IMPROVING BROILER PERFORMANCE THROUGH BREEDER NUTRITIONAL MANAGEMENT

Martin J. Zuidhof
410 Agriculture & Forestry Centre, University of Alberta, Edmonton, AB T6G 2P5, Canada; martin.zuidhof@ualberta.ca

Abbreviated Title: MATERNAL NUTRITION

Summary

Recent research on the subject of maternal nutrition has focused largely on the effects of vitamins, antioxidants, and most recently the complex interactions of dietary macronutrients (energy and protein) on economically important broiler traits: primarily growth rate and breast meat yield. Vitamin E and organic Se show great promise for improving hatchability and chick health. Recommendations for ideal maternal levels of dietary protein and energy are not as clear. The concept of ideal protein has greatly advanced the discipline of livestock nutrition, and it is likely that a quest for an optimal ratio of dietary energy to ideal protein will simplify our understanding of maternal nutrition. The fit of published research on maternal dietary energy and protein levels, ratios, and intakes are tested against several hypotheses. The most consistent alignment is with regard to the relationship between low maternal protein intake and progeny fatness. Lack of congruency with other hypotheses underscores a need for further research. It is clear that a more strategic approach is needed to improve understanding of this multifaceted question. Rather than studying offspring performance as an afterthought, experimental designs must prioritize discovery of the impact of maternal nutrition on offspring performance. Combined with carefully executed control of breeder experiments to reduce variation in breeder feed allocation, and thoughtful standardization of hen age, egg handling, incubation, hatching, and brooding conditions, carefully considered experiments will add greater value to our knowledge of transgenerational effects of maternal nutrition.

Key words: Maternal nutrition, broiler breeder, offspring, progeny, growth, yield, review

Introduction

Broiler growth rate, yield and efficiency are of key economic importance for chicken meat production. Understandably, most nutrition studies are conducted to understand the direct impact of broiler diet composition on broiler growth, development, and efficiency. Far less information is available on the effects of maternal nutrition on broiler productivity, though minor improvements in broiler productivity achieved through maternal nutrition may have significant economic benefit (Calini and Sirri, 2007). A major challenge with maternal nutrition is that its effects on broiler performance tend to be confounded by a large temporal lag during which many environment factors can influence the results.

Broiler breeder nutrition can directly affect chick health and productivity through egg composition (reviewed by Wilson, 1997; Kidd, 2003). Currently, however, a new and fascinating field of influence is being discovered. There is increasing disparity between the growth potential of broilers and the degree of expression allowed in broiler breeders to maximize reproductive output. In mammals, it is well established that maternal undernutrition can have negative metabolic effects in offspring and even grand offspring. Many of these insights originated from studying the impact of malnutrition on offspring growth, development and health during the ‘Dutch famine’ at the end of the Second World War (Roseboom et al., 2011). These and subsequent studies with various animal models have led to discovery of epigenetic (non-DNA sequence) moderators of progeny gene expression. The list of potential epigenetic mechanisms is expanding rapidly. Though not well understood, it is probable that aspects of broiler performance may be influenced indirectly by epigenetic mechanisms. Given the severe degree of feed restriction practiced in meat type chicken parent stocks, the importance of maternal nutrition may be increasing.

The objectives of the current paper are 1) to identify maternal nutritional strategies with potential to increase broiler health and performance, 2) to serve as an update on previous reviews, particularly of maternal dietary energy and protein effects on offspring performance, and 3) to outline key considerations for future research aimed at maternal nutrition strategies for optimal broiler productivity.

