ABSTRACT

BLACK, SAMANTHA ANN. Performance and Economic Impacts of Hatchery and Post-Hatch Constraints on Poult Quality. (Under the direction of Dr. Peter Ferket and Dr. Robert Beckstead).

Turkey production is important to the agricultural economy in the United States, valued at approximately $6 billion in 2016. Mortality at any point in the production scheme means a loss in profitability. Successful transition of poults from hatcheries to farms is essential to maximizing performance and minimizing mortality. Turkey embryos and poults are exposed to a variety of constraints that may impact subsequent survivability and performance, which when it is assessed is known as poult quality (PQ). Three live animal experiments, and one field data analysis project was conducted to assess various factors impacting poult quality and early poult mortality.

A preliminary study was conducted to determine body weight loss in turkey poults from young and peak stage of lay breeder hen from hatch to 48-hours post-hatch. Poults from young breeders had smaller hatching weights and lost body weight at a faster rate than poults from peak breeders. The greatest differences in body weight losses occurred from 36 to 48 hours post-hatch.

First, a battery cage experiment was conducted to assess the effects of breeder age and placement time in turkey poults to 2 weeks of age. A factorial design was used with breeder age (first week of lay = FOL and 14th week of lay = PEAK) and placement time (immediate or 48-hour holding period without feed or water). Body weights and feed intake were measured along with jejunum and ileum mucosal samples. In this study, poults hatched from young breeders weighed less than poults hatched from peak breeders. Intestinal growth as determined by villus height and villus surface area indicated a compensatory effort in

poults from young breeders and delayed placement poults, which trended towards normalization by 2 weeks of age. Mortality rates were higher in delayed placement poults and poults from young breeders. PQ measurements that were found to be accurate predictors of performance in this experiment were hock vein, shank color, and shank length.

Second, a market turkey hen experiment was conducted to elucidate the effect of breeder age on PQ assessment (Tona score or individual measured parameters) and turkey hen performance. A split-plot block design with 12 dietary treatments as the whole plot factor and two poult populations originating from young or old breeder hen eggs. Poults were placed in pine shaving floor pens and reared to 14 weeks of age. A PQ assessment was conducted on 2 poults per pen from the young breeder population. Body weight was measured, along with intestinal mucosal morphometrics and bone density. In this experiment, poults hatched from young breeders had lower hatchling weights and remained smaller than poults hatched from peak production breeders until market age. Poults from young breeders also had increased villus surface area at 5 weeks of age, suggesting compensatory growth effort. However, this compensatory growth did not result in market-age performance benefits as poults from young breeders had reduced breast muscle yield at processing as compared to poults from peak production breeders. Hatchling length was observed as the PQ assessment factor that was the most accurate predictors of performance in this experiment.

In the last study, factors related to pre-placement mortality (PPM) and first week mortality (FWM), and cumulative early mortality (CEM) were investigated. Field data collected from 6 US hatcheries over a 22-month period were analyzed. Data were analyzed using regression on dummy variables. PPM was significantly related to hatchery, season and distance traveled from hatchery to farm. FWM and CEM were significantly related to

hatchery, season, hatch day, gender, breeder age and distance traveled. Potential cost savings of eliminating poult mortality associated with young breeders is $60 to $128 per average truck delivery per farm and the potential cost savings of eliminating poult mortality by reducing distance traveled by 500 miles is $170 average truck delivery per farm. In summary, elucidating the impacts of hatchery and transportation constraints on PQ and early poult mortality can have a significant impact profitability of commercial turkey production.

Performance and Economic Impacts of Hatchery and Post-Hatch Constraints on Poult Quality

by
Samantha Ann Black

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APPROVED BY:

Dr. Peter Ferket Co-Committee Chair	Dr. Robert Beckstead Co-Committee Chair
Dr. Frank Edens	Dr. Tom Vukina

Dr. Meghan Schwartz
External Member

DEDICATION

I would like to dedicate this work to those who preserve. I have and so can you.

BIOGRAPHY

Samantha Ann Black was born in Annapolis, Maryland. She earned her Bachelor's degrees in Poultry Science and Biological Sciences from North Carolina State University. As an undergraduate student, she worked at the University Feed Mill, as an undergraduate teaching assistant for introductory Poultry Science classes, and as a student technician at the Turkey Educational Unit. Samantha earned her M.S. in Poultry Science in 2015 under the direction of Dr. Jesse Grimes studying the use of probiotics in chicks and poults. After earning this degree Samantha decided to continue her education by pursuing her Ph.D. in Nutrition and Animal Sciences under the direction of Dr. Peter Ferket and Dr. Robert Beckstead. Samantha has a passion for science policy and agricultural advocacy and has relocated to Washington, DC to pursue a career in this field.

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CHAPTER 1.

LITERATURE REVIEW

INTRODUCTION

Early poult mortality is an endemic problem for the worldwide turkey industry (Schultz-Cherry et al., 2000). Managing stress during the first week of life and minimizing distress and mortality is critical for maximizing livability and performance (Decuypere & Bruggeman, 2007; Lilburn, 1998; Willemsen et al., 2010). Due to the intensive husbandry practiced on farms today, the exposure of farm animals (including poultry) to greater variety of stressors has become more frequent (Dantzer & Mormède, 1983). The goal of this literature review was to identify factors and stressors during embryonic and early poult development, which are predictive of survivability and future growth performance. These predictive indicators collectively allow for the assessment of poult quality (PQ). Identification of biological elements and management practices influencing PQ should provide data and strategies for managing stressors and the successful transition of turkey embryos to turkey poults.

A high incidence of early poult mortality has often been an indicator of welfare problems (*Humane Farm Animal Care Standards for Turkeys*, 2013), which has become an increasingly more important issue for producers and consumers. According to Veissier & Boissy (2007) animal well-being is the absence of stress or only low levels of stress. Stress is an adaptive response by an animal to stressors that pose threats to an animal's homeostatic condition (Dohms & Metz, 1991). Hans Selye (1946) proposed the General Adaptation Syndrome (GAS), which consists of three stages: alarm, resistance and exhaustion. The immediate and involuntary alarm phase involves the immediate secretion of the catecholamines, primarily epinephrine, which is needed to sequester energy substrates to either resist a stressor or to flee the stressor. This is called the neurogenic response. It has an

instant component in which the sympathetic nervous system reacts instantly when an animal is exposed to as stressor. The second component of the neurogenic response is the fast response, which involves massive secretion of epinephrine and norepinephrine from the adrenal medulla. The neurogenic response is often referred to as the alarm phase of the GAS (Mailyan, 2016; Scanes, 2016). If the stressor persists, the resistance/adaptation phase is initiated, which is a physiological coping reaction to the increased demands of maintaining homeostasis at a higher level of activity (Dohms & Metz, 1991). The stage of resistance is characterized by induction of the hypothalamic-anterior pituitary- adrenal cortex (HPA) axis. In the HPA, the animal will begin increased secretion of the avian stress response hormone called corticosterone. This is a nonspecific reaction, since all animals will secrete large concentrations of the glucocorticoid hormone in response to chronic exposure to a stressor. An additional response to systemic and cellular stressors is the production of heat shock proteins (HSP), which have strong cytoprotective effects (Kregel, 2002). HSP acts as molecular chaperones – overproduction of HSP reduces stress-induced denaturation and aggregation of proteins (Rokutan, 2000), maintains structural proteins, and degradation of unstable proteins (Kregel, 2002). HSP also protect against reactive oxygen species (ROS), which cause cellular damage to DNA and mitochondria, by inhibiting NADPH oxidase activity to prevent apoptosis (Jacquier-Sarlin et al., 1994). Prior stressful experiences may have long-term benefits in helping an individual to cope with subsequent stressors (Zulkifli & Siegel, 1995). Nevertheless, prolonged exposure to the stressor is associated with development of pathological states that ultimately can lead to decreased performance or death (Mailyan, 2016).

The genetic selection for poultry species for increased body weight and maximal muscle mass has led to decreased disease resistance (Graczyk, 2003; Huff et al., 2007). Huff et al. (2001 and 2007) reported that turkeys were far more responsive to the environment and more socially interactive than broiler chickens. These behavioral differences between chickens and turkeys suggest the stress response in the two species differ. Due to excessive growth and blunted HPA axis (Huff et al., 2007) young turkey poults may be more sensitive to environmental stressors than chickens (Graczyk, 2003). Due to the complexity of stress mechanisms and complex neuroendocrine and lymphatic system interactions, classification of stress responses in turkeys remains open to further investigation.

Early poult mortality also negatively impacts the economic profitability of turkey producers. Turkey production is important to the agricultural economy in the United States. In 2016, 5.98 billion pounds of turkey meat were produced, valued at $6.18 billion (USDA-ERS, 2017). Mortality in young poults infers a loss of profits due to the costs associated with production (feed, electricity, man power, etc.) that cannot be recouped. There are currently no published studies evaluating the costs associated with specific factors and stressors that trigger early mortality in turkeys.

The following literature review illustrates the current understanding of the biological elements and management practices involved in embryonic and early post-hatch development of poultry.

FACTORS THAT INFLUENCE EMBRYONIC DEVELOPMENT

Energy Metabolism

Tight control of energy metabolism during embryonic development is essential for success of the hatching process. Glycogen is synthesized from glucose produced by

gluconeogenesis using amniotic fluid amino acids as substrate (Muramatsu et al., 1990), and it is in turn used to fuel the hatching process (Donaldson & Christensen, 1991). The hatching process is supported predominantly by glycogen synthesis (glycogenesis) between E24 and E26, and predominantly by glycogen degradation (glycogenolysis) after E26 (de Oliveira et al., 2013) during pipping and hatching (Picardo & Dickson, 1982). This use of glycogen requires a switch from lipid-based to carbohydrate-based metabolism as oxygen becomes limited around the time of internal pipping (Donaldson et al., 1991; Foye, 2005; Moran, 2007; Noy & Sklan, 1999; Romanoff, 1967; Uni & Ferket, 2004). The embryo depends on glycogenesis of the amnion and anaerobic metabolism of carbohydrates for energy during this time (de Oliveira et al., 2008; Moran, 2007). The liver has an important role in providing energy to tissues from different substrates based on oxygen supply to fuel increased physical activity during hatching. The liver's post-transcriptional control connects antagonistic pathways, such as glycolysis and gluconeogenesis with glycogenesis, glycogenolysis, and the pentose-phosphate pathway to regulate protein levels during critical periods of embryonic development (de Oliveira et al., 2013). If tissue growth, energy stores, or energy use is compromised due to *in ovo* stress, the embryo may not be able to emerge from the shell and will die as a consequence of metabolic energy constraints (de Oliveira et al., 2013). Additionally, reduced eggshell conductance, oxygen consumption, and energy budget in modern turkey eggs has resulted in increased embryonic mortality due to insufficient availability of carbohydrate (Christensen et al., 1993). Embryonic stress leads to the reduced ability to utilize energy stores resulting in depressed embryonic growth and/or early embryonic mortality.

Hormone Status

Insulin and Glucagon

Insulin and glucagon are closely related to energy metabolism in all avian embryonic tissues (Lu et al., 2007). Insulin and glucagon are hormones produced by the pancreas, and their release into blood circulation depends on the animal's energy status (McNabb & Darras, 2015). During embryogenesis, insulin levels increase with concomitant development of the pancreas (de Pablo et al., 1982; Lu et al., 2007). Insulin levels reach a plateau during pipping and hatching (Lu et al., 2007) as the embryo consumes the amnion and glycogen accumulates in liver and muscle tissues (McNabb & Darras, 2015). Embryonic plasma glucose levels follow the insulin pattern, increasing throughout incubation, and plateau at 4 days pre-hatch, during pipping, and at hatching (Franssens et al., 2014; Lu et al., 2007; F. McNabb & Darras, 2015). Plasma levels of glucagon remain low until mid-incubation, then gradually rise until they peak during pipping and hatching (Lu et al., 2007).

The insulin gene is expressed early during embryonic development in extra-pancreatic tissues prior to development of the pancreas, showing specific regulation (de la Rosa & de Pablo, 2011). Because embryo growth hormone levels are low during incubation, it has been hypothesized that insulin has a role as an embryonic growth factor (Lu et al., 2007) by accelerating embryonic growth and morphological development (de Pablo et al., 1991). The addition of insulin (or insulin-like growth factors, IGFs) to other specific factors in an incubation medium cocktail stimulates somite cell proliferation and myogenesis (Pirskanen et al., 2000). This effect is blocked by an insulin antibody and insulin receptor antibody, illustrating that insulin acts as a myogenic signal early in embryogenesis. A fine tuning of insulin gene expression is required to sustain adequate morphogenesis (McNabb &

Darras, 2015). In some cases, proinsulin is expressed but not cleaved, whereas in other cases it acts through hybrid insulin-IGF receptor (de la Rosa & de Pablo, 2011). Once insulin receptors are expressed and after binding with insulin during embryogenesis, they both have important roles in developmental events, such as the regulation of protein and carbohydrate metabolism (Franssens et al., 2014) by promoting protein deposition during rapid embryonic growth (Lu et al., 2007).

Glucagon and glycogen play a critical role in providing the glucose requirement of chick embryos during embryogenesis (de Oliveira et al., 2008; Lu et al., 2007). Glucose is the primary energy substrate needed during the first stage of embryonic development and is used in anaerobic glycolysis (Moran, 2007). Glucose is also stored in the liver and muscle as glycogen as the substrate for glycogenesis, which fuels energy metabolism during pipping and hatching (Picardo & Dickson, 1982). These processes are consistent with increased glucose levels and increased glucagon level just prior to pipping on E18 in chicken embryos (Lu et al., 2007). The embryonic hormonal changes indicate the importance of glucagon during hatching (Franssens et al., 2014; Lu et al., 2007; McNabb & Darras, 2015) and are associated with the sudden depletion of hepatic and muscle glycogen to provide glucose during the transition from lipid-based to carbohydrate-based metabolism (Christensen et al., 2001; Lu et al., 2007; Picardo & Dickson, 1982). Disruptions in energy metabolism of embryos, as discussed above, can result in depressed development or early embryonic mortality.

Thyroid Hormones

Thyroid hormones, triiodothyronine (T_3) and thyroxine (T_4), are critically important in maintaining normal growth and development during embryogenesis (Lu et al., 2007). In

general, thyroid hormones appear to act permissively and indirectly with other control substances to stimulate growth in birds (McNabb & King, 1993). These hormones, along with growth hormone, are important in tissue differentiation and maturation. In mammals, their roles in development have been studied in the various tissues (Pascual & Aranda, 2013). They are known to be crucial for development of the brain, eyes, and ears (Bernal, 2007; Forrest & Swaroop, 2012; Rusch et al., 2001). The information available on birds suggests similar roles for thyroid hormones during embryogenesis. For example, in the chicken embryonic gut, thyroid hormones alone can stimulate cellular differentiation and induce digestive enzymes, and in combination with glucocorticoids, thyroid hormones are necessary for the maturation of intestinal glucose transport (McNabb, 2006).

Hormone synthesis in avian species precedes organization of the thyroid gland into follicles and production begins in the first quarter of incubation (McNabb & Darras, 2015). Hypothalamic-pituitary control is established in the latter half of embryonic development (McNabb & Darras, 2015). The feedback loop of the hypothalamic-pituitary-thyroid axis (HPT) begins when the hypothalamus produces thyroid-releasing hormone (TRH) and/or corticotropin-releasing hormone (CRH) to stimulate the release of thyroid stimulating hormone (TSH) from the anterior pituitary. TSH is the major controller of production and release of thyroid hormones from the thyroid gland. Thyroid hormones then exert negative feedback on the pituitary and hypothalamus. Maturation of this feedback response is established by E19 of incubation in chicken embryos. The hypothalamus can also produce somatostatin, to block the production of TSH by the pituitary. This feedback response matures between E19 and hatch in chicken embryos (De Groef et al., 2007). Plasma T_4 rises during the latter half of embryonic life, but plasma T_3 remains low until late embryonic

development (Lu et al., 2007; McNabb & Darras, 2015). The highest levels of T_3 and T_4 during embryonic development are observed at internal pipping (Lu et al., 2007; Reyns et al., 2003). At this time, the embryo penetrates the air sac and switches from chorioallantoic respiration to lung respiration (Decuypere et al., 1990). The peak of T_3 and T_4 is also associated with initiation of thermoregulatory responses to cooling (McNabb & Darras, 2015). Hypothyroidism in turkey embryos at pipping contributes to late embryonic mortality (Christensen & Biellier, 1982). Hypothyroidism due to exposure to environmental thyroid-disrupting chemicals, such as PCB and dioxin, delays hatching in chicken embryos (Bruggeman et al., 2003; Roelens et al., 2005). Thyroid hormones are critical to achieve successful hatching, with roles in thermoregulation and respiration.

Insulin-Like Growth Factors

Insulin-like growth factors, IGF-I and IGF-II, produced in the liver under the influence of growth hormone and modulated by thyroid hormones, are mitogenic peptides that play an important role in regulation of cellular proliferation, differentiation, and metabolism in different tissues (Stewart & Rotwein, 1996). IGF-I and IGF-II stimulate hepatic glycogen, DNA and protein synthesis in embryonic hepatocytes, cellular proliferation, and cartilage growth (McMurtry et al., 1997, 1998). The roles of IGF-I and IGF-II in poultry are similar to those in mammals (McMurtry et al., 1998). Plasma IGF-I levels are lower than IGF-II levels during incubation (Lu et al., 2007; McMurtry et al., 1998). Plasma IGF-II concentrations are 5- to 10-fold higher than IGF-I during incubation (Lu et al., 2007). Circulating IGF-II levels peak at 26E in turkey embryos, then decline after hatch (McMurtry et al., 1998). High levels of hepatic and brain IGF-II gene expression before hatching are consistent with an important role for IGF-II during embryonic development

(Richards et al., 2005). IGF-II is a key regulator of embryonic development in mammals (Stewart & Rotwein, 1996) and poultry (McMurtry et al., 1998). Stewart & Rotwein (1996) showed the importance of IGF-II for embryonic development using mice lacking IGF-II, which diminished embryonic growth yet allowing normal growth rates after birth. IGF-II is almost exclusively expressed in embryonic and neonatal tissues, as seen with decreasing levels post-hatch (Lu et al., 2007; Stewart & Rotwein, 1996). Thus, IGF-II may play a predominant role in growth and development during incubation, whereas IGF-I becomes predominant after hatch (Lu et al., 2007). IGF-I is important in postnatal development by mediating the growth-promoting effects of growth hormone (GH) (Richards et al., 2005). GH is unlikely to affect embryonic development because circulating concentrations of GH are very low in the embryo (Hazelwood, 2000). GH and IGF-I are not correlated during embryonic development (Richards et al., 2005). This is supported by low IGF-I gene expression throughout embryonic development (Richards et al., 2005) that peaks at mid-incubation for chicken and turkey embryos (Lu et al., 2007). IGF-knockout mice have profound embryonic and postnatal growth retardation as well as high early mortality rates (Woods et al., 1996). Targeted disruption of the mouse IGF-II gene produces proportional dwarfs, which are 60% of normal birthweight (DeChiara et al., 1990). The onset of the mutational effect leading to this growth deficiency occurs during early embryogenesis due to loss of the growth-promoting function of IGF-II mediated through signaling via type-I IGF and insulin receptors (Baker et al., 1993; Burns & Hassan, 2001; Louvi et al., 1997). These genetic mutations show the importance of IGF factors in embryonic development.

The developmental profile of plasma levels of insulin, glucagon, T_3, T_4, IGF-I, and IGF-II in embryos indicates that a close relationship exists among circulating levels of

metabolic hormones and known developmental events (Lu et al., 2007). If disruption of these hormones occurs during embryogenesis, developmental and hatching success are threatened.

Stage of Lay

As breeder hens age, egg characteristics also change along with egg production and influence turkey embryonic development. Breeder hen age and stage of egg production affect egg weight and composition (Applegate et al., 1999; Vierira and Moran, 1998; O'Dea et al., 2004; Christensen et al., 2001). Christensen et al. (1996) reported that old breeder hens (45 to 50 weeks of age) laid heavier eggs than young (33 to 38 weeks of age) and peak (39 to 44 weeks of age) breeder hens. This weight difference is in part due to a smaller proportion of yolk deposition and decreased fat content in eggs from old turkey breeder hens than eggs from young or peak turkey breeder hens (Applegate et al., 1996). Consequently, there is decreased lipoprotein transfer from yolk to embryo (Latour et al., 1996) and lower circulating cholesterol from lipoproteins in hatchlings from old breeder hens (Applegate et al., 1996; Vieira et al., 1999), resulting in decreased embryonic fat metabolism (McNaughton et al., 1978; Applegate et al., 1996). In a similar manner to turkeys, egg yolks from old broiler breeder hens (62 weeks of age) have been shown to have lower concentrations of phospholipids and free cholesterol than eggs despite the increased size proportion of yolk compared to young broiler breeder hens (27 weeks of age) (Vieira et al., 1998), implying reduced potential for energy metabolism. O'Dea et al. (2004) observed higher embryonic metabolism, as measured by increased CO_2 production during incubation, in eggs from older breeder hens. Increased embryonic heat (significantly increased eggshell temperature) is observed in eggs from older breeder hens (Hamidu et al., 2007). Therefore, increased embryonic heat is correlated to increased embryonic metabolism that produces larger

hatchlings with longer body lengths (O'Dea et al., 2004; Hamidu et al., 2007). The reduced potential for energy metabolism and decreased embryonic metabolism in turkey embryos from young breeder hens may compromise poult hatchability and livability.

Breeder stage of lay can influence post-hatch development. Generally, the larger the yolk, the greater amount of IgG is available for absorption by the chick (Li et al., 1998; Vieira et al., 1999). Therefore, older breeder hens producing larger eggs with heavier yolks have increased potential to positively influence immunity in their progeny as compared to young breeder hens. Improved immune responses in young birds may be beneficial when stressors are encountered during the first days of life. Alternatively, hatchlings from young breeders have slower small intestine villus growth and enterocyte migration during the first week of life than hatchlings from older breeders, resulting in shorter villus height (Applegate et al., 1999; Schaefer et al., 2006), crypt depth and reduced surface area (Schaefer et al., 2006). These factors may provide a mechanism for decreased nutrient absorption, resulting in decreased body weights for poults from young breeder flocks (Schaefer et al., 2006). However, Applegate et al. (1999) noted that reductions in villus height of poults from young breeders during the first week did not affect small intestine maturation after placement as compared to poults from older breeders. Breeder stage of lay is an epigenetic factor that may act to modify the physiology of embryos and young poults, reducing metabolism and growth rates with increasing breeder age.

Egg Storage

A potential stressor that influences turkey embryonic development is length of egg storage. At oviposition, embryos are at stage VII or VIII of early embryonic development (total of XI stages), with complete hypoblast formation (Gupta & Bakst, 1993). After

oviposition, embryo development becomes latent until the egg is incubated (Bergoug et al., 2013). Development of embryonic poults occurs in 3 major stages: germ establishment (during the first week of incubation), embryo completion (until day 22E of turkey incubation), and emergence from the eggshell (de Oliveira et al., 2008). Eggs storage at 18 to 20°C for up eight days (Bergoug et al., 2013) is a normal management practice (Tona et al., 2003) that prevents bacterial growth and stops the development of the embryo (Fasenko & O'Dea, 2008). Increased egg storage duration slows embryonic growth rate and increases the time from the first hatched egg to the last (hatch window) (Fasenko et al., 2002; Tona et al., 2003). A large hatch window results in non-uniform body weight at 4 days post-hatch, where early hatched birds had significantly lower body weights than late hatched birds (Careghi et al., 2005). Additionally, hatchability of fertile eggs cumulatively decreases by 0.2% up to seven days of storage, after which it decreases by 0.5% for each day up to day 14 (Yassin et al., 2008). By slowing embryonic development, egg storage length impacts the hatchability and uniformity of hatched turkey poults.

Incubation Conditions

Incubation conditions influence turkey embryonic development. It is suggested that incubation temperature, or more specifically embryo temperature, is the most important factor affecting chick quality (Lourens, 2003). Temperature determines embryonic metabolic rate and use rate of yolk and albumen utilization, and therefore, impacts embryogenesis (Lourens et al., 2007). Optimal incubation temperature for turkeys varies between 34°C and 37°C (Visschedijk, 1991). Extremely high incubation temperatures (greater than 43°C) can decrease chick hatchability by up to 50% (Ande & Wilson, 1981; Thompson et al., 1976; Wilson, 1991). High incubation temperatures (41.2°C) can also result in variable chick organ

size, such as smaller heart weight and larger liver weights (Leksrisompong et al., 2007). Low incubation temperatures (35.8°C) results in smaller kidneys and lungs weights, and larger heart weight (Givisiez et al., 2001). Overheating embryos may negatively impact their survival and hatched quality (Hamidu et al., 2007). However, thermal conditioning during embryonic development can lead to epigenetic adaptation to heat stress post-hatch. Chicken eggs submitted to heat stress of 39°C at days 13, 14, 15, 16 and 17 of incubation had proximate benefits on the efficiency of thermoregulation during post-hatch life (Moraes et al., 2003). Maintaining temperature profiles within a narrow range during incubation is critical to successful hatching and hatch quality.

Ideal incubation results can be obtained with 50% relative humidity but may range from 40% to 70% relative humidity depending on egg quality and breeder age (Lundy, 1969; Robertson, 1961). Egg weight loss, due to incubator relative humidity, must be between 6.5 and 14% of initial egg weight to obtain an adequate air cell size before internal pipping (Molenaar et al., 2010). Any egg weight loss above 14% puts the embryo at risk for dehydration or death (Tullett & Burton, 1982). High relative humidity increases mortality during early incubation by disrupting embryonic development due to disturbance of organizing centers (responsible for controlling chromosome segregation during cell division) or reduced gas exchange (Peebles et al., 1987; Robertson, 1961). Alternatively, lowering incubator relative humidity shortens hatching time by promoting extra metabolic water loss, resulting in increased metabolic rates and increased embryo temperatures (Peebles et al., 1987).

Decreasing hatchability with high humidity (75% to 80% RH) can be associated with high early and late embryo mortality (Robertson, 1961). The exchange of gases in the egg is

facilitated by the chorioallantoic membrane, which permits the diffusion of O_2 and CO_2 between the environment and the embryo (Tullett & Deeming, 1982). Oxygen becomes a limiting factor when embryos switch from yolk fat to carbohydrate as the primary substrate for energy metabolism (de Oliveira et al., 2008). Oxygen concentrations decline as embryonic development progresses. Low eggshell conductance (limited availability of O_2 and difficulty removing of CO_2) can trigger the hatching process and cause early pipping (Nangsuay et al., 2016). Additionally, an early rise in CO_2 during incubation, results in accelerated embryonic growth and earlier hatching (Bruggeman et al., 2007). Incubation conditions that do not provide proper O_2 and CO_2 levels to the developing embryos will alter the rate of embryonic development and affect viability and organ development, resulting in compromised poults. To optimize hatchability and survivability, egg incubation conditions specific to various management factors, such as breeder stage of lay and egg storage, must be developed and implemented in hatcheries.

FACTORS THAT INFLUENCE POST-HATCH DEVELOPMENT

Post-Hatch Organ Growth and Maturation

Growth is an increase in mass that involves cell proliferation (hyperplasia) and increase in cell size (hypertrophy) (Velleman & McFarland, 2015). As hatch approaches, the remaining yolk is internalized into the abdominal cavity and transported to the intestine through the yolk stalk (Noy & Sklan, 1998). Yolk utilization for energy metabolism functions during the first 48 hours post-hatch (Noy & Sklan, 1998). Thereafter birds utilize exogenous feed as the only source of nutrients. Energy balance, or energy homeostasis, is the regulation and control of energy expenditure and energy intake (Boswell, 2005). Negative energy balance is typically experienced during periods of feed deprivation (Boswell, 2005),

especially during growth when excess nutrients are needed to support fat and muscle tissue during growth, tissue development, and higher basal energy expenditure (Tam & Ravussin, 2012). The increased energy need is satisfied by increased feed intake during early life (Richards, 2003). There is direct competition between organ systems for nutrients to meet metabolic requirements during the first week of life when rapid growth and development occur (Moss et al., 1964; Noy & Sklan, 1998). These processes are often complex and susceptible to environmental stressors that can cause depressed growth rates or death.

Genetic selection for fast growth rates in poultry species are fueled by increased feed intake to maintain homeostasis and growth during early post-hatch life. Small intestine growth takes precedence over all other organ growth during the first days post-hatch (Noy et al., 2001), as young birds begin utilizing lipids and amino acids as substrates in energy metabolism (Lu et al., 2007). Unlike mammals, poultry enterocyte proliferation and differentiation is not localized in the crypt region, but also occurs along the villi (Uni et al., 1998) developing absorptive functions towards the tip of the villi (Simon & Gordon, 1995). Hypertrophy in the duodenum occurs for up to 9 days post-hatch, and until 6-days post-hatch in the jejunum, whereas enterocytes in the ileum may be relatively mature at hatch (Geyra et al., 2001). The maturity of the ileum at hatch indicates that the ileum may be the primary absorptive site immediately after hatching. The fast rate of proliferation of the duodenum and jejunum allows for increased surface area and therefore increase nutrient absorption capability in the future (Geyra et al., 2001). Disruptions to metabolic homeostasis in young birds can cause a shift towards negative energy balance when energy stores and feed intake for growth cannot be supported (Richards, 2003). Another stressor that causes increased feed intake without subsequent growth is cold environmental temperatures (Richards, 2003). Cold

stress shifts physiological signals (high glucagon concentrations) towards catabolism to maintain homeostasis instead of growth (Scanes, 2009). However, it is interesting to note that even in feed-deprived birds, the small intestine still matures during the first 5 days post-hatch (Uni et al., 1998), indicating the premier importance for the development of absorptive capacity for maintaining homeostasis and growth in young birds.

Development of the gastrointestinal tract supports the establishment of the microbiome and immune system in poultry. The colonization of the gut by microbiota in young animals occurs simultaneously with the development of the gut tissue, which is an ideal niche environment for microorganisms that are carried into the system as riders on feed or litter. These indigestible components escape digestion and absorption serve as substrates for growth of intestinal bacteria (Pan & Yu, 2014). Metabolic processes of gut microbial species produce short chain fatty acids that increase the rate of enterocyte growth and proliferation (Blottiere et al., 2003; Fukunaga et al., 2003; Le Blay et al., 2000) to support overall growth of post-hatch birds. Colonization of pathogenic organisms, such as *Salmonella spp.* (Potturi et al., 2005) or *Eimeria* (Barnes et al., 2000), can cause enteric disease leading to decreased productivity and increased mortality (Patterson & Burkholder, 2003). Alternatively, the use of exogenous probiotics can positively affect intestinal morphology in poultry species (Pan & Yu, 2014) by competitively excluding pathogenic bacteria and promoting colonization of beneficial species (Patterson & Burkholder, 2003). The synchronized and balanced development of the microbiome and immune system in poults, allows them to elicit an immune response when pathogens threaten homeostasis.

The immune system is composed of organs, cellular components and soluble components that can defend against infectious agents in two ways: innate (non-specific) and

adaptive (specific) immunity (Panda et al., 2008). The innate response is characterized by direct reaction after exposure and absence of immunological memory (Schokker et al., 2009). In the gut, the yolk sac is an important transfer mechanism for passive immunity until the post-hatch humoral immune system is initiated around 2 weeks of age (Glick, 2000). The adaptive immune response has a lag time between exposure and response and does generate immunological memory. First, pro-inflammatory cytokines promote inflammatory responses, which can be observed immediately after hatch and are dependent on the presence of microorganisms and feed (Schokker et al., 2009). Within the first 2 days post-hatch there is granulocytopoiesis and lymphocytosis (white blood cells) in the small intestine (Bar-Shira & Friedman, 2006). Additionally, at hatch immature T- and B-cells, components of adaptive immunity, are already observed in the gut associated lymphoid tissues (GALT), but functional maturation occurs during the first 2 weeks of life (Bar-Shira et al., 2003).

The immune function is essential to protect newly hatched poults when they are most vulnerable immediately after hatching. Immunosuppression in poultry can be induced by a variety of events, including infectious and non-infectious agents (Umar et al., 2017). Non-infectious agents are stressors that suppress innate immunity in poultry species including: incubation and hatching stresses, chronic handling, extended transportation times, suboptimal brooding (Hoerr, 2010). Infections may lead to immunosuppression by destroying immune cells or decreasing their functionality. Immunosuppression is characterized by increased mortality and decreased performance in the poultry industry (Hoerr, 2010).

Immunosuppressed flocks are susceptible to secondary infections, they respond poorly to vaccines, and they do not perform as well as non-affected birds (Calcagni & Elenkov, 2006). The outcomes of immunosuppression in poultry can be controlled by

implementing biosecurity measures that reduce or prevent exposure to the causes of immunosuppressive diseases, immunization that increase the resistance to challenge from immunosuppressive agents, and genetic selection (Hoerr, 2010). Immunosuppression can have negative impacts on early poult growth, and commercial operations should implement strategies for preventing immunosuppression to protect flock health.

Gastrointestinal microbiome and immune development in poultry are interconnected and can positively or negative influence the development of other systems. Infections by enteric pathogens can negatively impact intestinal morphology (Pan & Yu, 2014) by cross-talk between pathogens and epithelial tissues that results in extensive rearrangement of epithelial cells upon colonization by pathogens (Goosney et al., 2000; Sansonetti, 2001). The microbiome is also integrated with the immune system in the gastrointestinal tract. The mucus layer is part of the innate immune system where microorganisms colonize. The mucus layer prevents harmful gut microorganism from penetrating the intestinal epithelium (Brisbin et al., 2008). The microbiome can also increase the cell-mediated response to specific pathogens by increasing IgG and IgM production. These antibodies produced by B-cells as part of the adaptive immune response (Haghighi et al., 2006). Growth of the gastrointestinal tract is supported by concomitant development of the gastrointestinal immune system and the microbiome (Schokker et al., 2009). This is independent of feed and bacteria exposure. These two responses suggest innate immune preparedness in young poultry species during early post-hatch.

Muscle growth is directly tied to satellite cell activity in post-hatch birds. The first week post-hatch is the most important for muscle production (Moss et al., 1964). Skeletal muscle growth (myogenesis) in post-hatch birds is determined by hypertrophy and

accumulation of nuclei in muscle fibers (Sklan et al., 2003). At hatch, skeletal muscle contains a high percentage of proliferating satellite cells that supports post-hatch muscle fiber hypertrophy and DNA accretion through fusion of myofibers (Halevy et al., 2000). Satellite cells play a crucial role in DNA accretion of muscle fibers (Moss & Leblond, 1971). Located beneath the basal lamina, satellite cells are capable of proliferating, differentiating and joining existing fibers or fusing with each other to form new fibers (Halevy et al., 2000). Growth factors that influence satellite cell proliferation and differentiation are the insulin-like growth factors (IGF), fibroblast growth factors (FGF), platelet-derived growth factors (PDGF), hepatocyte growth factor (HGF), transforming growth factor-beta (TGF-β), and myostatin (Velleman & McFarland, 2015) growth hormone (GH) (Halevy et al., 2000) and thyroid hormones (McNabb & Darras, 2015). These factors act as mitogens (encouraging cell division), differentiation inhibitors (allows time for division). For example, insulin also supports in vitro multiplication of 1 day old broiler muscle satellite cells and delays their differentiation (Sato et al., 2012). T_3 stimulates chondrocyte hypertrophy but does not influence cell proliferation, which is only stimulated in the presence of IGF-1 (McNabb & Darras, 2015). At hatch, skeletal muscle consists of a high percentage of proliferating satellite cells, which decrease rapidly at the end of the growth period (Cardasis & Cooper, 1975). Despite the inactive state and diminished numbers of satellite cell in mature bird muscles, they also have the capacity to become active and proliferate (Allen et al., 1980), suggesting their importance in damaged muscle repair.

Delayed Placement

The transition time from hatch until placement in brooder barns can be stressful to newly hatched poults. Poults often experience delayed placement times of greater than 48

hours post-hatch as a cumulative result of normal hatchery and transportation practices. Delayed access to feed 48h post-hatch results in higher yolk weight and depleted glycogen stores as compared to birds given free access to feed and water within a 6-hour period after hatch (Donaldson et al., 1991; Noy & Sklan, 1999). The altered metabolic status and subsequent negative energy balance of poults leads to significantly increased mortality on the second day of holding (Pinchasov & Noy, 1993).

