Review

Developmental specificity in skeletal muscle of late-term avian embryos and its potential manipulation

W. Chen,*†‡ Y. T. Lv,§ H. X. Zhang,*†‡ D. Ruan,*†‡ S. Wang,*†‡ and Y. C. Lin*†‡1

*Institute of Animal Science, Guangdong Academy of Agricultural Sciences, Guangzhou 510640, China; †State Key Laboratory of Livestock and Poultry Breeding, Guangzhou 510640, China; ‡The Key Laboratory of Animal Nutrition and Feed Science (South China) of Ministry of Agriculture, Guangzhou 510640, China; and §College of Animal Science, South China Agricultural University, Guangzhou 510640, China

ABSTRACT Unlike the mammalian fetus, development of the avian embryo is independent of the maternal uterus and is potentially vulnerable to physiological and environmental stresses close to hatch. In contrast to the fetus of late gestation in mammals, skeletal muscle in avian embryos during final incubation shows differential developmental characteristics: 1) muscle mobilization (also called atrophy) is selectively enhanced in the type II fibers (pectoral muscle) but not in the type I fibers (biceps femoris and semimembranosus muscle), involving activation of ubiquitin-mediated protein degradation and suppression of $S6K_1$-mediated protein translation; 2) the proliferative activity of satellite cells is decreased in the atrophied muscle of late-term embryos but enhanced at the day of hatch, probably preparing for the postnatal growth. The mobilization of muscle may represent an adaptive response of avian embryos to external (environmental) or internal (physiological) changes, considering there are developmental transitions both in hormones and requirements for glycolytic substrates from middle-term to late-term incubation. Although the exact mechanism triggering muscle fiber atrophy is still unknown, nutritional and endocrine changes may be of importance. The atrophied muscle fiber recovers as soon as feed and water are available to the hatchling. In ovo feeding of late-term embryos has been applied to improve the nutritional status and therein enhances muscle development. Similarly, in ovo exposure to higher temperature or green light during the critical period of muscle development are also demonstrated to be potential strategies to promote pre- and posthatch muscle growth.

Key words: muscle atrophy, late-term avian embryo, manipulation

2013 Poultry Science 92:2754–2764
http://dx.doi.org/10.3382/ps.2013-03099

INTRODUCTION

Prenatal myogenesis is a complex process involving proliferation of myoblast precursors, differentiation, alignment, and subsequent fusion to form multinucleated myotubes then finally forming mature muscle fibers (Ordahl et al., 2000; Picard et al., 2002). Primary myofibers form during the initial stage of myogenesis, and secondary myofibers subsequently form during the second wave of myogenesis, the latter accounting for the majority of muscle fibers (as reviewed in Du et al., 2010). The molecular events of the myogenic pathway are controlled by myogenic regulatory factors (e.g., MyoD, Myf-5, Myogenin, and Mrf-4) and their upstream factors, Wnt and Pax, the expression of which coordinately regulates the timing of proliferation and differentiation (Anakwe et al., 2003; Kassar-Duchossoy et al., 2005). Postnatal muscle is typically characterized by the increasing size of the myofibers (hypertrophy) rather than the number of myofibers because the total number of myofibers is fixed at birth in most species (Stickland, 1978; Wigmore and Stickland, 1983; Stockdale and Miller, 1987). In avian species, the skeletal muscle undergoes development and maturation in structure and function during the incubation period. In chickens, primary myofibers form from about d 6 of incubation (as reviewed in Stockdale, 1992; Stickland et al., 2004). Secondary, or type II, myofibers are derived from a separate population of myotubes and begin differentiation between 12 to 16 d [embryonic d (**E**) 12E to 16E, hatching is 21 d; as reviewed in Stockdale, 1992; Stickland et al., 2004]. Differentiation of both primary and secondary fibers is complete by 75% of the incubation period, and the total number of myofibers is therefore determined before the final stage of avian embryonic development (Stockdale and Miller, 1987). Despite

Received February 5, 2013.
Accepted May 19, 2013.
[1]Corresponding author: lyc0123@tom.com

the similarities in myogenesis in the mammalian fetus and avian embryo, differences in function and metabolism would be expected because avian embryos grow in a more confined space and the process of hatching demands active participation of muscles and attendant metabolic support. Relatively little is known about the metabolism and its regulation in avian embryos during the last quarter of incubation. There has been, however, recent interest and progress in defining and understanding the molecular events in the skeletal muscle of avian embryos.

This review provides a summary of recent developments in the field of muscle development and function in late-term avian embryos with a focus on the process of atrophy, along with effects of nutritional and environmental manipulation on pre- and posthatch muscle development.

Pectoral Muscle Development in Maturing Avian Embryos Is Morphologically and Molecularly Characterized by Atrophy

After the completion of myogenesis, it was generally expected that a phase dominated by hypertrophy would occur in the final quarter of incubation. The unexpected findings of a decreased percentage of pectoral mass in duck embryos toward the end of incubation (Chen et al., 2012) was also supported by the demonstration of decreased cross-sectional area of myofibers in pectoralis muscle with age of late-term turkey embryos (Moore et al., 2005a). These results suggest that this muscle may undergo an atypical development in the final days of incubation from what occurs posthatch when there is obvious hypertrophy (Remignon et al., 1995). These surprising findings implied that muscle mobilization (or "atrophy," as used in this review) occurs in late-term avian embryos, particularly in the turkey and duck embryos.

Although there has been several recent studies on muscle atrophy, no definitive explanation for the process has been described. Atrophy is descriptive of decreased cell size mainly caused by loss of organelles, cytoplasm, and protein (as reviewed in Sandri, 2008), and muscle atrophy is morphologically indicated by the changes in the myofibers, especially the reduction in their diameter and overall muscle mass (Ebert et al., 2010). The transcription of E3-ligase, the group O family (**FoxO**) of forkhead transcription factors, and nuclear factor kappa B are key elements involved in the signaling pathway involved in the induction of skeletal muscle atrophy (Sandri et al., 2004; Van Der Heide et al., 2004; Glass and Roubenoff, 2010; Braun and Gautel, 2011; Nagatomo et al., 2011). Because of their importance in mediating or inducing muscle atrophy, these signaling proteins have been designated as "atrophy-related factors" in many reports. It is likely, therefore, that the changes both in histology and in these signaling proteins provide indicators of atrophy at the morphological and molecular levels.

We previously (Chen et al., 2012) found that myofiber size in pectoral muscle of duck embryos decreased by 55% from 22E to hatching (d 28); this is probable morphological evidence of muscle fiber atrophy in this species. We evaluated the muscle atrophy-related signaling protein in late-term embryos to confirm the incidence of muscle atrophy. In support of this finding, the transcript abundance of Atrogin-1, an atrophy-related gene, increased 70-fold from 22E to hatching. This finding is suggestive of enhanced protein degradation through the ubiquitin-proteasome system because Atrogin-1 is an important mediator of E3-ligase. This degradation system accounts for most (<80%) intracellular turnover of proteins in all tissues (Gomes et al., 2001; Lecker et al., 2004, 2006). Relative expression of one of the key transcriptional factors, $FoxO_1$, showed similar changes during the final incubation period (Chen et al., 2012). In contrast, the cytoplasmic Ser-Thr kinase, p70 S6, vital in protein synthesis and in determining cell size, decreased to 30% the activity at 22E during the same period, indicating impaired mRNA translation and protein synthesis. This kinase is activated by nutrient levels and insulin-like growth factors (**IGF**), and is essential for mediating the control of muscle cytoplasmic volume by the integrating regulator mTOR (Figure 1, as reviewed in Sandri, 2008; Shavlakadze et al., 2010). It phosphorylates ribosomal protein S6 and thus regulates the translation of specific mRNA that encode essential components of the protein synthetic apparatus (Holz et al., 2005). Depletion of S6K1 impairs muscle growth and induces atrophy (Ohanna et al., 2005; Aguilar et al., 2007), so it is an important regulator of muscle atrophy.

Our research, along with that of others, has demonstrated enhanced atrophy-related signaling and its morphological consequences in the pectoral muscle in the final stages of embryonic development in poultry. Muscle development in the late-term poultry embryo (chicken, turkey, duck) clearly contrasts with that in mammalian fetuses at corresponding stages, with their considerable increase in muscle mass (Swatland, 1973).

