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# Effect of Posthatch Feed Restriction on Broiler Breast Muscle Development and Muscle Transcriptional Regulatory Factor Gene and Heparan Sulfate Proteoglycan Expression

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**Abstract:** The effect of an immediate posthatch growth restriction mediated through a 20% growth restriction the first 2 wk posthatch was studied for its effect on pectoralis major muscle morphological structure and the expression of the myogenic transcriptional regulatory factors, MyoD and myogenin, and the heparan sulfate proteoglycans syndecan-4 and glypican-1. Broiler chicks at hatch were divided into a full fed (control) group or a treatment group with feed managed to maintain a body weight 80% of control weight through 2 wk posthatch. At the end of 2 wk, the growth restricted chicks were put on full feed diet without further restriction. By 1 d posthatch, the morphological structure of the pectoralis major muscle in the growth restricted birds was not well organized into muscle fiber bundles with distinct individual muscle fibers as observed in the control group. The difference in the morphological structure remained throughout the 42 d of the study. The growth restricted birds had increased fiber necrosis and larger and more extensive fat cell depots beginning at 28 d posthatch. The BW of the growth restricted birds was significantly reduced compared to control birds through 28 d. Pectoralis major weight was significantly reduced through 28 d. The expression of genes required for the proliferation and differentiation of muscle cells was affected by the growth restriction. MyoD and syndecan-4 are both genes expressed during proliferation and both of these genes had elevated expression during the first wk of the growth restriction whereas the differentiation genes, myogenin and glypican-1, were significantly reduced in their expression during this period of time. Together these results suggest that the immediate posthatch feeding regimen to chicks is critical for the appropriate morphological development of the pectoralis major muscle, the formation of intramuscular fat depots, and the expression of genes necessary for muscle cell proliferation and differentiation.

**Key words:** Broilers, growth restriction, heparan sulfate proteoglycans, muscle, myogenic transcriptional regulatory factors

## INTRODUCTION

Development of skeletal muscle occurs as a result of the proliferation and differentiation of myoblasts that fuse to form multinucleated myotubes. The myotubes further differentiate into muscle fibers expressing muscle specific contractile proteins. After hatching, continued skeletal muscle growth is dependent upon the proliferation and differentiation of myogenic satellite cells. During the immediate posthatch period, muscle growth occurs through the process of hypertrophy. Hypertrophy entails the fusion of satellite cells with existing myofibers leading to an increase in muscle fiber size through increased protein synthesis. In feed deprived turkey poults and chicks satellite cell mitotic activity is reduced (Halevy *et al*., 2000; Mozdziak *et al*., 2002). The immediate posthatch period most likely represents a period of maximal satellite cell activity. An important regulator of satellite cell behavior is the extracellular matrix. The extracellular matrix is composed of collagenous and noncollagenous proteins secreted by the cell into the extrinsic cellular environment. The members of the proteoglycan component of the extracellular matrix functions in cell signal transduction pathways including growth factor responsiveness. Satellite cells are extremely responsive to the mitogenic effects of certain growth factors on their proliferation and differentiation characteristics (Dodson *et al*., 1996). Fibroblast Growth Factor 2 (FGF2), for example, is a potent stimulator of satellite cell proliferation and a strong inhibitor of satellite cell differentiation (Dollenmeier *et al*., 1981). For FGF2 to bind to its high affinity cell surface tyrosine kinase receptors, it must interact with cell surface heparan sulfate proteoglycans which function as a co-receptor for FGF2 (Aviezer *et al*.,

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1994). Velleman and Mozdziak (2005) showed that the expression of heparan sulfate proteoglycans is reduced by feed deprivation immediately after hatch suggesting a possible change in FGF2 signal transduction which would affect the proliferation and differentiation of satellite cells.

Satellite cells are muscle stem cells that are multipotential in terms of their cellular fate. Satellite cells are derived from mesodermal cells which form the cellular lineage for skeletal muscle, adipocytes, and chondrocytes. Askura *et al*. (2001) demonstrated in an *in vitro* study that satellite cells could be induced to follow myogenic, osteogenic and adipogenic cellular pathways depending on the culture conditions. Since satellite cells are multipotential and their cellular fate can be modified *in vitro* by altering the culture medium. One must wonder if the same occurs *in vivo* during the period of immediate posthatch muscle growth. This is a period when satellite cell activity is affected by the nutritional status of the birds as previously shown in immediate posthatch nutrition studies (Halevy *et al*., 2000; Mozdziak *et al*., 2002).