If birds survive the holding period, they are often dehydrated and experience high initial body weight losses and depressed growth rates after placement (Bergoug et al., 2013; Careghi et al., 2005). Feed-deprived and water-deprived poults are in severe negative energy balance, when glucose requirement exceeds gluconeogenic capacity (Donaldson et al., 1992), and they have depressed gastrointestinal tract development due to poor yolk sac utilization (Gonzales et al., 2003). At this point, intestinal growth becomes the limiting factor in post-hatch development (Sell et al., 1991). Despite the lack of exogenous feed, poult intestine size still increases (Noy et al., 2001) because the small intestine matures during the first 5 days of life, regardless of nutritional status (Uni et al., 1998). However, these birds have decreased villus length and villus surface area until after feeding in optimal conditions compared to birds that are fed immediately after hatch (Noy et al., 2001).

Feed deprivation beyond 24 hours has a negative effect on the maturation of both cell-mediated and humoral immune responses in poultry (Bar Shira et al., 2005; Bhanja et al., 2009; Panda et al., 2015). Since microbes enter the gastrointestinal tract as riders on feed and litter, delay of feed access can prolong the establishment of gut microbial species (Pan & Yu, 2014). Similarly, physical exposure to feed is one key stimulator of the immune system, consequently feed deprivation delays the onset of gut development (Geyra et al., 2001; Panda

et al., 2008). Feeding immediately after hatch provides an early antigenic stimulus, facilitating rapid differentiation of the immune organs, such as the bursa and spleen (Bar Shira et al., 2005; Ekino et al., 1980).

When feed is withheld, muscle retardation occurs, suggesting that genetic control of muscle development is down-regulated during feed-deprivation and is only activated upon feed availability (Bigot et al., 2003). Immediate feed availability post-hatch is critical for satellite cell proliferation, skeletal muscle development and growth (Halevy et al., 2000), and to meet the demands of intestine development. When feed-deprived poults are re-alimented, there is an increase in satellite cell mitotic activity as an attempt to meet unanswered demand for development and myonuclear accretion. However, periods of feed deprivation and delayed muscular development can not be compensated once the developmental process has been interrupted, during the critical period immediately post-hatch (Moore et al., 2005).

Hatchery services stress

Commercial turkey hatcheries implement standard practices on the last day of incubation to prepare newly hatched poults for placement in brooder barns. First, hatchery personnel pull poults from hatchers based on a percentage of total hatched poults. This creates a hatch duration or hatch window (time of first hatched poult until poults are removed from hatcher) that ranges from 24 to 48 hours, with the number of feed-deprived birds increasing as the spread increases (Careghi et al., 2005; Decuypere et al., 2001). Mortality is higher in birds that hatch early in the hatch window than birds who hatch later (Joseph & Moran, 2005).

Poult services are applied after poults are removed from the hatcher but before poults are transported and may vary due to company objectives and standard operating procedures. During this time, poults are routinely sexed, beak-trimmed, toe-trimmed, vaccinated and de-snooded (Carver et al., 2002). Poults are sexed because they are typically placed in brooder barns according to gender. Beak trimming in the poultry industry is done to prevent pecking, cannibalism, and avoid feed wastage (Kuenzel, 2007). Trimmed birds are generally less active, show less behavior involving pecking, eat less and grow more slowly than non-trimmed birds. The practice of poult beak trimming, particularly infrared beak treatment, may reduce mortality by inhibiting feather pecking, cannibalism and, aggressive pecking (Dennis et al., 2009; Hughes & Gentle, 1995). Toe trimming results in poults who rest more and eat less during the first days of life because of the pain associated with amputation of the claws, and this practice ultimately leads to decreased carcass scratching, and in the long term, it is beneficial to poult welfare (Fournier et al., 2015). Behavioral responses, such as decreased feed intake, may be due to pain experienced after the procedure (Gentle, 2011). Since HSP are involved in the pain signaling pathways, they may be used as a physiological indicator of stress due to poult servicing (Hutchinson et al., 2009).

Once hatchery services are complete, poults are placed in a shipping dock and await transportation. The amount of time the poults are waiting to be placed on a truck can vary based on company transportation schedules (Bergoug et al., 2013), truck availability and driver reliability. This indiscriminate period, increases the length of time poults are left without access to feed and water, placing poults at further risk for dehydration and distress.

Cumulatively, these stressful hatchery practices require 2 to 4 hours (even longer with variable wait times). Stressors applied during post-hatch servicing have additive effects and

trigger a rise of 13% to 30% in blood glucose, through mobilization of glycogen (Donaldson & Christensen, 1991). These potentially fatal metabolic changes elicit negative energy balance in turkey poults.

Transportation stress

After poults are loaded on trucks, transport time can be greater than 72 hours because hatcheries are often one or more states away from brooder farms (Lilburn & Loeffler, 2015). Stressors during transportation include temperature extremes, sudden acceleration and braking of the vehicle, vibration, abrasion on the crates, fasting, lack of water, , noise, and crowding (Glatz & Rodda, 2013; Willemsen et al., 2010). Mortality rates of greater than 2% often are observed after transportation of more than 5 hours in broilers chicks (Bianchi et al., 2005), and these deaths are attributed to dehydration and the birds' inability to dissipate heat (Bergoug et al., 2013; Lin et al., 2005; Raup & Bottje, 1990). The sources of heat within an enclosed transportation unit are related to radiation from the bodies of animals and conduction of heat from the outside (Crowther et al., 2003). The inability to dissipate heat results in hyperventilation and respiratory alkalosis. During dehydration, intracellular and extracellular water becomes deficient and normal metabolic process cannot occur, despite hormonal stress responses. Standing during transportation requires birds to brace themselves against vehicular motion (Crowther et al., 2003). This can lead to physical injuries that prevent them from achieving optimal growth on the farm. Alternatively, prolonged physical exertion can lead to 'capture myopathy' which involves a breakdown of muscle tissues leading to brain damage, paralysis and possibly death (Crowther et al., 2003; Wotton & Hewitt, 1999). Noise during transportation can cause a physiological stress response. For example, noise stimuli of both 80 dB and 100 dB intensities (normal range between 30 dB

and 55 dB) for 10 min induced significantly elevated plasma corticosterone levels of broilers (Chloupek et al., 2009). Crowding results in significantly decreased levels of glucose and increased concentrations of corticosterone (crowding) (Glatz & Rodda, 2013). General responses to transportation stress include increased levels of creatine kinase, triglycerides, corticosterone, adrenaline and noradrenaline, as well as a decrease in the total lipid content (Glatz & Rodda, 2013). These hormonal changes can lead to depletion of glycogen reserves. The resulting negative energy balance can lead to fatigue and potentially to death (Crowther et al., 2003; Jacobs et al., 2017). Transportation stress in combination with hatchery stress can be fatal in newly hatched poults. Therefore minimizing stress responses during these periods of time can promote poult health and prevent excess mortality during brooding.

Brooding

Management of environment and poult physiology during the first weeks of post-hatch is critical to optimize poult performance, and when environmental conditions are suboptimal, they can act as stressors that cause stress responses in turkey poults. Environmental conditions, such as temperature, relative humidity, ventilation, lighting during the critical brooding period are important factors influencing post-hatch development and performance (Gencoglan et al., 2009). Depressed growth rates during the first week post-hatch are observed when birds are reared in cooler brooding temperatures (25.6-27.8 °C) than normal brooding temperatures (27-35 °C) (van der Pol et al., 2013). Shorter chick lengths were also observed in cool brooded chicks than normal brooded chicks (van der Pol et al., 2013). A low level of relative humidity should be maintained during bird brooding to maintain low ammonia levels and reduce the risk of pathogenic disease and to maximize growth rates (Fairchild, 2009) and body weights at processing . (Weaver & Meijerhof, 1991).

High relative humidity (over 70%) is undesirable and should be remediated by adequate ventilation in buildings (Corkery et al., 2013). Ventilation is needed to regulate temperature and remove carbon dioxide, ammonia, other gases, moisture, dust and odors (Bucklin et al., 2009; Fairchild, 2009). Proper ventilation is necessary to provide the fresh air required to maintain good air quality, as well as to promote efficient operation of the cooling and heating systems (Mendes et al., 2013). Chick activity is greater in bright light intensity than low light intensity. Therefore, during brooding the light should be at the brightest intensity to encourage chick activity, assisting them to locate feed and water (Fairchild, 2009). Control of environmental conditions, particularly temperature and ventilation will minimize their effect as stressors on turkey poults.

Turkey poults experience human contact throughout their life, particularly during brooding when it is essential to keep birds contained with easy access to feed and water. Excessive handling of birds during brooding can cause a significant reduction in growth rate (Freeman & Manning, 1979). Chronic handling causes a significant decrease in corticosterone (Pierson et al., 1981), potentially making it more difficult to mobilize energy metabolites for maintenance of the homeostatic condition. Huff et al. (2001) reported that the stress of gently handling male turkeys once or twice daily for the first 10 days post-hatch, although tending to increase BW, can result in both an increased or decreased resistance to a bacterial respiratory challenge, depending on the number of handling events and the number of prior treatments with dexamethasone. Ultimately, turkeys can adapt to the stressor or become overwhelmed by a stress response, leading to death.

Turkey poults, similar to other poultry species, do not have the ability to regulate their internal body temperatures until about 6 days post-hatch, so they are sensitive to temperature

extremes during brooding that can alter their physiological stress responsiveness and lead to mortality during the first week post-hatch. The net energy stored in the tissues of a bird equals the difference between energy intake and energy loss. Metabolism of food and high environmental temperature are potential sources of energy while low environmental temperatures and the maintenance of normal body temperature are potential expenditures of energy (Etches et al., 2008). If young birds get cold within the first few days post-hatch,they become hypothermic. Hypothermia results in torpor, a state of slowed metabolism, crowding and inactivity to conserve energy (Wentworth et al., 2009). The effects of hypothermia are exacerbated by water and wind, which cause vasoconstriction, increased blood viscosity, and decreased circulation of blood to the periphery as a means to conserve body heat (Minka & Ayo, 2017). Birds in the hypothermic condition may not move to feed or water, which can result in death from dehydration or starvation (Julian, 2005). It is possible to induce early heat conditioning by exposing chickens to high temperatures (36C) for 24 hours at 3 to 5 days of age, to induce heat tolerance (Arjona et al., 1988, 1990; Yahav & Hurwitz, 1996; Zhou et al., 1997). There is an initial period of weight loss in these chickens, followed by compensatory growth resulting in higher BW at market age compared to non-conditioned chickens (Yahav & McMurtry, 2001; Yahav & Plavnik, 1999). Suggested mechanisms of action include induction of IGF-I to stimulate sattelite cell myogenesis (Halevy et al., 2000), as well as enterocyte proliferation, and increased brush border membrane activity (Uni et al., 2001). However, if young birds become hyperthermic during transportation or at the brooder farm, they begin to pant in an effort to dissipate heat by evaporative cooling. Prolonged panting will cause respiratory alkalosis due hyperventilation during hyperthermia. Respiratory alkalosis is a physiological state when excessive loss of CO_2 from the lungs

reduces the partial pressure of CO_2, and bicarbonate in the blood, and therefore lowers hydrogen ion concentration and a raises plasma pH (Richards, 1970; Wang et al., 1989). An additional mechanism to dissipate heat during heat stress is vasodilation which decreases blood pressure and increases cardiac output (Darre & Harrison, 1987). Birds under heat stress have a dichotomy of physiological mechanisms associated with thermoregulation and respiratory alkalosis: dissipation of heat by evaporative cooling demands an increase in respiration, while respiratory alkalosis demands a decrease in respiration (Etches et al., 2008). Water becomes deficient in intracellular fluids due to loss of intracellular K+, and it also becomes deficient in extracellular fluid due to loss of plasma Na+ (Nose et al., 1988). In response to this physiological dehydration, water consumption increases and feed intake decreases during heat stress (Etches et al., 2008; Yalcin et al., 1997). Physiological stress responses due to temperature extremes have the potential to disrupt acid-based balance in the body and lead to depressed growth or death in turkey poults.

Stressors due to early exposure to infectious agents or excessive activation of the immune system can lead to distress or death in brooding turkey poults. Poult enteritis complex (PEC) is a general term for a group of multifactorial, transmissible, infectious diseases of young turkeys characterized by enteritis, stunting (depressed growth), runting (slowed development), decreased feed efficiency, and nutritional deficiencies (Barnes et al., 2000). These clinical signs are consistent with general indicators of stress responses and immunosuppression (Hoerr, 2010). Several etiological agents associated with PEC are rotaviruses (McNulty, 1997), TCV (Dea et al., 1986; Guy et al., 1997; Hawk, 2000; Nagaraja & Pomeroy, 1997), turkey enterovirus (Guy & Barnes, 1991; Hayhow et al., 1993; McNulty & Guy, 1997), astrovirus (McNulty, 1993; Reynolds, 1997; Thouvenelle, Haynes, &

Reynolds, 1995; Thouvenelle, Haynes, Sell, et al., 1995), *Salmonella spp.* (Gast, 1997), *E. coli* (Edens et al., 1997ab; Guy et al., 2000), and Cryptosporidium spp. (Arafa et al., 1997; Bermudez et al., 1988; Current, 1997; Goodwin et al., 1988). Within 24 hours of infection, most flocks develop diarrhea that results in excessive fluid loss and hyperosmolar fluid in the intestinal lumen due to alterations in osmotic balance (Barnes et al., 2000; Cantey, 1993; Kidd et al., 1997). Consequently, birds will become lethargic and dehydrated. Additionally, the distribution of nutrients during limited feed intake and diarrhea preferentially provides the limited amount of nutrients to supply organs instead of demand organs resulting in lost growth potential (Barnes et al., 2000). This stress response causes the bird to be in negative energy balance, with nutrients utilized for maintenance of the homeostatic condition instead of growth, resulting in inferior performance or death. Occurrence of PEC is greatly influenced by environmental conditions on brooder farms, with higher incidence during warmer seasons due to more rapid pathogen multiplication at higher temperatures which increases the risk of contamination, greater number of disease vectors, and increased disease susceptibility during heat stress (Barnes et al., 2000). In commercial turkey flocks, exposure to environmental stressors causes excess mortality due to enteric diseases.

POULT QUALITY ASSESSMENT

Poult quality (PQ) is typically determined by quickly assessing visual parameters, on a small sampling of poults at hatcheries and on farms, using the Tona score protocol (Willemsen et al., 2008). The Tona score is a qualitative scoring system that assesses total score index of 100 based on a wide variety of visual parameters, such as activity, appearance, retracted yolk, eye condition, leg condition, navel deformities, remaining egg membrane, and remaining yolk (Tona et al., 2003). This subjective evaluation seems logical because visual

traits of development are associated with subsequent superior growth rates, breast meat yield, feed conversion ratios and survivability (Decuypere & Bruggeman, 2007). However, a strong positive correlation between Tona score and broiler performance has not been clearly demonstrated (Mukhtar et al., 2013).

There is controversy concerning the accuracy of quantitative parameters like day-old body weight as a predictor of final body weight because some researchers have found it to be an accurate predictor (Proudfoot & Hulan, 1981; Sklan et al., 2003), whereas others have not (Tona et al., 2004; Wolanski et al., 2004). These quantitative parameters are often used in combination with qualitative parameters, which are subjective in nature and difficult to replicate among evaluators due to high interpersonal variation of perception. Additionally, parameters included in the assessment and scoring system may need to be revised (Tona et al., 2005), with an emphasis on correlating factors and stressors during development with future growth performance characteristics of economic importance. For this reason, researchers have moved towards a strictly quantitative approach to assess PQ, including chick or poult length (Hill, 2001; Meijerhof, 2006; Molenaar et al., 2007; Wolanski et al., 2004) and shank length (Wolanski et al., 2004), which have all been proven to be reliable predictors of growth performance of chicks and poults. Further investigation is needed to correlate PQ factors / stressors to survivability and future performance using a combination of qualitative and quantitative measures.

Dissertation Hypothesis

Evidence for the impacts of breeder age and delayed placement on PQ and growth performance is strongly supported in the literature. Yet the question remains concerning how each of these factors individually alter the physiological or physical status of poults. Also,

there are validated methods to accurately determine which measurements have the most predictive value or are the most trustworthy when predicting future growth performance of turkeys.

Each chapter of this dissertation was designed to test a specific hypothesis within the common framework of the PQ and developmental factors as potential stressors (breeder age and delayed placement). These two controlled variables were used as a means to create variability in order to detect differences in PQ analysis. These differences collectively allowed us to determine the predictive success of individual PQ measurements relative to the controlled variables. The first hypothesis is that critical BW loss due to feed and water deprivation immediately post-hatch can be achieved within 48-hours. Next it is hypothesized that young breeder age and delayed placement time can reduce physical hatch measurements that are highly correlated to depressed growth performance traits during brooding. The subsequent hypothesis is that individual components of the Tona scoring system, are constraints of influence on subsequent production traits and compromised PQ specifically associated with young breeder hen age. Lastly, it is hypothesized that early poult mortality is related hatchery management practices that apply excess stress to young poults, specifically, young stage of lay breeders and increased travel distances from hatcheries to brooder farms. Cumulatively, the goal of this dissertation was to test the hypothesis that biological elements and management practices during embryonic and early poult development uniquely influence PQ, either as a stimulator of growth or a constraint that depresses growth. Furthermore, individual PQ indicators are predictive of survivability and future growth, relative to contributing developmental factors.

REFERENCES

Allen, R. E., McAllister, P. K., & Masak, K. C. (1980). Myogenic potential of satellite cells in skeletal muscle of old rats. A brief note. *Mechanisms of Ageing and Development*, *13*(2), 105–109.

Ande, T. B., & Wilson, H. R. (1981). Hatchability of chicken embryos exposed to acute high temperature stress at various ages. *Poultry Science*, *60*(7), 1561–1566.

Arafa, E. A. A., Sahlab, A. A. M., & Ibrahim, A. I. (1997). Cryptosporidiosis in turkey poults developmental stages and sensitivity of oocysts to disinfectants. *Assiut Veterinary Medical Journal*, *37*, 186–200.

Arjona, A. A., Denbow, D. M., & Weaver Jr, W. D. (1990). Neonatally-induced thermotolerance: Physiological responses. *Comparative Biochemistry and Physiology - A Molecular and Integrative Physiology*, *95*(3), 393–399.

Arjona, A. A., Denbow, D. M., & Weaver, W. D. (1988). High environmental temperatures just prior to marketing. *Poultry Science*, *67*, 226–231.

Baker, J., Liu, J. P., Robertson, E. J., & Efstratiadis, A. (1993). Role of insulin-like growth factors in embryonic and postnatal growth. *Cell*, *75*(1), 73–82.

Bar-Shira, E., & Friedman, A. (2006). Development and adaptations of innate immunity in the gastrointestinal tract of the newly hatched chick. *Developmental and Comparative Immunology*, *30*(10), 930–941.

Bar-Shira, E., Sklan, D., & Friedman, A. (2003). Establishment of immune competence in the avian GALT during the immediate post-hatch period. *Developmental and Comparative Immunology*, *27*(2), 147–157.

Bar Shira, E., Sklan, D., & Friedman, A. (2005). Impaired immune responses in broiler hatchling hindgut following delayed access to feed. *Veterinary Immunology and Immunopathology*, *105*(1–2), 33–45.

Barnes, H. J., Guy, J. S., & Vaillancourt, J. P. (2000). Poult enteritis complex. *Revue Scientifique et Technique (International Office of Epizootics)*, *19*(2), 565–588.

Bergoug, H., Burel, C., Guinebretiere, M., Tong, Q., Roulston, N., Romanini, C. E. B., … Eterradossi, N. (2013). Effect of pre-incubation and incubation conditions on hatchability, hatch time and hatch window, and effect of post-hatch handling on chick quality at placement. *World's Poultry Science Journal*, *69*(02), 313–334.

Bermudez, A. J., Ley, D. H., Levy, M. G., Ficken, M. D., & James, S. (1988). Intestinal and bursal cryptosporidiosis in turkeys following inoculation with Cryptosporidium sp . isolated from commercial poults. *Avian Diseases*, *32*(3), 445–450.

Bernal, J. (2007). Thyroid hormone receptors in brain development and function. *Nature Clinical Practice Endocrinology & Metabolism*, *3*(3), 249–259.

Bhanja, S. K., Devi, C. A., Panda, A. K., & Sunder, G. S. (2009). Effect of post hatch feed deprivation on yolk-sac utilization and performance of young broiler chickens. *Asian-Australasian Journal of Animal Sciences*, *22*(8), 1174–1179.

Bianchi, M., Petracci, M., & Cavani, C. (2005). Effects of transport and lairage on mortality, liveweight loss and carcass quality in broiler chickens. *Italian Journal of Animal Science*, *4*, 516–518.

Bigot, K., Mignon-Grasteau, S., Picard, M., & Tesseraud, S. (2003). Effects of delayed feed intake on body, intestine, and muscle development in neonate broilers. *Poultry Science*, *82*(5), 781–788.

Blottiere, H. M., Buecher, B., Galmiche, J. P., & Cherbut, C. (2003). Molecular analysis of the effect of short-chain fatty acids on intestinal cell proliferation. *Proceedings of the Nutrition Society*, *62*, 101–106.

Boswell, T. (2005). Regulation of energy balance in birds by the neuroendocrine hypothalamus. *Poultry Science*, *42*, 161–181.

Brisbin, J. T., Gong, J., & Sharif, S. (2008). Interactions between commensal bacteria and the gut-associated immune system of the chicken. *Animal Health Research Reviews / Conference of Research Workers in Animal Diseases*, *9*(1), 101–110.

Bruggeman, V., Swennen, Q., De Ketelaere, B., Onagbesan, O., Tona, K., & Decuypere, E. (2003). Embryonic exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin in chickens: Effects of dose and embryonic stage on hatchability and growth. *Comparative Biochemistry and Physiology - C Toxicology and Pharmacology*, *136*(1), 17–28.

Bruggeman, V., Witters, A., De Smit, L., Debonne, M., Everaert, N., Kamers, B., … Decuypere, E. (2007). Acid-base balance in chicken embryos (Gallus domesticus) incubated under high CO2concentrations during the first 10 days of incubation. *Respiratory Physiology and Neurobiology*, *159*(2), 147–154.

Bucklin, R. A., Jacob, J. P., Mather, F. B., Leary, J. D., & Naas, I. A. (2009). Tunnel ventilation of broiler houses. *Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida*, *PS-46*.

Burns, J. L., & Hassan, A. B. (2001). Cell survival and proliferation are modified by insulin-like growth factor 2 between days 9 and 10 of mouse gestation. *Development*, *128*(19), 3819–3830.

Calcagni, E., & Elenkov, I. (2006). Stress system activity, innate and T helper cytokines, and susceptibility to immune-related diseases. *Annals of the New York Academy of Sciences*, *1069*, 62–76.

Cantey, J. R. (1993). Escherichia coli diarrhea. *Gastroenterology Clinics of North America*, *22*(3), 609–622.

Cardasis, C. A., & Cooper, G. W. (1975). An analysis of nuclear numbers in individual muscle fibers during differentiation and growth: A satellite cell-muscle fiber growth unit. *Journal of Experimental Zoology*, *191*(3), 347–357.

Careghi, C., Tona, K., Onagbesan, O., Buyse, J., Decuypere, E., & Bruggeman, V. (2005). The effects of the spread of hatch and interaction with delayed feed access after hatch on broiler performance until seven days of age. *Poultry Science*, *84*, 1314–1320.

Carver, D. . K., Fetrow, J., Gerig, T., Krueger, T., & Barnes, H. J. (2002). Hatchery and transportation factors associated with early poult mortality in commercial turkey flocks. *Poultry Science*, *81*(12), 1818–1825.

Chloupek, P., Voslářová, E., Chloupek, J., Bedáňová, I., Pištěková, V., & Večerek, V. (2009). Stress in broiler chickens due to acute noise exposure. *Acta Veterinaria Brno*, *78*(1), 93–98.

Christensen, V. L., & Biellier, H. V. (1982). Physiology of turkey embryos during pipping and hatching. IV. Thyroid function in embryos from selected hens. *Poultry Science*, *61*(12), 2482–2488.

Christensen, V. L., Donaldson, W. E., & Nestor, K. E. (1993). Embryonic viability and metabolism in turkey lines selected for egg production or growth. *Poultry Science*, *72*, 829–838.

Christensen, V. L., Wineland, M. J., Fasenko, G. M., & Donaldson, W. E. (2001). Egg storage effects on plasma glucose and supply and demand tissue glycogen concentrations of broiler embryos. *Poultry Science*, *80*(12), 1729–1735.

Corkery, G., Ward, S., Colum Kenny, & Hemmingway, P. (2013). Monitoring environmental parameters in poultry production facilities. In *Computer Aided Process Engineering - CAPE Forum 2013, 2013*. Institute for Process and Particle Engineering, Graz University of Technology, Austria.

Crowther, C., Davies, R., & Glass, W. (2003). The effect of night transportation on the heart rate and skin temperature of ostriches during real transportation. *Meat Science*, *64*(4), 365–370.

Current, W. L. (1997). Cryptosporidiosis. In B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of Poultry* (10th ed., pp. 883–890). Ames, IA: Iowa State University Press.

Dantzer, R., & Mormède, P. (1983). Stress in farm animals: a need for reevaluation. *Journal of Animal Science*, *57*(1), 6–18.

Darre, M. J., & Harrison, P. C. (1987). Heart rate, blood pressure, cardiac output, and total peripheral resistance of single comb White Leghorn hens during an acute exposure to 35 C ambient temperature. *Poultry Science*, *66*(3), 541–547.

De Groef, B., Grommen, S. V. H., & Darras, V. M. (2007). Feedback control of thyrotropin secretion in the chicken: Thyroid hormones increase the expression of hypophyseal somatostatin receptor types 2 and 5. *General and Comparative Endocrinology*, *152*(2–3), 178–182.

de la Rosa, E. J., & de Pablo, F. (2011). Proinsulin: from hormonal precursor to neuroprotective factor. *Frontiers in Molecular Neuroscience*, *4*(20), 1–7.

de Oliveira, J. E., Druyan, S., Uni, Z., Ashwell, C. M., & Ferket, P. R. (2013). Metabolic profiling of late-term turkey embryos by microarrays. *Poultry Science*, *92*(4), 1011–1028.

de Oliveira, J. E., Uni, Z., & Ferket, P. R. (2008). Important metabolic pathways in poultry embryos prior to hatch. *World's Poultry Science Journal*, *64*(4), 488–499.

de Pablo, F., Robcis, H. L., Caldes, T., Alemany, J., Scavo, L., & Serrano, J. (1991). Insulin-like growth factor-I and insulin as growth and differentiation factors in chicken embryogenesis. *Poultry Science*, *70*, 1790–1796.

de Pablo, F., Roth, J., Hernandez, E., & Pruss, R. M. (1982). Insulin is present in chicken eggs and early chick embryos. *Endocrinology*, *111*(6), 1909–1916.

Dea, S. ., Marsolais, G. ., Beaubien, J. ., & Ruppanner, R. . (1986). Coronaviruses associated with outbreaks of transmissible enteritis of turkeys in quebec : hemagglutination properties and cell cultivation. *Avian Diseases*, *30*(2), 319–326.

DeChiara, T. M., Efstratiadis, A., & Robertson, E. J. (1990). A growth-deficiency phenotype in heterozygous mice carrying an insulin-like growth factor II gene disrupted by targeting. *Nature*, *345*(6270), 78–80.

Decuypere, E., & Bruggeman, V. (2007). The endocrine interface of environmental and egg factors affecting chick quality. *Poultry Science*, *86*, 1037–1042.

Decuypere, E., Dewil, E., & Kuhn, E. R. (1990). The hatching process and the role of hormones. In S. G. Tullett (Ed.), *Avian Incubation* (pp. 239–256). Kent: Butterworth & Co Ltd.

Decuypere, E., Tona, K., Bruggeman, V., & Bamelis, F. (2001). The day-old chick: a crucial hinge between breeders and broilers. *World's Poultry Science Journal*, *57*(2), 127–138.

Dennis, R. L., Fahey, A. G., & Cheng, H. W. (2009). Infrared beak treatment method compared with conventional hot-blade trimming in laying hens. *Poultry Science*, *88*(1), 38–43.

Dohms, J. E., & Metz, A. (1991). Stress - mechanisms of immunosuppression. *Veterinary Immunology and Immunopathology*, *30*, 89–109.

Donaldson, W. E., Brewer, C. E., Ferket, P. R., & Christensen, V. L. (1992). Posthatch carbohydrate feeding and subsequent performance of turkey poults. *Poultry Science*, *71*, 128–132.

Donaldson, W. E., & Christensen, V. L. (1991). Dietary carbohydrate level and glucose metabolism in turkey poults. *Comparative Biochemistry and Physiology, 98A*(2), 347–350.

Donaldson, W. E., Christensen, V. L., & Krueger, K. K. (1991). Effects of stressors on blood glucose and hepatic glycogen concentrations in turkey poults. *Comparative Biochemistry and Physiology Part A: Physiology*, *100*(4), 945–947.

Edens, F. W., Parkhurst, C. R., Qureshi, M. A., Casas, I. A., & Havenstein, G. B. (1997). Atypical Escherichia coli strains and their association with poult enteritis and mortality syndrome. *Poultry Science, 76*(7), 952–960.

Edens, F. W., Qureshi, R. A., Parkhurst, C. R., Qureshi, M. A., Havenstein, G. B., & Casas, I. A. (1997). Characterization of two Escherichia coli isolates associated with poult enteritis and mortality sundrome. *Poultry Science*, *76*, 1665–1673.

Ekino, S., Nawa, Y., Tanaka, K., Matsuno, K., Fujii, H., & Kotani, M. (1980). Suppression of immune response by isolation of the bursa of fabricius from environmental stimuli. *Australian Journal of Experimental Biology and Medical Science*, *58*(3), 289–296.

Etches, R. J., John, M. T., & Verrinder Gibbons, A. M. (2008). Behavioral, physiological, neuroendocrine and molecular responses to heat stress. In N. J. Daghir (Ed.), *Poultry Production In Hot Climates* (2nd ed., pp. 48–79). Cambridge, MA: CABI.

Fairchild, B. (2009). Environmental factors to control when brooding chicks. *University of Georgia College of Agricultural and Environmental Sciences, Cooperative Extension, Learning for Life*, (1287), 1–5.

Fasenko, G. M., & O'Dea, E. E. (2008). Evaluating broiler growth and mortality in chicks with minor navel conditions at hatching. *Poultry Science*, *87*, 594–597.

Fasenko, G. M., Robinson, F. E., & Christensen, V. L. (2002). How long-term hatching egg storage affects the egg, the embryo, and the chick. In D. C. Deeming (Ed.), *Practical Aspects of Commercial Incubation in Poultry* (pp. 33–39). Oxford, UK: Ratite Conf. Books.

Forrest, D., & Swaroop, A. (2012). Minireview: The role of nuclear receptors in photoreceptor differentiation and disease. *Molecular Endocrinology*, *26*(6), 905–915.

Fournier, J., Schwean-Lardner, K., Knezacek, T. D., Gomis, S., & Classen, H. L. (2015). The effect of toe trimming on behavior, mobility, toe length and other indicators of welfare in tom turkeys. *Poultry Science*, *94*(7), 1446–1453.

Foye, O. T. (2005). *The biochemical and molecular effects of amnionic nutrient administration , "in ovo feeding" on intestinal development and function and carbohydrate metabolism in turkey embryos and poults*. North Carolina State University.

Franssens, L., Buyse, J., Decuypere, E., & Everaert, N. (2014). Relationship between glucose and pancreatic hormones during the embryonic and postnatal phase in chickens. *Avian Biology Research*, *7*(1), 33–38.

Freeman, B. M., & Manning, A. C. (1979). Stressor effects of handling on the immature fowl. *Research in Veterinary Science*, *26*(2), 223–226.

Fukunaga, T., Sasaki, M., Araki, Y., Toshihiko, O., Yasuoka, T., Tsujikawa, T., … Bamba, T. (2003). Effects of the soluble fibre pectin on intestinal cell proliferation, fecal short chain fatty acid production and microbial population. *Digestion*, *67*, 42–49.

Gast, R. K. (1997). Paratyphoid infections. In B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of Poultry* (10th ed., pp. 97–121). Ames, IA: Iowa State University Press.

Gencoglan, S., Gencoglan, C., & Akyuz, A. (2009). Supplementary heat requirements when brooding tom turkey poults. *South African Journal of Animal Sciences*, *39*(1), 1–9.

Gentle, M. J. (2011). Pain issues in poultry. *Applied Animal Behaviour Science*, *135*(3), 252–258.

Geyra, A., Uni, Z., & Sklan, D. (2001). Enterocyte dynamics and mucosal development in the posthatch chick. *Poultry Science*, *80*(6), 776–782.

Givisiez, P. E. N., Silva, M. M., Mazzi, C. M., Ferro, M. I. T., Ferro, J. a, Gonzales, E., & Macari, M. (2001). Heat or cold chronic stress affects organ weights and Hsp70 levels in chicken embryos. *Canadian Journal of Animal Science*, *81*, 83–88.

Glatz, P., & Rodda, B. (2013). Turkey farming: welfare and husbandry issues. *African Journal of Agricultrural Research*, *8*(48), 6149–6163.

Glick, B. (2000). Immunophysiology. In G. C. Whittow (Ed.), *Avian Physiology* (pp. 657–670). San Diego: Academic Press.

Gonzales, E., Kondo, N., Saldanha, E. S. P. B., Loddy, M. M., Careghi, C., & Decuypere, E. (2003). Performance and physiological parameters of broiler chickens subjected to fasting on the neonatal period. *Poultry Science*, *82*(8), 1250–1256.

Goodwin, M. A., Steffens, W. L., Russell, I. D., & Brown, J. (1988). Diarrhea associated with intestinal cryptosporidiosis in turkeys. *Avian Diseases*, *32*(1), 63–67.

Goosney, D. L., Gruenheid, S., & Finlay, B. B. (2000). Gut feelings: enteropathogenic E. coli (EPEC) interactions with the host. *Annual Review of Cell and Developmental Biology*, *16*, 173–189.

Graczyk, S. (2003). Examinations of hematological and metabolic changes mechanisms of acute stress in turkeys. *Electronic Journal of Polish Agricultural Universities, Veterinary Medicine*, *6*(1).

Gupta, S. K., & Bakst, M. R. (1993). Turkey embryo staging from cleavage through hypoblast formation. *Journal of Morphology*, *217*(3), 313–325.

Guy, & Barnes, H. J. (1991). Partial Characterization of a Turkey Enterovirus-Like Virus. *Avian Diseases*, *35*(1), 197–203.

Guy, J. S., Barnes, H. J., Smith, L. G., & Breslin, J. (1997). Antigenic characterization of a turkey coronavirus identified in poult enteritis- and mortality syndrome-affected turkeys. *Avian Diseases*, *41*(3), 583–590.

Guy, J. S., Smith, L. G., Breslin, J. J., Vaillancourt, J. P., & Barnes, H. J. (2000). High mortality and growth depression experimentally produced in young turkeys by dual infection with enteropathogenic Escherichia coli and turkey coronavirus. *Avian Diseases*, *44*(1), 105–113.

Haghighi, H. R., Gong, J., Gyles, C. L., Hayes, M. A., Zhou, H., Sanei, B., … Sharif, S. (2006). Probiotics stimulate production of natural antibodies in chickens. *Clinical and Vaccine Immunology*, *13*(9), 975–980.

Halevy, O., Geyra, A., Barak, M., Uni, Z., & Sklan, D. (2000). Early posthatch starvation decreases satellite cell proliferation and skeletal muscle growth in chicks. *The Journal*

of Nutrition, *130*(4), 858–864.

Hamidu, J. A., Fasenko, G. M., Feddes, J. J. R., O'Dea, E. E., Ouellette, C. A., Wineland, M. J., & Christensen, V. L. (2007). The effect of broiler breeder genetic strain and parent flock age on eggshell conductance and embryonic metabolism. *Poultry Science*, *86*(11), 2420–2432.

Hawk, M. V. (2000). *Effects of turkey Coronavirus (NC95) on commercial turkey poults*. North Carolina State University, Raleigh, North Carolina.

Hayhow, C. S., Saif, Y. M., Kerr, K. M., & Whitmoyer, R. E. (1993). Further observations on enterovirus infection in specific-pathogen-free turkey poults. *Avian Diseases*, *37*(1), 124–134.