Factors Possibly Triggering Muscle Atrophy

Atrophy of skeletal muscle is usually a debilitating response to malnourishment (Ebert et al., 2010), disuse (Nagano et al., 2008; Levine et al., 2011), cancer (Burckart et al., 2010), or other systemic diseases (Price et al., 2010; Sishi et al., 2011). Its induction is associated with increased protein degradation and suppressed or inhibited protein synthesis, leading to net muscle mobilization or wastage. The capacity to mobilize protein from skeletal muscle is an important resource of amino acids, playing a role during fasting and in some diseases. In the catabolic state, protein from atrophy-

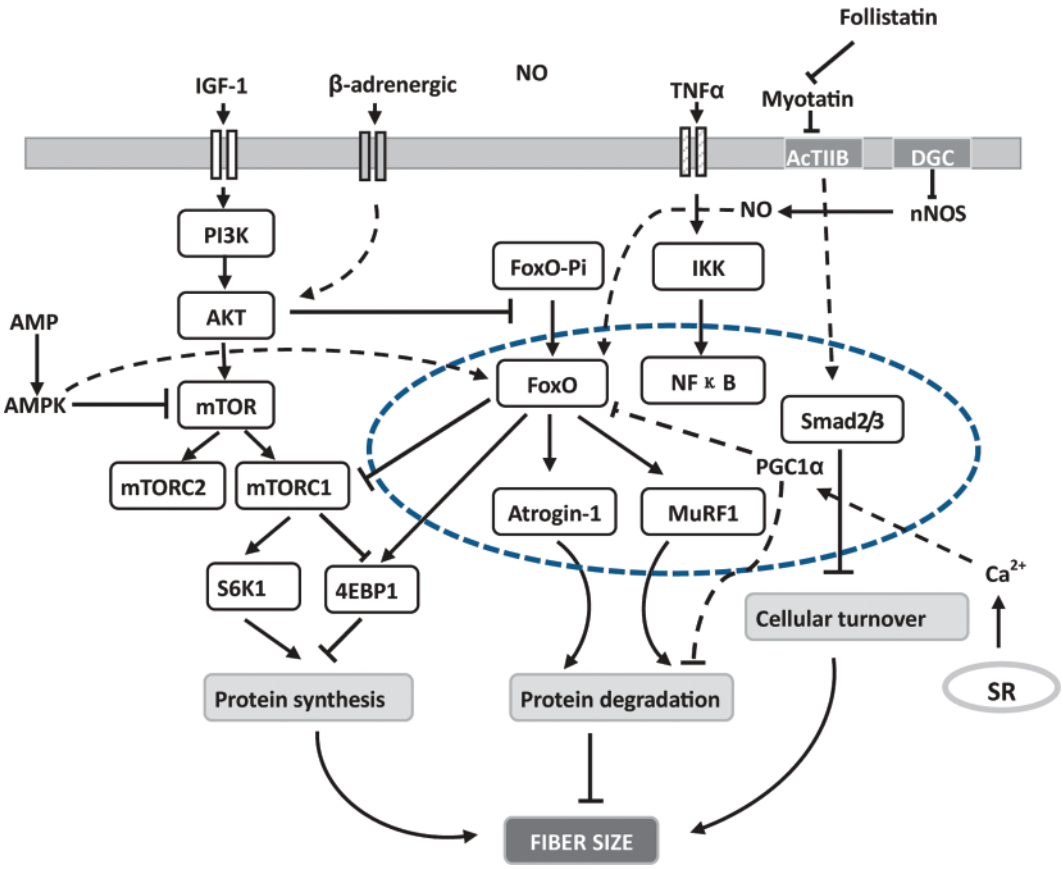

Figure 1. Scheme illustrating the major signaling pathways that control fiber size (adapted from Sandri et al., 2008). Dotted lines depict pathways whose molecular mechanisms and role in skeletal muscle have yet to be completely defined. PI3K, phosphatidylinositol-3 kinase; mTOR, mammalian target of rapamycin; mTORC, mTOR complex; Akt, protein kinase B; S6K1, p70 S6 kinase-1; 4EBP1, 4E binding protein 1; FoxO, group O family of forkhead transcription factors; ActRIIB, activin receptor; DGC, dystrophin glycoprotein complex; IKK, inhibitor of nuclear transcription factor κB kinase; NFκB, nuclear factor kappa B; MuRF1, muscle ring factor-1; PGC1α, peroxisome proliferator-activated receptor γ coactivator-1 α; NO, nitric oxide; nNOS, neuronal nitric oxide synthase; AMPK, AMP activated kinase; SR, sarcoplasmic reticulum. Color version available in the online PDF.

ing muscle is mobilized primarily by activating signaling of the ubiquitin-proteasome pathway (Mitch and Goldberg, 1996; Lecker et al., 2006) to provide energy via gluconeogenesis and essential amino acids for the synthesis of acute proteins in splanchnic tissues (Nair et al., 1995). Mobilization of muscle protein is therefore an adaptive response that enables provision of critical energy and protein precursors in catabolic states.

Generally, muscle atrophy is not common in the developing mammalian fetus, but long-term maternal nutrient restriction alters the developmental profile of muscle in the offspring (Zhu et al., 2004, 2006; Fahey et al., 2005; Daniel et al., 2007), suggesting that nutritional support, along with genetics, contributes to the developmental characteristics of muscle in the fetus. Similarly, the remaining yolk sac appears to be inadequate in meeting the full nutritional requirement of very mature avian embryos with their hatching activities (Uni et al., 2005; Kornasio et al., 2011). In this regard, it is likely that the development of pectoralis muscle, with 90% type-IIB fibers (Wiskus et al., 1976), may be vulnerable to the nutritional status of late-term avian embryos.

Glucose-Derived Energy Deficiency

The phosphorylation status of the AMP-activated kinase protein, a key sensor of cellular energy status, was enhanced in the pectoralis muscle of late-term duck embryos (Chen et al., 2012), which indicates energy deficiency because AMP-activated kinase protein is phosphorylated and activated by a low adenosine triphosphate/adenosine diphosphate ratio (Xiao et al., 2011). This change may reflect an energy crisis induced by restricted oxygen availability and increasing somatic activity with the approach or progress of hatching. Although lipids, mostly in the yolk, are the main energy source during incubation (as reviewed in De Oliveira et al., 2008), there is increased anaerobic glycolysis during the hatching process, because oxygen availability is limited (as reviewed in Moran, 2007; De Oliveira et al., 2013). Some tissues, especially the pipping muscle that is most active at this time, exclusively use glycolysis from glucose provided from glycogen reserves; a high demand for glucose occurs at this time. Increased plasma concentrations of glucose, and therefore net glucose output from other tissues (liver, muscle, yolk sac),

becomes necessary and is critical for energy shuttling and the very survival of the hatching embryo (Lu et al., 2007; Chen et al., 2010; Yadgary and Uni, 2012). Embryos, at this time, use their energy reserves, glycogen in particular, as the fuel for hatching activities (Christensen et al., 2001; Uni et al., 2005) and to proliferate lymphocytes (Rudrappa and Humphrey, 2007). These physiological demands for glucose during this vital transition cannot be met from yolk reserves (<1% remaining), necessitating increased gluconeogenesis (Foye et al., 2007; Moran, 2007) and mobilization of stored glycogen from liver and pectoral muscle (Vieira and Moran, 1999; Zhai et al., 2011). In this case, the glycolytic energy was balanced under the coordination of increased activity of gluconeogeneic enzymes (Foye et al., 2007; Moran, 2007).

The energy deficiency experienced by the hatching avian embryo triggers the signaling pathway, leading to muscle atrophy. Further supporting this hypothesis was the demonstration that supplying exogenous glucose substrate to late-term turkey embryos spares mobilization from the pectoral muscle (Uni et al., 2005). An additional burden during the final period of the incubation stems from the rapid development of the small intestine (Uni et al., 2003) with increased need for the amino acid glutamine, the main energy substrate for proliferating enterocytes (van der Schoor et al., 2010); pectoral muscle is driven to mobilize protein to provide endogenous glutamine, accounting for 90% of the release to blood (as reviewed in Newsholme, 2001).

Hormonal Factors

Hormones also play a potential role in affecting skeletal muscle in late-term avian embryos. During the final stage of avian embryos, there are increased plasma triiodothyronine (**T3**) and thyroxin (**T4**) levels (Piestun et al., 2009a; Willemsen et al., 2010), as well as increased insulin concentrations (Lu et al., 2007). These increased hormones seem to be able to promote the hypertrophy of myofibers. However, circulating levels of IGF-1 (mainly the extrahepatic origin, Serrano et al., 1990; Kikuchi et al., 1991; Burnside and Cogburn, 1992; McMurtry et al., 1997) as well as gene expression in pectoral muscle are steadily decreased toward hatch (McMurtry et al., 1998; Guernec et al., 2003; Lu et al., 2007). The IGF-1 is shown to be an important anabolic growth factor stimulating hypertrophy of myofibers in both avian species (Stitt et al., 2004; Liu et al., 2012) and mammals (Shavlakadze et al., 2010). In part, IGF-1 stimulates growth of myofibers by suppressing expression of Atrogin-1 and activating the phosphatidylinositol 3-kinase (PI3K)–Akt pathway, whereas inhibition to this pathway increased expression of Atrogin-1 and proteolysis (Sacheck et al., 2004). The reduction in circulating IGF-1 in late-term embryos would be expected to remove suppression to protein breakdown and enhance mobilization of the muscle. Additionally, lack of IGF-1 secretion has been demonstrated to impair the insulin sensitivity in skeletal muscle, an important activator of the protein synthesis signaling pathway (Yakar et al., 2001). Possible involvement of other hormones and growth factors in late-term avian embryos, for example a catabolic role for increasing concentrations of glucocorticoids at this time, cannot be dismissed in accounting for the atrophic changes described above.

