly, there were indications that Original XPC™ could improve gut health, as indicated by higher BW and improved FCR. However, the improvements in growth performance were not detectable during the finishing period. There was no effect on progeny performance seen with supplementing the breeder hens with Original XPC™. It is very likely that the two weeks of dietary Original XPC™ supplementation was an insufficient duration of time for the hens to express to any nutritional or immunological advantage. Perhaps if the hens were supplemented earlier and for a longer period of time, there could have been a carry–over effect from the breeder hen to their progeny. The inclusion of the yeast fermentation product (Original XPC™) in a standard corn-soy ration improved the growth performance of male turkeys until 14 weeks of age.

With the dietary inclusion of mannose-rich yeast cell wall fraction (Actigen™) during the starter period, there were indications of earlier gut maturation. Dietary supplementation of Actigen™ increased villous surface area and decreased villous height to crypt depth ratio, indicating less enteric stress and improved nutrient uptake. However, there were no improvements seen in growth performance parameters during the starter-developer phase. Perhaps with a higher dosage in the starter period and the dosage gradually decreased there would be not only an advantage seen at the gut level but also a response seen in growth performance. It is also possible that there could be a carry-over effect of the dietary Actigen™ inclusion from the starter-developer phases to the grow-finisher phase. However, due to the constraints of our experimental design we did not have birds that received Actigen™ supplementation in the early feeding phases and not the later phases, so a residual effect of the additive could not be measured. There was also not a clear advantage seen in the of Actigen™ inclusion in the grower-finisher diets as compared to dietary virginiamycin inclusion or the no additive control. It was unexpected that the virginiamycin treated birds were similar to the no-additive control, so perhaps in the environment of this trial, we were not able to measure a growth advantage with Actigen™ supplementation during the grower-finisher period. Dietary inclusion of Actigen™ improved intestinal health of turkey poults during the starter period. Actigen™ supplementation to birds during the grower-finisher period was not clearly different than either the virginiamycin-supplemented birds or the control birds. Actigen™ could have the potential to be an alternative to antibiotic inclusion in turkey feed during the grower-finisher phase, but the experimental conditions were not appropriate to express this potential.

Our interest with these two very different products was to observe if there is an advantage of using a highly refined yeast cell wall product or a yeast fermentation product that is an unknown mixture of products. Based on the results from these trials, either of these yeast products could be potential alternatives to antibiotic growth promotants in turkey feed and that they could both improve early enteric health of poults. From this research it is not clear if one approach is more advantageous than the other: a refined or an undefined yeast product. A bird's response to any additive, including antibiotic growth promoters, is influenced by management, environment and health status of the flock. In different environments, the birds may have a different response to these additives than was seen in these trials. The growth-promoting effects observed from antimicrobial additives are much more evident when birds are exposed to suboptimal conditions, which were not experienced in these trials. Even in the study where the poults were placed on used litter was not as intense of a microbial challenge as birds could experience in the industry.

It is very challenging to find one alternative product to replace all the benefits associated with antibiotics, especially because the exact mode of action of many growth promoting feed additives is not understood. Likely the solution for replacing antibiotics in animal feed will be a combination of multiple products or types of products, but prebiotic products derived from the yeast *Saccharomyces cerevisiae*, such as Original XPC™ or Actigen™ should be seriously considered as possibilities.