Hazelwood, R. L. (2000). Pancreas. In G. Whittow (Ed.), *Sturkie's Avian Physiology* (fifth, pp. 539–555). New York: Academic Press.

Hill, D. (2001). Chick length uniformity profiles as a field measurement of chick quality. *Avian Poultry Biological Reviews*, *12*, 188.

Hoerr, F. J. (2010). Clinical aspects of immunosuppression in poultry. *Avian Diseases*, *54*, 2–15.

Huff, G., Huff, W., Balog, J., & Rath, N. (2001). Effect of early handling of turkey poults on later responses to multiple dexamethasone-Escherichia coli challenge. 1. Production values and physiological response. *Poultry Science*, *80*(9), 1314–1322.

Huff, G., Huff, W., Rath, N., Donoghue, A., Anthony, N., & Nestor, K. (2007). Differential effects of sex and genetics on behavior and stress response of turkeys, (April), 1294–1303.

Hughes, B. O., & Gentle, M. J. (1995). Beak trimming of poultry: Its implications for welfare. *World's Poultry Science Journal*, *51*(1), 51–61.

Humane Farm Animal Care Standards for Turkeys. (2013). Herndon, VA. Retrieved from http://certifiedhumane.org/wp-content/uploads/Std14.Turkeys.2A-2.pdf%0D

Hutchinson, M. R., Ramos, K. M., Loram, L. C., Wieseler, J., Paige, W., Kearney, J. J., …

Watkins, L. R. (2009). Evidence for a role of heat shock protein-90 (HSP90) in TLR4 mediated pain enhancement in rats. *Neuroscience*, *164*(4), 1821–1832.

Jacobs, L., Delezie, E., Duchateau, L., Goethals, K., Ampe, B., Buyse, J., & Tuyttens, F. A. M. (2017). Impact of transportation duration on stress responses in day-old chicks from young and old breeders. *Research in Veterinary Science*, *112*(April), 172–176.

Jacquier-Sarlin, M. R., Fuller, K., Dinh-Xuan, A. T., Richard, M. J., & Polla, B. S. (1994). Protective effects of hsp70 in inflammation. *Experientia*, *50*(11–12), 1031–1038.

Joseph, N. S., & Moran, E. T. (2005). Effect of flock age and postemergent holding in the hatcher on broiler live performance and further-processing yield. *Journal of Applied Poultry Research*, *14*(3), 512–520.

Julian, R. J. (2005). Production and growth related disorders and other metabolic diseases of poultry - A review. *Veterinary Journal*, *169*(3), 350–369.

Kidd, M. T., Ferket, P. R., & Garlich, J. D. (1997). Nutritional and osmoregulatory functions of betaine. *World's Poultry Science Journal*, *53*, 125–139.

Kregel, K. C. (2002). Invited Review: Heat shock proteins: modifying factors in physiological stress responses and acquired thermotolerance. *Journal of Applied Physiology*, *92*(5), 2177–2186.

Kuenzel, W. J. (2007). Neurobiological basis of sensory perception: welfare implications of beak trimming. *Poultry Science*, *86*, 1273–1282.

Le Blay, G., Blottière, H. M., Ferrier, L., Le Foll, E., Bonnet, C., Galmiche, J. P., & Cherbut, C. (2000). Short-chain fatty acids induce cytoskeletal and extracellular protein modifications associated with modulation of proliferation on primary culture of rat intestinal smooth muscle cells. *Digestive Diseases and Sciences*, *45*(8), 1623–1630.

Leksrisompong, N., Romero-Sanchez, H., Plumstead, P. W., Brannan, K. E., & Brake, J. (2007). Broiler incubation. 1. Effect of elevated temperature during late incubation on body weight and organs of chicks. *Poultry Science*, *86*(12), 2685–2691.

Lilburn, M. S. (1998). Practical aspects of early nutrition for poultry. *The Journal of Applied Poultry Research*, *7*(4), 420–424.

Lilburn, M. S., & Loeffler, S. (2015). Early intestinal growth and development in poultry. *Poultry Science*, *94*(7), 1569–1576.

Lin, H., Zhang, H. F., Du, R., Gu, X. H., Zhang, Z. Y., Buyse, J., & Decuypere, E. (2005). Thermoregulation responses of broiler chickens to humidity at different ambient temperatures. I. One Week of Age. *Poultry Science*, *84*(8), 1173–1178.

Lourens, A. (2003). Residual yolk and egg weight loss during incubation under controlled eggshell temperatures. *Avian and Poultry Biology Reviews*, *14*(4), 209–211.

Lourens, A., van den Brand, H., Heetkamp, M. J. W., Meijerhof, R., & Kemp, B. (2007). Effects of eggshell temperature and oxygen concentration on embryo growth and metabolism during incubation. *Poultry Science*, *86*(10), 2194–2199.

Louvi, A., Accili, D., & Efstratiadis, A. (1997). Growth-promoting interaction of IGF-II with the insulin receptor during mouse embryonic development. *Developmental Biology*, *189*(1), 33–48.

Lu, J. W., McMurtry, J. P., & Coon, C. N. (2007). Developmental changes of plasma insulin, glucagon, insulin-like growth factors, thyroid hormones, and glucose concentrations in chick embryos and hatched chicks. *Poultry Science*, *86*, 673–683.

Lundy, H. (1969). A review of the effects of temperature, humidity, turning and gaseous environment in the incubator on the hatchability of hen's egg. In T. Carter & B. Freeman (Eds.), *The Fertility and Hatchability of the Hen's Egg* (pp. 143–176). Edinburgh, Scotland: Oliver and Boyd.

Mailyan, E. S. (2016). Stress in poultry: focus on turkeys causes, mechanism, consequences, diagnostics and prevention. *Journal of Dairy, Veterinary & Animal Research*, *3*(6).

McMurtry, J. P., Francis, G. L., & Upton, Z. (1997). Insulin-like growth factors in poultry. *Domestic Animal Endocrinology*, *14*(4), 199–229.

McMurtry, J. P., Rosebrough, R. W., Brocht, D. M., Francis, G. L., Upton, Z., & Phelps, P. (1998). Assessment of developmental changes in chicken and turkey insulin-like growth factor-II by homologous radioimmunoassay. *Journal of Endocrinology*, *157*(3), 463–473.

McNabb, & Darras, V. M. (2015). Thyroids. In C. G. Scanes (Ed.), *Sturkie's Avian*

Physiology (Sixth, pp. 535–547). London: Academic Press.

McNabb, F., & Darras, V. M. (2015). Thyroids. In *Sturkie's Avian Physiology* (Sixth, pp. 535–547).

McNabb, F., & King, D. B. (1993). Thyroid hormone effects on growth, development and metabolism. In M. P. Schreibman, C. G. Scanes, & P. K. T. Pang (Eds.), *The Endocrinology of Growth, Development, and Metabolism of Vertebrates* (pp. 393– 417). New York: Academic Press.

McNabb, F. M. A. (2006). Avian thyroid development and adaptive plasticity. *General and Comparative Endocrinology*, *147*(2), 93–101.

McNulty, M. S. (1993). Astrovirus infection of turkeys. In J. B. McFerran & M. S. McNulty (Eds.), *Vims infections of birds* (pp. 509–511). Amsterdam: Elsevier Science Publishers.

McNulty, M. S. (1997). Rotavirus infections. In B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of Poultry* (10th ed., pp. 692–701). Ames, IA: Iowa State University Press.

McNulty, M. S., & Guy, J. S. (1997). Avian enteroviruslike viruses. In B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of poultry* (10th ed., pp. 706–710). Ames, IA: Iowa State University Press.

Meijerhof, R. (2006). Chick size matters. *World Poultry*, *22*, 30–31.

Mendes, A. S., Moura, D. J., Naas, I. . A., Morello, G. M., Carvalho, T. M. R., Refatti, R., & Paixao, S. J. (2013). Minimum ventilation systems and their effects on the initial stage of turkey production. *Brazilian Journal of Poultry Science*, *15*(1), 7–14.

Minka, N. S., & Ayo, J. O. (2017). Severe hypothermia in transported pullets: case study of its occurrence , diagnosis and treatment using active external rewarming technique. *Veterianry Medicine and Science*, *3*, 115–122.

Molenaar, R., Reijrink, I. A. M., Meijerhof, R., & Brand, H. (2007). Relationship between chick length and chick weight at hatch and slaughter weight and breast meat yield in broilers. In *3rd Combined Workshop on Fundamental Physiology and Perinatal Development in Poultry*.

Molenaar, R., Reijrink, I., Meijerhof, R., & Van den Brand, H. (2010). Meeting embryonic requirements of broilers throughout incubation: a review. *Brazilian Journal of Poultry Science*, *12*(3), 137–148.

Moore, D. T., Ferket, P. R., & Mozdziak, P. E. (2005). Early post-hatch fasting induces satellite cell self-renewal. *Comparative Biochemistry and Physiology, 142A*(3), 331–339.

Moraes, V. M. B., Malheiros, R. D., Bruggeman, V., Collin, A., Tona, K., Van As, P., … Macari, M. (2003). Effect of thermal conditioning during embryonic development on aspects of physiological responses of broilers to heat stress. *Journal of Thermal Biology*, *28*(2), 133–140.

Moran, E. T. (2007). Nutrition of the developing embryo and hatchling. *Poultry Science*, *86*(5), 1043–1049.

Moss, F. P., & Leblond, C. P. (1971). Satellite cells as the source of nuclei in muscles of growing rats. *The Anatomical Record*, *170*(4), 421–435.

Moss, F. P., Simmonds, R. A., & McNary, H. W. (1964). The growth and composition of skeletal muscle in the chicken 2. The relationship between muscle weight and the number of nuclei. *Poultry Science*, *43*(5), 1086–1091.

Mukhtar, N., Khan, S. H., & Anjum, M. S. (2013). Hatchling length is a potential chick quality parameter in meat type chickens. *World's Poultry Science Journal*, *69*(04), 889–896.

Muramatsu, T., Hiramoto, K., Koshi, N., Okumura, J., Miyoshi, S., & Mitsumoto, T. (1990). Importance of albumen content in whole-body protein synthesis of the chicken embryo during incubation. *British Poultry Science*, *31*(1), 101–106.

Nagaraja, K., & Pomeroy, B. S. (1997). Coronaviral enteritis of turkeys (bluecomb disease). In B. W. Calnek, B. H.J, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of Poultry* (10th ed., pp. 686–692). Ames, IA: Iowa State University Press.

Nangsuay, A., Meijerhof, R., van den Anker, I., Heetkamp, M. J. W., Morita, V. D. S., Kemp, B., & van den Brand, H. (2016). Effects of breeder age, broiler strain, and eggshell temperature on development and physiological status of embryos and hatchlings. *Poultry Science*, *95*, 1666–1679.

Nose, H., Mack, G. W., Shi, X. R., & Nadel, E. R. (1988). Shift in body fluid compartments after dehydration in humans. *Journal of Applied Physiology*, *65*(1), 318–324.

Noy, Y., Geyra, A., & Sklan, D. (2001). The effect of early feeding on growth and small intestinal development in the posthatch poult. *Poultry Science*, *80*(7), 912–919.

Noy, Y., & Sklan, D. (1998). Yolk utilisation in the newly hatched poult. *British Poultry Science*, *39*(3), 446–451.

Noy, Y., & Sklan, D. (1999). Energy utilization in newly hatched chicks. *Poultry Science*, *78*(12), 1750–1756.

Pan, D., & Yu, Z. (2014). Intestinal microbiome of poultry and its interaction with host and diet. *Gut Microbes*, *5*(1), 108–119.

Panda, A. K., Bhanja, S. K., & Sunder, G. S. (2015). Early post hatch nutrition on immune system development and function in broiler chickens. *World's Poultry Science Journal*, *71*, 285–296.

Panda, A. K., Rama Rao, S. V., Raju, M. V. L. N., Reddy, M. R., & Shyam Sunder, G. (2008). Early gut development: Its importance in broiler production. In *Proceedings of the seminar on Sustainable poultry production: Rural and commercial approach* (pp. 79–83).

Pascual, A., & Aranda, A. (2013). Thyroid hormone receptors, cell growth and differentiation. *Biochimica et Biophysica Acta - General Subjects*, *1830*(7), 3908–3916.

Patterson, J. A., & Burkholder, K. M. (2003). Application of prebiotics and probiotics in poultry production. *Poultry Science*, *82*, 627–631.

Peebles, E. D., Brake, J., & Gildersleeve, R. P. (1987). Effects of eggshell cuticle removal and incubation humidity on embryonic development and hatchability of broilers. *Poultry Science*, *66*(5), 834–840.

Picardo, M., & Dickson, A. J. (1982). Hormonal regulation of glycogen metabolism in hepatocyte suspensions isolated from chicken embryos. *Comparative Biochemistry and Physiology*, *71B*(4), 689–693.

Pierson, F. W., Hester, P. Y., & Wilson, E. K. (1981). The effects of chronic handling on the incidence of leg abnormalities and several blood parameters in turkeys. *Poultry Science*, *60*(6), 1333–1339.

Pinchasov, Y., & Noy, Y. (1993). Comparison of post-hatch holding time and subsequent early performance of broiler chicks and turkey poults. *British Poultry Science*, *34*(1), 111–120.

Pirskanen, A., Kiefer, J. C., & Hauschka, S. D. (2000). IGFs, insulin, Shh, bFGF, and TGF-β1 interact synergistically to promote somite myogenesis in vitro. *Developmental Biology*, *224*(2), 189–203.

Potturi, P. V. L., Patterson, J. a, & Applegate, T. J. (2005). Effects of delayed placement on intestinal characteristics in turkey poults. *Poultry Science*, *84*(5), 816–824.

Proudfoot, F. G., & Hulan, H. W. (1981). The influence of hatching egg size on the subsequent performance of broiler chickens. *Poultry Science*, *60*(10), 2167–2170.

Raup, T. J., & Bottje, W. G. (1990). Effect of carbonated water on arterial ph, pco2 and plasma lactate in heat-stressed broilers. *British Poultry Science*, *31*(2), 377–384.

Reynolds, D. L. (1997). Astrovirus infections. In B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of Poultry* (10th ed., pp. 701–705). Ames, IA: Iowa State University Press.

Reyns, G. E., Venken, K., Morreale De Escobar, G., Kühn, E. R., & Darras, V. M. (2003). Dynamics and regulation of intracellular thyroid hormone concentrations in embryonic chicken liver, kidney, brain, and blood. *General and Comparative Endocrinology*, *134*(1), 80–87.

Richards. (1970). Physiology of thermal panting in birds. *Annales de Biologie Animale Biochimie Biophysique*, *10*(2), 151–168.

Richards. (2003). Genetic regulation of feed intake and energy balance in poultry. *Poultry Science*, *82*(6), 907–916.

Richards, M. P., Poch, S. M., & McMurtry, J. P. (2005). Expression of insulin-like growth factor system genes in liver and brain tissue during embryonic and post-hatch development of the turkey. *Comparative Biochemistry and Physiology - A Molecular*

and Integrative Physiology, *141*(1), 76–86.

Robertson, I. S. (1961). Studies on the effect of humidity on the hatchability of hen's eggs I. The determination of optimum humidity for incubation. *The Journal of Agricultural Science*, *57*(2), 185–914.

Roelens, S. A., Beck, V., Maervoet, J., Aerts, G., Reyns, G. E., Schepens, P., & Darras, V. M. (2005). The dioxin-like PCB 77 but not the ortho-substituted PCB 153 interferes with chicken embryo thyroid hormone homeostasis and delays hatching. *General and Comparative Endocrinology*, *143*, 1–9.

Rokutan, K. (2000). Role of heat shock proteins in gastric mucosal protection. *Journal of Gastroenterology and Hepatology*, *15 Suppl*(October 1999), D12–D19.

Romanoff, A. L. (1967). Biochemistry of the avian embryo: A quantitative analysis of prenatal development. *Interscience Publishing*.

Rusch, A., Ng, L., Goodyear, R., Oliver, D., Lisoukov, I., Vennstrom, B., … Forrest, D. (2001). Retardation of cochlear maturation and impaired hair cell function caused by deletion of all known thyroid hormone receptors. *The Journal of Neuroscience*, *21*(24), 9792–9800.

Sansonetti, P. J. (2001). Rupture, invasion and inflammatory destruction of the intestinal barrier by Shigella, making sense of prokaryote-eukaryote cross-talks. *FEMS Microbiology Reviews*, *25*, 3–14.

Sato, K., Aoki, M., Kondo, R., Matsushita, K., Akiba, Y., & Kamada, T. (2012). Administration of insulin to newly hatched chicks improves growth performance via impairment of myod gene expression and enhancement of cell proliferation in chicken myoblasts. *General and Comparative Endocrinology*, *175*(3), 457–463.

Scanes, C. (2009). Perspectives on the endocrinology of poultry growth and metabolism. *General and Comparative Endocrinology*, *163*(1–2), 24–32.

Scanes, C. G. (2016). Biology of stress in poultry with emphasis on glucocorticoids and the heterophil to lymphocyte ratio. *Poultry Science*, *95*(9), 2208–2215.

Schokker, D., Hoekman, A. J. W., Smits, M. A., & Rebel, J. M. J. (2009). Gene expression patterns associated with chicken jejunal development. *Developmental and Comparative*

Immunology, *33*(11), 1156–1164.

Schultz-Cherry, S., Kapczynski, D. R., Simmons, V. M., Matthew, D., Brown, C., Barnes, H. J., … Barnesd, H. J. (2000). Identifying agent(s) associated with poult enteritis mortality syndrome : Importance of the thymus. *Avian Diseases*, *44*(2), 256–265.

Sell, J. L., Angel, C. R., Piquer, F. J., Mallarino, E. G., & Al-Batshan, H. A. (1991). Developmental patterns of selected characteristics of the gastrointestinal tract of young turkeys. *Poultry Science*, *70*(5), 1200–1205.

Selye, H. (1946). The general adptation syndrome and disease of adaptation. *Journal of Clinical Endocrinology*, *6*, 117–231.

Simon, T. C., & Gordon, J. I. (1995). Intestinal epithelial cell differentiation: new insights from mice, flies and nematodes. *Current Opinion in Genetics and Development*, *5*, 577–586.

Sklan, D., Heifetz, S., & Halevy, O. (2003). Heavier chicks at hatch improves marketing body weight by enhancing skeletal muscle growth. *Poultry Science*, *82*, 1778–1786.

Stewart, C. E., & Rotwein, P. (1996). Growth, differentiation, and survival: multiple physiological functions for insulin-like growth factors. *Physiological Reviews*, *76*(4), 1005–1026.

Tam, C. S., & Ravussin, E. (2012). Energy balance: An overview with emphasis on children. *Pediatric Blood and Cancer*, *58*(1), 154–158.

Thompson, J. B., Wilson, H. R., & Voitle, R. A. (1976). Influence of high temperature stress of 16-day embryos on subsequent hatchability. *Poultry Science*, *55*(3), 892–894.

Thouvenelle, Haynes, A. J. S., & Reynolds, D. L. (1995). Astrovirus infection in hatchling turkeys : histologic , morphometric , and ultrastructural findings. *Avian Diseases*, *39*(2), 328–336.

Thouvenelle, Haynes, J., Sell, J., & Reynolds, D. (1995). Astrovirus infection in hatchling turkeys : Alterations in intestinal maltase activity. *Avian Diseases*, *39*(2), 343–348.

Tona, K., Bamelis, F., De Ketelaere, B., Bruggeman, V., Moraes, V., Buyse, J., …

Decuypere, E. (2003). Effects of egg storage time on spread of hatch, chick quality, and chick juvenile growth. *Poultry Science*, *82*(5), 736–741.

Tona, K., Bruggeman, V., Onagbesan, O., Bamelis, F., Gbeassor, M., Mertens, K., & Decuypere, E. (2005). Day-old chick auality: relationship to hatching egg quality, adequate incubation practice and prediction of broiler performance. *Avian and Poultry Biology Reviews*, *16*(2), 109–119.

Tona, K., Onagbesan, O., De Ketelaere, B., Decuypere, E., & Bruggeman, V. (2004). Effects of age of broiler breeders and egg storage on egg quality, hatchability, chick quality, chick weight, and chick posthatch growth to forty-two days. *Journal of Applied Poultry Research*, *13*(1), 10–18.

Tullett, S. G., & Burton, F. G. (1982). Factors affecting the weight and water status of the chick at hatch. *British Poultry Science*, *23*(4), 361–369.

Tullett, S. G., & Deeming, D. C. (1982). The relationship between eggshell porosity and oxygen consumption of the embryo in the domestic fowl. *Comparative Biochemistry and Physiology*, *72A*(3), 529–533.

Umar, S., Munir, M. T., Ahsan, U., Raza, I., Chowdhury, M. R., Ahmed, Z., & Shah, M. A. A. (2017). Immunosuppressive interactions of viral diseases in poultry. *World's Poultry Science Journal*, *73*(1), 121–135.

Uni, Z., & Ferket, R. P. (2004). Methods for early nutrition and their potential. *World's Poultry Science Journal*, *60*(01), 101–111.

Uni, Z., Gal-Garber, O., Geyra, A., Sklan, D., & Yahav, S. (2001). Changes in growth and function of chick small intestine epithelium due to early thermal conditioning. *Poultry Science*, *80*(4), 438–445.

Uni, Z., Platin, R., & Sklan, D. (1998). Cell proliferation in chicken intestinal epithelium occurs both in the crypt and along the villus. *Journal of Comparative Physiology*, *168B*(4), 241–247.

USDA-ERS. (2017). Turkey Sector: Background & Statistics. Retrieved December 4, 2017, from https://www.ers.usda.gov/newsroom/trending-topics/turkey-sector-background-statistics/

van der Pol, C. W., van Roovert-Reijrink, I. A. M., Maatjens, C. M., van den Brand, H., & Molenaar, R. (2013). Effect of relative humidity during incubation at a set eggshell temperature and brooding temperature posthatch on embryonic mortality and chick quality. *Poultry Science*, *92*(8), 2145–2155.

Veissier, I., & Boissy, A. (2007). Stress and welfare: Two complementary concepts that are intrinsically related to the animal's point of view. *Physiology and Behavior*, *92*(3), 429–433.

Velleman, S. G., & McFarland, D. C. (2015). Skeletal Muscle. In C. G. Scanes (Ed.), *Sturkie's Avian Physiology* (Sixth, pp. 379–402). London: Academic Press.

Visschedijk, A. H. J. (1991). Physics and Physiology of incubation. *British Poultry Science*, *32*(1), 3–20.

Wang, S., Bottje, W. G., Kinzler, S., Neldon, H. L., & Koike, T. I. (1989). Effect of heat stress on plasma levels of arginine vasotocin and mesotocin in domestic fowl (Gallus domesticus). *Comparative Biochemistry and Physiology -- Part A: Physiology*, *93*(4), 721–724.

Weaver, W. D., & Meijerhof, R. (1991). The effect of different levels of relative humidity and air movement on litter conditions, ammonia levels, growth, and carcass quality for broiler chickens. *Poultry Science*, *70*(4), 746–755.

Wentworth, B., Cigan, J., & Schaaf, T. (2009). Tolerance of Japanese quail embryos and young chicks to hypothermia. *Poultry Science*, *88*(5), 1040–1043.

Willemsen, H., Debonne, M., Swennen, Q., Everaert, N., Careghi, C., Han, H., … Decuypere, E. (2010). Delay in feed access and spread of hatch: importance of early nutrition. *World's Poultry Science Journal*, *66*(02), 177–188.

Willemsen, H., Everaert, N., Witters, A., De Smit, L., Debonne, M., Verschuere, F., … Bruggeman, V. (2008). Critical assessment of chick quality measurements as an indicator of posthatch performance. *Poultry Science*, *87*(11), 2358–2366.

Wilson, H. R. (1991). Interrelationships of egg size, chick size, posthatching growth and hatchability. *World's Poultry Science Journal*, *47*(1), 5–20.

Wolanski, N. J., Luiten, E. J., Meijerhof, R., & Vereijken, A. L. J. (2004). Yolk utilisation

and chick length as parameters for embryo development. *Avian and Poultry Biology Reviews, 15*(3–4), 233–234.

Woods, K. A., Camacho-Hubner, C., Savage, M. O., & Clark, A. J. L. (1996). Intrauterine growth retardation and postnatal growth failure associated with deletion of the insulin-like growth factor I gene. *The New England Journal of Medicine, 335*(18), 1363–1367.

Wotton, S. B., & Hewitt, L. (1999). Transportation of ostriches--a review. *The Veterinary Record, 145*, 725–731.

Yahav, S., & Hurwitz, S. (1996). Induction of thermotolerance in male broiler chickens by temperature conditioning at an early age. *Poultry Science, 75*(3), 402–406.

Yahav, S., & McMurtry, J. P. (2001). Thermotolerance acquisition in broiler chickens by temperature conditioning early in life -The effect of timing and ambient temperature. *Poultry Science, 80*(12), 1662–1666.

Yahav, S., & Plavnik, I. (1999). Effect of early-age thermal conditioning and food restriction on performance and thermotolerance of male broiler chickens. *British Poultry Science, 40*(1), 120–126.

Yalcin, S., Settar, P., Ozkan, S., & Cahaner, A. (1997). Comparative evaluation of three commercial broiler stocks in hot versus temperate climates. *Poultry Science, 76*, 921–929.

Yassin, H., Velthuis, A. G. J., Boerjan, M., Van Riel, J., & Huirne, R. B. M. (2008). Field study on broiler eggs hatchability. *Poultry Science, 87*(11), 2408–2417.

Zhou, W. T., Fujita, M., Ito, T., & Yamamoto, S. (1997). Effects of early heat exposure on thermoregulatory responses and blood viscosity of broilers prior to marketing. *British Poultry Science, 38*(3), 301–306.

Zulkifli, I., & Siegel, P. B. (1995). Is there a positive side to stress? *World's Poultry Science Journal, 51*(1), 63–76.

CHAPTER 2.

RESEARCH NOTE: PRELIMINARY RESEARCH ON THE EFFECTS OF VARIOUS BREEDER HEN AGES ON BODY WEIGHT LOSS DURING THE FIRST 48-HOURS POST-HATCH IN TURKEY POULTS

S. A. BLACK[1]

NORTH CAROLINA STATE UNIVERSITY

KEYWORDS: BREEDER AGE, TURKEY POULTS, DEHYRDATION

PRIMARY AUDIENCE: HATCHERY PERSONNEL

[1] Corresponding author: sablack@ncsu.edu

SUMMARY

Time from hatching to placement is an important factor for post-hatch development of turkey poults. During this time, access to feed and water is withheld. Depending on the environmental conditions during exposure and length of exposure to these conditions, body weight (BW) loss and death can occur. In this experiment, when poults were feed- and water-deprived during a 48-hour holding period, both poults hatched from young and old breeder hens had BW losses of over 14%. Delayed placement can prolong the development of the gastrointestinal tract and skeletal muscle. Dehydration during the first 48-hours post-hatch may result in growth retardation and negative carry-over effects on market age performance in turkey poults.

DESCRIPTION OF PROBLEM

Delayed placement time can be associated with hatch spread (time from the first hatched poult to hatch pull), poult servicing (routine sexing, beak-trimming, toe-trimming, vaccination and de-snooding), hatchery wait times (time after servicing when poults await transportation in the holding room), and transportation (up to 72 hours). BW losses of over 10% during holding periods result in dehydration of chicks and poults [1-2]. Extended placement times can result in moisture loss of 13 to 23% over a 48-hour period (without or with poult servicing) [3]. Chick dehydration during the first days of life can have a negative impact on subsequent growth rates [4]. Nir and Levanon (1993) observed a total of 6 grams of BW, with 33% reduction in yolk sac (2 grams) and 67% reduction in other body tissues (4 grams) during a 48-hour holding period. Birds hatched from young breeder hens are often small and more susceptible to mortality than birds hatched from peak breeder hens due to reduced utilization of yolk lipid [5–7]. It was hypothesized that critical BW loss due to feed

and water deprivation immediately post-hatch can be achieved within 48-hours. To test this hypothesis, poults from two different aged breeder hens were individually weighed every 6 hours for 48 hours to determine cumulative and period BW losses.

MATERIALS AND METHODS

200 male poults were obtained from a commercial hatchery within one hour of the time that hatch was pulled [8]. 100 poults from first of lay breeder hens (FOL = first week of lay) and 100 poults were from peak production breeder hens (PEAK = 10^{th} week of lay) were weighed at the hatchery and transported one hour (Scott Hall, NCSU Prestage Department of Poultry Science, Raleigh, NC), then held in a temperature-controlled room at 32°C for 48 hours. All birds were individually weighed at 6, 12, 18, 24, 30, 36, 42, and 48 hours. The percentage of total and period BW loses were calculated [9]. Data were analyzed using ANOVA procedure in JMP Pro 13 (SAS Institute Inc., Cary, NC). Least square means were used to determine significance at less than or equal to 0.05.

RESULTS AND DISCUSSION

At the time of hatch, FOL poults weighed significantly less than PEAK poults (56.25 g versus 60.39 g) and remained smaller over the course of 48 hours (47.51 g versus 51.46 g) (Table 2.1). By 48-hours post hatch, FOL poults had significantly higher BW losses (15.01%) than PEAK poults (14.18 %). Feed- and water-deprived poults have lower placement weights and depressed growth rates than poults with immediate access to feed and water during the same period post-hatch [10, 11]. Depressed growth may occur due to BW losses over 10% and subsequent dehydration [1, 2, 12]. Dehydrated poults lack intracellular fluid but have excess extracellular fluid, which is found in the intestinal lumen and consequently leaves the body as urine or diarrhea resulting in further BW loss [13]. Absolute

carcass water losses parallel carcass weight losses during holding periods, increasing over time [3]. Therefore, any moisture loss greater than 10% will result in dehydration, and greater than 20% may lead to mortality [12].

Interestingly, the rates of BW loss over 48 hours were similar among FOL and PEAK poults (Figure 2.1). However, the greatest drop in BW for FOL poults occurred between 42 to 48 hours without feed and water (2.15%), whereas the greatest drop in BW for PEAK poults occurred between 36 to 42 hours (1.70%) (Table 2.1; Figure 2.2). Turkey poults held without feed and water lose more BW from 24 to 48 hours than from 0 to 24 hours post-hatch [14]. To maintain energy balance, protein is spared during holding periods compared to moisture and fat [7], however negative energy balance ultimately occurs in feed- and water-deprived birds due to accelerated depletion of body protein reserves [3]. Negative energy balance leads to altered body composition, reduced subsequent growth rate [14], and increased mortality [15]. BW losses and dehydration during the first days of life can have a negative impact on subsequent growth rates, equaling a loss of up to 2 days of BW gain at market age in broilers [4]. Additionally, feed- and water-deprivation reduces the rate of yolk uptake in poults [7]. The yolk is necessary to fuel intermediary metabolism during the first days of life, and the extent of availability of this nutrient supply may be influential in determining tissue capacity for expansion [7]. The limiting factor in post-hatch growth may be gastrointestinal tract development, which increases in size regardless of access to food [16], indicating the importance of supply organs and nutrients that are necessary to fuel growth and development.

CONCLUSIONS AND APPLICATIONS

1. All turkey had BW losses of over 14% by 48-hours post-hatch. Poults from FOL breeder hens had a greater negative response to feed- and water- deprivation post-hatch than poults from PEAK breeder hens.
2. BW loss due to feed- and water- deprivation, reaches a critical point beyond 10%, can lead to dehydration and negative energy balance. These metabolic responses can negatively impact future growth performance of turkey poults.

Table 2.1 The effect of breeder hen age on body weight loss from 0-48 hours post-hatch in turkey poults.

Time Point (hr)	**0**	**6**	**12**	**18**	**24**	**30**	**36**	**42**	**48**
Average Body Weight (g)[1]					*(g)*				
FOL	56.25[b]	54.99[b]	54.12[b]	53.18[b]	52.00[b]	51.12[b]	50.09[b]	49.16[b]	47.51[b]
PEAK	60.39[a]	59.16[a]	57.95[a]	57.01[a]	56.00[a]	54.68[a]	53.66[a]	52.36[a]	51.46[a]
SEM	0.51	0.48	0.49	0.49	0.47	0.47	0.46	0.46	0.56
Total Body Weight Loss (%)					*(%)*				
FOL		1.40	3.08	4.77	6.83	8.46	10.26	12.09	15.01
PEAK		1.36	3.40	5.20	6.62	8.84	10.51	12.66	14.18
SEM		1.2	1.1	1.1	1.2	1.2	1.2	1.1	1.3
Period (hrs)		**0-6**	**6-12**	**12-18**	**18-24**	**24-30**	**30-36**	**36-42**	**42-48**
Period Body Weight Loss (%)					*(%)*				
FOL		1.40	0.87	0.99	1.40	0.77	1.23	0.96	2.15
PEAK		1.36	1.44	0.92	0.99	1.60	1.35	1.70	1.08
SEM		1.2	1.2	1.2	1.3	1.2	1.2	1.3	1.6

[a,b] Means within a row with different superscripts are significantly different ($p<0.05$).

[1]Mean values are averages of 100 poults per stage of lay (FOL = first week of lay or PEAK = 10th week of lay)

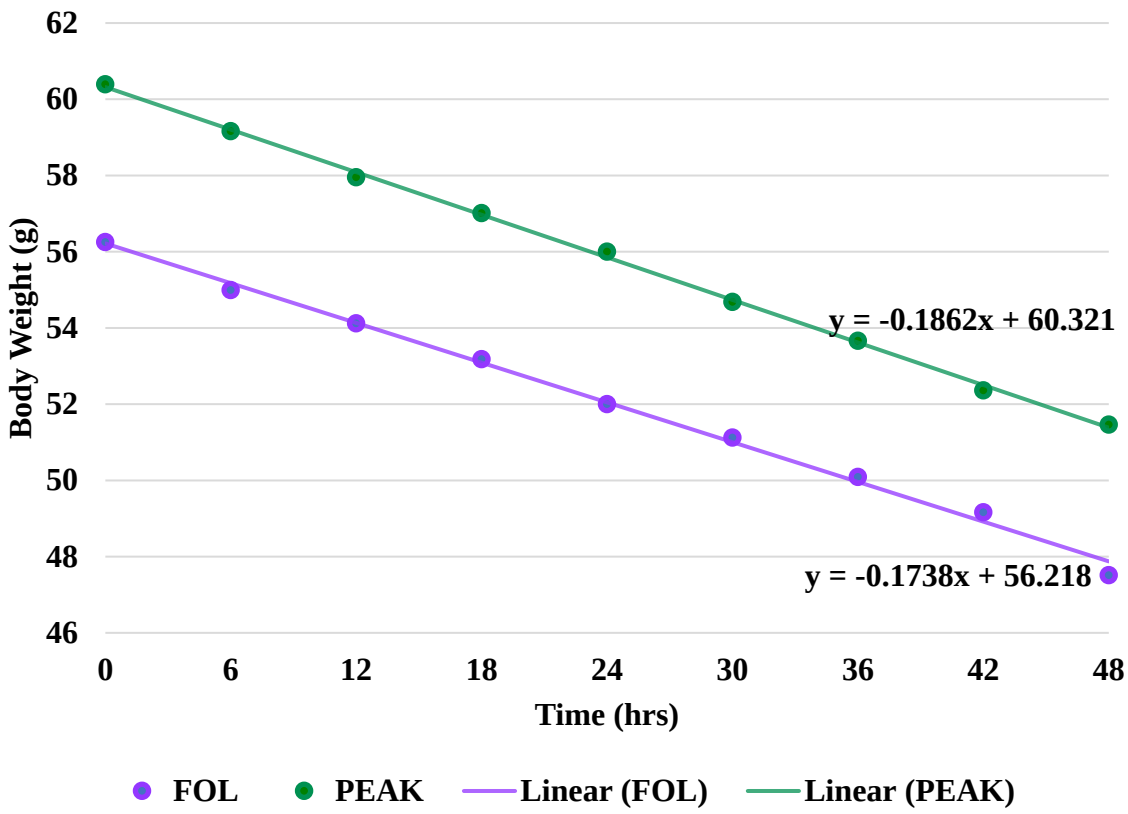

Figure 2.1. Body Weight Loss of FOL and PEAK turkey poults from 0 to 48 hours post-hatch

REFERENCES AND NOTES

[1] J. E. Hager and W. L. Beane, “Posthatch incubation time and early growth of broiler chickens,” *Poult. Sci.*, vol. 62, no. 2, pp. 247–254, 1983.