SPECIFICITY OF CHANGES IN THE PECTORAL MUSCLE DURING LATE-TERM INCUBATION

Selective Atrophy in Type II Pectoral Muscle

It is of great interest to determine if the growth dynamics of biceps femoris and semimembranosus muscle contrast to that already described in pectoralis. The biceps femoris and semimembranosus consist mainly of type-I fibers and display hypertrophy toward to the end of incubation, as reflected by dramatically increased diameter of the myofibers from 15E (Stojanović et al., 2009) or 16E (Henry and Burke, 1998) to hatching in chickens. This differential growth of biceps femoris and semimembranosus occurs despite the glucose deficit or the decreased circulating concentrations of IGF-1, already noted. It might be suggested that there is selective mobilization of muscle with predominantly type-IIB myofibers during the late incubation period of the avian embryo. In the relevant medical literature, type-II muscle fibers are more susceptible to atrophy than are type-I fibers in muscle denervation (Bakou et al., 1996), osteoporosis (Terracciano et al., 2013), chronic heart failure (Li et al., 2007), and obstructive pulmonary disease (Gosker et al., 2002). The content of satellite cells is also specifically reduced in the atrophied type-II muscle fiber (Verdijk et al., 2007). It seems, therefore, that type-I fibers are resistant to atrophy-inducing factors and that type-II fibers are preferentially affected.

It remains a puzzle why muscle atrophy predominantly involves glycolytic, type-II myofibers and typically spares the oxidative, type-I fibers in a range of conditions. Type-I fibers contain more mitochondria and have higher rates of oxidative metabolism compared with fast-twitch type-II fibers, especially the type-IIB fibers, which have relatively few mitochondria and are predominately glycolytic. The 2 types of fibers show differential functional and structural responses to exercise-training and energy metabolism through a calcium-induced and nuclear-signaling pathway (Pearen et al., 2012). For type-IIB fibers, there may be more oxidative stress because mitochondrial production and leakage of free radicals is 2- to 3-fold higher than in type-I fibers, while also possessing lower H_2O_2-scavenging capacity (Anderson and Neufer, 2006). In contrast, compared with type-II fibers, type-I fibers possess an intact antioxidant system that could protect the myofibers from oxidative stress (Yu et al., 2008). A novel Fyn/STAT3/

Vps34 signaling pathway was recently demonstrated to be responsible for fiber-type-specific regulation of macroautophagy and atrophy in muscle (Yamada et al., 2012). Activation of this signaling pathway primarily involves the glycolytic fibers with little effect on oxidative muscle fibers, giving some insight into the selective atrophy noted in type-II fibers. The relative preservation of type-I fiber size is advantageous because energy expenditure per unit tension developed is slowest in slow-twitch fibers (Henriksson, 1990). Oxidative myofibers possess an intact antioxidant system, mediated by NO signaling and transcription system (Yu et al., 2008) that serves to protect the myofibers from oxidative stress. Additional protection against atrophy exists in slow-twitch type-I fibers such as peroxisome proliferator-activated receptor γ coactivator-1 α (**PGC-1α**), examples of transcriptional coactivators. With high expression in type-I fibers, PGC-1α controls mitochondrial biogenesis in muscle (Rowe et al., 2012) and mediates fiber type switching and determination (Lin et al., 2002; Arany et al., 2007). Activation of PGC-1α promotes cellular defense of antioxidant (Geng et al., 2011) and reduces $FoxO_3$-dependent transcription of Atrogin-1 and muscle atrophy, indicating its involvement in resistance to muscle atrophy (Sandri et al., 2006).

The Declined Mitotic Ability of Satellite Cells in Late-Term Muscle Atrophy

The growth and repair of skeletal muscle postnatally are enabled primarily by proliferation of mononucleated satellite cells, located between the basal lamina and sarcolemma of the myofibers (Kuang et al., 2007). Hyperplasia of satellite cells is crucial for muscle repair and myonuclear accretion (Kawano et al., 2008) and underlie hypertrophy of the postnatal myofiber (as reviewed in Yablonka-Reuveni, 2011) because that satellite cells are capable of fusing into existing or forming new muscle fibers (Starkey et al., 2011). Proliferation and differentiation of satellite cells are affected by an array of intrinsic and extrinsic factors including nutrition (Halevy et al., 2000, 2003; Moore et al., 2005b), IGF-1 (McFarland et al., 1993), transforming growth factor-β (**TGF-β**; Li et al., 2006), myostatin (Leshem et al., 2000; McFarland et al., 2006), hepatocyte growth factor (Zeng et al., 2002), fibroblast growth factors (**FGF**; Velleman et al., 2008), and estradiol (McFarland et al., 2013).

The decreased numbers of satellite cells and their declined activation is commonly encountered in various models of muscle atrophy (Mitchell and Pavlath, 2004; Zhang et al., 2010; Morgan and Zammit, 2010; Verdijk et al., 2012). The dysfunction of satellite cells in atrophied muscle may lead to a delay in the repair and subsequent regrowth of muscle. Similarly, the proliferative activity and number of satellite cells in the pectoral muscle decreased with the age of late-term avian embryos (Henry and Burke, 1998; Piestun et al., 2009b). But the mitotic activity of satellite cells was enhanced at the day of hatch (Moore et al., 2005a). The increased proliferation of satellite cells at this time more likely is in preparation for the dramatic hypertrophy that occurs posthatch, when exogenous feed becomes available (Remignon et al., 1995; Chen et al., 2012). In contrast to this situation, there is a delayed recovery of muscle mass in other models of muscle atrophy (Lang et al., 2012). An interesting study with eared grebes (*Podiceps nigricolllis*) that spend several months at Mono Lake, California, during spring migration showed atrophy of the flight muscle in birds that become flightless during this period. In late autumn, when feed supply declines, the birds engage in conspicuous flapping and the flight muscles are rebuilt to full size before the birds emigrate (Gaunt et al., 1990). Given the previous analysis in this review, it appears that selective enhancement of the muscle atrophy signaling pathway in late-term avian embryos is an adaptive response and does not reflect anything dysfunctional or pathological. The pectorals play no critical life-support role immediately upon hatching, they have mainly type-II myofibers and readily undergo atrophy that is subsequently reversible. The pectorals serve as a source of glucose and amino acids from osmotically favorable storage forms as glycogen and protein, and they support the higher priority needs of other tissues at this time of transition from embryonic to posthatch life.

Manipulation of Muscle Dynamics

Muscle development in embryos, to a large extent, predetermines its postnatal growth. Given the specific characteristics of muscle growth, development and turnover in the final stage of incubation, discussed above, there is interest in the opportunities for modifying this normal process of atrophy to potentially promote muscle growth of poultry both pre-and posthatching.

In Ovo Feeding

Uni et al. (2005) established methodology for providing nutrients and other regulatory materials to the amniotic fluid in late-term avian embryos, also called in ovo feeding (**IOF**). Administration of exogenous sugar and amino acids (glutamine and arginine) via IOF of late-term poultry embryos improves energy status (Chen et al., 2010) and offsets muscle loss (Uni et al., 2005). Administration of carbohydrates and β-hydroxy-β-methylbutyrate-calcium increased hepatic content of glycogen in late-term embryos and protected liver glycogen from the severe depletion caused by a 36-h fast immediately after hatching (Kornasio et al., 2011). As a consequence of improved energy status from IOF, there was enhanced growth of myofibers in the embryos and it was sustained to d 35 (Kornasio et al., 2011), indicating a long-term supportive effect on posthatch muscle

growth. Considering the stimulatory role of glucose in regulating IGF-1 and insulin secretion (Martinez et al., 2006), the exogenous carbohydrate probably downregulates the ubiquitin pathway (Figure 2, unpublished data in our laboratory) and enhances muscle hypertrophy (Mikura et al., 2009). Exogenous carbohydrate, provided by IOF, enhanced the proliferation of myoblasts at 19E in chick embryos, and this effect was pronounced, especially in the birds receiving early feeding (6 h posthatch, feeding) and remained the high even as myoblast proliferation declined (Kornasio et al., 2011).