In commercial poultry operations, it is common during the immediate posthatch period for chicks to rely on nutrients from the yolk during shipping. In addition, control of metabolic disorders such as those resulting in leg problems and ascites has led to the industry recommendation of early feed restriction during the first 2 wk posthatch (Arce *et al*., 1992; Acar *et al*., 1995). The logic is that short term feed restriction applied early in life would allow the chicken to restore balance between supply and demand organs (Katanbaf *et al*., 1988; Acar *et al*., 1995). Final processing body weights would be achieved through compensatory gain. The phenomenon of compensatory gain in poultry has been studied for years (Auckland, 1972; Cherry *et al*., 1978; Malone *et al*., 1980; Ferket and Sell, 1989; Washburn, 1990) with mixed results. It appears that factors such as timing, duration and intensity of the restriction all impact the compensatory response of the bird. It is unclear, however, if muscle generated from a post restriction recovery period is the same as that of muscle from birds reared in unrestricted conditions.

To address the effects of an immediate posthatch growth restriction on muscle formation, fertile eggs were obtained from a commercial broiler breeder company and hatched. Resultant chicks were divided into a full fed group and a growth restricted group that maintained 80% body weight compared to the control for the first 2 wk posthatch. Samples of the pectoralis major muscle, the most economically valuable muscle, were collected for RNA analysis and histology. The muscle transcriptional regulatory factors MyoD and myogenin, and the heparan sulfate proteoglycans syndecan-4 and glypican-1 were studied for their expression. These genes were selected because of their role in regulating satellite cell proliferation and differentiation. MyoD is necessary for proliferation whereas myogenin is a marker of differentiation. The expression of MyoD and myogenin has been shown to be affected by posthatch feeding in turkeys (Halevy *et al*., 2003). The expression of syndecan-4 and glypican-1 were also measured as these are heparan sulfate proteoglycans that differentially regulate FGF2 signal transduction in satellite cells (Velleman *et al*., 2007) and whose expression is affected by selection for muscle mass accretion (Liu *et al*., 2006). Syndecan-4 has been hypothesized to play a role during proliferation whereas glypican-1 is postulated to be a regulator of differentiation due to their increased expression during these phases of muscle development (Brandan and Larraín, 1998; Cornelison *et al*., 2004).

## MATERIALS AND METHODS

**Birds:** Fertile eggs from a Cobb broiler line were shipped to the University of Arkansas Poultry facility and hatched. In two trials males were raised with 180 birds per trial. At d 0, the birds were divided into a control standard commercial regimen and a growth-restricted group. The growth restriction was designed to obtain a feed restricted treatment group that maintained 80% BW as compared to the full fed control for the first 2 wk posthatch. This was accomplished by daily weighing of all birds and adjusting the feed allocation of the restricted birds based on the weights of both groups. After the separation into the experimental groups, 10 birds from each group were sacrificed with BW and pectoralis muscle weight recorded, and a sample removed for histological analysis of muscle structure. The remainder of the pectoralis major muscle was immediately frozen and stored at -70°C for RNA analysis. Every 3 d for the 2 wk restriction period, 10 birds from each group were sacrificed and pectoralis major weights recorded, a sample removed for histology, and the pectoralis major muscle was frozen at -70°C. After the initial 2 wk, the growth restricted birds were returned to *ad-libitum* consumption of a standard commercial diet and every 7 d for a period of 6 wk (42 d) 10 birds from each group were sacrificed with BW and pectoralis major weights recorded, a sample removed for histology and the pectoralis major muscle were frozen and stored at -70°C. At the completion of the trials, all the muscle samples were shipped to The Ohio State University.