[2] L. T. Reis Gama and L. H. Chaveiro Soares, M., “Effects of Short Storage Conditions and Broilder Breeder Age on Hatchiability, Haching Time and Chicken Weights,” *Poult. Sci.*, vol. 76, pp. 1459–1466, 1997.

[3] W. E. Donaldson, J. Clark, and V. L. Christensen, “Protein, lipid and glycogen stores in newly-hatched turkey (Meleagris gallopavo) poults as affected by post-hatch Stressors and holding time,” *Comp. Biochem. Physiol. -- Part A Physiol.*, vol. 107, no. 3, pp. 559–562, 1994.

[4] I. Nir and M. Levanon, “Effect of posthatch holding time on performance and on residual yolk and liver composition,” *Poult. Sci.*, vol. 72, pp. 1994–1997, 1993.

[5] R. . C. Noble, F. Lonsdale, K. Connor, and D. Brown, “Changes in the lipid metabolism of the chick embryo with parental age,” *Poult. Sci.*, vol. 65, pp. 409–416, 1986.

[6] E. D. Peebles, R. W. Keirs, L. W. Bennett, T. S. Cummings, S. K. Whitmarsh, and P. D. Gerard, “Relationships among post-hatch physiological parameters in broiler chicks hatched from young breeder hens and subjected to delayed brooding placement,” *International Journal of Poultry Science*, vol. 3, no. 9. pp. 578–585, 2004.

[7] E. T. Moran and B. S. Reinhart, “Poult yolk sac amount and composition upon placement: effect of breeder age, egg weight, sex, and subsequent change with feeding or fasting,” *Poult. Sci.*, vol. 59, no. 7, pp. 1521–1528, 1980.

[8] “Nicholas Super Select tom poults were hatched in Goldsboro, NC at Butterball L.L.C.” .

[9] “Percent BW Loss = [(Starting BW – Ending BW) / Starting BW] x 100 .” .

[10] C. Careghi, K. Tona, O. Onagbesan, J. Buyse, E. Decuypere, and V. Bruggeman, “The effects of the spread of hatch and interaction with delayed feed access after hatch on broiler performance until seven days of age,” *Poult. Sci.*, vol. 84, pp. 1314–1320, 2005.

[11] H. Bergoug *et al.*, "Effect of pre-incubation and incubation conditions on hatchability, hatch time and hatch window, and effect of post-hatch handling on chick quality at placement," *Worlds. Poult. Sci. J.*, vol. 69, no. 02, pp. 313–334, 2013.

[12] G. D. Butcher and A. H. Nilipour, "Broiler management -The first 24 hours," *Univ. Florida Coop. Ext. Serv. Inst. Food Agric. Sci. EDIS*, 2002.

[13] H. Nose, G. W. Mack, X. R. Shi, and E. R. Nadel, "Shift in body fluid compartments after dehydration in humans," *J. Appl. Physiol.*, vol. 65, no. 1, pp. 318–324, 1988.

[14] Y. Pinchasov and Y. Noy, "Comparison of post-hatch holding time and subsequent early performance of broiler chicks and turkey poults," *Br. Poult. Sci.*, vol. 34, no. 1, pp. 111–120, 1993.

[15] W. E. Donaldson, C. E. Brewer, P. R. Ferket, and V. L. Christensen, "Posthatch carbohydrate feeding and subsequent performance of turkey poults," *Poult. Sci.*, vol. 71, pp. 128–132, 1992.

[16] J. L. Sell, C. R. Angel, F. J. Piquer, E. G. Mallarino, and H. A. Al-Batshan, "Developmental patterns of selected characteristics of the gastrointestinal tract of young turkeys," *Poult. Sci.*, vol. 70, no. 5, pp. 1200–1205, May 1991.

CHAPTER 3.

Effects of Breeder Hen Age and Placement Time on Poult Quality and Early Performance in Turkey Poults

S. Black[†], R. Malheiros, P. Ferket

Section: Management and Production

[†] Corresponding author: sablack@ncsu.edu

ABSTRACT

A battery cage experiment was conducted to assess early performance and development effects of breeder hen age and placement time in turkey poults to two weeks of age. The experiment was designed as a factorial, completely randomized arrangement of two breeder hen age groups (first week of lay = **FOL** and 14th week of lay = **PEAK**) and two placement times (immediate = **IP** or 48-hour holding period without feed or water = **DP**). BW, feed intake, and FCR, along with jejunum and ileum mucosal histology samples were analyzed by ANOVA procedure. Poult quality (**PQ**) was assessed at placement and correlated to BW using MANOVA procedure. FOL poults weighed significantly less than PEAK poults, while IP poults weighed significantly more than DP poults. Increased hock vein protrusion (**HV**) and shank color (**SC**) in DP poults predicted lower future BWs than IP poults. Increased navel buttons in FOL poults, decreased activity level in PEAK poults and placement BW may be indicators of suboptimal embryonic development. However, placement hatchling weight was not correlated significantly with future BWs (negative correlations). Villus height (**H**) and villus surface area (**SA**) in FOL and DP poults were significantly larger in the ileum and smaller in the jejunum at days 2 and 7 compared to PEAK and IP poults. Other mucosal measurements were inconsistent among treatment groups, but at 14 days of age there were no significant differences in intestinal mucosal tissues. In brief, day 2 and 7 BWs, navel buttons, HV and SC were successful PQ indicators of decreased growth of FOL and DP poults during the first 2 weeks of life.

Key Words: breeder age, delayed placement, turkey poults, brooding, growth performance

INTRODUCTION

Current production schemes expose poultry to numerous stressors during development and early life (Dantzer & Mormède, 1983). Minimizing stress during the first week of life is critical for maximizing livability and performance in poultry flocks (Decuypere & Bruggeman, 2007; Lilburn, 1998; Willemsen et al., 2010). Poult quality (**PQ**) is an indicator of survivability and future growth performance characteristics of young turkey poults. PQ is evaluated both qualitatively and quantitatively in turkey hatcheries and brooding farms. The Tona score is the most commonly used method for hatchling evaluation (Tona et al., 2003; Willemsen et al., 2008). This qualitative scoring measures a wide variety of parameters including activity, appearance, retracted yolk, eye condition, leg condition, navel deformities, remaining egg membrane, and remaining yolk (Tona et al., 2003). However, it is difficult to replicate. Therefore, many poultry scientists have moved towards a more quantitative approach to assess PQ including day-old BW (Decuypere et al., 2002; Deeming, 2000) and shank length (**SL**) (Wolanski et al., 2004). A majority of the hatchling quality data available is generated from studies using broiler chicks. While chicks and poults are anatomically and physiologically similar, research is needed to define the unique characteristics and standards of PQ.

Breeder hen age is associated with variable egg weight and composition (Applegate et al., 1999; Christensen et al., 2001; O'Dea et al., 2004; Vieira & Moran, 1998). Young breeder hens deposit a smaller proportion of yolk than older breeders (Applegate et al., 1999) resulting in malfunction of yolk assimilation, reduced yolk mobilization, and increased embryonic mortality (Noble et al., 1986). Young breeder hens also produce smaller eggs than peak breeder hens, and consequentially smaller hatchlings (O'Dea et al., 2004). Moreover,

the first 3 weeks of lay (young breeder hens) produce the most morphologically defective hatched poults (Mróz et al., 2013). One example of a morphological defect in poults is navel buttons. Navel buttons observed at hatch are indicative of impaired absorption of the residual yolk sac content and decreased intestinal villi growth (Kawalilak et al., 2010). This is consistent with slower villus elongation and enterocyte migration in hatchlings with navel buttons (Applegate et al., 1999; Schaefer et al., 2006). Therefore, developmental constraints caused by breeder age, particularly with very young breeder hens, may impact the developmental status of hatchlings (Roberts et al., 2011).

Turkey poults are often exposed to long holding periods before placement resulting in delayed access to feed and water. During these periods poults experience BW losses due to dehydration or failure to gain BW due to decreased yolk sac consumption (Noy & Sklan, 1999; Vieira & Moran, 1999). BW loss negatively impacts subsequent growth as evidenced by Pinchasov & Noy (1993) and Bhanja et al. (2009) who found that birds placed within 24 hours post-hatch had greater BW gains compared to birds placed from 32 to 48-hours post-hatch. Decreased yolk sac utilization in feed-deprived chicks leads to depressed gastrointestinal tract development and decreased overall growth (Gonzales et al., 2003). Feed-deprived birds have decreased villus surface area (**SA**), until feed consumption, when they exhibit accelerated intestinal growth (Noy et al., 2001), thereby overcoming initial deficiencies. Further investigation is required to determine if the effects of feed-deprivation in turkey poults are similar to those observed in chicks.

It was hypothesized that young turkey breeder age and delayed placement time of turkey poults can reduce physical hatch measurements including poult weight and poult length, which are individually correlated to depressed performance parameters such as BW

gain and intestinal growth during brooding. An aim of this research is to determine if a PQ evaluation protocol utilizing both qualitative and quantitative measures is accurate in predicting survivability and early growth in turkey poults. To test the predictive success of individual post-hatch poult measurements on early brooding performance, poults from young breeder hens and delayed placement poults were used as constraints on development (and therefore compromised PQ).

MATERIALS AND METHODS

Turkey Poults and Husbandry.

468 Nicholas Super Select hen poults (Aviagen Turkeys, Lewisburg, WV) provided by Butterball, LLC (Goldsboro, NC) were randomly distributed among 36 Petersime battery cages (13 birds per cage; 4 battery cages with 9 cages spread throughout 3 decks) and reared to 14 days of age. Half of the turkey poults were hatched from eggs laid by young breeder hens (1st week of lay, FOL) and the other half were hatched from eggs laid by peak breeder hens (14th week of lay, PEAK). The groups were split in half again into those that would be placed within 6 hours of hatch (IP) and those that would be placed after a 48-hour holding period (DP). During the 48-hour holding period, birds were held without access to feed and water in a climate-controlled room at 32°C (Scott Hall, NCSU Prestage Department of Poultry Science, Raleigh, NC). Each bird was identified by a neck tag and placed in cages. Poults were weighed individually at placement (0 or 2 days of age based on treatment) and at 7 and 13 days of age. Feed consumption was determined by pen. Mortality weights were recorded daily and used to adjust feed conversion. All birds were provided a common starter diet and water *ad libitum* throughout the experiment. All bird handling procedures were approved by the NCSU Institutional Animal Care and Use Committee.

PQ Assessment.

After determining placement BW, all poults were scored using a modified Tona score (Tona et al., 2003). The parameters that were evaluated to determine PQ in this experiment are listed in Table 3.1. Birds were placed and evaluated within 6 hours of hatch for IP treatments and after the 48-hour holding period for DP treatments. Activity, HV, and shank color (**SC**) were scored subjectively using a relative scoring index, while navel strings, navel buttons, SLs (hock joint to the end of the nail of the third toe), and hatchling length (tip of the beak to the end of the end of the third toe) were measured by linear length in centimeters. Relative asymmetry (**RA**) was calculated by (|L-R| / [(L+R)/2]) x 100 where L is left SL and R is right SL (Møller et al., 1999; Yalçin & Siegel, 2003; Yang et al., 1998).

Sample Collection and Histology.

After PQ evaluation, each bird was euthanized and intestinal samples were collected. On days 7 and 14 two poults per pen were randomly selected, euthanized and sampled. Histology samples were collected by removing one inch of tissue from the middle of the jejunum and ileum. The jejunum and ileum were differentiated at Meckel's diverticulum. These tissues were gently flushed with saline, placed into common conical tubes containing 10% formalin according to treatment, then stored at room temperature.

1 cm segments were embedded in paraffin. A microtome was used to produce three to four thin sections (2 microns) for each sample. These sections were mounted on microscope slides and stained with hematoxylin-eosin. Measurements per section per bird were collected for villus height (**H**), villus top width (**TW**), villus bottom width (**BW**), crypt depth (**CD**), and muscularis thickness by an image analyzer (MU1000 Microscope Digital Camera,

AmScope, Orange County, CA), according to the protocol of de Verdal et al. (2010). Villus surface area (**SA**) was calculated as the product of the height multiplied by the width.

Statistical Analysis.

Growth Performance and morphometric measurements were analyzed using the ANOVA procedure with means separated by LS Means in JMP Pro 13 (SAS Institute Inc., Cary NC). The experimental design was a 2 X 2 factorial. Means were considered significant at less than or equal to 0.05. Individual and pen average PQ assessment correlations were analyzed using the MANOVA procedure in JMP Pro 13 (SAS Institute Inc., Cary, NC). Significance was determined by using the critical value based on correlation coefficients.

RESULTS

Growth Performance.

Significant differences in BW were observed between breeder age and placement time (Table 3.2). At 14 days of age, IP-PEAK poults outperformed the rest of the treatments. On hatch day, FOL poults weighed significantly less than PEAK poults. There were no significant differences for BW at day 7 for FOL and PEAK poults, but at day 14 PEAK poults weighed more than FOL poults. DP poults had significantly smaller BWs as compared to IP poults at both 7 and 14-days of age. Placement BW of poults from different breeder ages is negatively correlated with week 2 BW (Table 3.3). However, day 2 and day 7 BW was strongly correlated with day 13 BW. Mortality rates in this battery experiment were high relative to industry standards. Higher mortality rates during week 1 were observed for FOL poults (6.67%) and DP poults (5.88%) relative to PEAK poults (3.94%) and IP poults (4.74%), but not statistically different.

IP-FOL consumed more feed during week 1 relative to DP-FOL poults. Increased consumption of IP poults compared to DP poults during week 1 is expected because these birds consumed feed for 2 additional days. During week 2, DP-FOL poults consumed significantly less feed than IP-FOL poults, but not different than DP-PEAK or IP-PEAK poults. Cumulative feed consumption was significantly greater in FOL-IP poults compared to all other treatments. There were no significant differences for main or interaction effects of feed conversion ratio.

PQ Assessment.

Several significant correlations were observed for the main effects (Table 3.4) and interaction effects (Table 3.5) of breeder age and placement time. FOL poults with longer shank length (**SL**) are associated with increased shank color (**SC**) (r=0.48) and hock vein (**HV**) (r=0.56). PEAK poults with longer SL are associated with increased HV (r>0.29). Moreover, decreased activity of PEAK poults is associated with increased navel strings (r=-0.44), increased placement BW (r=-0.42), decreased SL (r=0.42), and increased RA (r=-0.36). Lighter BWs in FOL and PEAK poults are associated with increased SC (r=-0.49 and -0.40, respectively). Decreased BW in DP poults is associated with increased SC (r=-0.33), while the decreased activity of IP poults is associated with increased navel strings (r=-0.45), increased placement BW (r=-0.30), decreased SL (r=0.44), and increased RA (r=-0.32).

Decreased DP poult activity is associated with increased SC (r=-0.30). Furthermore, there is a significant negative association of SC and activity in DP-FOL poults (r = -0.75). Increased SC is correlated to increased HV in DP, FOL, and PEAK poults (r=0.42, 0.59, and 0.64, respectively). This parallels to the significant interaction correlation between increased SC and HV in DP-FOL poults (r=0.38) and DP-PEAK poults (r=0.47). Lighter BWs in DP,

FOL, and PEAK poults are associated with increased HV (r=-0.34, -0.41, and -0.51, respectively). HV was also correlated negatively to BW for IP-FOL poults (r = -0.48) and DP-PEAK poults (r = -0.43). Heavier BW DP poults are associated with longer SLs (r=0.47, 0.40). Significant interaction positive associations were found for left and right SL and BW in FOL-DP, FOL-IP, and PEAK-DP poults (r>0.30). FOL poults with larger navel buttons have shorter SL (r>-0.30). Moreover, there is a negative correlation of left and right SL and buttons in DP-FOL poults (r = -0.64 and 0.55, respectively). Lastly, there is a positive correlation between HV and strings in IP-FOL poults (r = 0.57).

Histological morphometrics.

In the jejunum on day 0, FOL poults had greater H, TW, muscularis and H to crypt depth ratio than those found in PEAK poults (Table 3.6). Alternatively, on day 2 FOL poults had smaller jejunum TW than PEAK poults (Table 3.7). At day 7 FOL poults have smaller jejunum H, TW, BW, and SA than PEAK poults. Alternatively, at day 14 FOL poults had larger jejunum TW than PEAK poults. In the ileum on day 0, PEAK poults had greater H, CD, muscularis, and SA as compared to FOL poults (Table 3.6). Conversely, on day 2 FOL poults had larger ileal H, CD, muscularis, and SA than PEAK poults (Table 3.8). At day 7, FOL poults had larger H, but smaller BW than PEAK poults. At day 14 FOL poults had larger ileal H and SA than PEAK poults (438 µm, 56,221 μm^2 versus 386 µm, 46,396 μm^2, respectively).

In the jejunum on day 2, DP poults had smaller TW, BW, muscularis and SA than IP poults (Table 3.7). There is a significant interaction effect on day 2, where FOL-IP poults had larger ileal BW and SA than all other treatment groups. Conversely, at day 7 DP poults had larger jejunum H, CD and SA than IP poults. The only effect of placement time at day 14

was larger jejunum CD and muscularis in DP poults compared to IP poults. In the ileum on day 2 DP poults had smaller H, TW, muscularis and SA than IP poults (Table 3.8). But at day 7 DP poults had larger ileal H, CD, muscularis and SA than IP poults (528 µm, 88 µm, 205 µm, 52,177 μm^2 versus 401 µm, 69 µm,173 µm, 37,720 μm^2, respectively). There were no effects due to placement time at day 14.

DISCUSSION

There is controversy concerning the accuracy of day-old BW as a predictive indicator of final BW. Some have found it to be an accurate predictor (Proudfoot & Hulan, 1981; Sklan et al., 2003), while others have not (Tona et al., 2004; Wolanski et al., 2004). Moreover, differences in incubation, management, environment, and disease can greatly influence the weight of a broiler at market age (Hulet et al., 2007). Determining the individual impacts of breeder age and delayed placement on placement BW and early BWs (between 1-2 weeks of age) relative to market age performance is of interest to poultry companies. Further investigation utilizing turkey poults is needed to characterize unique problems in the turkey industry and optimize market age performance, which requires much longer growth periods than the broiler industry. Based on the research presented, day 2 BW and day 7 BW for all treatment groups were positively correlated with day 13 BW while placement BW was not suggesting that placement BW is not a good predictor of early performance in turkey poults.

Breeder Age

As part of the PQ assessment, navel buttons and placement BWs were associated negatively with right and left SL among FOL poults indicating that smaller birds are more likely to have unhealed navels. This conclusion is supported by research in the chicken

industry where Fasenko & O'Dea (2008) observed that chicks with buttons had lower day 7 BWs than chicks with healthy navels. Furthermore, young broiler breeder age is also associated with hatchlings with infected yolk sacs (Salahi et al., 2012).

In this experiment, FOL poults weighed significantly less than PEAK poults. This agreed with research conducted by Proudfoot et al. (1982)and Hulet et al. (2007) which found that chicks from larger eggs (older breeder flock) had significantly greater BW at market age (49-days of age). Stress or subliminal infection (manifested by slightly higher mortality) may be responsible for decreased weight gain or lack of development in FOL poults (Hulet et al., 2007). Alternatively, Joseph & Moran (2005) found that poults hatched from young breeders had lower BW at week 1 than poults from peak breeders. But at 6 weeks of age BWs of poults from young breeders were not statistically different from BWs of poults from peak breeders. Tona et al. (2004) also observed no significant differences in BWs between chicks from young or older breeders at the time of processing (42-days of age).

PEAK poults had larger jejunum TW and BW and ileal H and SA than FOL poults at hatch. By 14 days of age growth rates diverge with more rapid jejunum and ileal development observed in FOL compared to PEAK poults at 7 and 14 days of age indicating that gut development among poults from different breeder flocks is inherently variable. There is inconsistent data reported from previous research regarding intestinal development. Schaefer et al. (2006) observed that FOL poults start with greater H and TW, but the influence of breeder hen age became less noticeable over time (Schaefer et al., 2006). Increased rates of mucosal development in poults from young breeders may be required to provide optimal continued growth (Hulet et al., 2007). Alternatively, El Sabry et al. (2013) found that chicks from older breeder hens had increased SA during brooding. In summary,

early gastrointestinal tract development is initially greater in FOL relative to PEAK poults. However, larger jejunum and ileum mucosal measurements at 7 and 14 days of age indicate increased growth rates in FOL poults compared to PEAK poults.

Delayed Placement

Results of the PQ assessment indicated that HV protrusion and shank discoloration were correlated negatively to placement BW in association with DP for FOL and PEAK poults. IP poults did not exhibit HV protrusion. “Red hocks”, a discoloration of the shanks (in this study is a high SC score) and HV protrusion, are commonly recognized signs of dehydration and compromised PQ (de Jong et al., 2004). Increased HV protrusion is more common in early hatched birds that are subjected to extended holding periods in the incubator (Kingston, 1979), during poult servicing, and during transportation. Furthermore, hatch weight largely depends on hydration status (Deeming, 2005) and a large amount of water lost during incubation results in smaller birds (FOL) that may be susceptible to further dehydration than larger birds (PEAK).

In this experiment there was an effect of placement time, where DP poults were not able to recover BW by 14 days of age. Delayed access to feed and water post-hatch may stunt growth when early cell development occurs (Gonzales et al., 2003; Winick & Noble, 1966). Birds will reallocate nutrients to organs for survival (intestinal development) instead of growth (muscle deposition) (Sell et al., 1991). Furthermore, DP poults consumed significantly more feed than IP poults during the 2-week period (accounting for the days that they were held without feed and water). During this time, intestinal growth may be further limited when energy requirements exceed gluconeogenic capacity impeding growth and causing morbidity or mortality (Donaldson et al., 1992). Interaction effects showed that while

there were no differences in feed consumption between IP poults, IP-FOL poults did not gain as much BW as IP-PEAK poults (Table 3.2). Therefore, PEAK poults may have a higher potential for BW gain than FOL poults.

Jejunum and ileum mucosal development of IP poults was greater at day 2 relative to DP poults. However, feed consumption in DP poults initiate more rapid development and nutrient utilization to overcome deficiencies detected at placement (Careghi et al., 2005). Based on observations in the jejunum and ileum between days 2 and 7, delayed placement may result in stunted mucosal development with greater effects in PEAK poults than FOL poults. Delayed placement and dehydration may contribute to delayed growth and development of nutrient supply organs (intestines, pancreas, liver) and may have carry-over effects on early performance (Corless & Sell, 1999) during the first 2 weeks of life.

In conclusion, placement BW was not correlated with early growth performance of turkey poults. We found no correlation between neurological condition, activity level, and eye conditions with early growth performance. However, we found that navel buttons for FOL poults, and SC and HV for DP poults were negatively associated with growth performance in the first two weeks of life. Young breeder age and delayed placement time in current turkey production schemes uniquely impact embryonic development and early post-hatch growth. Early development and growth of poults from young breeders are inferior to poults from older breeders during the first two weeks of life. Delayed access to feed and water depressed early growth performance but growth rates may be alleviated after feed intake. This study was conducted until two weeks of age which was chosen because it is a benchmark used to determine hatchery prices and welfare quality in brooder barns. However, further investigation is needed to elucidate the effects of young breeder age and delayed

placement at market age in turkeys. In this experiment, day 2 and day 7 BW were successful PQ indicators which, taken together with other PQ indicators and intestinal development demonstrated that young breeder age and delayed placement have unique impacts during the critical brooding period which may carry over to market age performance of turkeys.

Table 3.1. Poult quality assessment parameters and descriptions.[1]

Activity	0 = no activity, completely stagnant 1= alert but no movement 2 = displays pecking behavior and some movement 3 = alert and curious, quick movement to target
String	Measured in cm
Button	Measured in cm
Left & Right Shank Length	Measured in cm
Shank Color	0 = pale pink color 1 = slight red color with scales slightly visible 2 = bright red color and scales prevalent
Hock Vein	0 = no visual evidence of vein 1 = slight visibility, but not raised above the skin 2 = very visible and protruded

[1]Neurological condition, omphalitis, yolk condition, and eyes were also measured, but not included in assessment due to low incidence.

Table 3.2. The effect of breeder age and placement time on poult performance to 2 weeks of age.[1]

Period		Average BW (g)			Feed Intake Per Bird (g)			FCR (g:g)[2]		
		Day 0	Day 7	Day 14	Wk 1	Wk 2	Cum.[3]	Wk 1	Wk 2	Cum.
(Main Effects)										
Breeder Age	**FOL[4]**	52.51^{B}	103.84	220.27^{B}	77.97^{a}	168.5	246.68	1.955	1.355	1.435
	PEAK	61.13^{A}	109.29	246.6^{A}	69.57^{b}	158.74	228.11	1.572	1.678	1.498
Placement Time	**IP**	56.32^{b}	116.64^{A}	248.9^{A}	57.25^{B}	153.34^{b}	211.25^{B}	1.871	1.507	1.466
	DP	57.32^{a}	96.49^{B}	217.97^{B}	90.29^{A}	173.91^{a}	263.53^{A}	1.655	1.526	1.467
(Breeder Age X Placement Time Interactions)										
FOL	**IP**	.	110.37	227.00^{b}	55.67^{C}	148.42^{b}	205.21^{B}	1.956	1.437	1.448
FOL	**DP**	.	97.3	213.55^{b}	100.27^{A}	188.58^{a}	288.14^{A}	1.954	1.273	1.423
PEAK	**IP**	.	122.91	270.82^{a}	58.83^{C}	158.25^{ab}	217.29^{B}	1.355	1.616	1.486
PEAK	**DP**	.	95.67	222.39^{b}	80.32^{B}	159.23^{ab}	238.93^{B}	1.788	1.741	1.509
Source of Variation		---------- *(P-Values)* ----------								
Breeder Age		**<0.01**	0.37	**<0.01**	**0.05**	0.26	0.07	0.18	0.1	0.37
Placement Time		**0.03**	**<0.01**	**<0.01**	**<0.01**	**0.02**	**<0.01**	0.45	0.92	0.98
Breeder Age*Placement Time		.	0.25	**0.03**	**0.01**	**0.03**	**<0.01**	0.44	0.45	0.73
SEM(30)[5]		0.4	4.5	5.6	2.9	6.4	7.5	0.25	0.15	0.05

[A,B] Means within a row with different superscripts are significantly different ($p<0.01$).

[a,b,c] Means within a column with different superscripts are significantly different ($p<0.05$).

[1]Mean values are averages of 9 replicate pens of 13 turkey hen poults per pen.

[2]Values were calculated using BW gain

[3]Cum. = cumulative

[4]FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens, IP = placed immediately within 6 hours of hatch, DP = placed after a 48-hour holding period.

[5]SEM = Standard Error of the Mean with 30 degrees of freedom.

Table 3.3. Average post-hatch BW correlations main and interactions effects of turkey poults.

		Day 0 BW	Day 2 BW	Day 7 BW
			(Main Effects)	
	FOL[1]	-0.084	0.788*	0.791*
	PEAK	-0.579*	0.571*	0.465*
	DP	.	0.541*	0.492*
	IP	.	0.669*	0.560*
Day 13 BW			*(Interaction Effects)*	
	FOL-DP	.	0.693*	0.652*
	FOL-IP	.	-0.413*	0.343*
	PEAK-DP	.	-0.636*	0.584*
	PEAK-IP	.	0.365*	-0.349*

[1]FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens, DP = placed after a 48-hour holding period, IP = placed immediately within 6 hours of hatch.

*Correlation coefficients at 95% confidence interval (35 degrees of freedom) that have critical values greater than 0.325 or less than -0.325.

Table 3.4. Poult quality assessment correlations of breeder age and placement time main effects on turkey poults.[1]

		String	Button	Plc BW	L SL	R SL	SC	HV	RA
Activity	**FOL**[2]	-0.191	-0.004	0.064	0.232	0.249	-0.252	0.000	-0.196
	PEAK	-0.440*	-0.077	-0.416*	0.422*	0.035	0.230	0.280+	-0.364*
	DP	-0.183	-0.250	-0.035	-0.253	-0.135	-0.311+	-0.038	-0.059
	IP	-0.449*	0.149	-0.302+	0.436*	0.066	0.000	-0.147	-0.321+
String	**FOL**		-0.176	-0.070	-0.076	-0.112	-0.018	-0.010	0.247
	PEAK		-0.045	0.255	0.009	-0.127	0.137	-0.088	0.013
	DP		-0.093	0.237	-0.118	-0.219	0.186	-0.131	0.276+
	IP		-0.086	0.129	0.045	0.017	0.000	0.143	0.014
Button	**FOL**			0.027	-0.306+	-0.299+	-0.087	-0.173	-0.027
	PEAK			0.145	0.199	0.174	0.011	0.038	-0.153
	DP			0.022	-0.065	-0.003	0.063	0.100	0.031
	IP			0.035	0.114	-0.120	0.000	-0.107	-0.146
Plc BW	**FOL**				-0.086	-0.074	-0.489*	-0.412*	-0.087
	PEAK				-0.142	0.088	-0.398*	-0.511*	0.247
	DP				0.472*	0.398*	-0.333*	-0.335*	0.112
	IP				-0.097	0.344*	0.000	-0.295+	0.211
L SL	**FOL**					0.931*	0.483*	0.563*	-0.587*
	PEAK					0.371*	0.189	0.293+	-0.528*
	DP					0.917*	-0.037	-0.032	-0.155
	IP					-0.024	0.000	-0.048	-0.529*
R SL	**FOL**						0.518*	0.615*	-0.523*
	PEAK						0.071	0.325*	-0.412*
	DP						-0.019	0.058	-0.392*
	IP						0.000	-0.209	-0.365*
SC	**FOL**							0.593*	-0.281+
	PEAK							0.638*	-0.090
	DP							0.420*	0.067
	IP							0.000	0.000
HV	**FOL**								-0.295+
	PEAK								-0.156
	DP								-0.080
	IP								0.001

[1]Activity (score of 0, 1, 2, or 3), String (measured in cm), Button (measured in cm), L SL = left shank length (measured in cm), R SL = right shank length (measured in cm), SC = Shank Color (score of 0, 1, or 2), HV = Hock vein (score of 0, 1, or 2), RA = relative asymmetry.
[2]FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens, DP = placed after a 48-hour holding period, IP = placed immediately within 6 hours of hatch.
*Correlation coefficients at 95% confidence interval (35 degrees of freedom) that have critical values greater than 0.325 or less than -0.325.
+Correlation coefficients at 90% confidence interval (35 degrees of freedom) that have critical values greater than 0.275 or less than -0.275.

Table 3.5. Poult quality assessment correlations of breeder age and placement time interactions on turkey poults.[1]

		String	Button	Plc BW	L SL	R SL	SC	HV	RA
Activity	**FOL-DP[2]**	0.086	-0.167	0.330*	-0.345*	-0.356*	-0.754*	-0.493*	0.219
	FOL-IP	-0.319+	0.189	0.008	0.327*	0.333*	.	-0.230	-0.189
	PEAK-DP	-0.280+	-0.303+	-0.338*	-0.212	-0.051	0.240	0.318+	-0.210
	PEAK-IP	-0.538*	0.164	-0.503*	0.542*	-0.130	.	.	-0.410*
String	**FOL-DP**		-0.325	0.010	0.228	0.181	0.132	-0.072	0.034
	FOL-IP		-0.218	-0.318+	-0.062	-0.110	.	0.565*	0.263
	PEAK-DP		-0.084	0.323+	-0.196	-0.272	0.302+	-0.139	0.301+
	PEAK-IP		.	0.236	0.080	0.011	.	.	-0.018
Button	**FOL-DP**			-0.355*	-0.637*	-0.548*	0.227	0.358*	0.201
	FOL-IP			0.033	-0.032	-0.045	.	-0.161	-0.246
	PEAK-DP			0.200	0.295+	0.309+	-0.029	-0.004	-0.095
	PEAK-IP			0.131	0.254	-0.149	.	.	-0.257
Plc BW	**FOL-DP**				0.296+	0.358*	-0.444*	-0.181	-0.083
	FOL-IP				0.407*	0.488*	.	-0.483*	-0.608*
	PEAK-DP				0.582*	0.404*	-0.297+	-0.434*	0.264
	PEAK-IP				-0.173	0.248	.	.	0.324+
L SL	**FOL-DP**					0.951*	0.153	0.089	-0.169
	FOL-IP					0.597*	.	-0.446*	-0.591*
	PEAK-DP					0.915*	-0.302+	-0.144	-0.084
	PEAK-IP					-0.057	.	.	-0.508*
R SL	**FOL-DP**						0.218	0.131	-0.229
	FOL-IP						.	-0.262	-0.311+
	PEAK-DP						-0.324	-0.025	-0.388*
	PEAK-IP						.	.	-0.545*
SC	**FOL-DP**							0.379*	-0.071
	FOL-IP							.	.
	PEAK-DP							0.471*	0.277+
	PEAK-IP							.	.
HV	**FOL-DP**								0.053
	FOL-IP								0.224
	PEAK-DP								-0.116
	PEAK-IP								.

[1]Activity (score of 0, 1, 2, or 3), String (cm), Button (cm), L SL = left shank length (cm), R SL = right shank length (cm), SC = Shank Color (score of 0, 1, or 2), HV = Hock vein (score of 0, 1, or 2), RA = relative asymmetry.

[2]FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens, DP = placed after a 48-hour holding period, IP = placed immediately within 6 hours of hatch.

*Correlation coefficients at 95% confidence interval (35 degrees of freedom) that have critical values greater than 0.325 or less than -0.325.

+Correlation coefficients at 90% confidence interval (35 degrees of freedom) that have critical values greater than 0.275 or less than -0.275.

Table 3.6. The main effect of breeder age on jejunum and ileum histology morphometrics at day 0 in turkey poults.[1]

	H[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm^2)
			(Main Effects: Jejunum)			
FOL[3]	335.37^A	48.07^B	49.67^B	47.08	116.42^A	16467
PEAK	274.94^B	56.27^A	60.49^A	42.98	94.18^B	16159
Source of Variation			*(P-Value)*			
Breeder Age	**<0.01**	**<0.01**	**<0.01**	0.06	**<0.01**	0.80
SEM(71)[4]	10.2	1.5	2.5	1.5	4.7	838
			(Main Effects: Ileum)			
FOL	189.61^B	41.78	45.92	43.96^A	99.15^B	8571^B
PEAK	276.95^A	43.84	45.15	59.55^B	137.06^A	12404^A
Source of Variation			*(P-Value)*			
Breeder Age	**<0.01**	0.29	0.73	**<0.01**	**<0.01**	**<0.01**
SEM(71)	12.5	1.4	1.6	2.3	6.0	696

^{A,B} Means within a row with different superscripts are significantly different ($p<0.01$).
[1]Mean values are averages of 8 replicate samples per treatment combination.
[2]H = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.
[3] FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens.
[4]SEM = Standard Error of the Mean with 71 degrees of freedom.