Muscle regulators are prospective candidates for modulating muscle development in avian embryos using in ovo treatment. Injection of exogenous TGF-β1, an extracellular matrix factor (as reviewed in Velleman, 2007), at 3E significantly reduced the weight of pectoralis major muscle at 1 wk posthatch and decreased the number of myofibers in the early posthatch days, probably through altering development of the perimysium in the embryos (Li and Velleman, 2009). Additionally, IOF of IGF-1 to late-term duck embryos, for example, promoted hypertrophy of muscle fibers in embryos and neonates through activation of satellite cells (Liu et al., 2012).

Of course, embryos can be stressed by in ovo injection procedures. This is particularly obvious in duck embryos because of their greater sensitivity to incubation humidity. Additionally, osmotic considerations when administering different substances, are important in influencing the survival of embryos (Ferket et al., 2005). At present, possible species specificity and substances used for in ovo injection must be given serious consideration if this method is to be applied to practical production.

ENVIRONMENTAL MANIPULATION

Light

The light spectrum affects growth performance of birds. Chickens selected for growth that were reared under blue or green fluorescent lamps had significantly higher BW than birds reared under red or white light (Wabeck and Skoglund, 1974). Embryonic development is also affected by in ovo illumination. Under regular conditions in hatcheries, eggs are incubated in darkness. Early studies indicate that white-illumination accelerates embryonic development of several avian species and continuous photostimulation during incubation augments embryonic development and accelerates hatching in birds (Coleman and McDaniel, 1976). When fertile eggs received monochromatic green light from 5E until hatching, relative growth of the pectoral muscle was enhanced throughout the incubation period and was sustained to 42-d market age (Rozenboim et al., 2004). This positive effect seems muscle-specific, considering that the positive effect of green-light stimulation on muscle growth preceded its effect on BW in the embryos and was evident at almost all days of incubation (Rozenboim et al., 2004).

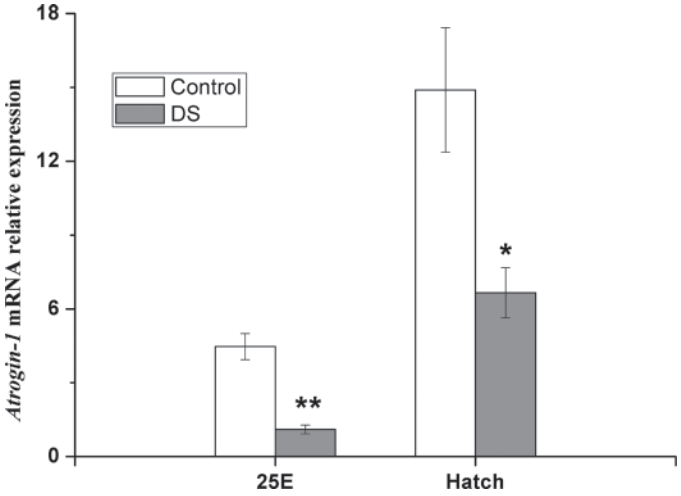

Figure 2. Changes in the Atrogin-1 mRNA expression in the pectoralis muscle following in ovo injection of DS (1.2 mL/egg) to duck embryos of 23 d of incubation. DS, disaccharides (consisting of 25 g of maltose/L, 25 g of sucrose/L, and 5 g of NaCl/L); 25E, 25 d of incubation. Values are means ± SE (n = 6). Asterisks indicate a significant difference from uninjected control ducks ($*P < 0.05$, $**P < 0.01$).

The transition from fetal- to adult-type myoblasts, normally occurring in late stages of chicken embryogenesis, is initiated earlier in embryos subjected to in ovo green-light illumination. It has been suggested that this stimulatory effect on posthatch muscle growth was probably due to enhanced proliferation of adult-type myoblasts and myofiber synchronization (Halevy et al., 2006a; Liu et al., 2010). Green light enhanced expression of pax7 and myogenin, which occurs sequentially only in activated satellite cells (Yablonka-Reuveni and Paterson, 2001; Allouh et al., 2008) and are considered to be early markers of myogenesis during posthatch muscle growth (Seale et al., 2000; Halevy et al., 2004; Oustanina et al., 2004). The available evidence indicates that exposure of eggs to green light during embryonesis affected both pre- and posthatch muscle growth by promoting proliferation of satellite cells. It has been suggested (Halevy et al., 2006a) that monochromatic green light penetrates the eggshell and has an indirect stimulatory effect on muscle in the embryos via the endocrine system. There is higher expression of growth hormone receptor in satellite cells and higher circulating concentrations of IGF-1 in green-light-illuminated chicks (Halevy et al., 1998; Liu et al., 2010).

Thermal Regulation

Incubation temperature is an important factor influencing embryo development, survival, and posthatch performance (Lourens et al., 2005). Thermal manipulation (**TM**) of chick embryos at critical phases of the development of various functional systems resulted in short-term (e.g., changed embryonic energy metabolism) (Willemsen et al., 2011) or long-term effects

(e.g., improved thermoregulatory function; Yahav et al., 2004; Piestun et al., 2009a). Treatment using TM of late-term embryos has the greatest effect on muscle growth. An increase of 1.7°C from normal conditions for 3 or 6 h daily from 16E to 18E promoted hypertrophy of muscle fibers at d 13 and 35 of age (Piestun et al., 2009b). The enhanced muscle growth following late-term embryonic TM is due to immediate, as well as long-lasting, effects on proliferation and differentiation of myogenic cells and subsequent hypertrophy (Piestun et al., 2009b).

The effects of TM on postnatal muscle growth are differential concerning the varied TM procedure. Intermittent TM (39.5°C for 12 h per day) from E7 to E16 increased the breast muscle weight in both male and female broilers at 70 d posthatch (Piestun et al., 2013a). Short-term TM (E0 to E5) increased the relative breast muscle weight in the female broilers of 35 d of age but had no effect in male broilers (Piestun et al., 2013b). Similarly, TM on E16 to E18, using 3 h at 39.5°C, increased the relative weight of pectoralis in female broilers at 42 d of age but exerted no effect in male broiler (Collin et al., 2007). This indicates that combinations of the timing and duration of the manipulation are critical for processes that underlie muscle growth regulation in avian species (as reviewed in Halevy et al., 2006b).

Because the avian embryos exhibit neuronal hypothalamic thermosensitivity (Tzschentke and Basta, 2002; Tzschentke, 2007), varied incubation temperature may lead to significant and long-term alternation in the postnatal behavior (Tzschentke and Plagemann, 2006) of the endocrine system (Loh et al., 2004). Increased incubation temperature during early or late-term embryonic period caused significant changes in plasma concentrations of thyroid hormone (Yahav et al., 2004; Piestun et al., 2009a) and IGF-1 level in the muscle (Halevy et al., 2001; Piestun et al., 2009b). Additionally, thermal regulation was reported to affect long-term thermosensitivity of neurons in the preoptic anterior hypothalamus (Loh et al., 2004). It is reasonable to speculate that TM may influence muscle hypertrophy or attenuate atrophy signaling pathway via an endocrine mechanism.

Strain or breed differences were shown to exist in the response to early TM (Al-Musawi et al., 2012). In layers, raising incubation temperature from 37.5°C to 38.5°C between 4E and 7E increased hypertrophy of the gastrocnemius muscle, increased numbers of fibers and nuclei and the nuclei:fiber ratio at 18E, preceded by increased hindlimb expression of *Myf5* (5E-8E), *Pax7* (5E-10E), bone morphogenesis protein-4 (*BMP4*; 6E-9E), and *IGF-1* mRNA (18E; Al-Musawi et al., 2012). In broilers, however, the same TM led to reduced cross-sectional area of the gastrocnemius with fewer fibers and nuclei without change in the nuclei:fiber ratio; peak expression of *Myf5* was delayed, that of *Pax7* increased (5E, 7–10E), as did *BMP4* (6–8E), but transcription of IGF-1 was reduced (8–10E; Al-Musawi et al., 2012).

CONCLUSIONS

Collectively, selective mobilization (atrophy) was enhanced specifically in type-II rather than type-I muscle fibers during the final stage of incubation. Though selective atrophy is reversible in neonates, the recovery of atrophied muscle would be delayed in the newly hatched birds that have no immediate access to feed or water in today's commercial hatchery, resulting in impaired growth potential of muscle (Halevy et al., 2000). While the precise mechanisms mediating response to manipulation of light, temperature, and nutrition during incubation are still uncertain, these environmental and nutritional treatments during embryonic development or in the early posthatch period deserve much further study. Clearly, the ease with which they can be applied and their noninvasive nature means that they can be readily transferred to practical application once their benefits in enhancing muscle growth are firmly established.