**Histology:** After the removal of the skin from the breast region, a sample of the breast muscle was obtained by carefully dissecting approximately a 0.3 to 0.5 cm wide section of the muscle following the orientation of the muscle fibers for a length of about 3 cm. The muscle samples were placed in 10% (vol/vol) buffered formalin fixative (pH 7.0) at 4°C for at least 17 h. After fixation, the samples were dehydrated through a series of graded

alcohols as previously described by Jarrold *et al.* (1999), cleared in Pro-Par Clearant (Anatech, Battle Creek, MI) for 1 h with one change at 30 min and then infiltrated with paraffin at 55°C for 4 h with one change at 1 h using a Leica TP1020 tissue processor (Leica, Nussloch, Germany). The samples were then embedded in paraffin and the resulting paraffin blocks were cross sectioned at 5 μm and mounted on Starfrost Adhesive slides (Mercedes Medical, Sarasota, FL). Hematoxylin and eosin staining was done as described in Velleman and Nestor (2004).

The stained muscle sections were analyzed for muscle morphology with an Olympus XI 70 microscope (Melville, KY) equipped with an Olympus Magna Fire digital camera linked to a computer with Image Pro Software (Media Cybernectics, Silverspring, MD). Each slide from each bird contained a minimum of 4 sections and 5 microscopic fields were evaluated from each section.

**Gene expression analysis:** The following genes were analyzed for their expression: MyoD, myogenin, syndecan-4, and glypican-1. MyoD and myogenin are muscle specific transcriptional regulatory factors expressed during the proliferation and differentiation, respectively, whose expression has been shown to be affected by posthatch feeding in turkeys (Halevy *et al.*, 2003). The syndecans and glypican are heparan sulfate proteoglycans whose expression is affected by growth selection (Liu *et al.*, 2006) and are required for FGF2 signal transduction. The expression of these genes was measured by real-time quantitative Polymerase Chain Reaction (PCR). The primers used for each gene are listed in Table 1. The amplified sequences were confirmed by DNA sequence analysis (data not shown). The real-time quantitative PCR analysis was done as previously described in Liu *et al.* (2006). In brief, total RNA was extracted from each individual bird using Trizol (Invitrogen, Carlsbad, CA). The isolated RNA was reverse transcribed into a cDNA using Maloney murine leukemia virus reverse transcriptase (Promega, Madison, WI). The real time quantitative PCR to measure the expression of each gene was performed using the DyNAmo Hot Start SYBR Green qPCR kit (Finnzymes, Beverly, MA). The final PCR products were analyzed on a 1.5% agarose gel to check for amplification specificity. Standard curves were constructed for MyoD, myogenin, syndecan-4, glypican-1 and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) with serial dilutions of the purified PCR products from each gene. The amount of cDNA for each gene was interpolated from the corresponding standard curve. The expression of MyoD, myogenin, syndecan-4, and glypican-1 was normalized to GAPDH expression.

**Statistical analysis:** The SAS PROC GLM (SAS Institute Inc., Cary, NC) was used for statistical analyses. Age and treatment were considered as the two main factors of variance. Differences among means in each experiment were evaluated using Fisher's least significant difference. Two-sided P-values of $P<0.05$ were considered statistically significant.

## RESULTS

**Effect of feed restriction on body weight and pectoralis major muscle weight:** The feed restriction resulted in a significant decrease in BW in the birds from 1 d posthatch through 28 d of age (Fig. 1A). During the first 2 wk posthatch, the restricted birds were maintained at 80% BW. Although not significant, the reduction in BW remained through the 42 d of the study. Pectoralis major muscle weight was significantly reduced by the feed restriction (Fig. 1B). Beginning at d 14 through d 28, the pectoralis major muscle was significantly decreased in weight and the trend continued throughout the trial. For both the BW and pectoralis major muscle weights there was no interaction between age and treatment.

**Effect of feed restriction on the expression of MyoD, Myogenin, Syndecan-4, and Glypican-1:** The expression of the myogenic regulatory factors was affected during the first 4 d posthatch by the feed restriction (Fig. 2A and B). The restricted birds had a significant increase in