Table 3.7. The effect of breeder age (BA) and placement time (PT) on jejunum histology morphometrics at 2, 7, and 14 days of age in turkey poults.[1]

	H[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm²)
			(Jejunum, Day 2)			
FOL[3]	391.63	49.94^{B}	61.96	53.15	84.65	21773
PEAK	385.40	56.19^{A}	68.44	50.58	88.17	24528
DP	401.87	48.25^{B}	55.36^{B}	51.05	95.61^{A}	20723^{B}
IP	374.93	57.87^{A}	75.04^{A}	52.67	77.22^{B}	25578^{A}
FOL-DP	415.68	45.05	54.09	56.12^{A}	99.53^{A}	20407
FOL-IP	366.89	54.84	69.83	50.18^{AB}	69.77^{B}	23139
PEAK-DP	388.06	51.46	56.63	45.99^{B}	91.68^{A}	21039
PEAK-IP	382.74	60.91	80.26	55.16^{A}	84.66^{AB}	28016
Source of Variation			*(P-Values)*			
BA	0.72	**<0.01**	0.06	0.26	0.43	0.10
PT	0.10	**<0.01**	**<0.01**	0.48	**<0.01**	**<0.01**
BA X PT	0.19	0.94	0.25	**<0.01**	**0.01**	0.20
SEM(29)[4]	14.1	1.8	2.9	2.0	3.8	1415
			(Jejunum, Day 7)			
FOL	681.50^{B}	85.52^{B}	127.88^{b}	86.67	152.65	71304^{B}
PEAK	782.97^{A}	99.70^{A}	145.81^{a}	83.30	144.25	94791^{A}
DP	768.56^{A}	93.00	140.39	90.37^{A}	149.86	88863^{A}
IP	695.90^{B}	92.21	133.29	79.59^{B}	147.04	77232^{B}
FOL-DP	710.09	80.74^{b}	121.74^{B}	91.90	155.44	69792^{B}
FOL-IP	652.91	90.29^{ab}	134.01^{AB}	81.44	149.87	72816^{B}
PEAK-DP	827.04	105.27^{a}	159.05^{A}	88.85	144.27	107934^{A}
PEAK-IP	738.89	94.13^{ab}	132.57^{AB}	77.75	144.22	81649^{B}
Source of Variation			*(P-Values)*			
BA	**<0.01**	**<0.01**	**0.02**	0.39	0.29	**<0.01**
PT	**0.01**	0.88	0.35	**<0.01**	0.72	**<0.01**
BA X PT	0.59	**0.04**	**0.01**	0.93	0.73	**<0.01**
SEM(29)	24.4	4.3	6.4	3.3	6.8	3779
			(Jejunum, Day 14)			
FOL	649.25	137.52^{a}	185.45	65.54	146.85	105169
PEAK	676.73	123.26^{b}	182.18	71.95	142.72	101294
DP	648.78	129.99	191.92	65.25^{b}	133.18^{B}	104913
IP	677.20	130.80	175.71	72.24^{a}	156.39^{A}	101550
FOL-DP	644.64	136.33	198.70	63.18	138.39	109844
FOL-IP	653.85	138.71	172.20	67.89	155.31	100493
PEAK-DP	652.92	123.64	185.14	67.31	127.97	99982
PEAK-IP	700.54	122.88	179.22	76.59	157.47	102606
Source of Variation			*(P-Values)*			
BA	0.33	**0.02**	0.74	0.05	0.57	0.52
PT	0.32	0.89	0.10	**0.04**	**<0.01**	0.57
BA X PT	0.50	0.79	0.30	0.49	0.39	0.32
SEM(29)	24.2	5.1	8.5	2.8	6.2	5071

^{A,B} Means within a row with different superscripts are significantly different ($p<0.01$).
^{a,b} Means within a row with different superscripts are significantly different ($p<0.05$).
[1]Mean values are averages of 8 replicate samples per treatment combination.
[2]H = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.
[3]FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens, DP = placed after a 48-hour holding period, IP = placed immediately within 6 hours of hatch.
[4]SEM = Standard Error of the Mean with 29 degrees of freedom.

Table 3.8. The effect of breeder age (BA) and placement time (PT) on ileum histology morphometrics at 2, 7, and 14 days of age in turkey poults [1]

	H[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm^2)
			(Ileum, Day 2)			
FOL[3]	330.79^{A}	50.79	61.49	62.77^{A}	116.54^{A}	18607^{A}
PEAK	255.34^{B}	48.12	59.04	54.08^{B}	96.73^{B}	13809^{B}
DP	282.34^{b}	47.55^{b}	58.53	59.49	114.36^{a}	15013^{B}
IP	303.78^{a}	51.35^{a}	62.00	57.36	98.91^{b}	17403^{A}
FOL-DP	310.19	47.62	57.31^{b}	62.73	126.30	16251^{B}
FOL-IP	351.39	53.95	65.68^{a}	62.81	106.78	20964^{A}
PEAK-DP	254.50	47.49	59.74^{ab}	56.25	102.43	13776^{B}
PEAK-IP	256.18	48.74	58.33^{b}	51.91	91.03	13842^{B}
Source of Variation			*(P-Values)*			
BA	**<0.01**	0.10	0.32	**<0.01**	**<0.01**	**<0.01**
PT	**0.03**	**0.02**	0.16	0.41	**0.02**	**<0.01**
BA X PT	0.05	0.11	**0.05**	0.39	0.53	**<0.01**
SEM(29)[4]	8.5	1.4	2.1	2.2	5.5	642
			(Ileum, Day 7)			
FOL	482.53^{A}	85.20	103.46^{b}	79.94	190.18	45522
PEAK	446.82^{B}	83.62	115.21^{a}	77.56	187.77	44375
DP	528.46^{A}	85.71	111.39	88.34^{A}	205.04^{A}	52177^{A}
IP	400.88^{B}	83.11	107.28	69.16^{B}	172.91^{B}	37720^{B}
FOL-DP	539.39	87.14	102.19	84.05^{AB}	182.99^{BC}	50891
FOL-IP	425.66	83.26	104.73	75.83^{B}	197.38^{AB}	40153
PEAK-DP	517.53	84.29	120.58	92.63^{A}	227.09^{A}	53462
PEAK-IP	376.11	82.96	109.83	62.49^{C}	148.45^{C}	35287
Source of Variation			*(P-Values)*			
BA	**0.01**	0.64	**0.04**	0.43	0.81	0.60
PT	**<0.01**	0.44	0.47	**<0.01**	**<0.01**	**<0.01**
BA X PT	0.32	0.71	0.25	**<0.01**	**<0.01**	0.09
SEM(29)	9.8	2.3	4.0	2.1	7.0	1546
			(Ileum, Day 14)			
FOL	438.36^{A}	100.19	155.20	63.50	151.4972	56221^{A}
PEAK	386.49^{B}	94.91	146.44	64.76	167.4031	46396^{B}
DP	405.63	95.58	148.72	64.43	164.605	49708
IP	419.21	99.53	152.92	63.83	154.2954	52910
FOL-DP	415.08	98.41	151.21	59.37	164.6838	52177
FOL-IP	461.63	101.98	159.20	67.63	138.3105	60266
PEAK-DP	396.18	92.75	146.23	69.49	164.5261	47239
PEAK-IP	376.79	97.07	146.65	60.03	170.2802	45554
Source of Variation			*(P-Values)*			
BA	**<0.01**	0.22	0.17	0.75	0.06	**<0.01**
PT	0.47	0.36	0.50	0.88	0.22	0.26
BA X PT	0.08	0.93	0.55	**0.03**	0.06	0.09
SEM(29)	13.2	3.0	4.4	2.8	5.9	2016

^{A,B} Means within a row with different superscripts are significantly different ($p<0.01$).
^{a,b} Means within a row with different superscripts are significantly different ($p<0.05$).
[1]Mean values are averages of 8 replicate samples per treatment combination.
[2]H = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.
[3]FOL = poults hatched from first of lay breeder hens, PEAK = poults hatched from peak production breeder hens, DP = placed after a 48-hour holding period, IP = placed immediately within 6 hours of hatch.
[4]SEM = Standard Error of the Mean with 29 degrees of freedom.

REFERENCES

Applegate, T. J., Dibner, J. J., Kitchell, M. L., Uni, Z., & Lilburn, M. S. (1999). Effect of turkey (Meleagridis gallopavo) breeder hen age and egg size on poult development. 2. Intestinal villus growth, enterocyte migration and proliferation of the turkey poult. *Comparative Biochemistry and Physiology - B Biochemistry and Molecular Biology*, *124*(4), 381–389.

Bhanja, S. K., Devi, C. A., Panda, A. K., & Sunder, G. S. (2009). Effect of post hatch feed deprivation on yolk-sac utilization and performance of young broiler chickens. *Asian-Australasian Journal of Animal Sciences*, *22*(8), 1174–1179.

Careghi, C., Tona, K., Onagbesan, O., Buyse, J., Decuypere, E., & Bruggeman, V. (2005). The effects of the spread of hatch and interaction with delayed feed access after hatch on broiler performance until seven days of age. *Poultry Science*, *84*, 1314–1320.

Christensen, V. L., Wineland, M. J., Fasenko, G. M., & Donaldson, W. E. (2001). Egg storage effects on plasma glucose and supply and demand tissue glycogen concentrations of broiler embryos. *Poultry Science*, *80*(12), 1729–1735.

Corless, A. B., & Sell, J. L. (1999). The effects of delayed access to feed and water on the physical and functional development of the digestive system of young turkeys. *Poultry Science*, *78*(8), 1158–1169.

Dantzer, R., & Mormède, P. (1983). Stress in farm animals: a need for reevaluation. *Journal of Animal Science*, *57*(1), 6–18.

de Jong, I. C., Fillerup, M., Riedstra, B. J., Boerjan, M., & Hopster, H. (2004). The relationship between characteristics, broiler welfare and growth. *Proceedings of the 38th International Congress of the ISAE, Helsinki, Finland*.

de Verdal, H., Mignon-Grasteau, S., Jeulin, C., le Bihan-Duval, E., Leconte, M., Mallet, S., … Narcy, A. (2010). Digestive tract measurements and histological adaptation in broiler lines divergently selected for digestive efficiency. *Poultry Science*, *89*(9), 1955–1961.

Decuypere, E., & Bruggeman, V. (2007). The endocrine interface of environmental and egg factors affecting chick quality. *Poultry Science*, *86*, 1037–1042.

Decuypere, E., Tona, K., Bamelis, F., Careghi, C., Kemps, B., Ketelaere, B. De, … Bruggeman, V. (2002). Broiler breeders and egg factors interacting with incubation conditions for optimal hatchability and chick quality. *Archive Für Geflügelkunde*, *66*, 56–57.

Deeming, D. C. (2000). What is chick quality. *World Poultry*, *11*(2), 34–35.

Deeming, D. C. (2005). Yolk sac, body dimensions and hatchling quality of ducklings, chicks and poults. *British Poultry Science*, *46*(5), 560–564.

Donaldson, W. E., Brewer, C. E., Ferket, P. R., & Christensen, V. L. (1992). Posthatch carbohydrate feeding and subsequent performance of turkey poults. *Poultry Science*, *71*, 128–132.

El Sabry, I. M., Yalçın, S., & Turgay-İzzetoğlu, G. (2013). Interaction between breeder age and hatching time affects intestine development and broiler performance. *Livestock Science*, *157*, 612–617.

Fasenko, G. M., & O'Dea, E. E. (2008). Evaluating broiler growth and mortality in chicks with minor navel conditions at hatching. *Poultry Science*, *87*, 594–597.

Gonzales, E., Kondo, N., Saldanha, E. S. P. B., Loddy, M. M., Careghi, C., & Decuypere, E. (2003). Performance and physiological parameters of broiler chickens subjected to fasting on the neonatal period. *Poultry Science*, *82*(8), 1250–1256.

Hulet, R., Gladys, G., Hill, D., & Meijerhof, R. (2007). Influence of Egg Shell Embryonic Incubation Temperature and Broiler Breeder Flock Age on Posthatch Growth Performance and Carcass Characteristics. *Poultry Science*, *86*, 408–412.

Joseph, N. S., & Moran, E. T. (2005). Effect of flock age and postemergent holding in the hatcher on broiler live performance and further-processing yield. *Journal of Applied Poultry Research*, *14*(3), 512–520.

Kawalilak, L. T., Ulmer Franco, A. M., & Fasenko, G. M. (2010). Impaired intestinal villi growth in broiler chicks with unhealed navels. *Poultry Science*, *89*(1), 82–87.

Kingston, D. J. (1979). Some hatchery factors involved in early chick mortality. *Australian Veterinary Journal*, *55*(9), 418–421.

Lilburn, M. S. (1998). Practical aspects of early nutrition for poultry. *The Journal of Applied Poultry Research*, *7*(4), 420–424.

Møller, A. P., Sanotra, G. S., & Vestergaard, K. S. (1999). Developmental instability and light regime in chickens (Gallus gallus). *Applied Animal Behaviour Science*, *62*(1), 57–71.

Mróz, E., Stępińska, M., Orłowska, A., & Michalak, K. (2013). Frequency of occurrence of physical defects in turkey poults. *Annals of Animal Science*, *13*(3), 543–552.

Noble, R. . C., Lonsdale, F., Connor, K., & Brown, D. (1986). Changes in the lipid metabolism of the chick embryo with parental age. *Poultry Science*, *65*, 409–416.

Noy, Y., Geyra, A., & Sklan, D. (2001). The effect of early feeding on growth and small intestinal development in the posthatch poult. *Poultry Science*, *80*(7), 912–919.

Noy, Y., & Sklan, D. (1999). Energy utilization in newly hatched chicks. *Poultry Science*, *78*(12), 1750–1756.

O'Dea, E. E., Fasenko, G. M., Feddes, J. J. R., Robinson, F. E., Segura, J. C., Ouellette, C. A., & Middelkoop, J. H. Van. (2004). Investigating the eggshell conductance and embryonic metabolism of modern and unselected domestic avian genetic strains at two flock ages. *Poultry Science*, *83*, 2059–2070.

Pinchasov, Y., & Noy, Y. (1993). Comparison of post-hatch holding time and subsequent early performance of broiler chicks and turkey poults. *British Poultry Science*, *34*(1), 111–120.

Proudfoot, F. G., & Hulan, H. W. (1981). The influence of hatching egg size on the subsequent performance of broiler chickens. *Poultry Science*, *60*(10), 2167–2170.

Proudfoot, F. G., Hulan, H. W., & Mcrae, K. B. (1982). Effect of hatching egg aize from semi-dwarf and normal maternal meat parent genotypes on the performance of broiler chickens. *Poultry Science*, *61*, 655–660.

Roberts, J. R., Souillard, R., & Bertin, J. (2011). Improving the safety and quality of eggs and egg products: egg chemistry, production and consumption. In *Food Science, Technology and Nutrition* (pp. 376–393). Woodhead Publishing.

Salahi, A., Khabisi, M. M., Pakdel, A., & Baghbanzadeh, A. (2012). Effects of cold stress during transportation on hatchability and chick quality of broiler breeder eggs. *Turkish Journal of Veterinary and Animal Sciences*, *36*(2), 159–167.

Schaefer, C. M., Corsiglia, C. M., Mireles, A., & Koutsos, E. A. (2006). Turkey breeder hen age affects growth and systemic and intestinal inflammatory responses in female poults examined at different ages posthatch. *Poultry Science*, *85*, 1755–1763.

Sell, J. L., Angel, C. R., Piquer, F. J., Mallarino, E. G., & Al-Batshan, H. A. (1991). Developmental patterns of selected characteristics of the gastrointestinal tract of young turkeys. *Poultry Science*, *70*(5), 1200–1205.

Sklan, D., Heifetz, S., & Halevy, O. (2003). Heavier chicks at hatch improves marketing body weight by enhancing skeletal muscle growth. *Poultry Science*, *82*, 1778–1786.

Tona, K., Bamelis, F., De Ketelaere, B., Bruggeman, V., Moraes, V., Buyse, J., … Decuypere, E. (2003). Effects of egg storage time on spread of hatch, chick quality, and chick juvenile growth. *Poultry Science*, *82*(5), 736–741.

Tona, K., Onagbesan, O., De Ketelaere, B., Decuypere, E., & Bruggeman, V. (2004). Effects of age of broiler breeders and egg storage on egg quality, hatchability, chick quality, chick weight, and chick posthatch growth to forty-two days. *Journal of Applied Poultry Research*, *13*(1), 10–18.

Vieira, S. L., & Moran, E. T. (1998). Eggs and chicks from broiler breeders of extremely different age. *Journal of Applied Poultry Research*, *7*(4), 372–376.

Vieira, S. L., & Moran, E. T. (1999). Effects of egg of origin and chick post-hatch nutrition on broiler live performance and meat yields. *World's Poultry Science Journal*, *55*, 125–142.

Willemsen, H., Debonne, M., Swennen, Q., Everaert, N., Careghi, C., Han, H., … Decuypere, E. (2010). Delay in feed access and spread of hatch: importance of early nutrition. *World's Poultry Science Journal*, *66*(02), 177–188.

Willemsen, H., Everaert, N., Witters, A., De Smit, L., Debonne, M., Verschuere, F., … Bruggeman, V. (2008). Critical assessment of chick quality measurements as an indicator of posthatch performance. *Poultry Science*, *87*(11), 2358–2366.

Winick, M., & Noble, A. (1966). Cellular response in rats during malnutrition at various ages. *The Journal of Nutrition*, *89*(3), 300–306.

Wolanski, N. J., Luiten, E. J., Meijerhof, R., & Vereijken, A. L. J. (2004). Yolk utilisation and chick length as parameters for embryo development. *Avian and Poultry Biology Reviews*, *15*(3–4), 233–234.

Yalçin, S., & Siegel, P. B. (2003). Exposure to cold or heat during incubation on developmental stability of broiler embryos. *Poultry Science*, *82*(9), 1388–1392.

Yang, A., Dunnington, E. A., & Siegel, P. B. (1998). Developmental stability in different genetic stocks of white rock chickens. *Journal of Heredity*, *89*(3), 260–264.

CHAPTER 4.

The Effect of Stage of Lay on Poult Quality and the Use of Poult Quality as an indicator of Post-Hatch Performance of Market Turkey Hens

S. Black*, M. Herchler, V. Schramm, R. Crivellari, R. Malheiros, P. Ferket

Section: Management and Production

* Corresponding author: sablack@ncsu.edu

ABSTRACT

Post-hatch survival and growth performance of market turkeys may be influenced by maternal breeder age, and certain poult quality (PQ) measurements may be a predictive tool for these economically-important traits. A split-plot block experiment was designed with 12 dietary treatments as the whole plot factor and two poult populations originating from young breeder hens (first week of lay = FOL) or old breeder hens (7^{th} week of lay = PEAK). Poults were placed in 48 pine shaving floor pens and reared to 14 weeks of age. A PQ assessment was conducted on two FOL poults per pen. BW was recorded on day 0, and weeks 1, 3, 5, 8, and 10. Jejunum and ileum histology samples were collected at weeks 1 and 5. Breast muscle yield (**BMY**) was determined at week 14. Data were analyzed by ANOVA and MANOVA using JMP Pro 13. FOL poults had lower body weight gains (**BWG**) than PEAK poults until 10 weeks of age (3.71 kg *vs.* 3.95 kg, respectively, $p<0.01$). FOL poults had increased mortality rates during the starter period compared to PEAK poults (3.8% *vs.* 0.6% respectively, $p<0.01$). BMY were smaller in FOL hens than PEAK hens (24.25% *vs.* PEAK = 24.90%, $p<0.01$). Hatchling length (**HL**) was more highly correlated to performance than hatchling weight (**HW**). A subjective scoring rubric provided information about the condition of the poult at the time of assessment, but was not a good predictor of growth performance for commercial turkeys. Jejunum and Ileum mucosa histological morphometrics were similar among poult sources at week 1, but FOL poults had greater villi surface area at week 5, which may be a compensatory effort to overcome earlier growth handicaps. In conclusion, growth performance of market turkey hens from young breeders was inferior to hens from older breeders. Moreover, hatchling and shank length (**SL**) were observed to be most predictive of subsequent growth performance in turkey hens.

Key words: breeder age, poult quality, growth performance, breast muscle yield

INTRODUCTION

Reaching genetic potential for growth performance at market age is important for maximizing profit and understanding the factors that influence survivability and performance to help producers achieve economic and performance objectives. Poult quality (PQ) is an indicator of survivability and future growth performance characteristics of young turkey poults. Compromised PQ may adversely affect subsequent responses to environmental and management constraints, resulting in depressed growth and increased mortality. It is unknown if the adverse consequences of compromised PQ can be remediated by dietary treatments after placement on the farm.

Variable PQ is associated with breeder hen age (stage of lay) (Boerjan, 2002; Tona et al., 2001, 2004). Breeder age alters egg weight and composition (Applegate et al., 1999; Christensen et al., 2001; O'Dea et al., 2004; Vieira & Moran, 1998), which dictates the BW and metabolic status of poults at hatch (Ulmer-Franco et al., 2010). Body weight at market age is negatively influenced by young breeder flocks (Christensen et al., 2001; Schaefer et al., 2006; Ulmer-Franco et al., 2010) that produce smaller eggs with smaller proportion of yolk at the beginning of egg production than breeder hens at peak production (Applegate et al., 1999). These deficiencies at the breeder level, particularly with very young hens, will negatively impact hatchability, PQ (Roberts et al., 2011) and hatchling size (O'Dea et al., 2004).

PQ is typically determined by quickly assessing visual quality, on a small sampling of poults at hatcheries and on farms, using a subjective scoring rubric commonly known as the Tona score protocol (Willemsen et al., 2008). The Tona score is a qualitative scoring system that assesses total score index of 100 based on a wide variety of visual indicators, such as

activity, appearance, retracted yolk, eye condition, leg condition, navel deformities, remaining egg membrane, and remaining yolk (Tona et al., 2003). However, a strong positive correlation between the Tona score values and performance has not been demonstrated (Mukhtar et al., 2013). Therefore, many researchers have moved towards a more quantitative approach to assess PQ. These indicators include hatchling weight (**HW**) (Decuypere et al., 2002; Deeming, 2000), hatchling length (**HL**) (Hill, 2001) and shank length (**SL**) (Wolanski et al., 2004). HW is the most widely used indicator of chick and poult quality (Decuypere et al., 2002), but it may not a good predictor of subsequent growth due to differences in abdominal residual yolk and the efficiency rate at which the hatchling utilizes this residual yolk during post-hatch development (Mukhtar et al., 2013). Young breeder age negatively influences yolk deposition, hence a negative association with HW. More recent research has shown that the HL is a better method of evaluating PQ (Joseph et al., 2006). The length of a hatchling is related more to the size of the egg than to its body weight (Geidam et al., 2007), and egg size is related to the age of the breeder (Nangsuay et al., 2011; Ulmer-Franco et al., 2010). Therefore, it appears that there is a positive association between HL and breeder age (Hill, 2001). Additionally, SL is strongly correlated to HL, and may be used as an alternative indicator of HL (Meijerhof, 2005).

The aim of this experiment was to determine if the Tona score, a visually subjective quality evaluation protocol, is feasible for assessing the potential growth performance in market turkey hens. This experiment was designed to test the hypothesis that individual components of the Tona scoring system are constraints of influence on subsequent production traits and correlated to compromised PQ specifically associated with young breeder hen age. Furthermore, we hypothesize that compromised PQ due to young breeder

age has lasting adverse effects on progeny growth performance, that cannot be overcome by dietary intervention. To test the effectiveness of the Tona score and PQ indicators associated with breeder hen age, correlations to overall production performance parameters, such as BW and breast muscle yield (**BMY**), were examined.

MATERIALS AND METHODS

Turkey Poults and Husbandry.

One thousand-seven hundred-fifty eight Nicholas Super Select turkey hen poults (Aviagen Turkeys, Lewisburg, WV) provided by Butterball, LLC (Goldsboro, NC) were placed in 48 floor pens containing pine shavings (Turkey Educational Unit, North Carolina State University (NCSU) Prestage Department of Poultry Science, Raleigh, NC). Within each pen, two populations of poults were placed as a sub-plot factor: 25 poults from a population of eggs laid from breeder hens in their 7th week of production (**PEAK**) and 11 poults from a population of eggs laid from breeder hens in their first week of production (**FOL**). There were two blocks in the barn, each containing 24 pens, with similar environmental exposures. Each poult was weighed and tagged according to population, then placed into pens assigned to one of 12 dietary treatments (whole-plot factor). Dietary treatments were fed to whole pens and assigned randomly among the 48 pens, resulting in four replications per dietary treatment. Light was provided 24 hours per day for the first three days, followed by 14 hours of light / 10 hours of dark per day for the remainder of the experiment. Group BWs were recorded by pen at placement and weeks 1, 3, and 5. Individual BWs were recorded at weeks 8, 10, 12, and 14. Mortality (and weight) were recorded daily. All bird handling procedures were approved by the NCSU Institutional Animal Care and Use Committee.

Dietary Treatments.

The 12 dietary treatments consisted as a 4 X 3 factorial arrangement of 4 diet formulations and 3 dietary Azomite® inclusion levels. The corn-soybean meal diet formulations consisted of; 1) positive control diet (**PC**) formulated to meet 90% of breeder standards; 2) a negative control diet (**NC**) formulated with reduced available phosphorus, calcium, and metabolizable energy (-0.145% AP, -0.1245% Ca, -45 kcal/lb); 3) a NC + standard enzyme diet (**E1**) with 250 FTU/kg phytase and 1500 EPU/kg xylanase; and 4) a NC + super-dose enzyme diet (**E2**) with 1500 FTU/kg phytase and 1500 EPU/kg xylanase (Optiphos® and Hostazym® P, Huvepharma Inc., Sofia, Bulgaria). The three dietary inclusion levels of Azomite® (AZOMITE Mineral Products, Inc., Nephi, UT), a volcanic mineral source, included 0 g/kg, 2.5 g/kg or 5 g/kg of feed. All feed was manufactured at the NCSU Feed Mill Education Unit (Raleigh, NC) and was formulated to meet or exceed the Aviagen Nicholas turkey nutrient specifications, throughout the six phases of production (Table 3.1). Birds were fed starter 1 crumble diet until three weeks of age, a small pellet starter 2 diet from 3 to 5 weeks of age, and pelleted diets for the remainder of the experiment: grower 1 from 5 to 8 weeks, grower 2 diet from 8 to 10 weeks, finisher 1 from 10 to 12 weeks and finisher 2 from 12-14 weeks. All diets in all phases were manufactured according to individual treatment formulations. Diets were blended in a 2-ton double shaft mixer (Model: TRBD126-0604, Hayes & Stolz, Fort Worth, TX) and the addition of enzymes and/or mineral supplements was verified by the feed mill staff and two graduate students. The diets were then pelleted below 180° F in a 10-ton double pass conditioner and pellet mill (Model: 60-130, Bliss, Ponca City, OK). All the feed was bagged according to treatment and

transported to the NCSU Turkey Education Unit. Feed and water were provided *ad libitum*. Feed consumption per pen was determined at weeks 3, 5, 8, and 10.

PQ Assessment.

On placement day, PQ assessment was performed on two randomly-selected birds per pen from the FOL population. These poults were scored as described by Tona et al. (2003) and HL and SL were also determined for each bird. To determine HL, the poult was laid on its ventral side next to a ruler and the measurement was recorded from the tip of the beak to the end of the end of the third toe. To measure SL, each leg was extended and a caliper was used to measure from the hock joint to the beginning of the footpad. Relative asymmetry (**RA**) was calculated by (|L-R| / [(L+R)/2]) x 100 where L is left SL and R is right SL (Møller et al., 1999; Yalçin & Siegel, 2003; Yang et al., 1998). Individual PQ indicators and performance correlations were calculated on an individual bird basis and pen PQ assessments and performance correlations were calculated on a pen average basis.

Sample Collection.

Turkeys were sampled on weeks 1, 5, and 14. At all sample time points, two PEAK poults and two FOL poults were randomly selected for sampling and were euthanized by cervical dislocation (weeks 1 and 5) or by a two-step method of electrocution (week 14) (Leary et al., 2013). At weeks 1 and 5, histology samples were collected by removing one inch of tissue from the middle of the jejunum and ileum. The jejunum and ileum were differentiated at Meckel's diverticulum. These tissues were gently flushed with saline, placed into common conical tubes containing 10% formalin according to treatment, then stored in labeled tubes at room temperature. At week 14, the entire breast muscle, *pectoralis major*, was removed from the carcass and weighed to calculate relative BMY.

Histology.

Intestinal samples were cut into 1 cm segments and embedded in paraffin. A microtome was used to produce three to four 2-micron thin sections of each sample. These sections were mounted on microscope slides and stained with hematoxylin-eosin. Measurements per section per bird were collected for villus height, villus top width, villus bottom width, crypt depth (**CD**), and muscularis thickness by an image analyzer (MU1000 Microscope Digital Camera, AmScope, Orange County, CA), according to the protocol of de Verdal et al. (2010). Subsequently, villus surface area (**SA**) was calculated as the product of the height multiplied by the width.

Statistical Analysis.

Growth Performance, morphometric measurements, and BMY were analyzed using the REML procedure with means separated by LS Means in JMP Pro 13 (SAS Institute Inc., Cary NC). The experimental design was a complete block split-plot design with a 4 X 3 factorial arrangement. The experimental unit was the split-plot factor (poult population). Means were considered significant at less than or equal to 0.05. PQ assessment correlations were analyzed using the MANOVA procedure in JMP Pro 13 (SAS Institute Inc., Cary, NC). Significance was determined by using the critical value based on correlation coefficient.

RESULTS

Growth Performance

There were no interactions due to either Azomite® and breeder age or diet and breeder age on body weight gain (**BWG**) of turkey hen poults (Table 4.2). Significant differences were observed in breeder age populations, where FOL poults weighed significantly less than PEAK poults. Additionally, there was a significant diet effect where

E2, the super dosing phytase enzyme combination, consistently had increased BWG compared to the positive and negative control diets from weeks 3 to 5 and weeks 5 to 8, and consequently cumulative BWGs to 5 weeks and 8 weeks of age. E2 had significantly higher BWG over E1 from weeks 5 to 8 and consequently cumulative BWG to 8 weeks of age. There was no interaction between diet and breeder age, but there was a consistent nominal trend due to a percentage increase in cumulative BWG of PEAK poults as compared to FOL poults fed any diet ($p<0.05$) to weeks 3, 5, 8, and 10 (3.8%, 9.9%, 24.4% and 22.3%, respectively) and period BWG from 3 to 5 and 5 to 8 weeks (14.5%, 14.5%, respectively). This same trend was observed between Azomite® and breeder age, with increased cumulative percentage BWG of PEAK poults than FOL poults fed any level of Azomite® to weeks 3, 5, 8 and 10 (3.8%, 9.7%, 24.4%, and 22.0%, respectively) and period BWG from 3 to 5 and 5 to 8 weeks (14.5%, 14.5%, respectively). Observable differences in BWG decreased from 5 to 10 weeks of age, with no observable differences in any treatments from 8 to 10 weeks. There were no Azomite® or diet main effects on mortality throughout the course of the experiment (Table 4.3). However, during the starter period, there were significantly more mortalities in FOL poults than PEAK poults (3.83% versus 0.59%). There were no main effects of breeder age during the grower period, but the significance of the starter period resulted in similar significance for cumulative mortality.

Relative BMY at 14 weeks of age, expressed as a percent of live BW, was significantly increased by E1 or E2 treatments in comparison to the NC treatment by 1.27% and 0.96%, respectively (Table 4.4), with the PC treatment having an intermediate response. Additionally, PEAK hens had 0.65% greater BMY then FOL hens. There were no main effects of Azomite® on relative BMY. BMY is a common physiological indicator of growth

and had variable effects in this experiment, indicating that developmental factors may uniquely affect development.

PQ Assessment

Total Tona score of individual FOL poults was correlated significantly to activity, retracted yolk, eyes, legs, and navel (r = 0.24, 0.44, 0.29, 0.74, and 0.32, respectively) (Table 4.5). Tona score was not correlated to appearance, remaining membrane or remaining yolk. At the 90% confidence level, remaining yolk had a significant and negative correlation to appearance (r = -0.15), and legs were correlated significantly to retracted yolk (r = 0.18). Tona score was not correlated to post-hatch performance (Table 4.6). Retained yolk was correlated significantly to week 1 BW, indicating heavier birds had higher yolk scores. Alternatively, eyes were correlated negatively and significantly to all BWs after 3 weeks of age (Table 3.8; r < -0.174). Remaining yolk was correlated positively and significantly to both week 8 and week 10 BWs. HL was positively and significantly correlated to BWs at all time points from week 1 to 10. Both left and right SL were correlated significantly to week 1 and week 3 BWs. However, only the left SL was correlated significantly to 5 and 8-week BWs. RA was correlated negatively to BWs at only week 5, 8 and 10, indicating that poults with highly asymmetric shanks had lower BWs. All BWs were correlated positively and significantly to each other, with the strength of the correlation increasing as the poults grow older. Relative BMY was correlated positively to all BWs at weeks 3, 5, and 10 (Table, 4.7; r = 0.33, 0.27, 0.38). Hens with lower relative BMY had a significantly lower mortality rate during the starter period and lower cumulative mortality rate than hens with higher relative BMY (r = -0.25, -0.24, respectively). HW was correlated positively and significantly to mortality during the grower phase (r = 0.28). Smaller birds at week 1 had lower mortality

than heavier birds during the grower period (r = -0.33), resulting in the same effect for cumulative mortality (r = -0.29).

Histological Morphometrics

No main effects of breeder hen age were observed for jejunum morphometrics at week 1 (Table 4.8). However, there was an interaction between Azomite® and breeder age observed in the jejunum at week 5, where CON-PEAK hens (0 g/kg Azomite®) had increased villus top width as compared to 0-FOL hens, as opposed to 2.5-PEAK hens (2.5 g/kg Azomite®) who had decreased villus top width as compared to 2.5-FOL (Table 4.9). Hens fed 2.5 g/kg Azomite® had increased villus SA, villus bottom width, and villus height than control hens (0 g/kg Azomite®). FOL hens had greater jejunum villus SA, and villus bottom width than PEAK hens. PC hens had increased villus area and villus top width as compared to NC hens.

There were several ileum mucosa interactions effects observed at week 1 between Azomite® and breeder age, where 0-FOL poults (0 g/kg Azomite®) had significantly larger villus SAs and villus bottom width than 0-PEAK poults (Table 4.10). Similarly, interaction effects of diet and breeder age were observed, where NC-FOL poults had significantly larger villus SAs and villus bottom width than NC-PEAK poults. Poults fed 2.5 g/kg Azomite® had significantly greater villus top width than control poults (0 g/kg Azomite®). Lastly, PEAK poults had significantly thicker muscularis layer than FOL poults. There were Azomite® and breeder age interaction effects at week 5 in the ileum, where 0-PEAK had significantly greater CDs than 0-FOL hens (Table 4.11). Another interaction effect of diet and breeder age was observed, where NC-FOL had greater CDs than NC-PEAK, but PC-FOL had significantly smaller CDs than PC-FOL. Villus height in PEAK hens was significantly lower

compared to FOL hens. Also, the villus top width of PEAK hens was significantly less than that of FOL hens. There were no main effects of Azomite® or diet on 5 week ileal mucosa morphometrics.

DISCUSSION

Growth Performance

The FOL poults had a significantly lower hatchling BW and cumulative 10 week BWG than PEAK poults. In agreement, O'Dea et al. (2004) reported young breeder hens produced eggs and chicks with weights than older breeder hens. Likewise, Ulmer-Franco et al. (2010) and Christensen et al. (2001) demonstrated that PEAK breeder hens produce poults with heavier BWs. In contrast, Tona et al. (2004) observed no difference in BWs between chicks from young or older chicken dams at the time of processing. We observed no differences in BWG between 8 and 10 weeks among the FOL and PEAK hens, which resulted normalized final BWs at processing. Schaefer et al. (2006) suggests that when there are no differences in BWs between different aged breeder flocks, it might indicate that FOL poults were eventually able to compensate for initial growth deficiency. Because there were no significant breeder age X diet interaction effects observed throughout the study, the effects of breeder age and diet are independent. Evidently, the inferior growth performance effects of FOL poults could not be overcome by either dietary enzyme or Azomite® supplementation. Regardless of the source of poults, the dietary enzyme supplementation treatments increased BWG in comparison to the PC or NC treatments, and the PC diet birds consistently gained more weight than the NC birds, confirming the birds responded to increased dietary nutrient intake. A similar effect was observed in numerous enzyme-based feeding trials (Francesch et al., 2012; Lü et al., 2009; Tatara et al., 2015).

The progeny from the FOL breeders had significantly higher 0-10 week mortality rate than the progeny from the peak breeders (3.83% versus 0.59%,, $P<0.001$). In agreement, Vieira & Moran (1999) reported higher mortality rates during the first week post-hatch of chicks from 27-week old broiler breeders than chicks from 62-week old breeders. Noble et al. (1986) suggested that early FOL mortality is caused by reduced yolk lipid utilization, while Latour et al. (1996) concluded that it is a result of elevated cholesterol and depressed glucose levels. Regardless of the cause of this early mortality, if FOL poults survived beyond the starter period, they were not able to overcome initial growth deficiencies.