ACKNOWLEDGMENTS

We sincerely thank W. Bruce Currie (emeritus professor, Cornell University, Ithaca, NY) for his help in presentation of this manuscript. This work was supported by the Fund for China Agricultural Research System (CARS-43-13) and Presidential Foundation of the Guangdong Academy of Agricultural Sciences (201212), Guangzhou, P. R. China.

REFERENCES

Aguilar, V., S. Alliouachene, A. Sotiropoulos, A. Sobering, Y. Athea, F. Djouadi, S. Miraux, E. Thiaudière, M. Foretz, B. Viollet, J. Bastin, P. Benit, and P. Rustin. 2007. S6 kinase depletion suppresses muscle growth adaptions to nutrient availability by activating AMP kinase. Cell Metab. 6:476–487.

Allouh, M. Z., Z. Yablonka-Reuveni, and B. W. C. Rosser. 2008. Pax7 reveals a greater frequency and concentration of satellite cells at the ends of growing skeletal muscle fibers. J. Histochem. Cytochem. 56:77–87.

Al-Musawi, S. L., N. C. Stickland, and S. A. Bayol. 2012. In ovo temperature manipulation differentially influences limb musculoskeletal development in two lines of chick embryos selected for divergent growth rates. J. Exp. Biol. 215:1594–1604.

Anakwe, K., L. Robson, J. Hadley, P. Buxton, V. Church, S. Allen, C. Hartmann, B. Harfe, T. Nohno, A. M. Brown, D. J. Evans, and P. Francis-West. 2003. Wnt signalling regulates myogenic differentiation in the developing avian wing. Development 130:3503–3514.

Anderson, E. J., and P. D. Neufer. 2006. Type II skeletal myofibers possess unique properties that potentiate mitochondrial H_2O_2 generation. Am. J. Physiol. Cell Physiol. 290:844–851.

Arany, Z., N. Lebrasseur, C. Morris, E. Smith, W. Yang, Y. H. Ma, S. Chin, and B. M. Spiegelman. 2007. The transcriptional coactivator PGC-1β drives the formation of oxidative type IIX fibers in skeletal muscle. Cell Metab. 5:35–46.

Bakou, S., Y. Cherel, B. Gabinaud, L. Guigand, and M. Wyers. 1996. Type-specific changes in fibre size and satellite cell activation following muscle denervation in two strains of turkey (*Meleagris gallopavo*). J. Anat. 188:677–691.

Braun, T., and M. Gautel. 2011. Transcriptional mechanisms regulating skeletal muscle differentiation, growth and homeostasis. Nat. Rev. Mol. Cell Biol. 12:349–361.

Burckart, K., S. Beca, R. J. Urban, and M. Scheffield-Moore. 2010. Pathogenesis of muscle wasting in cancer cachexia: Targeted anabolic and anticatabolic therapies. Curr. Opin. Clin. Nutr. Metab. Care 13:410–416.

Burnside, J., and L. A. Cogburn. 1992. Developmental expression of hepatic growth hormone receptor and insulin-like growth factor-I mRNA in the chicken. Mol. Cell. Endocrinol. 89:91–96.

Chen, W., M. Tangara, J. Xu, and J. Peng. 2012. Developmental transition of pectoral muscle from atrophy in late-term duck embryos to hypertrophy in neonates. Exp. Physiol. 97:861–872.

Chen, W., J. Xu, M. Tangara, and J. Peng. 2010. Effects of in ovo injecting disaccharides and alanyl-glutamine dipeptide on the energy status in duck embryos and neonates. Anim. Reprod. Sci. 122:29–35.

Christensen, V. L., M. J. Wineland, G. M. Fasenko, and W. E. Donaldson. 2001. Egg storage effects on plasma glucose and supply and demand tissue glycogen concentration of broiler embryos. Poult. Sci. 80:1729–1735.

Coleman, M. A., and G. R. McDaniel. 1976. Light alterated changes in the embryonic age versus incubation age of white leghorn embryos. Poult. Sci. 55:2483–2485.

Collin, A., C. Berry, S. Tesseraud, F. E. Redon, S. Skiba-Cassy, S. Crochet, M. J. Duclos, N. Rideau, K. Tona, J. Buyse, V. Bruggeman, E. Decuyper, M. Picard, and S. Yahav. 2007. Effects of thermal manipulation during early and late embryogenesis on thermotolerance and breast muscle characteristics in broiler chickens. Poult. Sci. 86:795–800.

Daniel, Z. C. T. R., J. M. Brameld, J. Craigon, N. D. Scollan, and P. J. Buttery. 2007. Effect of maternal dietary restriction during pregnancy on lamb carcass characteristics and muscle fiber composition. J. Anim. Sci. 85:1565–1576.

De Oliveira, J. E., S. Druyan, Z. Uni, C. M. Ashwell, and P. R. Ferket. 2013. Metabolic profiling of late-term turkey embryos by microarray. Poult. Sci. 92:1011–1028.

De Oliveira, J. E., Z. Uni, and P. R. Ferket. 2008. Important metabolic pathways in poultry embryos prior to hatch. World's Poult. Sci. 64:488–499.

Du, M., J. Tong, J. Zhao, K. R. Underwood, M. Zhu, S. P. Ford, and P. W. Nathanielsze. 2010. Fetal programming of skeletal muscle development in ruminant animals. J. Anim. Sci. 88:E51–E60.

Ebert, S. M., A. M. Montevs, D. K. Fox, K. S. Bongers, B. E. Shields, S. E. Malmberg, B. L. Davidson, M. Suneja, and C. M. Adams. 2010. The transcription factor ATF4 promotes skeletal myofiber atrophy during fasting. Mol. Endocrinol. 24:790–799.

Fahey, A. J., J. M. Brameld, T. Parr, and P. J. Buttery. 2005. The effect of maternal undernutrition before muscle differentiation on muscle fiber development of the newborn lamb. J. Anim. Sci. 83:2564–2571.

Ferket, P. R., J. D. E. Oliveier, A. Ghane, and Z. Uni. 2005. Effect of in ovo feeding solution osmolality on hatching turkeys. Poult. Sci. 84:118. (Abstr.)

Foye, O. T., P. R. Ferket, and Z. Uni. 2007. The effects of in ovo feeding arginine, beta-hydroxy-beta-methyl-butyrate, and protein on jejunal digestive and absorptive activity in embryonic and neonatal turkey poults. Poult. Sci. 86:2343–2349.

Gaunt, A. S., R. S. Hikida, J. R. Jehl, and L. Fenbert. 1990. Rapid atrophy and hypertrophy of an avian flight muscle. Auk 107:649–659.

Geng, T. Y., P. Li, X. H. Yin, and Z. Yan. 2011. PGC-1α promotes nitric oxide antioxidant defenses and inhibits FoxO signaling against cardiac cachexia in mice. Am. J. Pathol. 178:1738–1748.

Glass, D., and R. Roubenoff. 2010. Recent advances in the biology and therapy of muscle wasting. Ann. N. Y. Acad. Sci. 1211:25–36.

Gomes, M. D., S. H. Lecker, R. T. Jagoe, A. Navon, and A. L. Goldberg. 2001. Atrogin-1, a muscle-specific F-box protein highly expressed during muscle atrophy. Proc. Natl. Acad. Sci. USA 98:14440–14445.

Gosker, H. R., M. P. Engelen, H. van Mameren, P. J. van Dijk, G. J. van der Vusse, E. F. Wouters, and A. M. Schols. 2002. Muscle fiber type IIX atrophy is involved in the loss of fat-free mass in chronic obstructive pulmonary disease. Am. J. Clin. Nutr. 76:113–119.

Guernec, A., C. Berri, B. Chevalier, N. Wacrenier-Cere, E. Le Bihan-Duval, and M. J. Duclos. 2003. Muscle development, insulin-like growth factor-I and myostatin mRNA levels in chickens selected for increased breast muscle yield. Growth Horm. IGF Res. 13:8–18.

Halevy, O., I. Biran, and I. Rozenboin. 1998. Various light source treatments affect body and skeletal muscle growth by affecting skeletal muscle satellite cell proliferation in broilers. Comp. Biochem. Physiol. A 120:317–323.

Halevy, O., A. Geyra, M. Barak, Z. Uni, and D. Sklan. 2000. Early posthatch starvation decreases satellite cell proliferation and skeletal muscle growth in chicks. J. Nutr. 130:858–864.