Mechanisms for maternal nutrition effects

There are two main ways that maternal nutrition can affect broiler performance. The first is through differential incorporation of nutrients into the egg, which directly impacts progeny development. The second involves epigenetic ef-
fects that indirectly affect progeny growth development by changing the regulation of gene expression in the progeny. Epigenetic mechanisms that impact skeletal muscle growth and development, for example, has been reviewed by Saccone and Puri (2010). For many years, we have known that the phenotype of an animal is the result of its genome and its environment. This has often been referred to as the G x E interaction. The environment dictates the degree of gene expression that influences among other things, the animal’s size and composition. There is increasing evidence that the environment can influence gene expression later in the life of an individual, but also trans-generationally through DNA or chromatin modifications (reviewed by Jirtle and Skinner, 2007) or diffusible RNA factors (Daxinger and Whitelaw, 2012). This provides a new framework for understanding the impact of broiler breeder nutrition on broiler performance. Many studies of maternal nutrition in many species have shown an impact on progeny body weight (BW) and carcass composition. Unfortunately, there is often little in the way of an obvious pattern in these effects. However, since many epigenetic mechanisms are influenced by the availability of nutrients to the parents, ever-increasing degrees of feed restriction in broiler breeders suggests direct relevance of epigenetics to the field of broiler maternal nutrition. Though important to note the significance, a review of possible epigenetic mechanisms is beyond the scope of the current analysis.

Challenges of measuring impact of maternal nutrition on broiler performance

It is often difficult to determine patterns in the effects of maternal nutrition on progeny performance. Part of the challenge is the lengthy delay between feeding the pullet and the hen and the resulting progeny performance. There is a time lag of months during which many confounding environmental influences can complicate the interpretation of the effect of maternal nutrition on progeny performance. The following effects summarize some of the important research into maternal nutritional management that may be applied to increase broiler health, livability, growth rate, and yield.

Hatching egg composition, hatchability and chick quality

**Vitamins and antioxidants**

There is clear evidence that incorporation of vitamins and minerals, many of which are antioxidants, into hatching eggs is highly influenced by maternal intake. Vitamin and mineral deficiencies, or large excesses can harm or kill the embryo, reducing hatchability (reviewed by Wilson, 1997). In reviewing the subject of maternal vitamin and mineral nutrition, Hocking (2007) concluded that organic forms of minerals, potentially in slightly higher than recommended concentrations appear to be beneficial for progeny performance, and since that time further research has provided more evidence for this hypothesis. Surai (2000) reported that supplementation of broiler breeder diets with selenium (200 and 400 μg/kg) and vitamin E (40 to 200 mg/kg α-tocopherol) increased egg concentrations of these antioxidants. The liver antioxidant system in 1- and 5-day-old chicks, but not in 10-day-old chicks was enhanced, suggesting a direct effect via increased antioxidant levels in the egg. Rajashree et al. (2014) fed up to 500 μg/kg of organic Se, and observed both an increase in hatchability of over 4%, and increased antioxidant status of eggs. Urso et al. (2015) found that 120 mg/kg of vitamin E increased 29 week hatchability by 3.2 chicks per 100 eggs set, and high organic Se increased hatching weight by approximately 0.4 to 0.7 g until 33 weeks of age. In terms of effects on progeny growth, An et al. (2012) found that high maternal vitamin E (100 mg/kg vs. 20 mg/kg) increased FCR to 1.75 vs. 1.68, respectively, but reduced drip loss from 2.60 to 1.85%. Vitamin E levels of 120 mg/kg and organic selenium at 400 μg/kg are recommended to increase chick quality and hatchability.