Early growth deficiencies of progeny from FOL breeders may adversely affect BMY at market age. We observed the poults from FOL hens had significantly less BMY as a percentage of BW at 10 week market age than poults from PEAK hens. In agreement, other reseearchers observed pectoral muscle yield of broilers from young breeders was significantly less than broilers from peak breeders which had larger hatching weights (Sklan et al., 2003; Vieira & Moran, 1999). Christensen et al. (2001) suggested that perinatal turkeys from young breeder hens develop at a slower rate and they have lower hatch weights with inferior survival. Moreover, reduced muscle development and early growth of progeny from FOL hens may be a result of a priority on post-hatch intestinal development at the expense of muscle satellite cell proliferation (Vieira & Moran, 1999). In contrast, Joseph & Moran (2005) found no statistical differences in relative BMY between chickens hatched from young or peak breeder hens, but this may be because the birds were processed at the physiological age beyond the maximum rate of breast muscle development and there was an opportunity for delayed compensatory growth. As observed in our experiment, the turkey hens were marketed at an age (10 weeks) that was too early for the FOL progeny to

compensate for early deficiencies in muscle development to achieve their genetic potential for breast meat yield and body weight as observed in the PEAK progeny

PQ Assessment

Hatchling weight was not correlated to subsequent BWs as observed by other researchers (Decuypere et al., 2002; Willemsen et al., 2008; Wolanski et al., 2006). However, after week 1, all BWs to future BWs were correlated highly and positively. This was consistent with Tona et al. (2004) who found no significant relationship between HW and processing weight, but they did find 7-day and 35-day BW were correlated significantly to processing weight. Therefore, HL has been determined to be a better method for assessing hatchling quality (Joseph et al., 2006) and has been demonstrated to be correlated positively to market BW in broiler chickens (Meijerhof, 2005). HL was found to be a better predictor of growth performance than HW because it is more strongly correlated with BW (Petek et al., 2010), which is confirmed our study results. Wolanski et al. (2006) found that SL was more strongly correlated to 14-day BW than HW or HL, indicating it might be another strong predictor of growth performance. RA was correlated positively to final BW in this experiment, which supports the work of Sanotra et al. (2001) in broiler chickens. In the current study, BMY also was correlated to weeks 3, 5 and 10 BWs, but not to HW. This observation opposes Wolanski et al. (2006) who found that HW and 14-day BW were correlated positively to breast muscle.

In contrast to expectations, the total Tona score was not predictive of subsequent growth performance, although some of the component indicators of the Tona score did. Large remaining yolks (assessed by remaining yolk score) were correlated to decreased appearance scores in poults, and normally retracted yolks (assessed by retracted yolk score)

were correlated to lack of infected legs. Moreover, birds with fully absorbed yolks (retracted yolk score) were significantly heavier at market age compared to bird with large yolk (remaining yolk score). Total yolk absorption and utilization supplies the required energy for metabolism and development at the expense of supplying phospholipids necessary for building mucosal cellular membranes if dietary calorie consumption is deficient during the first days of life (Noy & Sklan, 1999). The rate of membrane turnover is greatest in mucosal epithelial tissues of the intestinal tract and eyes. Mucosal epithelial development of the eye can be determined by non-invasive observation, whereas enteric mucosal development cannot. We observed eye abnormalities (eye condition score) were correlated to decreased market age BWs, which may be associated with inferior enteric mucosa development and malabsorption of perinatal poults. Evidently, the prevalence of eye abnormalities is greatest in FOL breeders and occurrence is higher in poults than chicks (Mróz et al., 2013).

Intestinal Development

Although no significant breeder age effects on jejunum or ileum villi were observed in progeny at 1 week of age, the FOL poults were observed to have significantly greater jejunum villi SA associated with greater villus width than PEAK poults at 5 weeks of age. In contrast, Mahmoud & Edens (2012) and El Sabry et al. (2013) found the jejunum and ileum villus SAs to be less for day-old chicks from FOL breeders than PEAK breeders. At 5 weeks of age, PEAK poults had increased ileum villus height and villus top width, which agrees with the observations reported by other researchers (El Sabry et al., 2013; Schaefer et al., 2006). However, the effect of breeder flock age on intestinal mucosal villi SA diminishes as the poult increases in age (Schaefer et al., 2006).

In conclusion, young breeder stage of lay negatively impacts early growth of poults and subsequent growth performance of market turkey hens, despite dietary intervention. The most significant indicators for predicting the future growth performance of FOL poults were HL, SL and RA. In contrast to expectations, hatchling weight was not correlated to subsequent BWs, including the final market age BW at 14 weeks of age. Although, the Tona score protocol may be a reasonable indicator of poult condition at hatch, it is not an effective indicator of subsequent growth performance; rather, more specific quantitative indicators should be used to determine the effects of breeder hen age on market age performance in turkey hens. This research confirms that young breeder age can affect embryonic development and post-hatch growth rates likely by constraining egg characteristics. By using young breeder age to create differences in PQ indicators, these indicators were observed to be significantly correlated with depressed growth rates and reduced meat yield at market age.

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Table 4.1. Ingredients and nutrient composition of experimental diets fed to turkey hens for 14 weeks.

	Starter 1		Starter 2		Grower 1		Grower 2		Finisher 1		Finisher 2	
	Diet 1	**Diets 2-4**	**Diet 1**	**Diets 2-4**	**Diet 1**	**Diets 2-4**	**Diet 1**	**Diets 2-4**	**Diet 1**	**Diets 2-4**	**Diet 1**	**Diets 2-4**
Ingredients	*% of total diet*											
Corn	24.5	27.0	32.5	35.1	38.9	41.4	44.5	47.1	49.1	51.6	52.1	54.7
Wheat	15.0	15.0	15.0	15.0	15.0	15.0	15.0	15.0	15.0	15.0	15.0	15.0
DDGS corn 27%CP	3.0	3.0	3.0	3.0	3.0	3.0	5.0	5.0	5.0	5.0	5.0	5.0
Soybean meal	46.0	45.7	39.1	38.9	34.0	33.8	27.2	27.0	23.5	23.2	20.1	19.8
Poultry fat	4.66	3.13	3.90	2.36	3.35	1.83	2.95	1.40	2.76	1.23	3.08	1.53
Limestone	2.12	2.09	2.07	2.04	1.87	1.84	1.76	1.72	1.55	1.52	1.57	1.53
Dicalcium phosphate	2.72	2.02	2.47	1.77	2.16	1.49	1.93	1.22	1.61	0.91	1.63	0.93
Salt NaCl	0.20	0.20	0.22	0.21	0.24	0.24	0.22	0.22	0.12	0.13	0.16	0.16
Sodium bicarbonate	0.26	0.27	0.21	0.21	0.14	0.14	0.16	0.16	0.26	0.26	0.22	0.21
L-Lysine HCl 98%	0.40	0.40	0.35	0.35	0.32	0.32	0.31	0.32	0.27	0.27	0.26	0.26
DL-Methionine 99.0%	0.36	0.36	0.32	0.31	0.27	0.27	0.22	0.21	0.18	0.17	0.14	0.14
L-Threonine 98.5%	0.10	0.09	0.08	0.08	0.07	0.07	0.06	0.06	0.05	0.05	0.04	0.04
Vitamin Premix[1]	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15
Trace Mineral Premix[2]	0.20	0.20	0.20	0.20	0.20	0.20	0.20	0.20	0.20	0.20	0.20	0.20
Sodium Premix (0.06%)	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05
Choline chloride 60%	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Premix (A, B, or C)[3,4]	0.21	0.21	0.22	0.22	0.17	0.16	0.18	0.18	0.16	0.16	0.17	0.17
Nutrient Composition												
ME POULTRY, kcal/kg	2800	2755	2850	2805	2900	2855	2950	2905	3000	2955	3050	3005
CP, %	26.46	26.52	23.78	23.84	21.79	21.85	19.53	19.59	18.04	18.12	16.66	16.73
CA APP PHY, %	1.46	1.34	1.38	1.26	1.24	1.12	1.14	1.01	1.00	0.88	1.00	0.87
NON PHYT P, %	0.75	0.61	0.69	0.55	0.62	0.48	0.57	0.43	0.50	0.36	0.50	0.36
CA, %	1.46	1.34	1.38	1.26	1.24	1.12	1.14	1.01	1.00	0.88	1.00	0.87
P, %	1.02	0.88	0.94	0.80	0.86	0.73	0.80	0.66	0.72	0.58	0.71	0.57
CA:AV P	1.95	2.21	2.00	2.30	2.00	2.33	2.00	2.38	2.00	2.46	2.00	2.45
DM	89.13	88.84	88.89	88.60	88.66	88.38	88.49	88.19	88.33	88.04	88.32	88.03

[1]Vitamin premix provided the following per kg of diet: 13242 IU of vitamin A, 3973 IU of vitamin D, 66 IU of vitamin E, 0.40 mg/kg of vitamin B12, 0.25 mg/kg of biotin, 3.97 mg/kg of vitamin K, 13.24 mg/kg of riboflavin, 22.07 mg/kg of [2]Mineral premix provided the following per kg of diet: 5.00 mg/kg of Cu, 40.04 mg/kg of Fe, 60.07 mg/kg of Mn, 60.07 mg/kg of Zn, 1.25 mg/kg of I.

[3]Premix A = 0.1% corn - added to diets 1, 2, 5, 6, 9, 10 / Premix B = 0.025% Hostazym®, 0.0125% Optiphos®, and 0.0625% corn - added to diets 3, 7 and 11 / Premix C = 0.025% Hostazym® and 0.075% Optiphos® - added to diets 4, 8 and 12.

[4]Azomite® excluded from diets 1, 2, 3 and 4 - included at 0.025% in diets 5, 6, 7 and 8 - included at 0.50% in diets 9, 10, 11 and 12.

Table 4.2. Main effects and interactions of Azomite®, diet and breeder age on body weight gain (BWG) of turkey hens to 10 weeks of age.

Period (Weeks)		0-3	0-5	0-8	0-10	3-5	5-8	8-10
		(Main Effects: BWG, kg)[1]						
Azomite® (g/kg)	***0***	0.554	1.553	3.834	5.55	0.999	2.281	1.716
	2.5	0.553	1.564	3.829	5.54	1.011	2.264	1.683
	5	0.552	1.552	3.825	5.5	1.005	2.274	1.645
Diet	***NC***	0.528^{B}	1.480^{C}	3.681^{C}	5.38^{B}	0.953^{C}	2.200^{B}	1.701
	PC	0.547^{AB}	1.556^{B}	3.791^{BC}	5.51^{AB}	1.009^{B}	2.236^{B}	1.679
	E1	0.561^{A}	1.575^{AB}	3.839^{B}	5.55^{AB}	1.014^{AB}	2.264^{B}	1.675
	E2	0.570^{A}	1.615^{A}	4.007^{A}	5.68^{A}	1.045^{A}	2.392^{A}	1.669
Breeder Age	***PEAK***	0.570^{A}	1.606^{A}	3.951^{A}	5.64^{A}	1.036^{A}	2.345^{A}	1.691
	FOL	0.532^{B}	1.507^{B}	3.707^{B}	5.42^{B}	0.975^{B}	2.201^{B}	1.671
		(Azomite® X Breeder Age Interactions: BWG, kg)[2]						
0	***PEAK***	0.574	1.601	3.936	5.66	2.334	2.334	1.726
2.5	***PEAK***	0.569	1.608	3.945	5.62	2.337	2.337	1.679
5	***PEAK***	0.568	1.609	3.974	5.64	2.365	2.365	1.669
0	***FOL***	0.534	1.510	3.733	5.44	2.227	2.227	1.706
2.5	***FOL***	0.537	1.521	3.712	5.46	2.192	2.192	1.686
5	***FOL***	0.527	1.495	3.677	5.36	2.183	2.183	1.620
		(Diet X Breeder Age Interactions: BWG, kg)[3]						
NC	***PEAK***	0.542	1.527	3.784	5.5	2.257	2.257	1.718
PC	***PEAK***	0.565	1.602	3.914	5.61	2.312	2.312	1.698
E1	***PEAK***	0.582	1.634	4.000	5.68	2.367	2.367	1.684
E2	***PEAK***	0.592	1.662	4.107	5.77	2.445	2.445	1.665
NC	***FOL***	0.513	1.434	3.578	5.26	2.144	2.144	1.685
PC	***FOL***	0.529	1.509	3.668	5.41	2.159	2.159	1.660
E1	***FOL***	0.539	1.516	3.676	5.42	2.160	2.160	1.665
E2	***FOL***	0.548	1.569	3.907	5.58	2.339	2.339	1.673
Source of Variation		---------- *(P-Values)* ----------						
Azomite®		0.59	0.56	0.98	0.56	0.45	0.90	0.15
Diet		**<0.01**	**<0.01**	**0.01**	**<0.01**	**<0.01**	**<0.01**	0.84
Breeder Age		**<0.01**	**<0.01**	**<0.01**	**<0.01**	**<0.01**	**<0.01**	0.38
Azomite®*Breeder Age		0.56	0.58	0.59	0.38	0.64	0.63	0.62
Diet*Breeder Age		0.55	0.82	0.63	0.89	0.78	0.65	0.90
SEM(36)[4]		0.006	0.02	0.05	0.06	0.01	0.04	0.05

[A-C] Means within a row with different superscripts are significantly different ($p<0.01$).

[1]Mean values are averages of 16 replicate pens (Azomite®) and 12 replicate pens (Diet) of ~20 birds; 48 replicate pens (containing ~5 FOL and ~15 PEAK birds).

[2]Mean values are averages of 8 replicate pens of ~8 birds per FOL treatment combination and ~20 birds per PEAK treatment combination.

[3]Mean values are averages of 6 replicate pens of ~8 birds per FOL treatment combination and ~20 birds per PEAK treatment combination.

[4]SEM= Standard Error of the Mean with 36 degrees of freedom.

Table 4.3. Main effects of Azomite®, diet and breeder age on mortality of turkey hens to 10 weeks of age.[1]

Period[2]		Starter	Grower	Cumulative
		(Main Effects: Mortality %)[1]		
Azomite® (g/kg)	***0***	1.87	1.51	3.38
	2.5	2	1.34	3.35
	5	2.76	1.34	4.1
Diet	***NC***	2.92	0.46	3.38
	PC	1.48	2.16	3.64
	E1	2.71	1.64	4.35
	E2	1.73	1.32	3.06
Breeder Age	***PEAK***	0.59^{B}	1.25	2.13^{B}
	FOL	3.83^{A}	1.54	5.08^{A}
Source of Variation		---------- *(P-Values)* ----------		
Azomite®		0.6237	0.9769	0.8493
Diet		0.5101	0.4363	0.8883
Breeder Age		**<0.0001**	0.7184	**0.0043**
SEM(36)[3]		0.84	1.04	0.84

[A.B] Means within a row with different superscripts are significantly different ($p<0.01$).
[1]Mean values are averages of 16 replicate pens (Azomite®) and 12 replicate pens (Diet); 48 replicate pens (Breeder Age).
[2]Starter period is from 0-5 weeks of age, grower period is from 5-10 weeks, cumulative period is from 0-10 weeks of age.
[3]SEM= Standard Error of the Mean with 36 degrees of freedom.

Table 4.4. Main effects of Azomite®, diet and breeder age on relative breast muscle yield (BMY) of turkey hens at 14 weeks of age.

		(Main Effects: % breast muscle / total body weight) [1]
Azomite® (g/kg)	***0***	24.46
	2.5	24.85
	5	24.42
Diet	***NC***	23.83^{B}
	PC	24.58^{AB}
	E1	24.79^{A}
	E2	25.10^{A}
Breeder Age	***PEAK***	24.90^{A}
	FOL	24.25^{B}
Source of Variation		---------- *(P-Values)* ----------
Azomite®		0.15
Diet		**<0.01**
Breeder Age		**<0.01**
SEM(36)[2]		0.19

^{A,B} Means within a row with different superscripts are significantly different ($p<0.01$).
[1]Mean values are averages of 16 replicate pens (Azomite®) and 12 replicate pens (Diet) of ~20 birds; 48 replicate pens (containing ~5 FOL and ~15 PEAK birds).
[2]SEM= Standard Error of the Mean with 36 degrees of freedom.

Table 4.5. Individual correlations among day-old poult PQ qualitative and quantitative measurements.[1]

	APP	RTY	EYE	LEG	NAV	MEM	RMY	TONA	HL	L	R	RA
ACT	0.086	-0.069	-0.073	0.014	0.052	-0.056	-0.088	0.242*	0.073	0.110	0.013	-0.113
APP		-0.113	0.059	0.123	-0.023	-0.092	-0.145+	0.163	-0.160	0.001	-0.030	-0.085
RTY			-0.047	0.181+	-0.085	-0.036	0.110	0.437*	0.143	-0.006	0.003	0.083
EYE				0.010	-0.091	-0.038	-0.061	0.286*	-0.135	-0.045	-0.032	0.013
LEG					0.018	-0.104	0.034	0.741*	-0.069	-0.045	-0.069	-0.007
NAV						0.039	-0.109	0.318*	0.022	-0.124	-0.252*	-0.182+
MEM							-0.046	0.041	0.094	-0.038	0.048	0.210*
RMY								0.151	0.102	0.056	-0.037	-0.125
TONA									0.004	-0.058	-0.151	-0.071
HL										0.417*	0.336*	-0.062
L											0.826*	-0.241*
R												0.218*

[1]ACT = activity (score of 0, 3, or 6 based on altertness), APP = appearance (score of 0, 8, or 10 based on downs), RTY = retracted yolk (score of 0, or 12 based on condition of internal yolk), EYE = eye condition (score of 0, 8, or 16), LEG = leg infection condition (score of 0, 8, or 16), NAV = navel condition (score of 0, 6, or 12 based on extent of navel healing), MEM = membrane (score of 0, 4, 8, or 12 based on attached eggshell membrane), RMY = remaining yolk (score of 0, 8, 12 or 16 based on external yolk), TONA = total Tona score (0-100), HL = hatchling length (cm), L = left shank length (mm), R = right shank length (mm), RA = relative asymmetry.

*Correlation coefficients at 95% confidence interval (93 degrees of freedom) that have critical values greater than 0.202 or less than -0.202.

+Correlation coefficients at 90% confidence interval (93 degrees of freedom) that have critical values greater than 0.170 or less than -0.170.

Table 4.6. Individual correlations among day-old PQ measurements and post-hatch performance.[1]

	BW, wk 1	BW, wk 3	BW, wk 5	BW, wk 8	BW, wk 10
ACT	0.051	0.023	0.064	0.112	0.142
APP	-0.146	-0.165	-0.166	-0.022	-0.052
RTY	0.263*	0.131	0.154	0.124	0.127
EYE	-0.072	-0.237*	-0.257*	-0.246*	-0.174+
LEG	-0.015	0.130	0.086	0.130	0.038
NAV	-0.074	0.014	0.032	-0.025	-0.078
MEM	-0.078	0.134	0.029	0.045	-0.073
RMY	-0.029	0.070	0.090	0.246*	0.218*
TONA	0.014	0.074	0.052	0.118	0.047
HL	0.238*	0.553*	0.360*	0.360*	0.293*
L	0.251*	0.346*	0.185+	0.205*	0.132
R	0.222*	0.264*	-0.011	0.068	0.023
RA	-0.009	-0.035	-0.223*	-0.228*	-0.247*
BW, wk 1		0.388*	0.400*	0.285*	0.203*
BW, wk 3			0.599*	0.667*	0.562*
BW, wk 5				0.621*	0.513*
BW, wk 8					0.763*

[1]ACT = activity (score of 0, 3, or 6 based on altertness), APP = appearance (score of 0, 8, or 10 based on downs), RTY = retracted yolk (score of 0, or 12 based on condition of internal yolk), EYE = eye condition (score of 0, 8, or 16), LEG = leg infection condition (score of 0, 8, or 16), NAV = navel condition (score of 0, 6, or 12 based on extent of navel healing), MEM = membrane (score of 0, 4, 8, or 12 based on attached eggshell membrane), RMY = remaining yolk (score of 0, 8, 12 or 16 based on external yolk), TONA = total Tona score (0-100), HL = hatchling length (cm), L = left shank length (mm), R = right shank length (mm), RA = relative asymmetry.

*Correlation coefficients at 95% confidence interval (93 degrees of freedom) that have critical values greater than 0.202 or less than -0.202.

+Correlation coefficients at 90% confidence interval (93 degrees of freedom) that have critical values greater than 0.170 or less than -0.170.

Table 4.7. Average correlations between post-hatch performance parameters.[1]

	BMY	SM	GM	CM
HW	-0.116	-0.067	0.282[+]	0.168
BW, wk 1	0.085	-0.038	-0.331[*]	-0.290[*]
BW, wk 3	0.334[*]	0.027	-0.145	-0.092
BW, wk 5	0.265[+]	-0.03	-0.019	-0.039
BW, wk 8	0.084	0.035	0.025	0.048
BW, wk 10	0.383[*]	-0.23	0.023	-0.165
BMY[1]		-0.251[+]	-0.049	-0.238[+]
SM[1]			-0.202	0.638[*]
GM[1]				0.626[*]

[1]HW = hatchling weight, BW = average body weight, BMY = relative breast muscle yield (% total breast muscle / total body weight), SM = starter mortality, GM = grower mortality, CM = cumulative mortality.

[*]Correlation coefficients at 95% confidence interval (93 degrees of freedom) that have critical values greater than 0.202 or less than -0.202.

[+]Correlation coefficients at 90% confidence interval (93 degrees of freedom) that have critical values greater than 0.170 or less than -0.170.

Table 4.8. Main effects and interactions of Azomite®, diet and breeder age on jejunum morphometrics of turkey hens at 1 week of age. [1]

		VH[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm^2)
				(Main Effects)			
Azomite® (g/kg)	**0**	679.9	89	150.7	76.5	105	81574
	2.5	649.8	90.9	158	82.6	102.3	82028
Diet	**NC**	680	92.1	162.5^{A}	74.9^{B}	104.4	86875
	PC	649.8	87.8	146.1^{B}	84.1^{A}	103	76727
Breeder Age	**PEAK**	670.8	86.2	149.4	77.7	104.5	79660
	FOL	659	93.7	159.2	81.4	102.9	83942
				(Azomite® X Breeder Age Interactions)			
0	**PEAK**	677.2	84	142.3	73.8	108.2	76543
2.5	**PEAK**	664.4	88.3	156.5	81.7	100.8	82777
0	**FOL**	682.6	94	159	79.2	101.8	86605
2.5	**FOL**	635.3	93.4	159.4	83.5	103.9	81278
				(Diet X Breeder Age Interactions)			
NC	**PEAK**	664	88.5	157.3	75.2	107	82440
PC	**PEAK**	677.6	83.9	141.6	80.2	102	76880
NC	**FOL**	696	95.7	167.8	74.6	101.7	91310
PC	**FOL**	622	91.7	150.6	88.1	104	76573
Source of Variation		----------	----------	*(P-Values)*	----------	----------	----------
Azomite®		0.40	0.68	0.23	0.06	0.51	0.94
Diet		0.40	0.33	**<0.01**	**<0.01**	0.74	0.11
Breeder Age		0.74	0.09	0.11	0.25	0.69	0.49
Azomite®*Breeder Age		0.63	0.58	0.25	0.57	0.25	0.36
Diet*Breeder Age		0.23	0.95	0.91	0.57	0.37	0.46
SEM(45)[3]		25.3	3	4.2	2.2	3	4387

[A,B] Means within a row with different superscripts are significantly different ($p<0.01$).

[1]Mean values are averages of 8 replicate samples per treatment combination.

[2]VH = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.

[3]SEM = Standard Error of the Mean with 45 degrees of freedom.

Table 4.9. Main effects and interactions of Azomite®, diet and breeder age on jejunum morphometrics of turkey hens at 5 weeks of age. [1]

		VH[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm^2)
				(Main Effects)			
Azomite® (g/kg)	**0**	1207[b]	173	262	126.3	184.7	265103[b]
	2.5	1331[a]	179.1	285.2	123.4	187.3	312082[a]
Diet	**NC**	1218	169.9	264	122.8	186.7	266037[b]
	PC	1320	182.2	283.2	126.8	185.3	311147[a]
Breeder Age	**PEAK**	1259	165.9[b]	254.2[B]	123.6	184.1	267947[b]
	FOL	1279	186.2[a]	293.1[A]	126	187.9	309237[a]
				(Azomite® X Breeder Age Interactions)			
0	**PEAK**	1198	153.9	235.3	126.8	186.2	235826
2.5	**PEAK**	1320	177.9	273	120.5	182	300068
0	**FOL**	1216	192.1	288.7	125.7	183.3	294379
2.5	**FOL**	1342	180.3	297.4	126.4	192.5	324095
				(Diet X Breeder Age Interactions)			
NC	**PEAK**	1233	158.4	238.8	122.9	188.8	247919
PC	**PEAK**	1285	173.3	269.5	124.4	179.4	287976
NC	**FOL**	1203	181.4	289.2	122.8	184.7	284156
PC	**FOL**	1355	191	297	129.3	191.1	334318
Source of Variation		---------- *(P-Values)* ----------					
Azomite®		**0.02**	0.51	0.07	0.56	0.88	**0.03**
Diet		0.06	0.19	0.12	0.41	0.80	**0.03**
Breeder Age		0.70	**0.03**	**<0.01**	0.62	0.70	**0.05**
Azomite®*Breeder Age		0.97	0.06	0.24	0.47	0.49	0.40
Diet*Breeder Age		0.36	0.77	0.35	0.60	0.42	0.80
SEM(45)[3]		37.7	6.4	8.6	3.4	6.9	14281

[A,B] Means within a row with different superscripts are significantly different ($p<0.01$).

[a,b] Means within a row with different superscripts are significantly different ($p<0.05$).

[1]Mean values are averages of 8 replicate samples per treatment combination.

[2]VH = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.

[3]SEM = Standard Error of the Mean with45 degrees of freedom.

Table 4.10. Main effects and interactions of Azomite®, diet and breeder age on ileum morphometrics of turkey hens at 1 week of age. [1]

		VH[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm^2)
				(Main Effects)			
Azomite® (g/kg)	**0**	442.8	88.3^{a}	128.6	74.4	118.5	48134
	2.5	457.5	81.5^{b}	123.4	74.9	127.2	47347
Diet	**NC**	449.3	84.6	127.4	72.3	124.7	47663
	PC	451.0	85.2	124.6	77.0	121.0	47818
Breeder Age	**PEAK**	454.2	84.5	123.7	75.1	131.9^{A}	47582
	FOL	446.2	85.3	128.3	74.2	113.8^{B}	47899
				(Azomite® X Breeder Age Interactions)			
0	**PEAK**	431.6	85.6	119.4^{c}	72.4	127.7	44097^{B}
2.5	**PEAK**	476.8	83.5	128^{b}	77.8	136.0	51066^{A}
0	**FOL**	454.0	91.1	137.9^{a}	76.4	109.2	52170^{A}
2.5	**FOL**	438.3	79.5	118.7^{c}	72.0	118.4	43628^{B}
				(Diet X Breeder Age Interactions)			
NC	**PEAK**	442.9	82.7	119^{c}	73.2	135.2	44577^{B}
PC	**PEAK**	465.5	86.5	128.5^{b}	76.9	128.5	50586^{A}
NC	**FOL**	455.7	86.7	135.8^{a}	71.4	114.2	50748^{A}
PC	**FOL**	436.6	84.0	120.8^{c}	77.0	113.4	45049^{B}
Source of Variation		---------- *(P-Values)* ----------					
Azomite®		0.43	**0.02**	0.30	0.87	0.12	0.77
Diet		0.93	0.84	0.58	0.11	0.50	0.96
Breeder Age		0.67	0.78	0.37	0.75	**<0.01**	0.91
Azomite®*Breeder Age		0.11	0.11	**<0.01**	0.09	0.94	**<0.01**
Diet*Breeder Age		0.27	0.26	**0.02**	0.74	0.59	**0.04**
SEM(45)[3]		13.2	2	3.6	2	3.9	1935

[A,B] Means within a row with different superscripts are significantly different ($p<0.01$).
[a,b] Means within a row with different superscripts are significantly different ($p<0.05$).
[1]Mean values are averages of 8 replicate samples per treatment combination.
[2]VH = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.
[3]SEM = Standard Error of the Mean with 45 degrees of freedom.

Table 4.11. Main and interaction effects of Azomite®, diet and breeder age on ileum morphometrics of turkey hens at 5 weeks of age. [1]

		VH[2] (µm)	TW (µm)	BW (µm)	CD (µm)	M (µm)	SA (µm^2)
				(Main Effects)			
Azomite® (g/kg)	**0**	814.9	127.6	204.4	118.9	237.9	135194
	2.5	821.3	126.9	204.9	115.9	237.6	135445
Diet	**NC**	808.3	131.8	208.9	117.4	235.9	137368
	PC	827.9	122.7	200.3	117.3	239.7	133271
Breeder Age	**PEAK**	843.4	122.0	200.6	117.4	235.1	135522
	FOL	792.8	132.5	208.7	117.3	240.5	135117
				(Azomite® X Breeder Age Interactions)			
0	**PEAK**	833.3	120.7	196.2	123.3[a]	241.9	131718
2.5	**PEAK**	853.4	123.2	204.9	111.6[b]	228.2	139326
0	**FOL**	796.5	134.4	212.5	114.4[b]	233.9	138669
2.5	**FOL**	789.1	130.6	204.8	120.1[a]	247.1	131564
				(Diet X Breeder Age Interactions)			
NC	**PEAK**	829.2	123.4	202.5	113.3[b]	230.0	134719
PC	**PEAK**	857.6	120.8	198.6	121.6[a]	240.1	136326
NC	**FOL**	787.3	140.4	215.3	121.5[a]	241.8	140016
PC	**FOL**	798.2	124.7	202.0	113.1[b]	239.2	130217
Source of Variation		---------- *(P-Values)* ----------					
Azomite®		0.81	0.90	0.96	0.40	0.98	0.97
Diet		0.47	0.11	0.34	0.99	0.71	0.55
Breeder Age		0.07	0.06	0.37	0.96	0.58	0.95
Azomite®*Breeder Age		0.61	0.57	0.36	**0.02**	0.18	0.29
Diet*Breeder Age		0.75	0.23	0.60	**0.02**	0.52	0.41
SEM(45)[3]		19	3.9	6.3	2.5	6.9	4871

[a,b] Means within a row with different superscripts are significantly different ($p<0.05$).

[1]Mean values are averages of 8 replicate samples per treatment combination.

[2]VH = villus height, TW = villus top width, BW = villus bottom width, CD = crypt depth, M = muscularis layer, SA = villus surface area.

[3]SEM = Standard Error of the Mean with 45 degrees of freedom.

REFERENCES

Applegate, T. J., Dibner, J. J., Kitchell, M. L., Uni, Z., & Lilburn, M. S. (1999). Effect of turkey (Meleagridis gallopavo) breeder hen age and egg size on poult development. 2. Intestinal villus growth, enterocyte migration and proliferation of the turkey poult. *Comparative Biochemistry and Physiology - B Biochemistry and Molecular Biology*, *124*(4), 381–389.

Boerjan, M. L. (2002). Programs for single stage incubation and chick quality. *Avian and Poultry Biology Reviews*, *13*(4), 237.

Christensen, V., Grimes, J., & Wineland, M. (2001). Effects of turkey breeder hen age, strain, and length of the incubation period on survival of embryos and hatchlings. *Journal of Applied Poultry Research*, *10*, 5–15.

de Verdal, H., Mignon-Grasteau, S., Jeulin, C., le Bihan-Duval, E., Leconte, M., Mallet, S., … Narcy, A. (2010). Digestive tract measurements and histological adaptation in broiler lines divergently selected for digestive efficiency. *Poultry Science*, *89*(9), 1955–1961.

Decuypere, E., Tona, K., Bamelis, F., Careghi, C., Kemps, B., Ketelaere, B. De, … Bruggeman, V. (2002). Broiler breeders and egg factors interacting with incubation conditions for optimal hatchability and chick quality. *Archive Für Geflügelkunde*, *66*, 56–57.

Deeming, D. C. (2000). What is chick quality. *World Poultry*, *11*(2), 34–35.

El Sabry, I. M., Yalçın, S., & Turgay-İzzetoğlu, G. (2013). Interaction between breeder age and hatching time affects intestine development and broiler performance. *Livestock Science*, *157*, 612–617.

Francesch, M., Pérez-Vendrell, A. M., & Broz, J. (2012). Effects of a mono-component endo-xylanase supplementation on the nutritive value of wheat-based broiler diets Effects of a mono-component endo-xylanase supplementation on the nutritive value of wheat-based broiler diets. *British Poultry Science*, *53*(6), 809–816.

Geidam, Y. A., Bukar, M. M., & Ambali, A. G. (2007). Chick quality control: a key to sustainable poultry production in Nigeria. *Nigerian Veterinary Journal*, *27*(2), 1–6.

Hill, D. (2001). Chick length uniformity profiles as a field measurement of chick quality. *Avian Poultry Biological Reviews, 12*, 188.

Joseph, N. S., Lourens, A., & Moran, E. T. (2006). The effects of suboptimal eggshell temperature during incubation on broiler chick quality, live performance, and further processing yield. *Poultry Science, 85*(5), 932–938.

Joseph, N. S., & Moran, E. T. (2005). Effect of flock age and postemergent holding in the hatcher on broiler live performance and further-processing yield. *Journal of Applied Poultry Research, 14*(3), 512–520.

Latour, M. A., Peebles, E. D., Boyle, C. R., Doyle, S. M., Pansky, T., & Brake, J. D. (1996). Effects of breeder hen age and dietary fat on embryonic and neonatal broiler serum lipids and glucose. *Poultry Science, 75*(6), 695–701.

Leary, S., Underwood, W., Anthony, R., & Cartner, S. (2013). *AVMA Guidelines for the Euthanasia of Animals: 2013 Edition*. *American Veterinary Medical Association*.

Lü, M., Li, D., Gong, L., Ru, Y., & Ravindran, V. (2009). Effects of supplemental microbial phytase and xylanase on the performance of broilers fed diets based on corn and wheat. *Japan Poultry Science Association, 46*(3), 217–223.

Mahmoud, K. Z., & Edens, F. W. (2012). Breeder age affects small intestine development of broiler chicks with immediate or delayed access to feed. *British Poultry Science, 53*(1), 32–41.

Meijerhof, R. (2005). What counts for chick quality. *Hybro, BV*.

Møller, A. P., Sanotra, G. S., & Vestergaard, K. S. (1999). Developmental instability and light regime in chickens (Gallus gallus). *Applied Animal Behaviour Science, 62*(1), 57–71.

Mróz, E., Stępińska, M., Orłowska, A., & Michalak, K. (2013). Frequency of occurrence of physical defects in turkey poults. *Annals of Animal Science, 13*(3), 543–552.

Mukhtar, N., Khan, S. H., & Anjum, M. S. (2013). Hatchling length is a potential chick quality parameter in meat type chickens. *World's Poultry Science Journal, 69*(04), 889–896.

Nangsuay, A., Ruangpanit, Y., Meijerhof, R., & Attamangkune, S. (2011). Yolk absorption and embryo development of small and large eggs originating from young and old breeder hens. *Poultry Science*, *90*(11), 2648–2655.

Noble, R. . C., Lonsdale, F., Connor, K., & Brown, D. (1986). Changes in the lipid metabolism of the chick embryo with parental age. *Poultry Science*, *65*, 409–416.

Noy, Y., & Sklan, D. (1999). Energy utilization in newly hatched chicks. *Poultry Science*, *78*(12), 1750–1756.

O'Dea, E. E., Fasenko, G. M., Feddes, J. J. R., Robinson, F. E., Segura, J. C., Ouellette, C. A., & Middelkoop, J. H. Van. (2004). Investigating the eggshell conductance and embryonic metabolism of modern and unselected domestic avian genetic strains at two flock ages. *Poultry Science*, *83*, 2059–2070.

Petek, M., Orman, A., Dikmen, S., & Alpay, F. (2010). Physical chick parameters and effects on growth performance in broiler. *Archives Animal Breeding*, *53*(1), 108–115.

Roberts, J. R., Souillard, R., & Bertin, J. (2011). Improving the safety and quality of eggs and egg products: egg chemistry, production and consumption. In *Food Science, Technology and Nutrition* (pp. 376–393). Woodhead Publishing.

Sanotra, G. S., Lund, J. D., Ersboll, a K., Petersen, J. S., & Vestergaard, K. S. (2001). Monitoring leg problems in broilers : a survey of commercial broiler production in Denmark. *World's Poultry Science Journal*, *57*(March), 55–69.

Schaefer, C. M., Corsiglia, C. M., Mireles, A., & Koutsos, E. A. (2006). Turkey breeder hen age affects growth and systemic and intestinal inflammatory responses in female poults examined at different ages posthatch. *Poultry Science*, *85*, 1755–1763.

Sklan, D., Heifetz, S., & Halevy, O. (2003). Heavier chicks at hatch improves marketing body weight by enhancing skeletal muscle growth. *Poultry Science*, *82*, 1778–1786.

Tatara, M. R., Krupski, W., Kozlowski, K., Drazbo, A., & Jankowski, J. (2015). Effects of administration of four different doses of Escherichia coli phytase on femur properties of 16-week-old turkeys. *BMC Veterinary Research*, *11*(69), 66–71.