Halevy, O., A. Krispin, Y. Leshem, J. P. McMurtry, and S. Yahav. 2001. Early-age heat exposure affects skeletal muscle satellite cell proliferation and differentiation in chicks. Am. J. Physiol. Regul. Integr. Comp. Physiol. 281:R302–R309.

Halevy, O., Y. Nadel, M. Barak, I. Rozenboim, and D. Sklan. 2003. Early posthatch feeding stimulates satellite cell proliferation and skeletal muscle growth in turkey poults. J. Nutr. 133:1376–1382.

Halevy, O., Y. Piestun, M. Allouh, B. Rosser, Y. Rinkevitch, R. Reshef, I. Rozenboim, M. Wleklinski-Lee, and Z. Yablonka-Reuveni. 2004. The pattern of Pax7 expression during myogenesis in the posthatch chicken establishes a model for satellite cell differentiation and renewal. Dev. Dyn. 231:489–502.

Halevy, O., Y. Piestun, I. Rozenboim, and Z. Yablonka-Reuveni. 2006a. In ovo exposure to monochromatic green light promotes skeletal muscle cell proliferation and affects myofiber growth in posthatch chicks. Am. J. Physiol. Regul. Integr. Comp. Physiol. 290:R1062–R1070.

Halevy, O., I. Rozenboim, and S. Yahav. 2006b. Enhancement of meat production by environmental manipulation in embryo and young broilers. World's Poult. Sci. J. 62:485–497.

Henriksson, J. 1990. The possible role of skeletal muscle in the adaptation to periods of energy deficiency. Eur. J. Clin. Nutr. 44:55–64.

Henry, M. H., and W. H. Burke. 1998. Sexual dimorphism in broiler chick embryos and embryonic muscle development in late incubation. Poult. Sci. 77:728–736.

Holz, M. K., B. A. Ballif, S. P. Gygi, and J. Blenis. 2005. mTOR and S6K1 mediate assembly of the translation preinitiation complex through dynamic protein interchange and ordered phosphorylation events. Cell 123:569–580.

Kassar-Duchossoy, L., E. Giacone, B. Gayraud-Morel, A. Jory, D. Gomes, and S. Tajbakhsh. 2005. Pax3/Pax7 mark a novel population of primitive myogenic cells during development. Genes Dev. 19:1426–1431.

Kawano, F., Y. Takeno, N. Nakai, Y. Higo, M. Terada, T. Ohira, I. Nonaka, and Y. Ohira. 2008. Essential role of satellite cells in the growth of rat soleus muscle fibers. Am. J. Physiol. Cell Physiol. 295:C458–C467.

Kikuchi, K., F. C. Buonomo, Y. Kajimoto, and P. Rotwein. 1991. Expression of insulin-like growth factor-I during chicken development. Endocrinology 128:1323–1328.

Kornasio, R., O. Halevy, O. Kedar, and Z. Uni. 2011. Effect of in ovo feeding and its interaction with timing of first feed on glycogen reserves, muscle growth, and body weight. Poult. Sci. 90:1467–1477.

Kuang, S., K. Kuroda, F. Le Grand, and M. A. Rudnicki. 2007. Asymmetric self-renewal and commitment of satellite stem cells in muscle. Cell 129:999–1010.

Lang, S. M., A. A. Kazi, L. Hong-Brown, and C. H. Lang. 2012. Delayed recovery of skeletal muscle mass following hindlimb immobilization in mTOR heterozygous mice. PLoS ONE 7:e38910. http://dx.doi.org/10.1371/journal.pone.0038910.

Lecker, S. H., A. L. Goldberg, and W. Mitch. 2006. Protein degradation by the ubiquitin-proteasome pathway in normal and diseases states. J. Am. Soc. Nephrol. 17:1807–1819.

Lecker, S. H., R. T. Jagoe, A. Gilbert, M. Gomes, V. Baracos, J. Bailey, S. R. Price, W. E. Mitch, and A. L. Goldberg. 2004. Multiple types of skeletal muscle atrophy involved a common program of changes in gene expression. FASEB J. 18:39–51.

Leshem, Y., D. B. Spicer, R. Gal-Levi, and O. Halevy. 2000. Hepatocyte growth factor (HGF) inhibits skeletal muscle cell differentia-

tion: A role for the bHLH protein twist and the cdk inhibitor p27. J. Cell. Physiol. 184:101–109.

Levine, S., C. Biswas, J. Dierov, R. Barsotti, J. B. Shrager, T. Nguyen, S. Sonnad, J. C. Kucharchzuk, L. R. Kaiser, S. Singhai, and M. T. Budak. 2011. Increased proteolysis, myosin depletion, and atrophic AKT-FoxO signaling in human diaphragm disuse. Am. J. Respir. Crit. Care Med. 183:483–490.

Li, P., R. E. Waters, S. I. Redfern, M. Zhang, and L. Mao. 2007. Oxidative phenotype protects myofibers from pathological insult induced by chronic heart failure in mice. Am. J. Pathol. 170:599–608.

Li, X., D. C. McFarland, and S. G. Velleman. 2006. Effect of transforming growth factor-β on decorin and β1 integrin expression during muscle development in chickens. Poult. Sci. 85:326–332.

Li, X., and S. G. Velleman. 2009. Effect of transforming growth factor-β1 on decorin expression and muscle morphology during chicken embryonic and posthatch growth and development. Poult. Sci. 88:387–397.

Lin, J., P. T. Tarr, C. Y. Zhang, Z. Wu, O. Boss, L. F. Michael, P. Puigserver, E. Isotani, E. N. Olson, B. B. Lowell, R. Bassseldub y, and B. M. Spiegelman. 2002. Transcriptional co-activator PGC-1α drives the formation of slow-twitch muscle fibres. Nature 418:797–801.

Liu, H. H., J. W. Wang, R. P. Zhang, X. Chen, H. Y. Yu, H. B. Jin, L. Li, C. C. Han, F. Xu, B. Kang, H. He, and H. Y. Xu. 2012. In ovo feeding of IGF-1 to ducks influences neonatal skeletal muscle hypertrophy and muscle mass growth upon satellite cell activation. J. Cell. Physiol. 227:1465–1475.

Liu, W., Z. Wang, and Y. Chen. 2010. Effects of monochromatic light on developmental changes in satellite cell population of pectoral muscle in broilers during early posthatch period. Anat. Rec. 293:1315–1324.

Loh, B., I. Maier, A. Winar, O. Janke, and B. Tzschentke. 2004. Prenatal development of epigenetic adaptation processes in poultry: Changes in metabolic and neuronal thermoregulatory mechanisms. Avian Poult. Biol. Rev. 10:119–128.

Lourens, A., H. van den Brand, R. Meijerhof, and B. Kemp. 2005. Effect of eggshell temperature during incubation on embryo development, hatchability and post-hatch development. Poult. Sci. 84:914–920.

Lu, J. W., J. P. McMurtry, and C. N. Coon. 2007. Developmental changes of plasma insulin, glucagon, insulin-like growth factors, thyroid hormones, and glucose concentrations in chick embryos and hatched chicks. Poult. Sci. 86:673–683.

Martinez, S. C., C. Cras-Méneur, E. Bernal-Mizrachi, and M. A. Permutt. 2006. Glucose regulates FoxO1 through insulin receptor signaling in the pancreatic islet beta-cell. Diabetes 55:1581–1591.

McFarland, D. C., J. E. Pesall, C. S. Coy, and S. G. Velleman. 2013. Effects of 17β-estradiol on turkey myogenic satellite cell proliferation, differentiation, and expression of glypican-1, MyoD and myogenin. Comp. Biochem. Physiol. A Mol. Integr. Physiol. 164:565–571.

McFarland, D. C., J. E. Pesall, and K. K. Gilkerson. 1993. The influence of growth factors on turkey embryonic myoblasts and satellite cells in vitro. Gen. Comp. Endocrinol. 89:415–424.

McFarland, D. C., S. G. Velleman, J. E. Pesall, and C. Liu. 2006. Effect of myostatin on turkey myogenic satellite cells and embryonic myoblasts. Comp. Biochem. Physiol. A Mol. Integr. Physiol. 144:501–508.

McMurtry, J. P., G. L. Francis, and Z. Upton. 1997. Insulin-like growth factors in poultry. Domest. Anim. Endocrinol. 14:199–229.

McMurtry, J. P., R. W. Rosebrough, D. M. Broucht, G. L. Francis, Z. Upton, and P. Phelps. 1998. Assessment of developmental changes in chicken and turkey insulin-like growth factor-II by homologous radioimmunoassay. J. Endocrinol. 157:463–473.