Table 1: Primer sequences for real-time polymerase chain reaction

| Primer | Sequence[1] | Product size |
|---|---|---|
| MyoD | 5'-GACGGCATGATGGAGTACAG-3' (Forward) | |
| | 5'-AGCTTCAGCTGGAGGGAGTA-3' (Backward) | 201 bp |
| Myogenin | 5'-GGCTTTGGAGGAGAAGGACT-3' (Forward) | |
| | 5'-CAGAGTGCTGCGTTTCAGAG-3' (Backward) | 184 bp |
| SYN 4 | 5'- CCAACAGCAGCATCTTTGAA-3' (Forward) | |
| | 5'-GATGGGTTTCTTCCCAAGGT -3' (Backward) | 234 bp |
| GPC 1 | 5'-ACATCGGGAATGATGTGGAT-3' (Forward) | |
| | 5'-AAGAGGAGGAAGGCAGAAGG-3' (Backward) | 208 bp |
| GAPDH | 5'-GAGGGTAGTGAAGGCTGCTG-3' (Forward) | |
| | 5'-CCACAACACGGTTGCTGTAT-3' (Backward) | 200 bp |

[1]Primer sequences were designed from the following GenBank accession numbers: MyoD, L34006: Myogenin D90157; Syndecan-4 (SYN 4), NM001007869; Glypican-1 (GPC 1), L29089; glyceraldehyde-3-phosphate dehydrogenase (GAPDH), U94327

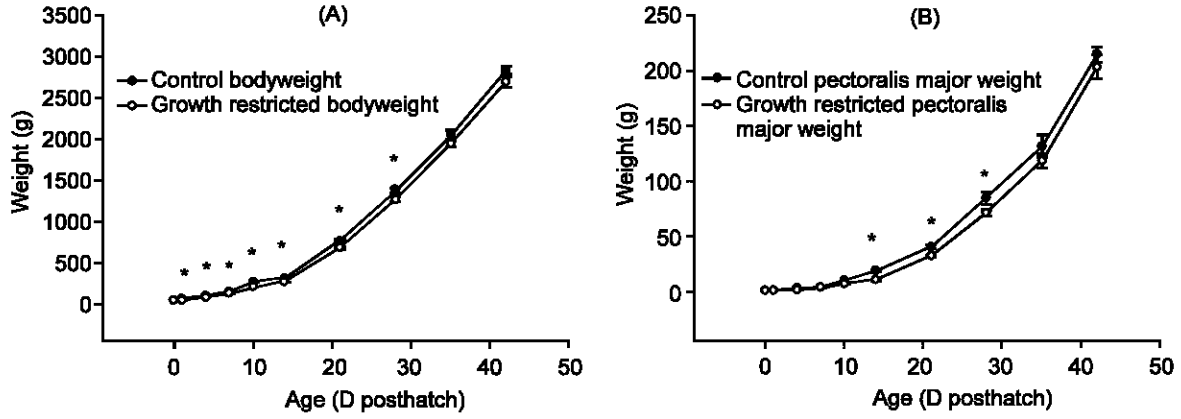


Fig. 1: Body weight and pectoralis major muscle weight in the control and growth restricted birds from hatch (d 0) through 42 d of age. A) Body weight measurements in g and B) pectoralis major muscle weight in g. Bars represent the standard error of the mean. *Indicates a significant difference ($p<0.05$)