**Immune function**

Quereshi et al. (1998) exposed broiler breeders to aflatoxin and observed reduced hatchability and compromised antibody response in broiler chicks. Studying maternal supplementation of antioxidants on broiler innate immunity, Johnson (2013) found that maternal dietary replacement of synthetic vitamin E with a natural vitamin E increased *in vitro* E. coli bactericidal capacity of chicks from older (58 wk old) breeders, and oxidative burst in chicks from 46 and 58 wk old breeders. Supplementation with 6 and 12 ppm of canthaxanthin increased plasma antioxidant capacity, while supplementation with 50 mg/kg L-carnitine decreased oxidative burst in 4-day old chicks (Johnson, 2013). Hen and chick age had large effects on immune status of broilers. The degree of variation due to hen and chick age in progeny responses to maternal nutrition were strongly established by Johnson (2013) and Torres (2013). Inconsistent reports of the effects of maternal nutrition on broiler performance is likely due to lack of consistency in hen age between experiments.
Omega-3 fatty acids
Supplementation of maternal diets with long-chain omega-3 fatty acids lowered egg weight, and reduced 11- and 28-day BW, and increased both feed conversion ratio and mortality of offspring (Koppenol et al., 2015b). Further, there was little benefit of omega-3 supplementation for the immune response of the progeny (Koppenol et al., 2015a).

Managing maternal nutrient intake

Maternal feed restriction
Waaij et al. (2011) conducted a study to investigate the effects of maternal feed intake on broiler growth, and to specifically match and mismatch the nutritional environments of hens and chicks. These authors fed 60-week-old broiler breeders at recommended or 35% higher intake, then fed the progeny ad libitum or at 70% of ad libitum. Of hens were fed restricted amounts, their male broiler progeny grew less quickly, while their ad libitum-fed female progeny were 8% fatter. This was a short-term hen feeding study, so it is possible that the nutritional status of the hen caused a shift in egg composition, leading in turn to modified performance of the broilers. However, it raises important questions because notably, they identified outcomes of maternal feed restriction that are undesirable commercially.

Among a suite of complex interactions, Eusebio-Balcazar et al. (2015) observed that maternal nutrient restriction at a rate of 89% of during weeks 20 to 26 followed by rapid growth from 26 to 29 weeks of age worsened the walking ability of corn-fed male broilers, but only when maternal feeder space increased 33% at 22 weeks of age.

Maternal dietary energy and protein
Quantitative restriction of calorie and protein intake is one of the major strategies employed to optimize broiler breeder BW for reproductive success. There is a growing body of information on the subject, of the impact of amino acid or crude protein (CP) and metabolizable energy (ME) levels on offspring performance, but the approach has not been systematic, and the results often paradoxical. To understand the effects of maternal protein and energy nutrition, the ratio of energy to amino acids, total balanced protein, and other nutrients must be taken into account – not simply dietary level. This is because the partitioning of energy to maintenance, growth, and egg production is dictated by the most limiting nutrient. Growth and reproduction follow the organism’s first metabolic priority, maintenance (survival). It is critical to understand maternal maintenance energy and amino acid requirements because the residual energy and amino acids remaining after maintenance is taken care of are used for growth and reproduction. Excess or insufficient energy or essential amino acids precedes complex metabolic decision making that dictates the productivity and health of the hen.

Low ME-high CP diets have generally reduced hatchability (Leeson and Summers, 1991; Pearson and Herron, 1982). These effects on hatchability could be explained by altered composition in the egg, directly affecting the developing embryo. Typically, higher maternal CP intake resulting from higher dietary CP or lower ME levels increases egg and chick weights (Joseph et al., 2000; Enting et al. 2007). Though weakly correlated, this typically translates into larger chicks and larger broilers, but these effects will not be the focus of the current review. The following summarizes much of the recent published literature on the effects of maternal dietary energy and protein levels on progeny performance in the context of some classical works. Because of the complexity of the subject, this section is organized as a series of general hypotheses, specifically supported or contradicted by the research that has been conducted in this area. Factors such as hen age, the timing of the maternal dietary treatment (rearing or laying phases, or both), and the sex of the progeny tested have been considered. Several inconclusive reports have been omitted from this classification scheme.

Hypothesis 1*: Low maternal protein intake leads to lipid accumulation in offspring.
Of all the propositions, this one had the clearest support from published studies. Several studies provided evidence supporting, and none contradicted this hypothesis.