Mikura, M., I. Yamaoka, M. Doi, Y. Kawano, M. Nakayama, R. Nakao, K. Hirasaka, Y. Okumura, and T. Nikawa. 2009. Glucose infusion suppresses surgery-induced muscle protein breakdown by inhibiting ubiquitin-proteasome pathway in rats. Anesthesiology 110:81–88.

Mitch, W. E., and A. L. Goldberg. 1996. Mechanisms of muscle wasting—The role of the ubiquitin-proteasome pathway. N. Engl. J. Med. 335:1897–1905.

Mitchell, P. O., and G. K. Pavlath. 2004. Skeletal muscle atrophy leads to loss and dysfunction of muscle precursor cells. Am. J. Physiol. Cell Physiol. 287:C1753–C1762.

Moore, D. T., P. R. Ferket, and P. E. Mozdziak. 2005a. Muscle development in the late embryonic and early post-hatch poult. Int. J. Poult. Sci. 3:138–142.

Moore, D. T., P. R. Ferket, and P. E. Mozdziak. 2005b. The effect of early nutrition on satellite cell dynamics in the young turkey. Poult. Sci. 84:748–756.

Moran, E. T., Jr. 2007. Nutrition of the developing embryo and hatchling. Poult. Sci. 86:1043–1049.

Morgan, J. E., and P. S. Zammit. 2010. Direct effects of the pathogenic mutation on satellite cell function in muscular dystrophy. Exp. Cell Res. 316:3100–3108.

Nagano, K., E. Suzaki, Y. Nagano, K. Kataoka, and K. Ozawa. 2008. The activation of apoptosis factor in hindlimb unloading-induced muscle atrophy under normal and low-temperature environmental conditions. Acta Histochem. 110:505–518.

Nagatomo, F., H. Fujino, H. Kondo, H. Suzuki, M. Kouzaki, I. Takeda, and A. Ishihara. 2011. PGC-1α and FOXO1 mRNA levels and fiber characteristics of the soleus and plantaris muscles in rats after hindlimb unloading. Histol. Histopathol. 26:1545–1553.

Nair, K. S., G. C. Ford, K. Ekberg, E. Fernqvist-Forbes, and J. Wahren. 1995. Protein dynamics in whole body and in splanchnic and leg tissues in type I diabetic patients. J. Clin. Invest. 95:2926–2937.

Newsholme, P. 2001. Why is L-glutamine metabolism important to cells of the immune system in health, postinjury, surgery or infection. J. Nutr. 131:2515S–2522S.

Ohanna, M., A. K. Sobering, T. Lapointe, L. Lorenzo, C. Praud, E. Petroulakis, N. Sonenberg, P. A. Kelly, A. Sotiropoulos, and M. Pende. 2005. Atrophy of S6K1−/− skeletal muscle cells reveals distinct mTOR effectors for cell cycle and size control. Nat. Cell Biol. 7:286–294.

Ordahl, C. P., B. A. Williams, and W. Denetclaw. 2000. Determination and morphogenesis in myogenic progenitor cells: An experimental embryological approach. Curr. Top. Dev. Biol. 48:319–367.

Oustanina, S., G. Hause, and T. Braun. 2004. Pax7 directs postnatal renewal and propagation of myogenic satellite cells but not their specification. EMBO J. 23:3430–3439.

Pearen, M. A., N. A. Eriksson, R. L. Fitzsimmons, J. M. Goode, N. Martel, S. Andrikopoulos, and G. E. Muscat. 2012. The nuclear receptor, Nor-1, markedly increases type II oxidative muscle fibers and resistance to fatigue. Mol. Endocrinol. 26:372–384.

Picard, B., L. Lefaucheur, C. Berri, and M. J. Duclos. 2002. Muscle fibre ontogenesis in farm animal species. Reprod. Nutr. Dev. 42:415–431.

Piestun, Y., S. Druyan, J. Brake, and S. Yahav. 2013a. Thermal manipulations during broiler incubation alter performance of broilers to 70 days of age. Poult. Sci. 92:1155–1163.

Piestun, Y., S. Druyan, J. Brake, and S. Yahav. 2013b. Thermal treatments prior to and during the beginning of incubation affect phenotypic characteristics of broiler chickens posthatching. Poult. Sci. 92:882–889.

Piestun, Y., O. Halevy, and S. Yahav. 2009a. Thermal manipulations of broiler embryos—The effect on thermoregulation and development during embryogenesis. Poult. Sci. 88:2677–2688.

Piestun, Y., M. Hare, M. Barak, S. Yahav, and O. Halevy. 2009b. Thermal manipulations in late-term chick embryos have immediate and longer term effects on myoblast proliferation and skeletal muscle hypertrophy. J. Appl. Physiol. 106:233–240.

Price, S. R., J. L. Gooch, S. K. Donaldson, and T. K. Roberts-Wilson. 2010. Muscle atrophy in chronic kidney disease results from abnormalities in insulin signaling. J. Ren. Nutr. 20:S24–S28.

Remignon, H., M. F. Gardahaut, G. Marche, and F. H. Ricard. 1995. Selection for rapid growth increases the number and the size of muscle fibers without changing their typing in chickens. J. Muscle Res. Cell Motil. 16:95–102.

Rowe, G. C., R. Ei-Khoury, I. S. Patten, P. Rustin, and Z. Arany. 2012. PGC-1α is dispensable for exercise-induced mitochondrial biogenesis in skeletal muscle. PLoS ONE 7:1–9. http://dx.doi.org/10.1371/journal.pone.0041817.

Rozenboim, I., Y. Piestun, N. Mobarkey, M. Barak, A. Hoyzman, and O. Halevy. 2004. Monochromatic light stimuli during embryogenesis enhance embryo development and posthatch growth. Poult. Sci. 83:1413–1419.

Rudrappa, S. G., and B. D. Humphrey. 2007. Energy metabolism in developing chicken lymphocytes is altered during the embryonic to posthatch transition. J. Nutr. 137:427–432.

Sacheck, J. M., A. Ohtsuka, S. C. McLary, and A. L. Goldberg. 2004. IGF-1 stimulates muscle growth by suppressing protein breakdown and expression of atrophy-related ubiquitin ligase, atrogin-1 and MuRF1. Am. J. Physiol. Endocrinol. Metab. 4:591–601.

Sandri, M. 2008. Signaling in muscle atrophy and hypertrophy. Physiology (Bethesda) 23:160–170.

Sandri, M., J. D. Lin, C. Handschin, W. L. Yang, Z. P. Arany, S. H. Lecker, A. L. Goldberg, and B. M. Spiegelman. 2006. PGC-1α protects skeletal muscle from atrophy by suppressing FoxO3 action and atrophy-specific gene transcription. Proc. Natl. Acad. Sci. USA 44:16260–16265.

Sandri, M., C. Sandri, A. Gilbert, C. Skurk, E. Calabria, A. Picard, K. Walsh, S. Schiaffino, S. H. Lecker, and A. L. Goldberg. 2004. FoxO transcription factors induce the atrophy-related ubiquitin ligase atrogin-1 and cause skeletal muscle atrophy. Cell 117:399–412.

Seale, P., L. A. Sabourin, A. Girgis-Gabardo, A. Mansouri, P. Gruss, and M. A. Rudnicki. 2000. Pax7 is required for the specification of myogenic satellite cells. Cell 102:777–786.

Serrano, J., A. R. Shuldiner Jr., C. T. Roberts, D. LeRoith, and F. DePablo. 1990. The insulin-like growth factor I (IGF-I) gene is expressed in chick embryos during early organogenesis. Endocrinology 127:1547–1549.

Shavlakadze, T., J. Chai, K. Maley, G. Cozens, G. Grounds, N. Winn, N. Rosenthal, and M. D. Grounds. 2010. A growth stimulus is needed for IGF-1 to induce skeletal muscle hypertrophy in vivo. J. Cell Sci. 123:960–971.

Sishi, B., B. Loos, B. Ellis, W. Smith, E. F. du Toit, and A. M. Engelbrecht. 2011. Diet-induced obesity alters signaling pathways and induces atrophy and apoptosis in skeletal muscle in a prediabetic rat model. Exp. Physiol. 2:179–193.

Starkey, J. D., M. Yamamoto, S. Yamamoto, and D. J. Goldhamer. 2011. Skeletal muscle satellite cells are committed to myogenesis and do not spontaneously adopt nonmyogenic fates. J. Histochem. Cytochem. 59:33–46.

Stickland, N. C. 1978. A quantitative study on muscle development in the bovine foetus (*Bos indicus*). Anat. Histol. Embryol. 7:193–205.