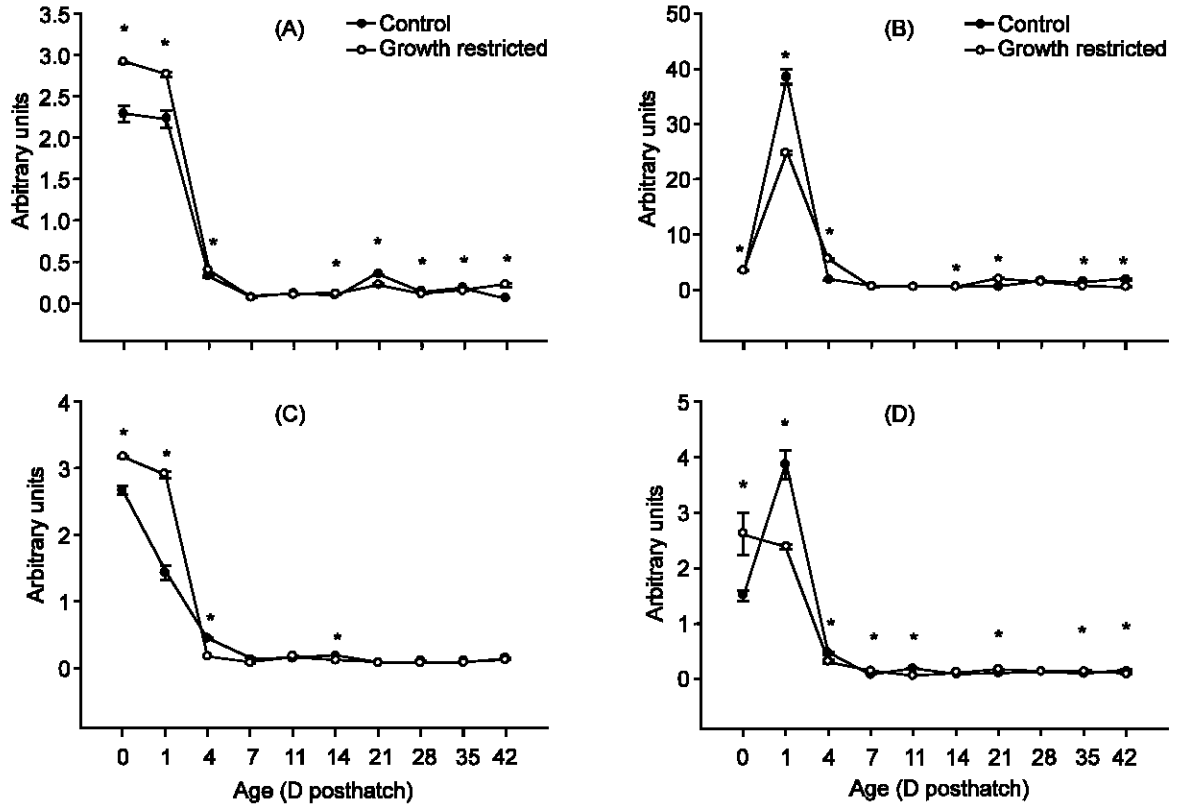


Fig. 2: Expression of MyoD (A), myogenin (B), syndecan-4 (C) and glypican-1 (D) from hatch (d 0) through 42 d of age. The bars represent the standard error of the mean. *Indicates a significant difference ($p<0.05$)

MyoD expression through d 4 posthatch compared to the unrestricted group (Fig. 2A). At 1 d posthatch, there was a spike in myogenin expression in both the fed and restricted groups (Fig. 2B). The fed group expressed about 50% more myogenin than the feed restricted birds. Similar to MyoD expression, syndecan-4 was upregulated during the first 4 d posthatch due to the growth restriction whereas glypican-1 was decreased in its expression (Fig. 2C and D). For MyoD, myogenin, and syndecan-4 expression, there was an age effect with the response to the treatment whereas with glypican-1 there was no age effect with response to the treatment.