---

3 Corollaries of Hypotheses – since studies almost always tested ratios of energy to protein:
A. High maternal energy intake leads to the same effect
B. High maternal protein intake leads to the opposite effect
C. Low maternal energy intake leads to the opposite effect
• Higher abdominal fat percentage in female broilers from hens fed low CP diets (10% compared 16% CP; Lopez and Leeson, 1995)
• Maternal dietary ME restriction during lay (20, 30, and 50%) increased abdominal fatpad, subcutaneous fat, liver fat, and breast muscle fat content (Li et al., 2014)
• Moraes et al. (2014) predicted a 4.8% larger fatpad in broilers from hens fed lower CP during their rearing period (13.8 vs. 15.5% maternal CP)
• Male broilers from hens with low maternal ME intake during the laying period (450 vs. 385 or 325 kcal/d) had more carcass fat (Spratt and Leeson, 1987)

**Hypothesis 2: Low maternal protein intake leads to lower offspring body weight.**

*In support of hypothesis 2:*

• Female broilers from hens with lower maternal CP intake during rearing had reduced BW (Moraes et al., 2014)
• At a low maternal pullet diet ME level (2,528 kcal/kg), a higher CP level (15.3 vs. 13.7%) increased male broiler BW to 22 day of age (Moraes, 2013)
• Male broilers from hens with higher BW (higher feed intake) had higher BW than those from standard BW hens (Emous et al., 2015)
• Broilers had higher BW when maternal diets contained low ME (ME:CP ratio of 18.0 kcal/g; Peebles et al., 2002)

Because the effect of rearing period nutrition was often more important than the laying phase, these findings point to epigenetic programming. Similarly, after the ‘Dutch famine’ increased neonatal adiposity was observed in children and grandchildren of women exposed prenatally to nutrient shortage (Painter et al., 2008). Heritable, transgenerational mechanisms for maternal nutrition to influence offspring growth and development are currently being explored (Jirtle and Skinner, 2007; Daxinger and Whitelaw, 2012).

**Contradicting hypothesis 2:**

• Ciacciariello and Tyler (2013) fed digestible maternal lysine levels ranging from 0.44 to 0.75% during the laying phase and found that progeny of young breeders (not beyond 38 weeks of age) fed the lowest lysine levels grew dramatically heavier and converted feed more efficiently
• Higher maternal pullet phase ME level (2,736 vs. 2,528 kcal/kg; 15.3% CP) decreased CP intake and increased female broiler BW to 22 day of age (Moraes, 2013)

Suggesting the possibility of a nonlinear relationship, an optimal maternal pullet phase ME:CP ratio of 18.25 kcal/g was proposed for maximizing female broiler BW (Moraes et al., 2014). For chick weight, a similar optimal maternal laying phase ME:CP ratio of 18.0 kcal/g can be inferred from Leeson and Summers (1991).

**Hypothesis 3: Low maternal CP intake leads to lower breast or total carcass yield.**

*In support of hypothesis 3:*

• Chicks from young broiler breeders (26 weeks of age) fed low digestible lysine (0.6 g/d) had lower BW, carcass weight, and breast weight, and higher drumstick and thigh weights (Mejia et al., 2013). This transient effect was not observed in progeny of 31 and 36 week-of-age broiler breeders.
• Li et al. (2013) suggested that myogenin (MYOG) gene expression was delayed in embryos from hens receiving low feed intake (75% of recommended)

**Contradicting hypothesis 3:**

• Emous et al. (2015) found that male broilers from pullets fed lower maternal dietary CP (ME:CP = 21.2 vs. 18.4 kcal/g) had higher breast yield than from pullets fed higher maternal dietary CP.
• Feeding higher levels of CP to broiler breeder pullets during the rearing phase reduced breast yield in male and female broilers (Moraes, 2013). Dietary combinations that increased CP intake of broiler breeders during rearing and lay had sex-specific additive effects on broilers: it decreased male broiler breast yield, but increased female breast and carcass yield.
Moraes (2013) observed that in pullets fed 15.3% CP during rearing, a higher ME level (2,736 vs. 2,528 kcal/kg), which decreased CP intake increased overall broiler carcass yield at 40 d of age.