Stickland, N. C., S. Bayol, C. Ashton, and C. Rehfeldt. 2004. Manipulation of muscle fibre number during prenatal development. Pages 69–79 in Muscle development of livestock animals. M. F. W. te Pas, M. E. Everts, and H. P. Haagsman ed. CABI Publishing, Cambridge, MA.

Stitt, T. N., D. Drujan, B. A. Clarke, F. Panaro, Y. Timofeyva, W. O. Kline, M. Gonzalez, G. D. Yancopoulos, and D. J. Glass. 2004. The IGF-1/PI3K/Akt pathway prevents expression of muscle atrophy-induced ubiquitin ligases by inhibiting FOXO transcription factors. Mol. Cell 14:395–403.

Stockdale, F. E. 1992. Myogenic cell lineages. Dev. Biol. 154:284–298.

Stockdale, F. E., and J. B. Miller. 1987. The cellular basis of myosin heavy chain isoform expression during development of avian skeletal muscles. Dev. Biol. 123:1–19.

Stojanović, S., Z. Kanački, D. Žikić, and G. Ušćebrka. 2009. Prenatal dynamic of development of skeletal musculature of broiler and layer chickens. Biotech. Anim. Husbandry 25:1063–1069.

Swatland, H. J. 1973. Muscle growth in the fetal and neonatal pig. J. Anim. Sci. 37:536–545.

Terracciano, C., M. Celi, D. Lecce, J. Baldi, E. Rastelli, E. Lena, R. Massa, and U. Tarantino. 2013. Differential features of muscle fiber atrophy in osteoporosis and osteoarthritis. Osteoporos. Int. 24:1095–1100.

Tzschentke, B. 2007. Attainment of thermoregulation as affected by environmental factors. Poult. Sci. 86:1025–1036.

Tzschentke, B., and D. Basta. 2002. Early development of neuronal hypothalamic thermosensitivity in birds: Influence of epigenetic temperature adaptation. Comp. Biochem. Physiol. A Mol. Integr. Physiol. 131:825–832.

Tzschentke, B., and A. Plagemann. 2006. Imprinting and critical periods in early development. World's Poult. Sci. J. 62:626–638.

Uni, Z., P. R. Ferket, E. Tako, and O. Kedar. 2005. In ovo feeding improves energy status of late-term chicken embryos. Poult. Sci. 84:764–770.

Uni, Z., E. Tako, O. Gal-Garber, and D. Sklan. 2003. Morphological, molecular, and functional changes in the chicken small intestine of the late-term embryo. Poult. Sci. 82:1747–1754.

Van Der Heide, L. P., M. F. Hoekman, and M. P. Smidt. 2004. The ins and outs of FoxO shuttling: Mechanisms of FoxO translocation and transcriptional regulation. Biochem. J. 380:297–309.

van der Schoor, S. R., H. Schierbeek, P. M. Bet, M. J. Vermeulen, H. N. Lafeber, J. B. van Goudoever, and R. M. van Elburq. 2010. Majority of dietary glutamine is utilized in first pass in preterm infants. Pediatr. Res. 67:194–199.

Velleman, S. G. 2007. Muscle development in the embryo and hatchling. Poult. Sci. 86:1050–1054.

Velleman, S. G., X. Li, C. S. Coy, and D. C. McFarland. 2008. The effect of fibroblast growth factor 2 on the in vitro expression of syndecan-4 and glypican-1 in turkey satellite cells. Poult. Sci. 87:1834–1840.

Verdijk, L. B., M. L. Dirks, T. Snjders, J. J. Prompers, M. Beelen, R. A. Jonkers, D. H. Tijssen, M. T. Hopman, and L. J. van Loon. 2012. Reduced satellite cell numbers with spinal cord injury and aging in humans. Med. Sci. Sports Exerc. 44:2322–2330.

Verdijk, L. B., R. Koopman, G. Schaart, K. Meijer, H. H. Savelberg, and L. J. van Loon. 2007. Satellite cell content is specifically reduced in type II skeletal muscle fibers in the elderly. Am. J. Physiol. Endocrinol. Metab. 292:E151–E157.

Vieira, S. L., and E. T. Moran. 1999. Effects of egg of origin and chick posthatch nutrition on broiler live performance and meat yields. World's Poult. Sci. J. 55:125–142.

Wabeck, C. J., and W. C. Skoglund. 1974. Influence of radiant energy from fluorescent light sources on growth, mortality, and feed conversion of broilers. Poult. Sci. 53:2055–2059.

Wigmore, P. M., and N. C. Stickland. 1983. Muscle development in large and small pig fetuses. J. Anat. 137:235–245.

Willemsen, H., B. Kamers, F. Dahlke, H. Han, Z. Song, Z. Ansari Pirsaraei, K. Tona, E. Decuypere, and N. Everaert. 2010. High- and low-temperature manipulation during late incubation: Effects on embryonic development, the hatching process, and metabolism in broilers. Poult. Sci. 89:2678–2690.

Willemsen, H., Y. Li, E. Willems, L. Franssens, Y. Wang, E. Decuypere, and N. Everaert. 2011. Intermittent thermal manipulations of broiler embryo during late incubation and their immediate effect on the embryonic development and hatching process. Poult. Sci. 90:1302–1312.

Wiskus, K. J., P. B. Addis, and R. T. Ma. 1976. Distribution of βR, αR and αW fibers in turkey muscles. Poult. Sci. 55:562–572.

Xiao, B., M. J. Sanders, E. Underwood, R. Heath, F. V. Mayer, D. Carmena, C. Jing, P. A. Walker, J. F. Eccleston, L. F. Haire, S. A. Howell, R. Aasland, S. R. Martin, D. Carling, and S. J. Gamblin. 2011. Structure of mammalian AMPK and its regulation by ADP. Nature 472:230–233.

Yablonka-Reuveni, Z. 2011. The skeletal muscle satellite cell: Still young and fascinating at 50. J. Histochem. Cytochem. 59:1041–1059.

Yablonka-Reuveni, Z., and B. M. Paterson. 2001. MyoD and myogenin expression patterns in cultures of fetal and adult chicken myoblasts. J. Histochem. Cytochem. 49:455–462.

Yadgary, L., and Z. Uni. 2012. Yolk sac carbohydrate level and gene expression of key gluconeogenic and glycogenic enzymes during chick embryonic development. Poult. Sci. 91:444–453.

Yahav, S., R. Sasson Rath, and D. Shinder. 2004. The effect of thermal manipulations during embryogenesis of broiler chicks (*Gallus domesticus*) on hatchability, body weight and thermoregulation after hatch. J. Therm. Biol. 29:245–250.

Yakar, S., J. L. Liu, A. M. Fernandez, Y. P. Wu, A. V. Schally, J. Frystyk, S. D. Chernausek, W. Mejia, and D. L. Roith. 2001. Liver-specific igf-1 gene deletion leads to muscle insulin insensitivity. Diabetes 50:1110–1118.

Yamada, E., C. C. Bastie, H. Koga, Y. Wang, A. M. Cuervo, and J. E. Pessin. 2012. Mouse skeletal muscle fiber-type specific macroautophagy and muscle wasting is regulated by a Fyn/STAT3/Vps34 signaling pathway. Cell Rep. 1:557–569.

Yu, Z., M. Zhang, M. Hannink, J. S. Stamler, and Z. Yan. 2008. Fiber type-specific nitric oxide protects oxidative myofibers against cachectic stimuli. PLoS ONE http://dx.doi.org/10.1371/journal.pone.0002086.

Zeng, C., J. E. Pesall, K. K. Gilkerson, and D. C. McFarland. 2002. The effect of hepatocyte growth factor on turkey satellite cell proliferation and differentiation. Poult. Sci. 81:1191–1198.

Zhai, W., L. W. Bennett, P. D. Gerard, R. Pulikanti, and E. D. Peebles. 2011. Effects of in ovo injection of carbohydrates on somatic characteristics and liver nutrient profiles of broiler embryos and hatchlings. Poult. Sci. 90:2681–2688.

Zhang, L., X. N. Wang, H. L. Wang, J. Du, and W. E. Mitch. 2010. Satellite cell dysfunction and impaired IGF-1 signaling cause CKD-induced muscle atrophy. J. Am. Soc. Nephrol. 21:419–427.

Zhu, M. J., S. P. Ford, W. J. Means, B. W. Hess, P. W. Nathanielsz, and M. Du. 2006. Maternal nutrient restriction affects properties of skeletal muscle in offspring. J. Physiol. 575:241–250.

Zhu, M. J., S. P. Ford, P. W. Nathanielsz, and M. Du. 2004. Effect of maternal nutrient restriction in sheep on the development of fetal skeletal muscle. Biol. Reprod. 71:1968–1973.