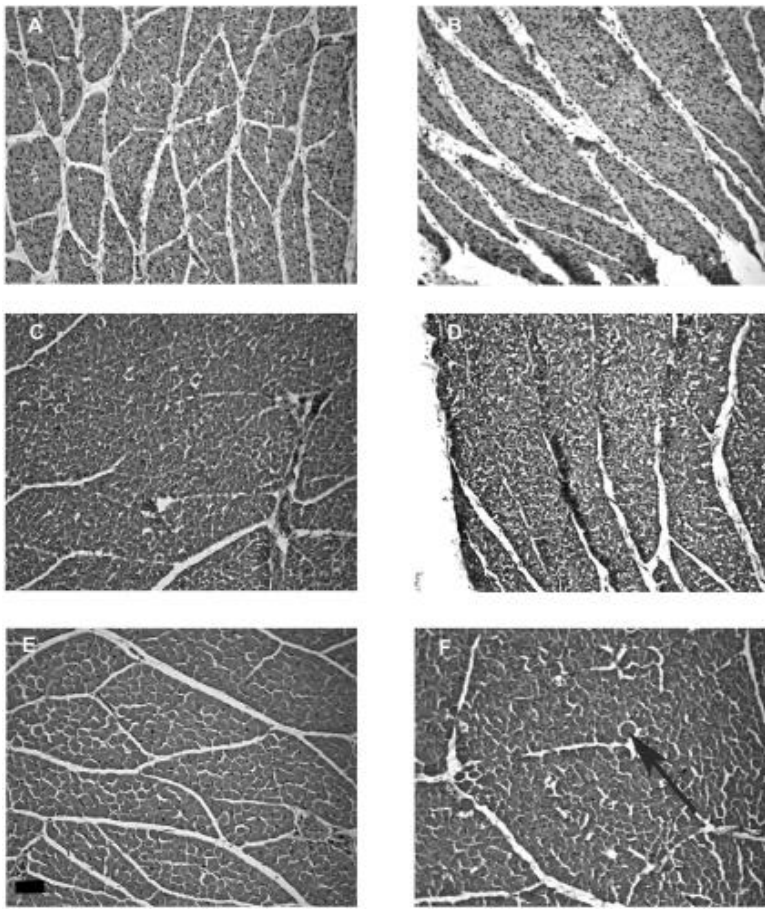


Fig. 3: Morphological structure of the pectoralis major muscle from 1 d through 7 d of age in control and growth restricted chicks. A, C and E are representative images of the control pectoralis major muscle at 1, 4 and 7 d of age. B, D and F contain representative images of the growth restricted pectoralis major muscle at 1, 4 and 7 d of age. The arrow highlights a hypercontracted muscle fiber. The scale bar represents 10 μm

**Development of the pectoralis major muscle morphological structure in the control and feed restricted groups:** Figure 3 contains representative pectoralis major muscle samples of the control and feed restricted birds at 1 d through 7 d of age. At 1 d posthatch in the control pectoralis major muscle samples the muscle fiber bundles were well organized with well defined connective tissue layers surrounding the bundles as well as the individual fibers. Day 4 showed further fiber and fiber bundle development in the control whereas fiber and fiber bundles were not well defined in the growth restricted muscle. By d 7, the individual muscle fibers and muscle fiber bundles are evenly spaced and symmetrical in the control. In the feed restricted pectoralis major muscle, the individual fibers and the fiber bundles were not uniformly spaced. At d 7 rounded muscle fibers were observed in the feed restricted pectoralis major muscle. These rounded fibers usually represent the formation of permanently contracted or hypercontracted fibers.

The difference in the developmental organization between the control and feed restricted birds continued throughout the duration of the study (data not shown). By d 35 of age the feed restricted birds had muscle fiber degeneration as well as the continued presence of hypercontracted fibers (Fig. 4 A and B). By d 42, the degeneration of the muscle fibers was increased. Muscle fiber degeneration and necrosis was not noted in the control pectoralis major muscle (Fig. 4 C and D).

**Intramuscular fat deposition in the control and feed restricted pectoralis major muscle:** The presence of intramuscular fat was noted in both the control and feed restricted birds. However, the characteristics of these fat depots were different between the two treatment groups. Intramuscular fat was measurable in the feed restricted birds beginning at d 28 (Fig. 5). In contrast, intramuscular fat was observed beginning at d 35 in the control pectoralis major muscle samples. The size of the fat cells was consistently larger in the feed restricted