Zhu et al. (2012) fed Chinese yellow breeders maternal diets containing ME ranging from 2,650 to 2,850 kcal/kg, with 15.5 to 17.5% CP. Hens were fed fixed amounts of feed daily. Moderate ME:CP ratios tended to optimize live performance, whereas yield was highest with low CP intake, and lowest at moderate CP intake. This result also points to a nonlinear relationship between CP intake and carcass yield.

Other notable findings
Several observations did not fit the relatively simple suite of hypotheses, and suggest that more complex nonlinear relationships should be investigated to explain interactions between maternal ME and CP on progeny performance. Leeson and Summers (1991) reported that a laying phase ME to CP ratio of 18 kcal/g maximized chick weight. Enting et al. (2007) fed normal and 12 or 23% lower maternal ME levels, and reported an increase in 38 d BW in the moderate ME level, which had a pullet phase ME:CP ratio of 16.0 kcal/g, increasing to 16.6 and 17.7 kcal/g during early and late lay periods. Enting et al. (2007) also reported reduced mortality with their lowest ME level. For male broiler breast and carcass yield, optimal maternal ME:CP ratios during rearing and lay were (average of 16.5 and 17.9) and 18.5 kcal/g, respectively (Moraes et al. 2014). For female broiler breast and carcass yield, they reported optimal maternal ME:CP ratios during rearing and lay of 17.2 and 19.4 kcal/g, respectively. Conversely, the worst maternal diet ME:CP for male broiler breast and carcass yield were 19.2 and 18.5 kcal/g, respectively. For female broiler breast and carcass yield, the worst ME:CP ratios during rearing and lay were 17.2 and 18.5 kcal/g, respectively. Interestingly, maternal diets that were optimal for male performance were worst for female broilers. Increasing the dietary ME:CP ratio from 17.2 to 19.4 kcal/kg appeared to be optimal for mixed sex flocks, as the yield performance of male progeny in this treatment was not significantly different from the optimal male scenario. A lower ME:CP ratio during lay should be avoided (Moraes et al., 2014). In other words, higher (over-) consumption of CP during the laying period appeared to have the most negative effects on broiler performance.

Moraes et al. (2014) found that breast yield increased when maternal ME:CP ratio increased during transition from pullet to laying phase diets (17.5 or 19.0 to 19.4; 17.5 to 18.5 kcal/g; Moraes et al., 2014). This is consistent with current recommended practice, where recommended ME levels are maintained and CP levels drop in later dietary phases.

Lopez and Leeson (1995) observed lower feed intake in male broiler offspring of parents fed 10 to 16% dietary CP, but the broilers compensated such that those from hens fed 10 and 12% dietary CP had lower feed conversion rates than those fed 14 or 16%.

Management of broiler breeder feeding

Metabolic stress
Reducing metabolic stress may be key to reproductive success. Zuidhof et al. (2007) demonstrated that radical target BW strategies during the pullet phase can yield similar reproductive outcomes. A key success strategy was adherence to the principle: “Don't let the bird's metabolism know you have made a change to the feed allocation”. Stable metabolism in that study was accomplished by weighing birds and determining feed allocations twice per week. Liver weight and composition is an indicator of the metabolic status of birds, and it is a key organ in storage and mobilization of energy. de Beer et al. (2007) reported similar baseline liver glycogen stores, which doubled in skip-a-day fed versus every-day fed broiler breeders. A similar pattern was observed for liver weight. Metabolic stress may also affect progeny. Portha et al. (2011) reviewed the effects of maternal metabolic stress on beta-cell programming. In rats, both undernutrition and obesity decreased the development of beta cells and increased the likelihood of diabetes in offspring and grand offspring. In birds, where there is no physiological contact with the mother during embryo development, epigenetic mechanisms are more likely to be involved in transgenerational inheritance than parental physiological mechanisms. The escalating severity of feed restriction in broiler breeders presents a great model to study maternal metabolic stress on the offspring. However, there are large gaps in the study of transgenerational effects of nutrition in meat-type chickens.