Tona, K., Bamelis, F., Coucke, W., Bruggeman, V., & Decuypere, E. (2001). Relationship between broiler breeder's age and egg weight loss and embryonic mortality during

incubation in large-scale conditions. *Journal of Applied Poultry Research*, *10*, 221–227.

Tona, K., Bamelis, F., De Ketelaere, B., Bruggeman, V., Moraes, V., Buyse, J., … Decuypere, E. (2003). Effects of egg storage time on spread of hatch, chick quality, and chick juvenile growth. *Poultry Science*, *82*(5), 736–741.

Tona, K., Onagbesan, O., De Ketelaere, B., Decuypere, E., & Bruggeman, V. (2004). Effects of age of broiler breeders and egg storage on egg quality, hatchability, chick quality, chick weight, and chick posthatch growth to forty-two days. *Journal of Applied Poultry Research*, *13*(1), 10–18.

Ulmer-Franco, A. M., Fasenko, G. M., & O'Dea Christopher, E. E. (2010). Hatching egg characteristics, chick quality, and broiler performance at 2 breeder flock ages and from 3 egg weights. *Poultry Science*, *89*, 2735–2742.

Vieira, S. L., & Moran, E. T. (1998). Eggs and chicks from broiler breeders of extremely different age. *Journal of Applied Poultry Research*, *7*(4), 372–376.

Vieira, S. L., & Moran, E. T. (1999). Effects of egg of origin and chick post-hatch nutrition on broiler live performance and meat yields. *World's Poultry Science Journal*, *55*, 125–142.

Willemsen, H., Everaert, N., Witters, A., De Smit, L., Debonne, M., Verschuere, F., … Bruggeman, V. (2008). Critical assessment of chick quality measurements as an indicator of posthatch performance. *Poultry Science*, *87*(11), 2358–2366.

Wolanski, N. J., Luiten, E. J., Meijerhof, R., & Vereijken, A. L. J. (2004). Yolk utilisation and chick length as parameters for embryo development. *Avian and Poultry Biology Reviews*, *15*(3–4), 233–234.

Wolanski, N. J., Renema, R. A., Robinson, F. E., Carney, V. L., & Fancher, B. I. (2006). Relationship between chick conformation and quality measures with early growth traits in males of eight selected pure or commercial broiler breeder strains. *Poultry Science*, *85*, 1490–1497.

Yalçin, S., & Siegel, P. B. (2003). Exposure to cold or heat during incubation on developmental stability of broiler embryos. *Poultry Science*, *82*(9), 1388–1392.

Yang, A., Dunnington, E. A., & Siegel, P. B. (1998). Developmental stability in different

genetic stocks of white rock chickens. *Journal of Heredity*, *89*(3), 260–264.

CHAPTER 5

ESTIMATING EFFECTS OF HATCHERY PRACTICES ON EARLY POU\LT MORTALITY USING TURKEY INDUSTRY FIELD DATA

S. BLACK[3] **AND T. VUKINA**

NORTH CAROLINA STATE UNIVERSITY

KEYWORDS: EARLY POULT MORTALITY, BREEDER AGE, HATCHERY MANAGEMENT

PRIMARY AUDIENCE: BREEDER AND HATCHERY PERSONNEL

[3] Corresponding author: sablack@ncsu.edu

SUMMARY

Early poult mortality is a widespread problem in the turkey industry resulting in potential loss of profits and animal welfare concerns. In this study, factors related to pre-placement mortality (PPM), first week mortality (FWM) and cumulative early mortality (CEM) were investigated. Field data collected from six US hatcheries, for a total of 3,562 shipments from various breeder flocks delivered to a total of 1,051 customers over a 22-month period, were analyzed using the ordinary least squares (OLS) regression analysis. PPM was found to be significantly related to hatchery, season and distance traveled from hatchery to farm and FWM and CEM were found to be significantly related to hatchery, season, hatch day, gender, breeder age and distance traveled. Assuming the average truck delivery of 17,000 poults and the average poult cost of $2.00, the counterfactual cost analysis showed that the extra production costs from poult mortality due to young stage breeders ranged from $60 to $128 and each 500 miles of travel from hatchery to brooder farm costs an extra $170 per truck delivery. The results indicate that modifications in management practices coordinated between breeder farms, hatcheries and brooder farms could reduce early poult mortality, leading to cost savings and increased profitability.

DESCRIPTION OF THE PROBLEM

Early poult mortality is an endemic problem for the worldwide turkey industry [1]. The first week is critical for maximizing livability and flock uniformity in turkey production [2] as modern day genetics allow for accelerated development and growth rates. Turkey production is important to the agricultural economy in the United States. In 2016, 5.98 billion pounds of turkey meat were produced valued at $6.18 billion [3]. Mortality at any point in the production scheme means a loss in profitability. In addition to its economic importance, high incidence of early poult mortality is often an indicator of animal welfare problems [4]. Stress during incubation or during the first days of life, due a variety of incubation or environmental factors, can cause spikes in embryonic and/or early poult mortality.

The first week of life is critical as poults transition from an extremely conditioned life at hatchery to independent life at farm [5]. Poult management is critical for optimizing the transition from the hatchery to the farm, therefore mortality rate can be an indicator of performance [5]. Poult quality, an indicator of survivability and growth of young turkey poults, is important when considering early poult mortality. Poult performance can be influenced by various embryonic developmental factors. Embryonic stressors during incubation can alter the rate of embryo development or lead to embryonic mortality [6, 7]. It is well documented that breeder age influences egg characteristics and alter turkey embryonic development rate impacting hatchability and livability [8–12]. Incubation conditions that do not provide adequate temperature [13], gas exchange [14], and humidity [15] to developing embryos will alter the rate of embryonic development and affect the viability and organ development resulting in compromised hatchlings. Post-hatch factors that impact poult growth and livability are distance and travel time, environmental conditions during transportation and brooding

conditions [16–18]. For example, delayed access to feed and water resulting in dehydration causes higher initial body weight losses and depressed growth rates after placement [7, 19]. Additionally, turkey poults cannot regulate their body temperatures post-hatch so they are sensitive to temperature extremes that can alter their physiological stress response and lead to mortality during the first week of life. Stressors that cause disruptions in embryonic or early poult development can lead to increased early poult mortality.

The aim of this study is to evaluate the relationship between hatchery and poult management practices and farm level early poult mortality using field data from a US turkey company. In the first part of the study we estimate a linear regression model linking different mortality measures to the variety of indicators reflective of hatchery management and poult quality attributes. In the second part of the study we perform a counterfactual cost analysis simulating potential cost savings associated with altering some of the management practices.

MATERIALS AND METHODS

Description of the Data

Our data set consists of the pre-placement mortality (PPM), first week mortality (FWM) and combined early mortality (CEM) measurements collected by a commercial US turkey company. Data was recorded for a 22-month period from December of 2015 to September of 2017. The unit of observation is a customer invoice. There was a total of 1,051 customers, some of which were repeat customers. Each invoice contained the data on the number of poults per breeder flock delivered to a farm. There were several lines of data for each invoice, generated from different breeder flocks but all delivered to the same customer on the same date. The original data set included 12,746 customer deliveries. To deal with the problem of missing

observations, the data set was cut down to 3,562 customer deliveries. The average number of poults per delivery 17,000.

The summary description of the data set is contained in Table 5.1. The data includes the following variables: hatchery code, hatch date, poult gender, breeder age (wk), truck hatchery start mileage, truck mileage at farm, number of birds found dead on arrival, number of birds killed in transit, number of bird killed at farm, number of birds intentionally destroyed at placement, days 1 through 7 individual mortality and total poults placed. The numbers in Table 5.1 show that in the data set of 3,562 deliveries, there were 1,075 hen poult deliveries (30%) and 2,487 tom poult deliveries (70%). Season code is determined by converting the hatch day to its respective month and then categorized into season (Winter = December to February, Spring = March to May, Summer = June to August, Fall = September to November). Hatch day is determined by converting the hatch date to its respective weekday. Breeder hens are sorted into 3 groups based on stage of lay: "young" during weeks 1 to 7 of egg production, "peak" during weeks 8 to 24, and "old" during weeks 25 to 31. Distance is calculated by taking the difference of truck mileage at farm and truck hatchery start mileage.

The percent of PPM is calculated as the proportion of the sum of the number of birds found dead on arrival, the number of birds killed in transit, the number of birds killed at a farm and the number of birds intentionally destroyed at placement relative to the total number of poults delivered, that is:

$$PPM = \frac{\#\ Dead\ poults\ before\ placement}{\#\ Total\ poults\ delivered} \times\ 100$$

The percent of FWM is calculated as the proportion of the total number of poults that died from days 1 through 7 after placement relative to the total number of poults delivered to a farm, i.e.:

$$FWM = \frac{\#\ Dead\ poults\ from\ day\ 1\ to\ 7\ on\ farm}{\#\ Total\ poults\ delivered} \times 100$$

The percent of CEM is calculated as the sum of pre-placement mortality and first week mortality.

The summary statistics of the mortality rates broken down by each categorical variable are presented in Table 5.2. The mean, standard deviation, minimum value and maximum value for PPM, FWM and CEM are detailed. The hatchery factor is a complex variable, including but not limited to the inclusion of equipment, personnel, standard operating procedures that may vary from hatchery to hatchery and other factors not disclosed in the data. The data indicates that the mean CEM is the highest for hatchery #2 (4.6%) and so is the FWM but not the PPM which is the highest in hatchery #1. The mean CEM was the lowest in hatchery #5 (1.52%) and so is the FWM but the lowest PPMs (0.0%) are recorded in both hatchery #4 and #5. The data also indicate that FWM is higher in all hatcheries than PPM, signaling that hatchery management practices most definitely influence early poult mortality.

The mortality outcomes also vary by season. The CEM was the highest in winter (3.7%) and the lowest in Spring (2.86%). The other two mortality measures exhibit the same patterns but the PPM is always significantly lower than the FWM. These numbers are reflective of the effect of environmental conditions that are more easily controlled in the hatcheries but difficult to control in brooder barns. The data also reveals the fact that the mortality rates vary significantly by the hatch day. For poorly understood reasons, the CEM is the highest on Tuesdays and the lowest on Mondays but the PPM is the highest on Tuesdays (0.16%) but the lowest on Fridays (0.06%). The mortality rates exhibit consistently higher mortality rates for hens than for toms. This is true for all three measured mortality rates. The differences are quite substantial: the CEM for hens is 4.11% whereas for toms only 2.77%.

Finally, the stage of lay factor has also an important influence on mortality rates with FWM always exceeding the PPM, indicating that some genetic factors may not have a significant impact during embryonic development, but can influence survivability and growth in young poults. The data shows that CEM is the highest in poults coming from young hens (3.66%) and the lowest in poults coming from old hens (2.86%). The same pattern is detectable for FWM but not in PPM where lowest mortality is associated with poults from old breeders and the highest with poults from peak breeders. In the rest of this analysis, CEM is utilized as the benchmark for early poult mortality and as such is the focus of most of the discussions.

Regression Model

Data were analyzed using JMP Pro 13 (SAS Institute Inc., Cary, NC) to investigate the impact of various hatchery management variables on early poult mortality. The estimation method was ordinary least squares (OLS). Three separate statistical models were estimated and the results are presented in Table 5.3 (Model 1). They all have the same functional form specified below but they have different dependent variables Y_i^k where k = 1 indicates percent pre-placement mortality, k = 2 percent first-week mortality and k = 3 percent combined early mortality:

$$\Upsilon_i^k = \beta_0 + \sum_{h=1}^{5} \beta_1^h H_i^h + \sum_{s=1}^{3} \beta_2^s S_i^s + \sum_{d=1}^{3} \beta_3^d D_i^d + \beta_4 G_i + \sum_{l=1}^{2} \beta_5^l L_i^l + \beta_6 m_i + \epsilon_i \quad (1)$$

In equation (1), subscript i denotes individual observations, $\beta_0 - \beta_6$ are parameters to be estimated, H_i is the hatchery indicator variable picking up fixed effects of 5 different hatcheries, S_i is the season indicator variable (Fall, Summer, Winter), D_i is the day indicator variable (Monday, Thursday, Friday), G_i is the gender dummy, L_i is the stage of lay (breeder age)

indicator variable (Peak, Young), m_i is the continuous variable measuring distance as previously defined and ϵ_i represents the error of regression with standard (OLS) distributional assumptions.

The construction of multidimensional categorical variables must be explained in detail. Notably, because there are 6 different hatcheries, 5 dummy variables were constructed representing 5 hatcheries, the 6th hatchery has been left out to avoid the problem of perfect multi-collinearity (dummy trap). Therefore, the performance of each of the five included hatcheries (1-5) will be compared relative to the 6th (left-out) hatchery whose performance is bundled in the estimated constant term. Concretely, the hatchery dummy variable H^1 is constructed by setting it equal to 1 if the poults came from hatchery No. 1 and 0 if poults came from any other hatchery. Similarly, the hatchery dummy variable H^2 is constructed as equal to 1 if the poults came from hatchery No. 2 and 0 if poults came from any other hatchery, and so on.

Other multi-level categorical variables were constructed in the similar fashion. For example, there are only 3 seasons dummy variables S_i^s because Spring was left out and the estimated coefficients β_2^s measure the performance of the corresponding season relative to Spring. Similarly, because in commercial turkey hatcheries, hatch is pulled on Mondays, Tuesdays, Thursdays and Fridays, four hatch days were represented by 3 dummy variables indicating Monday, Thursday and Friday. The estimated coefficients β_3^d measure the difference in mortality on Monday, Thursday or Friday all relative to Tuesday. Finally, breeder management in commercial turkey production classifies breeders into 3 general stages of lay: young, peak or old breeder hens. Hence, two dummy variables were always constructed, leaving the third one out to avoid the mentioned perfect collinearity problems. The estimated

coefficient β_5^l always measures the performance of the said breeder hens (in terms of the mortality of their progeny) relative to the left-out group. The exact specification varies depending on the model. Finally, the model includes one simple dummy variable for gender. It is defined as equal to 1 if poults were hens and 0 if they were toms.

In addition to the base model, we also estimate the augmented model that incorporates several interaction effects. The estimation results are presented in Table 5.4 (Model 2). Just like in equation (1), three statistical models with different dependent variables Y_i^k were specified where k = 1 indicates percent pre-placement mortality, k = 2 percent first-week mortality and k = 3 percent combined early mortality:

$$\Upsilon_i^k = \beta_0 + \sum_{h=1}^{5} \beta_1^h H_i^h + \sum_{s=1}^{3} \beta_2^s S_i^s + \sum_{d=1}^{3} \beta_3^d D_i^d + \beta_4 G_i + \sum_{l=1}^{2} \beta_5^l L_i^l + \beta_6 m_i + \sum_{l=1}^{2} \beta_7^l L_i^l G_i + \beta_8 G_i m_i + \epsilon_i \quad (2)$$

In equation (2), all parameters $\beta_0 - \beta_6$ and variables (H_i, S_i, D_i, G_i, L_i, m_i) are the same as in equation (1), where subscript i denotes individual observations and ϵ_i represents the error of regression with standard (OLS) distributional assumptions. The construction of multidimensional categorical variables is also identical to equation (1). However, in equation (2) cross-product effects between variables G_i and L_i^l (gender and stage of lay) and G_i and m_i (gender and distance traveled) are added to the original model specification. Model 2 specifies the interactions between tom poults and different stage of lay breeders (old and peak) and tom poults and distance. The interpretation of the estimated coefficients associated with the product of two dummy variables is that they measure the effect of peak (and old) stage of lay on tom poults that is not present for hen poults. The interpretation of the cross-product coefficients

between the gender dummy variable and distance measures the differential impact of distance traveled between tom and hen poults.

RESULTS AND DISCUSSION

Regression Analysis

The results of the OLS model estimation of our base model in equation (1) are reported in Table 5.3. In our discussions, we focus mainly on the CEM (combined early mortality) results because they are the most statistically significant and can also be used to explain the results of the PPM (pre-placement mortality) and FWM (first week mortality) in a similar fashion. The first independent variable in the model is hatchery, where hatchery #1 and hatchery #2 perform worse than the comparison hatchery #6. H^1 and H^2 had significantly higher mortality rate than H^6 by 0.375% and 1.901%, respectively. The remaining hatchery coefficients are not statistically different from zero indicating that the CEM of H^3, H^4 and H^5 are statistically indistinguishable from the H^6 (left-out).

Several researchers found that that mortality rates varied among different hatcheries [5, 18]. Incubation has multifactorial effects on embryonic development and hatch success. Hatcheries and hatchery personnel are responsible for accounting for a variety of factors which produce a spectrum of egg sizes and quality. Management practices during incubation are used to control the rate and success of embryonic development. These practices include egg storage [19], incubation temperature and humidity profiles, and adequate turning and ventilation throughout incubation [20]. Additional factors to consider are the age and type of equipment used (incubator and hatchers) and personnel efficiency and welfare to reduce stress. One example demonstrated by Kingston (1979) found that when chicks were held in the incubator

for 24 hours after hatching (simulating dehydration stress), there was a 12.9% mortality rate in chicks at 10 days of age.

In this study, there was no additional information provided about hatchery conditions or management practices, so no conclusions can be drawn about effects on performance, except that mortality varies among poults from different hatcheries. Further investigation is needed in this type of analysis, including more hatchery information to elucidate their effects on early poult mortality and turkey performance.

The next categorical variable in the model is the season. The results show that summer and winter have worse performance relative to spring. Summer has significantly higher mortality by 0.333% and winter has significantly higher mortality by 0.973%. Fall mortality is not statistically different from spring. Seasonality of the early poult mortality is caused by weather. The temperature and humidity extremes experienced during winter and summer could place heat or cold stress on poults thereby increasing early poult mortality rates [22]. Growth depression due to brooding temperature is observed during the first week of life when birds are exposures to high or low environmental temperatures [23]. Humidity should be controlled by ventilation [24], where proper ventilation maintains good air quality, as well as to promote efficient operation of the cooling and heating systems [25]. Control of environmental conditions during transportation and brooding, will minimize the deleterious effects of temperature extremes on early poult mortality.

The next indicator in our model is the hatch day. Mortality is higher by 0.357% on Fridays compared to mortality on Tuesdays and there are no statistically significant differences in mortality between Monday or Thursday hatch days compared to Tuesdays. It is hypothesized that less care is taken at the hatcheries on Friday, the last day of the week, as personnel rush

and attention to detail decreases. Likewise, poults delivered to farms on Fridays may suffer from grower neglect over the weekend, leaving poult vulnerable to mortality due to suboptimal environmental conditions or management. Further investigation is needed to elucidate the effects on hatch day on early poult mortality.

The impact of gender on early poult mortality is significant. The results show that hen poults have 1.112% greater early mortality than tom poults. High mortality in tom poults may be a result of genetic strain [26]. This is in agreement with Carver et al. (2002) who found that at 7 days post-placement, toms had increased cumulative mortality compared to hens. In the current study, 70% of customer invoices were toms, 30% were hens and there was increased mortality for FWM and CEM in toms versus hens.

Breeder stage of lay was also found to significantly explain the behavior of mortality rates. Poults from peak and young stage breeder hens had increased mortality, by 0.268% and 0.912%, respectively, compared to poults from old stage breeder hens. Our results are consistent with the findings of Vieira & Moran (1999). Increased mortality in poults from young stage breeders may be related to reduced embryo development [29, 30] and the direct relationship between the nutrients provided by the yolk sac and the subsequent performance of hatchlings [28]. Eggs from young breeder hens are also smaller and produce smaller hatchlings [31] that are more susceptible to decreased performance and mortality.

We were also interested in measuring the importance of distance that turkey poults travel from hatchery to the farm on explaining early mortality. The results show that increased distance significantly impacted CEM. For each additional mile traveled from the hatchery to the farm, mortality increased by 0.001%. The main factors affecting day-old birds during transportation are temperature, humidity, vibration, sound and delay of feed and water intake

[7]. Continuous noise, vibration, and movement are stressors arising during transportation, which can lead to fatigue due to depletion of glycogen reserves and mortality [32]. Poor ventilation can lead to increased body temperature and improper heat loss by evaporation [7]. Many authors have reported an inverse relationship between transportation duration and subsequent growth performance [33–38] so our findings are supportive of all those earlier results.

The results pertaining to the CEM in Model 1 can largely be described by the FWM, as most mortality differences across stratifications of the data set occurred during the first week of production (for hatchery, season, hatch day, gender, stage of lay, and distance). The only significant differences in PPM were between the two hatcheries: H^2 compared to H^6 (-0.101%), the two seasons: winter compared to spring (0.204%), and the distance (0.001% increase in mortality per mile).

In Table 5.4, we present the estimation results of our augmented model as specified in equation (2). In Model 2, the intercepts represent the means of various mortality rates (PPM, FWM and CEM) for the poults with code 0 on all categorical variables included. In this case, the intercept measures the mean mortality for poults from hatchery #6, spring, Tuesday, hens, young breeders, and 0 miles from the hatchery. Like in the previous model, the parameters belonging to single dummy variables that are not interacted with “Toms” show the controlled mortality (say CEM) difference with the related reference category. For example, H^2 coefficient measures the difference in mean CEM between hatchery #2 and reference hatchery #6 (1.876% higher) after controlling for all other variables. The same interpretation applies to season and hatch day dummies.

Because of interactions with “Toms”, stage of lay dummies coefficients relate to the mean CEM of hens. For instance, the estimated “Old” coefficient denotes the mean CEM difference between hen poults from old and young stage breeders. Hen poults from old breeders have 0.916% lower mortality than hen poults from young breeders. Similarly, hen poults from peak breeders have 0.646% lower mortality than hen poults from young breeders. By the same logic as above, due to the commutative law for multiplication, the single “Toms” dummy coefficient measures the difference in CEM between hens and toms poults coming from only young breeders. This is because “Young” is the reference variable in the stage of lay category which is interacted with “Toms”. So, tom poults from young breeders have 1.169% lower mortality then hen poults coming from young breeders.

To investigate if CEM varies among tom and hen poults from different breeder stage of lay, the gender and stage of lay dummy variables are interacted. The reference variables were hens and young breeder age. The mean CEM of each combination of gender and stage of lay, presented in Figure 5.1, were calculated for Model 2 utilizing equation (2) and the results from Table 5.4. The intercept from Table 5.4 (β_0= 3.530%) can be interpreted as the estimate of the CEM of hen poults from young breeders, that traveled 0 miles from the hatchery to the brooder farm. The remaining two points on the hens line are obtained as follows: the CEM of hens from peak breeders is 2.884% ($\beta_0 + \beta_5^{Peak}$) and of hens from old breeders is 2.614% ($\beta_0 + \beta_5^{Old}$). Similarly, the points on the toms line are obtained as follows: the CEM of tom poults from young breeders is 2.361% ($\beta_0 + \beta_4$), toms from peak breeders 1.299% ($\beta_0 + \beta_4 + \beta_5^{Peak} + \beta_7^{Toms*Peak}$) and toms from old breeders is 0.646% ($\beta_0 + \beta_4 + \beta_5^{Old} + \beta_7^{Toms*Old}$). Therefore, as clearly seen in the graph, the mortality gender gap between hen and tom poults is widening as we move from young to peak to old breeder hens because the gender

difference in mortality rates is increasing from 1.169% for young breeder to 1.585% for peak breeders to 1.968% for old breeders.

An alternative, equally interesting, interpretation of the same results is that the effect of the age of breeder hens, moving from young to peak to old, is stronger for tom poults than for hen poults. As seen from Figure 5.1, this is because the mortality for hen poults drops from 3.53% to 2.884% (0.646 percentage points drop between young an peak breeders) and then to 2.614% (0.916 percentage points drop between young and old breeders), whereas the mortality for tom poults is more pronounced from 2.361% to 1.299% (1.062 percentage points difference between young and peek breeders) and then to 0.646% (1.715 percentage points difference between young and old).

For completeness and better understanding of the results, it is worth emphasizing that the computed reduction in mortality for the hen poults moving from young to peak breeder hens (-0.646%) is exactly equal to the estimated stage of lay “Peak” coefficient for CEM in Table 5.4. Likewise, the computed reduction in mortality for hen poults moving from peak to old breeders (-0.916%) is exactly equal to the estimated stage of lay “Old” old coefficient for CEM in the same table. Finally, the interpretation of the interaction coefficients themselves is also interesting. For example, sticking with the definition of the gender gap in mortality as the hen poults mortality minus the tom poults mortality, then “Tom*Old” coefficient measures the difference in gender gaps between peak and young breeders because the gender gap for peak breeders (= -1.585 from Figure 5.1) is the sum of the gender gap for young breeder (coefficient on "Toms"= -1.169) plus the coefficient on "Toms*Old" (= -.0799).

Finally, the continuous variable “Distance” has been interacted with the dummy variable for gender. In this segment we can look at two sets of results. First, we can look at the

differential impact of miles travelled between tom and hen poults, assuming they both come from young breeders. Secondly, we can fix the distance travelled and look at the differential impact of distance travelled between tom and hen poults by varying the stage of lay variable from young to peak to old breeders.

The first set of results can be elegantly summarized in Figure 5.2. We plot the predicted CEM of tom and hen poults over distance traveled with y axis representing the predicted CEM and x axis represents the distance traveled. Because the reference gender variable is hens, the stand-alone "Toms" coefficient predicts the CEM difference between tom and hen poults when they travel 0 miles. For earlier explained reasons, this coefficient also pertains to poults coming from young breeders (as well as poults from hatchery #6, spring and Tuesday). At 0 miles traveled, tom poults from young breeders have an estimated 1.169% lower mortality than hen poults from young breeders. The predictions of CEM for different sexes is obtained as follows and are based on the results from Table 5.4:

$$y_{hens} = \beta_0 + \beta_6 x = 3.530 + 0.001x \quad (3)$$

$$y_{toms} = (\beta_0 + \beta_4) + (\beta_6 + \beta_8)x = 2.361 + 0.002x \quad (4)$$

As seen from Figure 5.2, the hen poults mortality is greater at distances traveled up to approximately 1,200 miles, whereas tom poults mortality is greater at distances beyond 1,200 miles. The obtained results are in line with Carver et al. (2002) who reported that tom poult mortality increased with every additional hour increase in travel time.

The second set of results are depicted in Figure 5.3 for a fixed distance set at the maximum distance traveled recorded in the data set (1,518 miles; see Table 5.1). To obtain the predicted CEM for tom and hen poults coming from peak and old breeders formulae (3) and (4) need to be modified as follows:

$$y_{hens} = \beta_0 + \beta_5^l + \beta_6 x \quad (5)$$

$$y_{toms} = (\beta_0 + \beta_4 + \beta_5^l + \beta_7^l) + (\beta_6 + \beta_8)x \quad (6)$$

It is easy to see that the superscript "l" in β_5 and β_7 coefficients will assume the value of "peak" coefficients in formulae for predicting the CEM for hens (5) and toms (6) coming from peak breeders and will assume the value of "old" coefficients in formulae for predicting the CEM for hens (5) and toms (6) coming from old breeders.

As seen from Figure 5.3, at a fixed and long distance travelled tom poults coming from young breeders exhibit higher mortalities then hen poults from young breeders. For poults coming from peak breeders the mortality lines become reversed and for poults coming from old breeders it becomes obvious that the predicted CEM for hen poults exceed that for tom poults. Lastly, regardless of distance traveled, poults from young breeders have significantly higher mortality than poults from old breeders, and nominally higher mortality than peak breeders at small distances traveled. So, CEM decreases as breeder age increases. Some researchers have found no differences in mortality due to breeder age [39, 40], while others have reported greater mortality in progeny from old breeders [41]. However, most researchers agree that higher mortality rates are observed in young breeder flocks compared to peak and old breeder flocks [28, 42, 43].

Cost Estimates

The estimated coefficients from the base model in Table 5.3 are used to perform some counterfactual analyses to determine how economically meaningful are the obtained results. In particular, whether some prescriptions for changing hatchery management practices would make economic sense. We focus on the effects of the stage of lay on increased mortality. All

counterfactual calculations are based on two plausible assumptions: (a) the average number of poults per truck load delivery is 17,000, and (b) the monetary cost of one poult is $2.00.

The first counterfactual experiment is designed as follows: take all 694 deliveries of poults coming from old breeders from Table 5.1 (observed in the dataset) and make them hypothetically young. Because there is n=3,562 deliveries, 694 deliveries of old breeder poults equals 19.5%. Next, the percentage of old poults in the data is applied to the assumed truck load of 17,000 poults, resulting in an estimated 3,312 poults from old breeders per truck delivery. If these poults became young, they would have higher mortality by 0.912% compared to poults coming from old breeders (see the estimated coefficient for "Young" in Table 5.3), roughly 30 additional poults per truck load would die with a total increase in cost of approximately $60. So, the hypothetical increase in the cost resulting from replacing poults from old breeders with poults from young breeders is $60 per truck load delivery.

The next counterfactual experiment is run as follows: take 694 old breeders poults and make them hypothetically peak. Again, this will give us 3,312 poults per truck delivery (19.5%*17,000). If this old poults became peak, they would have higher mortality by 0.268% compared to poults coming from old breeders (see the coefficient for "peak" in Table 5.3) and 9 additional mortalities per truck delivery would result. The total cost increase of replacing poults from old breeders with poults from peak breeders is $18 per truck delivery.

Using the same methodology and assumptions as before, the percentage of peak poults in the data set per assumed truck load can be made artificially young (58.5%*17,000) resulting in an estimated 9,937 poults from peak breeders per truck delivery. For this we need to estimate another model where the left-out stage of lay category is "Peak." Those results show (not presented here but available from author upon request) that young breeders produce poults

with 0.643% higher mortality than peak breeders. Applying the mortality rate for poults from young breeders to the estimated poults from peak breeders results in approximately 64 additional mortality. The total cost increase is $128 per truck delivery when replacing poults from peak breeders with poults from young breeders.

The importance of stage of lay on mortality reduction and the associated economic benefits were also ascertained utilizing the same methodology and assumptions as those discussed above. If the percentage of poults from peak breeders in the data set per assumed truck load are artificially made old (58.5%*17,000), then the estimated number of poults from peak breeders per truck delivery is 9,937. Because the mortality rate for old breeders is 0.268% lower than for peak breeder, switching old breeder poults to peak breeder poults will result in 27 less mortality. Therefore, the total monetary benefit of replacing poults from peak breeder with poults from old breeders is $54 per truck delivery.

Next, poults from young breeders can be artificially replaced by poults from old breeders. The percentage of poults from young breeders in the data per assumed truck load (22.1%*17,000) is artificially made old to estimate 3,751 poults from young breeders per truck delivery. When the mortality rate for old (-0.912%) is applied to the estimated number of poults from young breeders, the mortality is lowered by 34 poults. Therefore, the total cost savings of replacing poults from young breeders with poults from old breeders is $68 per truck delivery.

Lastly, if the percentage of poults from young breeders in the data set per assumed delivery size (22.1%*17,000) is artificially made peak, the estimated number of poults from young breeders is 3,751. When the mortality rate for peak (-0.643%) is applied to this estimate, the mortality decreases by 24 birds. Therefore, benefit of replacing poults from young breeders with poults from peak breeders is $48 per delivery.

The estimated distance coefficient can also be used to perform a counterfactual analysis to determine if the distance between hatcheries and brooder barns impacts the economic costs associated with increased early mortality in turkey poults. Using the same assumptions as in the stage of lay calculations, the increased costs associated with each additional 100 miles traveled is determined. The coefficient for CEM from Table 5.3 for 100 miles (0.001%*100 = 0.1%) is applied to the assumed truck load size, resulting in an extra 17 mortality for each additional 100 miles traveled. Therefore, the total cost increase of each additional 100 miles traveled is $34 per truck delivery. Similarly, the total cost increase due to additional 500 miles traveled would be $170 per truck delivery.

To the best of our knowledge, this is the first economic investigation into the extra costs and cost savings associated with specific poult development and hatchery factors in the turkey industry. Contrary to reported literature [44, 45], in the current data set, the cost savings estimations do not include factors such as feed costs, housing costs, labor and equipment costs, or processing costs accrued over the production period. If poults from young stage breeders were to survive, past 7 days of age, the literature suggests that their growth [40, 46, 47][11], [40], [47] and yield [48] will be stunted at market age resulting in inflated profit losses. This provides further evidence that identifying cost-saving centers during incubation and/or early production can have large impacts on performance and profitability at market age.

CONCLUSIONS AND APPLICATIONS

1. Young breeder age and increased distance traveled from the hatchery to brooder barns, are controllable factors that act as constraints on turkey poult performance and impact tom and hen poults uniquely. In this study, we investigate factors that impact early mortality in turkeys poults. We use field data collected from six US hatcheries, for a total of 3,562

shipments from various breeder flocks delivered to a total of 1,051 customers over a 22-month period.

2. Our most important results indicate that first, the cumulative early mortality is significantly related to a specific hatchery, season, hatch day, gender, breeder age and the distance traveled from the hatchery to the farm and, second, that certain modifications of management practices could reduce early poult mortality, leading to cost savings and increased profitability. Strategic implementation of management changes can lead to optimized poult quality and reduced early poult mortality. Cost increases due to additional mortality associated with poults from young breeder hens ranged from $60 to $128 per average truck delivery. We also found that for every additional 500 miles traveled from hatchery to brooder barns the cost increases by $170 per average truck delivery.
3. The main drawback of this study is the fact that the grow-out performance data for these turkey poults was not available. Therefore, the economic cost and benefits could only be calculated based on the first seven days mortality data, i.e., we don't know how various hatchery management practices and poult quality attributes would have been reflected further down the production channel via the possible differences in feed conversion, aggregate live weight produced, processing yields, etc. We believe that this study eloquently illustrates that data collection (from poultry companies and growers) as well as data management and analysis are becoming crucial factors in streamlining the process for receiving real-time information, necessary for effective management of a poultry enterprise. Concretely, by identifying which factors most effect early poult mortality and subsequent grow-out performance, the poultry industry can adjust management practices to improve animal welfare, thereby improving overall flock health and maximizing profits.

Table 5.1. Description of the data set

Variable	Unit	Range	Average*
PPM	%	0 – 36.95	0.11
FWM	%	0.06 – 40.60	3.07
CEM	%	0.06 – 41.45	3.18
Hatchery	Categorical		
		H^1	688
		H^2	45
		H^3	16
		H^4	32
		H^5	24
		H^6	2,757
Season	Categorical		
		Fall	604
		Spring	1,088
		Summer	1,149
		Winter	721
Hatch Day	Categorical		
		Monday	845
		Tuesday	1,315
		Thursday	596
		Friday	803
Gender	Categorical		
		Hen	1,075
		Tom	2,487
Stage of Lay	Categorical		
		Old	694
		Peak	2,082
		Young	786
Distance	Miles	6 – 1,518	462

*Entries for categorical variables indicate the number of observations

Table 5.2. Variation in mortality rates by hatchery and poult characteristics.