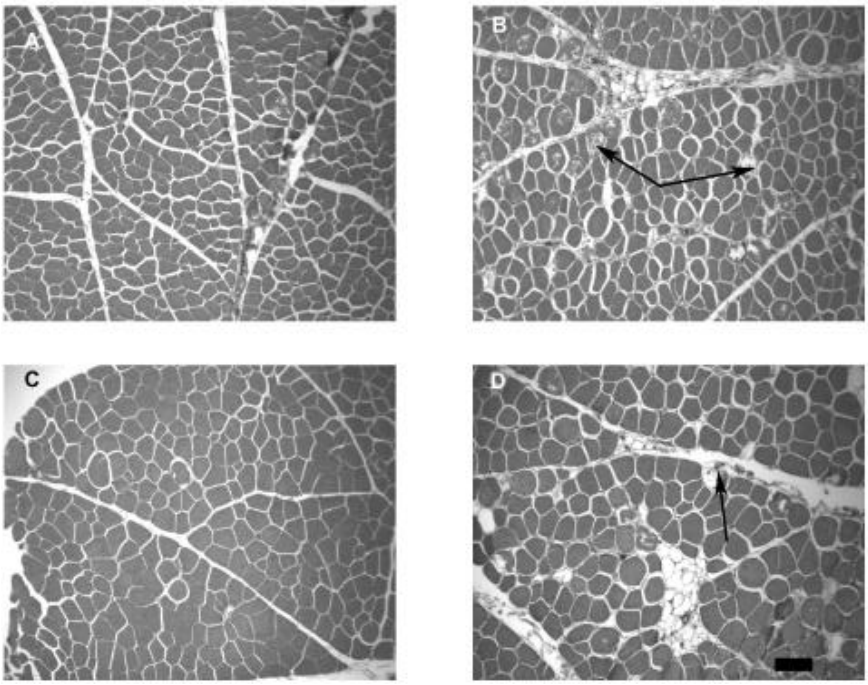


Fig. 4: Morphological structure of the pectoralis major muscle at 35 and 42 d of age in control and growth restricted chicks. A and C show the control pectoralis major muscle at 35 and 42 d of age, respectively. The morphological structure of the growth restricted pectoralis major muscle at 35 (B) and 42 (D) d of age. The arrows highlight muscle fibers undergoing lysis. The scale bar represents 10 μm

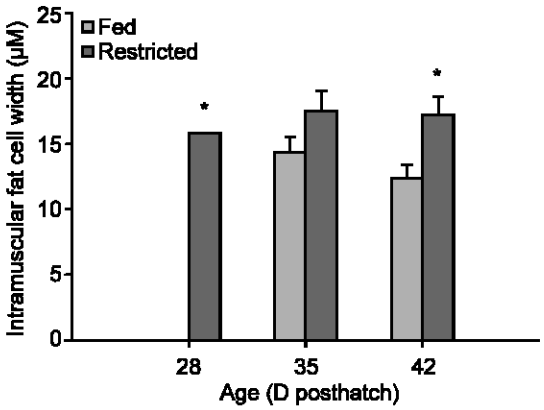


Fig. 5: Measurement of intramuscular fat cell width in control and growth restricted pectoralis major muscle at 28 through 42 d of age. Bars represent the standard error of the mean. *Indicates a significant difference ($p<0.05$)

birds compared to the control. The fat depots were also more expansive in the feed restricted muscle samples compared to the control as shown in Fig. 6 at 42 d of age. Intramuscular fat cell diameter was influence by treatment but not age and there was no interaction between age and treatment.

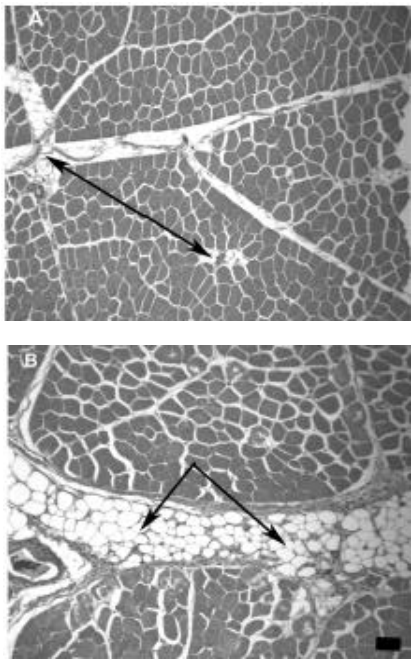


Fig. 6: Fat cell morphological distribution at 42 d of age in the A) control and B) growth restricted pectoralis major muscle. The arrows highlight fat cells. The scale bar is equal to 10 μm

## DISCUSSION

The immediate posthatch period for broilers is characterized by a period of rapid growth. During this time, muscle growth occurs through the process of hypertrophy. Hypertrophy entails the fusion of satellite cells with existing myofibers leading to increased muscle fiber size through increased protein synthesis. In conjunction with the rapid muscle growth, the bird's skeleton must develop. To allow skeletal development to occur prior to the onset of rapid muscle mass accretion to reduce the incidence of skeletal deformities like tibial dyschondroplasia, one approach used is to place the birds on a restricted diet to limit growth immediately following hatch. After the period of growth restriction, the accepted theory is that through compensatory growth mechanisms skeletal muscle growth including the pectoralis major muscle will reach normal weight levels and not be structural affected. In the present study after d 28, BW and pectoralis major muscle weight were not significantly different from the control group. If compensatory gain does occur in a manner which does not affect the biochemistry or morphological structure of the muscle, the following events must take place: 1) posthatch muscle growth mediated by satellite cell induced hypertrophy remains the same with the growth restriction; 2) growth restriction does not alter cellular fate and 3) inhibiting muscle hypertrophy will not change muscle fiber organization, mass, or impact meat quality. Each of these assumptions will be addressed individually and related to the results from the present study.