Precision feeding
Given the challenges around metabolic stress in broiler breeders, strategies for achieving more stable metabolism will likely improve breeder performance, and will likely influence progeny performance. A precision feeding system
for broiler breeders has been developed at the University of Alberta (Zuidhof, 2014). The system has been used to raise broiler breeder pullets to sexual maturity with a BW CV of less than 3%. The sequential feeding system is a paradigm shift for feeding breeders, as birds can eat multiple small meals per day rather than binging and fasting, which has become the norm for broiler breeder management. Such a system shows promise for accelerating research on maternal nutrition. It is now possible to control previously confounding effects of BW differences and lack of uniformity, and the true effects of nutrition on breeder and progeny performance will become clearer.

**Considerations for future maternal nutrition research**

Much research is still required to fill the knowledge gaps in this complex field. Better control of confounding sources of variation in studies of the effects of maternal nutrition on progeny growth and development is needed. The following categories should be strategically considered in the study of transgenerational impacts of maternal nutrition. Standardization within and between studies will aid the interpretation of experimental results.

**Maternal age**

Maternal age has a strong influence on broiler productivity. Effects of maternal nutrition tend to be amplified in progeny from young breeders.

**Timing of maternal nutrition**

The amount of time prior to egg and chick production that a diet or nutritional management is applied has significant influence on the outcome of many studies. Factorial arrangements of maternal nutrition treatments, including diet changes from the rearing to laying phases will help to identify epigenetic effects.

**Genetic factors**

The major components of hatching eggs (yolk, albumen and shell) vary in a predictable manner according to age, body composition, and maternal nutrition (Nonis and Gous, 2013), but there are strain- (Johnston and Gous, 2007) and likely hen-specific differences in egg composition that can and should be accounted for. Pedigree information should be used to account for genetic variation in broiler performance. This will allow for more precise estimation of the actual effects of maternal nutrition treatments on progeny performance.

Romero et al. (2009) characterized the efficiency of hens in terms of residual feed intake (RFI) and residual maintenance requirement (RME$_m$). RME$_m$ was particularly useful for explaining variation in egg and chick weights. Efficient hens (with low RME$_m$) partitioned more nutrients toward reproduction than inefficient hens (with high RME$_m$). Broilers from RFI-efficient hens grew at a higher rate from 21 to 28 days, and of age, and had a higher 28- and 38-day BW. Broilers from RME$_m$-efficient hens grew at a lower rate than less RME$_m$-efficient hens. In fact, the broilers with the highest BW originated from RFI-efficient and RME$_m$-inefficient hens. Breast meat yield in broilers from RME$_m$-efficient hens was lower, whereas no meaningful relationship between RFI and breast muscle yield was detected.

**Maternal body weight variation**

Body weight differences due to unequal feed distribution is a growing challenge in interpreting the complex effects of maternal nutrition on progeny performance. A more strategic approach to maternal nutrition research must start with rigid BW control and high breeder flock uniformity, achieved through better control of feed distribution. Precision feeding offers promise as a research tool to manage feed intake according to individual birds according to their body weight. Combined with adequate micronutrient supplementation, consistent hatching and brooding management, and better accounting for heritable maternal and paternal contributions to broiler growth, a more clear and consistent pattern of the effects of maternal nutrition on progeny performance will be possible.

**Conclusion**

The data currently available on the impact of maternal dietary ME and CP levels does not paint a simple picture of how to use maternal nutrition to optimize broiler performance and yield. More strategic experimental design, expert breeder management, control of ancillary sources of variation, and evolution of our understanding of epigenetics and nutritional programming will add resolution to our understanding of this difficult subject.
References