	PPM (%)				FWM (%)				CEM (%)			
	Mean	**STD**	**Min**	**Max**	**Mean**	**STD**	**Min**	**Max**	**Mean**	**STD**	**Min**	**Max**
TOTAL DATA	0.11	0.93	0.00	36.95	3.07	2.79	0.06	40.60	3.18	3.01	0.06	41.45
HATCHERY												
1	0.13	0.30	0.00	3.61	3.78	3.41	0.20	17.06	3.92	3.53	0.20	17.30
2	0.11	0.15	0.00	0.55	4.49	4.20	0.38	16.41	4.60	4.21	0.44	16.46
3	0.08	0.08	0.00	0.19	3.25	1.20	0.93	4.46	3.33	1.19	0.95	4.57
4	0.00	0.00	0.00	0.00	2.95	2.20	1.01	7.07	2.95	2.20	1.01	7.07
5	0.00	0.00	0.00	0.00	1.55	0.82	0.78	3.76	1.52	0.82	0.78	3.76
6	0.11	1.05	0.00	36.45	2.88	2.57	0.06	40.60	2.99	2.83	0.06	41.45
SEASON												
Fall	0.09	0.11	0.00	0.55	3.00	2.63	0.15	20.40	3.06	2.67	0.15	20.45
Spring	0.07	0.09	0.00	1.23	2.79	2.26	0.20	34.50	2.86	2.26	0.20	34.50
Summer	0.08	0.25	0.00	5.19	3.14	2.79	0.20	16.94	3.21	2.84	0.20	17.25
Winter	0.24	2.04	0.00	36.95	3.46	3.53	0.06	40.60	3.70	4.20	0.06	41.45
HATCH DAY												
Monday	0.11	0.32	0.00	3.61	2.83	2.28	0.06	28.60	2.94	2.39	0.06	28.60
Tuesday	0.16	1.50	0.00	36.95	3.26	3.12	0.20	34.50	3.42	3.53	0.20	41.45
Thursday	0.07	0.22	0.00	5.19	3.00	2.88	0.38	40.60	3.07	2.91	0.44	40.60
Friday	0.06	0.22	0.00	5.10	3.04	2.63	0.20	15.95	3.11	2.68	0.20	16.82
GENDER												
Hen	0.19	1.62	0.00	36.95	3.92	2.81	0.26	16.94	4.11	3.29	0.26	41.45
Tom	0.07	0.33	0.00	11.61	2.70	2.71	0.06	40.60	2.77	2.78	0.06	40.60
STAGE OF LAY												
Old	0.08	0.15	0.00	2.14	2.79	2.63	0.20	16.94	2.86	2.68	0.20	17.25
Peak	0.12	1.18	0.00	36.95	2.98	2.73	0.06	34.50	3.10	3.04	0.06	41.45
Young	0.10	0.52	0.00	11.61	3.56	3.05	0.15	40.60	3.66	3.13	0.15	40.60

Table 5.3. OLS Regression results (Base model #1):

	PPM (%)			FWM (%)			CEM (%)		
	Estimate	**Std Error**	**T Ratio**	**Estimate**	**Std Error**	**T Ratio**	**Estimate**	**Std Error**	**T Ratio**
INTERCEPT	-0.054	0.05	-0.99	**1.559**	0.16	10.04**	**1.505**	0.17	9.04**
HATCHERY									
1	**-0.101**	0.05	-2.16*	**0.476**	0.13	3.58**	**0.375**	0.14	2.63**
2	0.033	0.14	0.23	**1.868**	0.40	4.66**	**1.901**	0.43	4.42**
3	0.012	0.23	0.05	0.105	0.67	0.16	0.119	0.72	0.17
4	-0.126	0.17	-0.75	-0.097	0.48	-0.2	-0.223	0.52	-0.43
5	0.01	0.19	0.05	-0.865	0.55	-1.57	-0.855	0.59	-1.45
SEASON									
Fall	0.013	0.05	0.27	0.1	0.14	0.72	0.113	0.15	0.76
Summer	0.011	0.04	0.28	**0.322**	0.11	2.82**	**0.333**	0.12	2.72**
Winter	**0.204**	0.05	4.46**	**0.769**	0.13	5.87**	**0.973**	0.14	6.93**
HATCH DAY									
Mon	-0.015	0.04	-0.34	-0.076	0.12	-0.63	-0.091	0.13	-0.7
Thu	-0.069	0.05	-1.47	-0.119	0.13	-0.88	-0.188	0.14	-1.31
Fri	-0.03	0.05	-0.67	**0.387**	0.13	3.03**	**0.357**	0.14	2.61**
GENDER									
Hens	0.065	0.04	1.76	**1.048**	0.11	9.98**	**1.112**	0.11	9.88**
STAGE OF LAY									
Peak	0.054	0.04	1.32	0.214	0.12	1.83	**0.268**	0.13	2.14*
Young	0.041	0.05	0.85	**0.87**	0.14	6.25**	**0.912**	0.15	6.11**
DISTANCE	**0.0002**	0.00004	4.88**	**0.001**	0.0001	7.72**	**0.001**	0.0001	8.79**

*P<0.05, **P<0.01, R^2 = 0.02

Table 5.4. OLS regression results (Augmented model #2):

	PPM (%)			FWM (%)			CEM (%)		
	Estimate	**Std Error**	**T Ratio**	**Estimate**	**Std Error**	**T Ratio**	**Estimate**	**Std Error**	**T Ratio**
INTERCEPT	0.048	0.059	0.81	**3.483**	0.168	20.69**	**3.530**	0.181	19.54**
HATCHERY									
1	-0.091	0.047	-1.93	**0.326**	0.135	2.42*	0.235	0.144	1.62
2	0.038	0.140	0.27	**1.837**	0.399	4.60**	**1.876**	0.428	4.38**
3	0.026	0.234	0.11	-0.039	0.666	-0.06	-0.011	0.715	-0.02
4	-0.135	0.168	-0.80	-0.162	0.478	-0.34	-0.297	0.513	-0.58
5	0.001	0.193	0.00	-0.634	0.549	-1.15	-0.633	0.590	-1.07
SEASON									
Fall	0.012	0.048	0.24	0.142	0.138	1.03	0.154	0.148	1.04
Summer	0.014	0.040	0.35	**0.298**	0.114	2.62**	**0.312**	0.122	2.56*
Winter	**0.2**	0.046	4.37**	**0.799**	0.130	6.13**	**0.999**	0.140	7.14**
HATCH DAY									
Mon	-0.013	0.042	-0.30	-0.087	0.120	-0.72	-0.100	0.129	-0.77
Thu	-0.070	0.047	-1.48	-0.104	0.134	-0.78	-0.174	0.143	-1.21
Fri	-0.030	0.045	-0.67	**0.387**	0.127	3.04**	**0.356**	0.136	2.61**
GENDER									
Toms	-0.061	0.037	-1.65	**-1.109**	0.105	-10.57**	**-1.169**	0.113	-10.39**
STAGE OF LAY									
Old	-0.038	0.049	-0.78	**-0.878**	0.139	-6.34**	**-0.916**	0.149	-6.16**
Peak	0.016	0.039	0.40	**-0.662**	0.111	-5.96**	**-0.646**	0.119	-5.42**
DISTANCE	**0.0002**	0.00005	4.2**	**0.001**	0.0001	9.56**	**0.001**	0.0001	10.28**
Toms*Old	-0.069	0.106	-0.65	**-0.731**	0.303	-2.41*	**-0.799**	0.325	-2.46*
Toms*Peak	**-0.171**	0.086	-1.98*	-0.246	0.245	-1.01	-0.416	0.263	-1.58
Toms*Distance	-0.00006	0.0001	-0.80	**0.001**	0.0002	6.30**	**0.001**	0.0002	5.60**

*P<0.05, **P<0.01, R^2 = 0.02

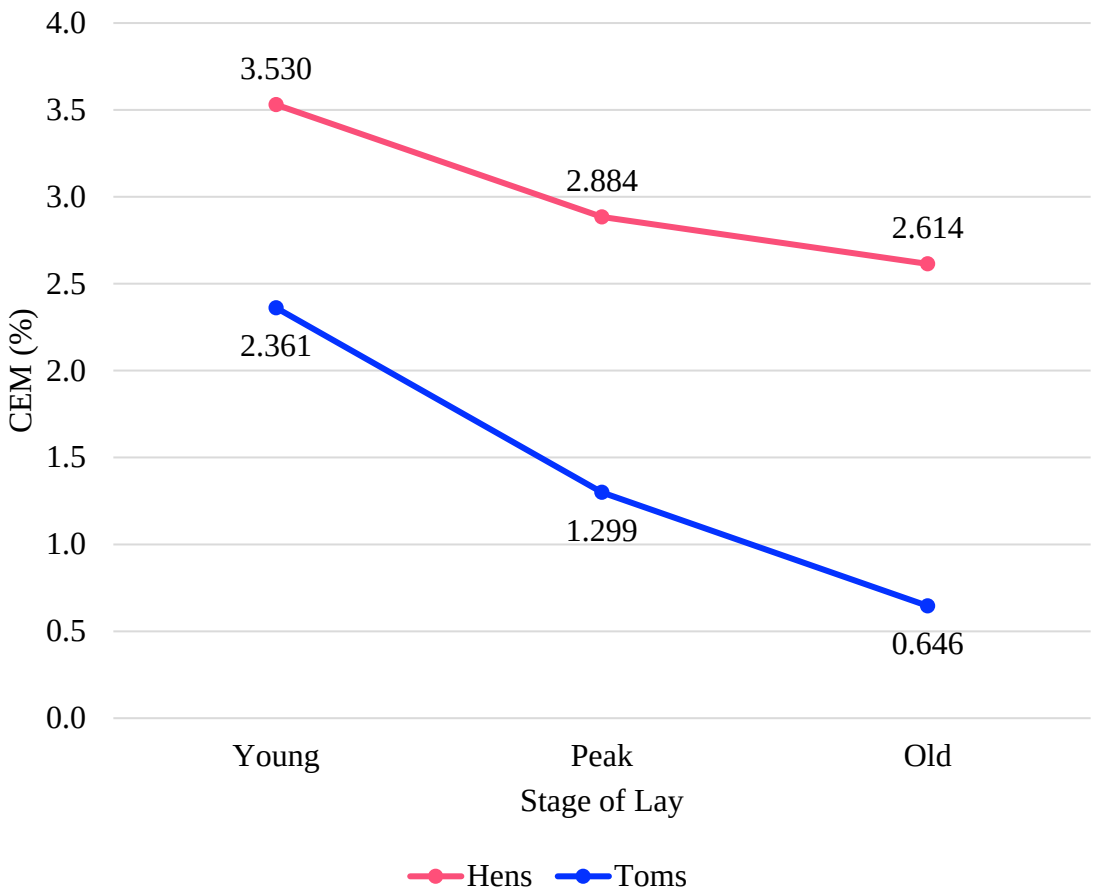

Figure 5.1. Predicted CEM (%) for tom and hen poults from different stage of lay breeders that have traveled 0 miles.

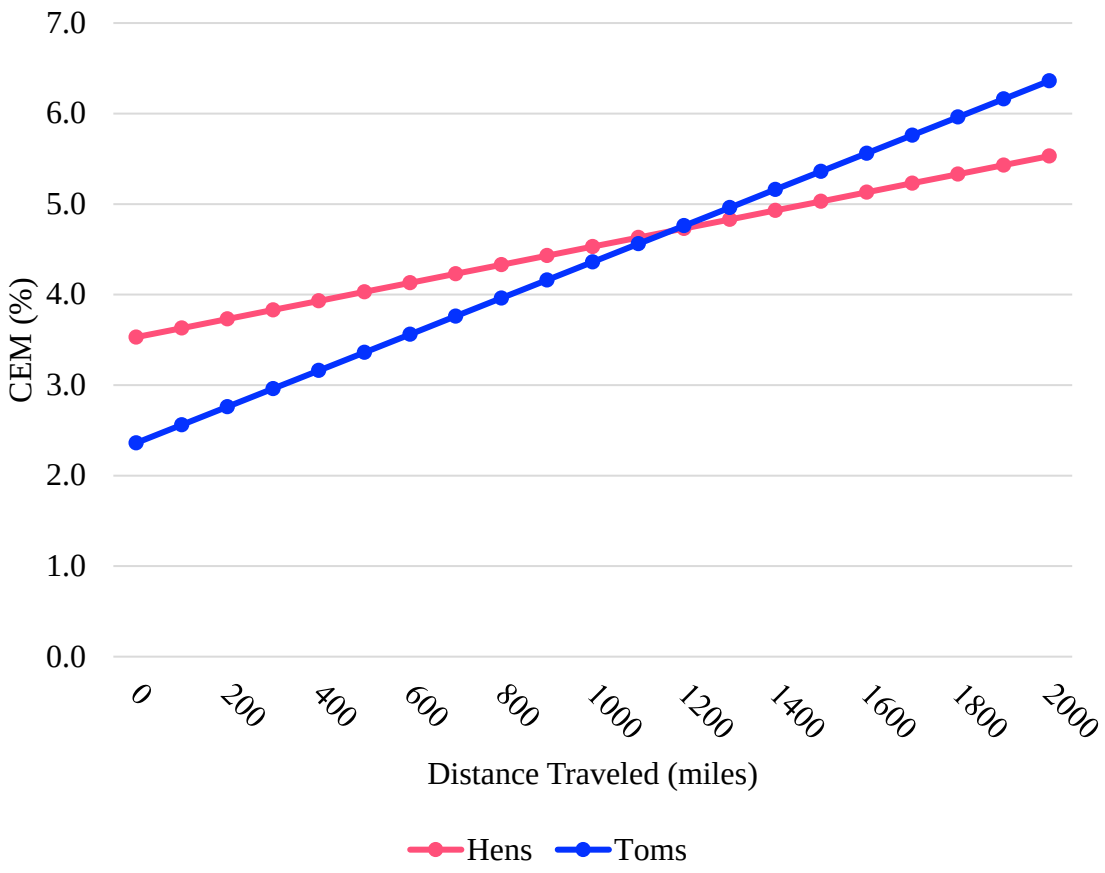

Figure 5.2. The predicted mean CEM (%) for tom and hen poults from young breeders by distance traveled

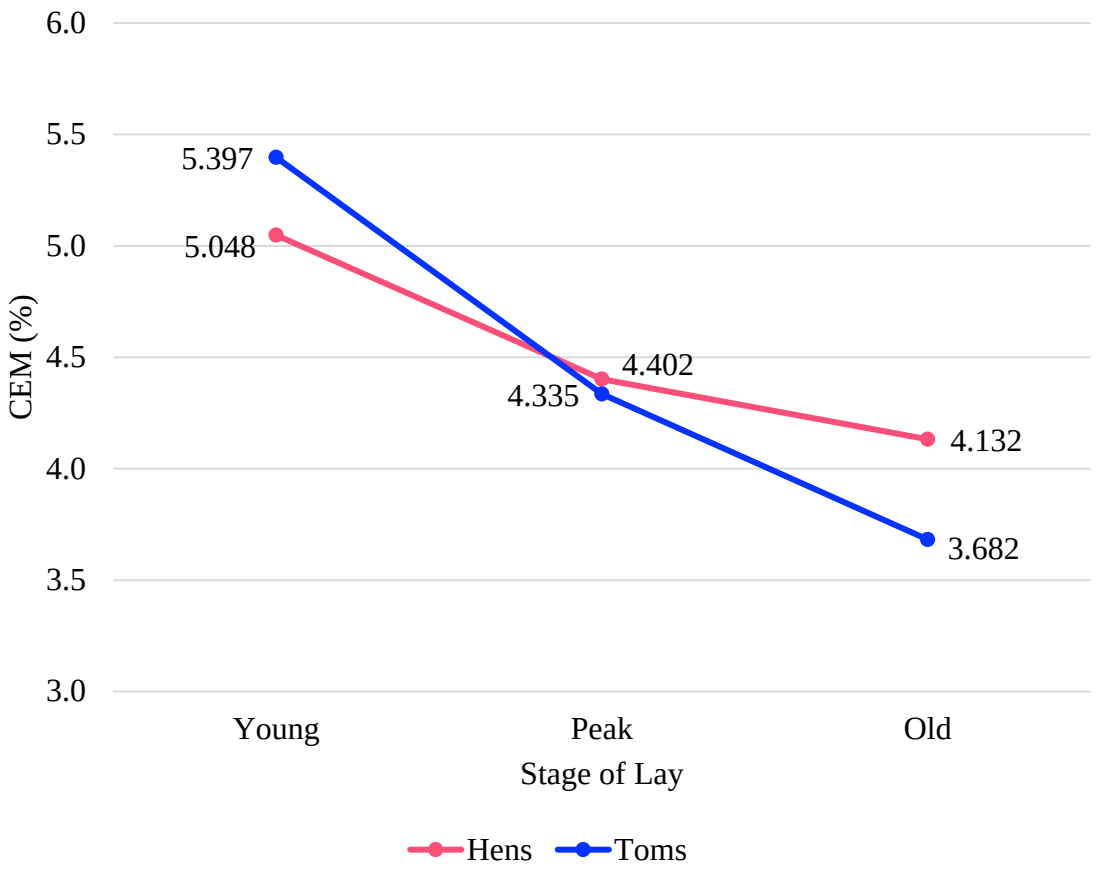

Figure 5.3. Predicted CEM (%) for tom and hen poults from different stages of lay breeders that have traveled 1,518 miles.

REFERENCES

1. S. Schultz-Cherry *et al.*, “Identifying agent(s) associated with poult enteritis mortality syndrome : Importance of the thymus,” *Avian Dis.*, vol. 44, no. 2, pp. 256–265, 2000.

2. M. S. Lilburn, “Practical aspects of early nutrition for poultry,” *J. Appl. Poult. Res.*, vol. 7, no. 4, pp. 420–424, 1998.

3. USDA-ERS, “Turkey Sector: Background & Statistics,” *Trending Topics*, 2017. [Online]. Available: https://www.ers.usda.gov/newsroom/trending-topics/turkey-sector-background-statistics/. [Accessed: 04-Dec-2017].

4. “Humane Farm Animal Care Standards for Turkeys,” Herndon, VA, 2013.

5. H. Yassin, A. G. J. Velthuis, M. Boerjan, J. Van Riel, and R. B. M. Huirne, “Field study on broiler eggs hatchability,” *Poult. Sci.*, vol. 87, no. 11, pp. 2408–2417, 2008.

6. E. Decuypere and V. Bruggeman, “The endocrine interface of environmental and egg factors affecting chick quality,” *Poult. Sci.*, vol. 86, pp. 1037–1042, 2007.

7. H. Bergoug *et al.*, “Effect of pre-incubation and incubation conditions on hatchability, hatch time and hatch window, and effect of post-hatch handling on chick quality at placement,” *Worlds. Poult. Sci. J.*, vol. 69, no. 02, pp. 313–334, 2013.

8. T. J. Applegate, J. J. Dibner, M. L. Kitchell, Z. Uni, and M. S. Lilburn, “Effect of turkey (Meleagridis gallopavo) breeder hen age and egg size on poult development. 2. Intestinal villus growth, enterocyte migration and proliferation of the turkey poult,” *Comp. Biochem. Physiol. - B Biochem. Mol. Biol.*, vol. 124, no. 4, pp. 381–389, 1999.

9. S. L. Vieira and E. T. Moran, “Eggs and chicks from broiler breeders of extremely different age,” *J. Appl. Poult. Res.*, vol. 7, no. 4, pp. 372–376, 1998.

10. E. E. O’Dea *et al.*, “Investigating the eggshell conductance and embryonic metabolism of modern and unselected domestic avian genetic strains at two flock ages,” *Poult. Sci.*, vol. 83, pp. 2059–2070, 2004.

11. V. L. Christensen, M. J. Wineland, G. M. Fasenko, and W. E. Donaldson, “Egg storage effects on plasma glucose and supply and demand tissue glycogen

concentrations of broiler embryos," *Poult. Sci.*, vol. 80, no. 12, pp. 1729–1735, 2001.

12. J. A. Hamidu *et al.*, "The effect of broiler breeder genetic strain and parent flock age on eggshell conductance and embryonic metabolism," *Poult. Sci.*, vol. 86, no. 11, pp. 2420–2432, 2007.

13. A. Lourens, H. van den Brand, M. J. W. Heetkamp, R. Meijerhof, and B. Kemp, "Effects of eggshell temperature and oxygen concentration on embryo growth and metabolism during incubation," *Poult. Sci.*, vol. 86, no. 10, pp. 2194–2199, 2007.

14. A. Nangsuay *et al.*, "Effects of breeder age, broiler strain, and eggshell temperature on development and physiological status of embryos and hatchlings," *Poult. Sci.*, vol. 95, pp. 1666–1679, 2016.

15. R. Molenaar, I. Reijrink, R. Meijerhof, and H. Van den Brand, "Meeting embryonic requirements of broilers throughout incubation: a review," *Brazilian J. Poult. Sci.*, vol. 12, no. 3, pp. 137–148, 2010.

16. N. Imaeda, "Influence of the stocking density and rearing season on incidence of sudden death syndrome in broiler chickens," *Poult. Sci.*, vol. 79, pp. 201–204, 2000.

17. G. C. Chou, D. D. Jiang, and Y. P. Hung, "Risk factors for cumulative mortality in broiler chicken flocks in the first week of life in Taiwan," *Br. Poult. Sci.*, vol. 45, no. 5, pp. 573–577, 2004.

18. B. T. Heier, H. R. Hogåsen, and J. Jarp, "Factors associated with mortality in Norwegian broiler flocks," *Prev. Vet. Med.*, vol. 53, pp. 147–158, 2002.

19. C. Careghi, K. Tona, O. Onagbesan, J. Buyse, E. Decuypere, and V. Bruggeman, "The effects of the spread of hatch and interaction with delayed feed access after hatch on broiler performance until seven days of age," *Poult. Sci.*, vol. 84, pp. 1314–1320, 2005.

20. K. Tona *et al.*, "Day-old chick auality: relationship to hatching egg quality, adequate incubation practice and prediction of broiler performance," *Avian Poult. Biol. Rev.*, vol. 16, no. 2, pp. 109–119, 2005.

21. D. J. Kingston, "Some hatchery factors involved in early chick mortality," *Aust. Vet. J.*, vol. 55, no. 9, pp. 418–421, 1979.

22. R. A. Ernst, W. W. Weathers, and J. Smith, “Effects of heat stress on day-old broiler chicks,” *Poult. Sci.*, vol. 63, pp. 1719–1721, 1984.

23. C. W. van der Pol, I. A. M. van Roovert-Reijrink, C. M. Maatjens, H. van den Brand, and R. Molenaar, “Effect of relative humidity during incubation at a set eggshell temperature and brooding temperature posthatch on embryonic mortality and chick quality,” *Poult. Sci.*, vol. 92, no. 8, pp. 2145–2155, 2013.

24. G. Corkery, S. Ward, Colum Kenny, and P. Hemmingway, “Monitoring environmental parameters in poultry production facilities,” in *Computer Aided Process Engineering - CAPE Forum 2013, 2013*, 2013.

25. A. S. Mendes *et al.*, “Minimum ventilation systems and their effects on the initial stage of turkey production,” *Brazilian J. Poult. Sci.*, vol. 15, no. 1, pp. 7–14, 2013.

26. M. Goliomytis, E. Panopoulou, and E. Rogdakis, “Growth curves for body weight and major component parts, feed consumption, and mortality of male broiler chickens raised to maturity,” *Poult. Sci.*, vol. 82, pp. 1061–1068, 2003.

27. D. . K. Carver, J. Fetrow, T. Gerig, T. Krueger, and H. J. Barnes, “Hatchery and transportation factors associated with early poult mortality in commercial turkey flocks,” *Poult. Sci.*, vol. 81, no. 12, pp. 1818–1825, 2002.

28. S. L. Vieira and E. T. Moran, “Effects of egg of origin and chick post-hatch nutrition on broiler live performance and meat yields,” *Worlds. Poult. Sci. J.*, vol. 55, pp. 125–142, 1999.

29. R. . C. Noble, F. Lonsdale, K. Connor, and D. Brown, “Changes in the lipid metabolism of the chick embryo with parental age,” *Poult. Sci.*, vol. 65, pp. 409–416, 1986.

30. M. A. Latour, E. D. Peebles, C. R. Boyle, S. M. Doyle, T. Pansky, and J. D. Brake, “Effects of breeder hen age and dietary fat on embryonic and neonatal broiler serum lipids and glucose,” *Poult. Sci.*, vol. 75, no. 6, pp. 695–701, 1996.

31. V. L. Christensen, W. E. Donaldson, and J. P. McMurtry, “Physiological differences in late embryos from turkey breeders at different ages,” *Poult. Sci.*, vol. 75, no. 2, pp. 172–178, Feb. 1996.

32. C. Crowther, R. Davies, and W. Glass, “The effect of night transportation on the heart rate and skin temperature of ostriches during real transportation,” *Meat Sci.*, vol. 64, no. 4, pp. 365–370, 2003.

33. Y. Pinchasov and Y. Noy, “Comparison of post-hatch holding time and subsequent early performance of broiler chicks and turkey poults,” *Br. Poult. Sci.*, vol. 34, no. 1, pp. 111–120, 1993.

34. G. Hackl and E. F. Kaleta, “No TitleMethods for the estimation of the age of broilers and effects of water and feed deprivation within the first days of life,” *Arch. fuer Gefluegelkd.*, vol. 61, pp. 280–286, 1997.

35. H. Xin and K. Lee, “Physiological evaluation of chick morbidity during extended posthatch holding,” *J. Appl. Poult. Res.*, vol. 6, no. 4, pp. 417–421, 1997.

36. Y. Noy and D. Sklan, “Energy utilization in newly hatched chicks,” *Poult. Sci.*, vol. 78, no. 12, pp. 1750–1756, 1999.

37. J. Dibner, “Feeding hatchling poultry. Avoid any delay,” *Feed Int.*, pp. 30–34, 1999.

38. E. Decuypere, K. Tona, V. Bruggeman, and F. Bamelis, “The day-old chick: a crucial hinge between breeders and broilers,” *Worlds. Poult. Sci. J.*, vol. 57, no. 2, pp. 127–138, 2001.

39. F. S. C. Vargas, T. R. Baratto, F. R. Magalhães, A. Maiorka, and E. Santin, “Influences of breeder age and fasting after hatching on the performance of broilers,” *J. Appl. Poult. Res.*, vol. 18, no. 1, pp. 8–14, 2009.

40. A. M. Ulmer-Franco, G. M. Fasenko, and E. E. O’Dea Christopher, “Hatching egg characteristics, chick quality, and broiler performance at 2 breeder flock ages and from 3 egg weights,” *Poult. Sci.*, vol. 89, pp. 2735–2742, 2010.

41. E. D. Peebles *et al.*, “Effects of breeder age and dietary fat on subsequent broiler performance . 1. Growth, mortality, and feed conversion,” *Poult. Sci.*, vol. 78, pp. 512–515, 1999.

42. J. L. McNaughton, J. W. Deaton, F. N. Reece, and R. L. Haynes, “Effect of age of parents and hatching egg weight on broiler chick mortality,” *Poult. Sci.*, vol. 57, no. 1, pp. 38–44, 1978.

43. G. M. Fasenko and E. E. O'Dea, "Evaluating broiler growth and mortality in chicks with minor navel conditions at hatching," *Poult. Sci.*, vol. 87, pp. 594–597, 2008.

44. X. Jiang, A. F. Groen, and E. W. Brascamp, "Economic values in broiler breeding," *Poult. Sci.*, vol. 77, no. 7, pp. 934–943, 1998.

45. B. J. Wood, "Calculating economic values for turkeys using a deterministic production model," *Can. J. Anim. Sci.*, vol. 89, no. 2, pp. 201–213, 2009.

46. V. Christensen, J. Grimes, and M. Wineland, "Effects of turkey breeder hen age, strain, and length of the incubation period on survival of embryos and hatchlings," *J. Appl. Poult. Res.*, vol. 10, pp. 5–15, 2001.

47. C. M. Schaefer, C. M. Corsiglia, A. Mireles, and E. A. Koutsos, "Turkey breeder hen age affects growth and systemic and intestinal inflammatory responses in female poults examined at different ages posthatch," *Poult. Sci.*, vol. 85, pp. 1755–1763, 2006.

48. S. Black, "Performance and economic impacts of hatchery and post-hatch constraints on poult quality: Chapter 3. Effects of breeder hen age and placement time on poult quality and early performance in turkey poults," North Carolina State University, 2018.

CHAPTER 6.

DISSERTATION CONCLUSIONS

CONCLUSIONS

High incidence of early poult mortality is often an indicator of welfare problems (*Humane Farm Animal Care Standards for Turkeys*, 2013), which is becoming an increasingly more important issue for consumers. According to Veissier & Boissy (2007) animal well-being is the absence of stress or low levels of stress. Distress, or physiological inability to cope with stress, occurs when elevated corticosterone levels in response to chronic stress transform metabolic processes to irreversible catabolic routes (Mailyan, 2016) resulting in decreased performance or death. Managing stress during the first week of life and minimizing distress and mortality is critical for maximizing livability and performance (Decuypere & Bruggeman, 2007; Lilburn, 1998; Willemsen et al., 2010). The goal of this dissertation was to identify factors and stressors during embryonic and early poult development and that are predictive of survivability and future growth, which collectively allow for the assessment of poult quality (PQ). Identification of biological elements and management practices influencing PQ will provide data and strategies for managing stress and the successful transition of turkey embryos to turkey poults.

In Chapter 3 an experiment was conducted to test the predictive success of individual post-hatch poult measurements on early performance. Poults from young breeders and delayed placement poults were used to simulate stress (and therefore compromised PQ) as compared to poults from peak breeder hens and poults that were placed immediately after hatch (Black, 2018a). Early post-hatch development is variable among poults from different aged breeder hens. Early development and growth of poults from young breeders are inferior to poults from older breeders. Delayed access to feed and water severely inhibited early growth performance in turkey poults. Poult measurements associated with growth

performance were: navel buttons for FOL poults; and shank color and hock vein for delayed placement poults; and shank length for all poults. PQ indicators successfully and distinctively linked early poult growth and survivability to a developmental factor (breeder age) and a management factor (delayed placement), providing insight to turkey producers for improved production and profitability.

Subsequently, in Chapter 4 an experiment was conducted to test the effectiveness of the Tona score and PQ measurements as associated with breeder hen age, correlations to overall production performance parameters, such as body weight and breast muscle yield, were examined (Black, 2018b). Successful indicators for predicting the future growth performance of poults from young breeders were poult length, shank length and relative asymmetry. Additionally, hatching weight was not correlated to subsequent body weights, including the final market age body weight at 14 weeks of age. Therefore, the Tona score may be a reasonable indicator of the condition of poults at hatch, but it is not an effective tool and more specific quantitative measurements must be used to determine the effects of breeder hen age on market age performance in turkey hens.

There are several limitations surrounding the research presented in Chapters 3 and 4. First, due to experimental design, only one or two factors can be tested at a time. Therefore, the complex interactions of hatchery factors and brooding factors that impact early poult development and early poult mortality cannot be simultaneously examined. Secondly, only one age of breeder (Black, 2018b) or two ages of breeder hens (Black, 2018a) coming from a single genetic line were utilized in these experiments. To provide true insight into the effects of breeder age on poult development, a spectrum of ages must be utilized, since the turkey industry uses breeders throughout the entire production cycle. Lastly, in a laboratory or

research setting, poults may not have the same stress responses as in normal commercial settings. Accounting for the variability of environmental factors and sub-optimal conditions that poults are normally exposed, is difficult to accomplish. This established the need for field research and real-time data collected from industry.

Early poult mortality negatively impacts the economic profitability of the turkey sector, which was valued at $6.18 billion in 2016 (USDA-ERS, 2017). To evaluate the costs associated with specific factors and stressors that trigger early poult mortality, a study in Chapter 5 was conducted using field data from a US turkey company (Black, 2018c). The results of this study concluded that there are external sources of variability that impact mortality such as the weather. Moreover, there are factors which act as stressors that when properly managed can optimize PQ and consequently reduce early mortality. These controllable factors include breeder and hatchery practices. It is interesting to note the results of this study found that a variety of these stressors impacted tom poults and hen poults differently. These results indicate the importance of adjusting post-hatch management based on gender. Of economic importance are the cost increase due to additional mortality associated with poults from young stage breeders which in this analysis ranged from $60 to $128. There is also a cost increase of $170 for every additional 500 miles traveled from hatchery to brooder barns. The limitations of this research are associated with the data set that is utilized. It is important to note that only one genetic line was analyzed, and the results may have changed if multiple genetic lines were included. Additionally, this data set only analyzed poults hatched in the Mid-West region of the United States, therefore the results might change if hatcheries from all regions in the United States were analyzed, particularly the Southeastern region, where humidity and temperature vary greatly in the summer season.

Despite these limitations, this data and analysis provides useful insights into which management practices need to be modified or customized to optimize PQ and minimize early poult mortality.

The research in Chapters 3 and 4 verifies that young breeder age can act as a stressor during embryonic development and post-hatch growth (Black, 2018a, 2018b). Chapter 3 confirms that holding time before placement, can also act as stressors during embryonic development and early post-hatch life. Individual quantitative PQ measurements are utilized to predict future growth of turkey poults in association with developmental factors and stressors (Black, 2018a). Looking forward, a new model for PQ is proposed in Figure 6.1, incorporating the concepts and effects from the chapters in this dissertation. To fully understand the impact of stressors / factors on performance, PQ needs to be quickly assessed at the hatchery and on the farm. Inputs into the model include 2 major stress categories: embryonic development and post-hatch development. Specific factors are known to impact poults performance and survivability as measured by PQ indicators and previously discussed in the previous chapters of this dissertation. Additionally, the combination of multiple developmental factors results in varying PQ of poults or an additive effect of mortality. In practice, only one or two physical parameters, such as alertness and appearance, are used as the main method for sorting hatchling in commercial hatcheries (Tona et al., 2005). Therefore, it is important to pick measurements that apply to the poults, and the developmental factors that created poults, to accurately predict future performance. However, it is important to note that many factors produce the same performance outcomes (i.e. lower body weight), therefore knowledge of specific input factors / stressors is critical for understanding the underlying causes of compromised PQ. To further investigate the causes

compromised PQ, several post-hoc tools to analyze physiological status can be utilized. These analyses should be done on a small sub-sample of poults with known input factors, as they often require blood collection or euthanasia. For example, a bird that has been transported for over 72 hours should be sampled for hematocrit to determine dehydration status or monitoring eggshell conductance during incubation of eggs of various breeder origins. In the future, stress / input factors should be extensively explored both individually and in combination with each other. This information can be used by integrated turkey companies to improve PQ in its facilities and identify compromised PQ poults with potential interventions.

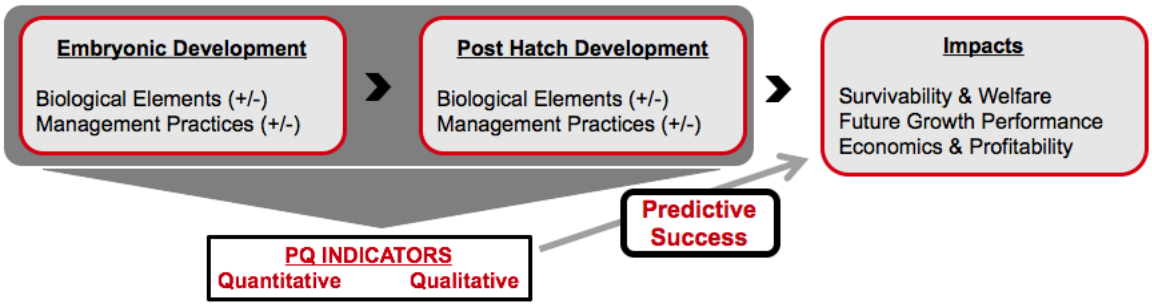

Figure 6.1. New Proposed PQ Model.

REFERENCES

Black, S. (2018a). *Performance and economic impacts of hatchery and post-hatch constraints on poult quality: Chapter 3. Effects of breeder hen age and placement time on poult quality and early performance in turkey poults*. North Carolina State University.

Black, S. (2018b). *Performance and economic impacts of hatchery and post-hatch constraints on poult quality: Chapter 4. The effect of stage of lay on poult quality and the use of poult quality as an indicator of post-hatch performance of market turkey hens*. North Carolina State University.

Black, S. (2018c). *Performance and economic impacts of hatchery and post-hatch constraints on poult quality: Chapter 5. Estimating effects of hatchery practices on early poult mortality using turkey industry field data*. North Carolina State University.

Decuypere, E., & Bruggeman, V. (2007). The endocrine interface of environmental and egg factors affecting chick quality. *Poultry Science, 86*, 1037–1042.

Humane Farm Animal Care Standards for Turkeys. (2013). Herndon, VA. Retrieved from http://certifiedhumane.org/wp-content/uploads/Std14.Turkeys.2A-2.pdf%0D

Lilburn, M. S. (1998). Practical aspects of early nutrition for poultry. *The Journal of Applied Poultry Research, 7*(4), 420–424.

Mailyan, E. S. (2016). Stress in poultry: focus on turkeys causes, mechanism, consequences, diagnostics and prevention. *Journal of Dairy, Veterinary & Animal Research, 3*(6).

Tona, K., Bruggeman, V., Onagbesan, O., Bamelis, F., Gbeassor, M., Mertens, K., & Decuypere, E. (2005). Day-old chick auality: relationship to hatching egg quality, adequate incubation practice and prediction of broiler performance. *Avian and Poultry Biology Reviews, 16*(2), 109–119.

USDA-ERS. (2017). Turkey Sector: Background & Statistics. Retrieved December 4, 2017, from https://www.ers.usda.gov/newsroom/trending-topics/turkey-sector-background-statistics/

Veissier, I., & Boissy, A. (2007). Stress and welfare: Two complementary concepts that are intrinsically related to the animal's point of view. *Physiology and Behavior, 92*(3), 429–

433.

Willemsen, H., Debonne, M., Swennen, Q., Everaert, N., Careghi, C., Han, H., … Decuypere, E. (2010). Delay in feed access and spread of hatch: importance of early nutrition. *World's Poultry Science Journal*, *66*(02), 177–188.