The results from the present study demonstrated that both BW and pectoralis major muscle weight were significantly reduced by the 2 wk 80% growth restriction following hatch for 28 d. The reduction in pectoralis major weight is likely due to reduced muscle hypertrophy. Myogenin is a transcriptional regulatory factor expressed during muscle cell differentiation. Myogenin expression was decreased by the feed restriction suggesting a reduction in muscle cell differentiation. Interestingly, MyoD expression was increased during the period of time that myogenin expression was reduced. Since, the muscle cells are not able to transition to differentiate it is likely that the muscle cells are compensating for this reduction in differentiation by increasing the number of muscle cells available to differentiate. Increased muscle cell proliferation as suggested by the elevation in MyoD levels does not always result in increased differentiation unless the cells receive the appropriate signals to undergo differentiation.

One type of muscle cell involved in the posthatch growth of muscle are satellite cells. Satellite cells have a limited period of activity and in broilers the first wk posthatch is the most critical for muscle growth (Moss *et al*., 1964). Halevy *et al*. (2000) showed in chicks deprived of feed for 48 h posthatch that both BW and pectoralis major muscle weight was reduced throughout the study duration of 41 d. The reduction in muscle growth was thought to be due to reduced satellite cell activity from the feed deprivation. In the current study, muscle cell and satellite cell proliferation and differentiation may be altered by the growth restriction as shown by the change in muscle transcriptional regulatory factor expression, but further studies are necessary to precisely define the proliferation and differentiation of these cells.

The second assumption of compensatory muscle growth is that restricting growth does not alter cellular fate. During development, tissues are formed from one of three germ layers: the endoderm, ectoderm, or mesoderm. These germs layers are not fixed in their cellular fate and can be induced to form a different cell type. Satellite cells are multipotential stem cells that can undergo myogenic, osteogenic, or adipogenic differentiation (Askura *et al*., 2001). The events leading to changes in cell fate are not well understood. However, it is clear that muscle cells can be induced to follow an adipogenic pathway leading to fat cell accretion and affecting the fat-to-lean muscle composition (Sordella *et al*., 2003; Quinn, 2008). It is also possible that muscle mesenchymal progenitor cells that are distinct from satellite cells may contribute to the fat cell formation observed in the growth restricted birds (Uezumi *et al*., 2010) which may have also occurred. However, the results from the current study demonstrated that intramuscular fat deposition in the pectoralis major muscle occurred in response to an immediate posthatch feed restriction.

The third assumption associated with compensatory muscle growth is that postnatal muscle growth is not altered by a growth restriction. The results from the present study show that muscle fiber organization and muscle mass accretion are affected by the feed restriction. Muscle growth after hatch occurs through hypertrophy. The gene expression, weight accretion, and morphological data all support a change in muscle cell activity with a growth restriction regimen.

The results from the present study demonstrated that using an approach to restrict growth by 20% immediately after hatch will change breast muscle development. The first 4 d after hatch appear to be the most affected with muscle transcriptional regulatory factor expression significantly modified. Halevy *et al*. (2000) used a 2 d fed deprivation to measure the effect on chick skeletal muscle growth and satellite cell proliferation. The timing after hatch of the feed deprivation influenced muscle growth. The closer the period of starvation was to hatch the lower the bird's ability to exhibit compensatory growth. The birds deprived feed from d 4 to 6 after hatch had a complete recovery in terms of muscle growth. Furthermore, Plavnik and Hurwitz (1988, 1990) reported that restrictive feeding in the second wk after hatch results in complete compensated growth.

In summary, the findings from the current study demonstrate that appropriate feeding immediately after hatch is necessary to maintain proliferation and

differentiation leading to muscle growth by hypertrophy, limiting intramuscular fat accretion, and maintaining muscle fiber and fiber bundle morphological structure. If growth restriction programs are implemented which do not achieve maximal muscle growth or maintain muscle structure, these changes could have economic implications. Further studies are needed to identify the optimal timing for growth restriction regimens after hatch in poultry that address satellite cell activity during proliferation and differentiation and the resulting morphological structure of the muscle.

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