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Review

Skeletal muscle development in vertebrate animals

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Abstract: This review covers the pre- and post-natal development of skeletal muscle of vertebrate animals with cellular and molecular levels. The formation of skeletal muscle initiates from paraxial mesoderm during embryogenesis of individuals which develops somites and subsequently forms dermomyotome derived myotome to give rise axial musculature. This process (myogenesis) includes stem and progenitor cell maintenance, lineage specification, and terminal differentiation to form myofibrils consequent muscle fibers which control muscle mass and its multiplication. The main factors of muscle growth are proliferation and differentiation of myogenic cells in prenatal stage and also the growth of satellite cells at postnatal stage. There is no net increase in the number of muscle fibers in vertebrate animals after hatch or birth except fish. The development of muscle is characterized by hyperplasia and hypertrophy in prenatal and postnatal stages of individuals, respectively, through Wnt signalling pathway including environment, nutrition, sex, feed, growth and myogenic regulatory factors. Therefore further studies could elucidate new growth related genes, markers and factors to enhance meat production and enrich knowledge on muscle growth.

Keywords: myogenesis; proliferation; differentiation; wnt; satellite cells; myoblast

1. Introduction

Skeletal muscle is a complex and heterogeneous tissue, and considered as an organ for the muscular system which consisting of the tissues of skeletal muscle, connective, nerve and blood or vascular to serve multitude of functions (Bentzinger *et al.*, 2012 and SEER, 2015). It derives from paraxial mesoderm and divides into somites and notochord (Christ and Ordahl, 1995) in vertebrate animals like mammals and avian species. The fetal stage not only involves the development of muscle cells (myogenesis) but also adipocyte and fibroblast (Du *et al.*, 2010) of animals. In adult period, skeletal muscles are composed of bundles of multinucleated myofibers which distributed tendon to tendon and provided contractile activity in skeletal muscle (Gunning and Hardeman, 1991).

Proliferation and differentiation of the muscle precursor cells or myoblasts are the key factors which regulate skeletal muscle development. The growth and myogenic regulatory factors are associated with skeletal muscle growth through cell proliferation and differentiation (Parakati and DiMario, 2013) in numerous tissues of animals. Growth rate and growth potential are undoubtedly heritable characteristics and also relate to their fiber-type composition, particularly in terms of contractile speed and metabolism, but different environmental factors like ambient temperature and food availability could also affect the growth of all species (Leatherland, 1994 and Rehfeldt *et al.*, 2011a). Skeletal muscle could regenerate and restore the cellular architecture completely (Shi and Garry, 2006 and Meadows *et al.*, 2008) in response to repeated injuries by one type muscle stem cell called satellite cells.

Therefore understanding the mechanisms of muscle growth and developmental is one of the most important areas in animal since to execute and facilitate further advance or standardization research works on meat production and quality.

2. Origin of skeletal muscle

The ectoderm, mesoderm and endoderm are subsequently initial embryonic patterns in which mesoderm is anatomically divided into paraxial, intermediate and lateral mesoderm (Bentzinger *et al.*, 2012). In vertebrate, skeletal muscle formation is derived from the paraxial mesoderm during embryogenesis (Buckingham *et al.*, 2003) and the accumulation of paraxial mesoderm forms segmented epithelial spheres (Pirskanen *et al.*, 2000 and Shi and Garry, 2006) which referred to somites. The somite undergoes differentiation, and subdivides into dorsal dermomyotome and ventral sclerotome (Wang *et al.*, 2013). Further differentiation of dermomyotome creates dorsal-lateral dermatome and myotome which give rise to dermis and axial musculature, respectively (Bumcrot and McMahon, 1995). The review of Rehfeldt *et al.*, (2011a) stated that the first phase of skeletal trunk muscle formation in mammals, poultry and fish is primary myotome which originates from the four epithelial borders of the dermomyotome. In addition, the central region of that part changes epithelial to mesenchymal transition in the second phase to release *paired box* (*PAX*) *3/7*-positive cells into primary myotome and these cells evolve into embryonic myoblasts or fetal myoblasts or satellite cells. But the limb muscles originate from muscle progenitors which delaminate from the ventrolateral border of the dermomyotome.

3. Structure of skeletal muscle

The basic structure of skeletal muscle (Figure 1) includes the cells of myocytes, adipocytes and fibroblasts which derive from the same pool of mesenchymal stem cells in fetal muscle of farm animals (Du *et al.*, 2010). The skeletal muscle formation begins during embryonic life including stem and progenitor cell maintenance, lineage specification and terminal differentiation (Bentzinger *et al.*, 2012). The process is completed after postnatal growth (Amthor *et al.*, 1999) when an animal is reached its adult stage. According to Scime *et al.*, (2009) skeletal muscle is made up of thousands of cylindrical multinucleated muscle fibers (myofibers) composing an array of stacked myofibrils running the entire length of the cell. Myofibrils consist of thick and thin filaments which are organized into a contractile unit called a sarcomere and surrounded by a basal lamina. A population of quiescent muscle progenitor called satellite cells is located beneath the basal lamina. Normally the secondary myofibers overlap with adipocytes and fibroblasts, and epigenetic control such as maternal nutrition in mammalian species influences the number of secondary myofibers but this factor cannot alter primary fibers (Dwyer and Stickland, 1991 and Picard *et al.*, 2002). Muscle mass and its multiplication mainly depend on the number and size of fibers, and also genetic and environmental factors (Rehfeldt *et al.*, 2000) which are capable to influence prenatal growth. Therefore postnatal muscle growth of any individuals mainly depends on its prenatal developmental status regarding muscle fiber number which ultimately grows in size rather than number. The most striking difference between striated fish and higher vertebrates is the separated fiber types into discrete layers in fish, in which more than 90 % muscle constitutes with high glycolytic and anaerobic type known as Fast- White fibers (Kiessling *et al.*, 2006).

4. Myogenesis or development of skeletal muscle

Myogenesis is the multifarious process of skeletal muscle formation in vertebrates including all mammals, birds and fish. The timing of different developmental stages varied significantly but the end products are same to form multinucleated myofibers with contractile capability (Knight and Kothary, 2011). Myogenesis is controlled by an elaborate interplay of extrinsic and intrinsic regulatory mechanisms (Bentzinger *et al.*, 2012) regulated by myogenic regulatory factors including 2-3 distinct phases, in addition, protein kinase family which transmits and executes signals originated by promyogenic stimuli (Rehfeldt *et al.*, 2011a and Knight and Kothary, 2011).

The key factors of muscle growth are proliferation and differentiation of myogenic cells (prenatal stage) (Figure 2) and satellite cells (postnatal stage) (Figure 3). Growth of muscle tissue is accomplished by two fundamental biological processes: protein accretion and cell proliferation which finally produce meat. According to quantal cell cycle theory, myoblasts produce during myogenesis as a result of the final quantal cell cycle which are no longer capable to proliferate further; therefore, they fuse and synthesize myofibrillar proteins for permanent withdrawal from the cell cycle (Picard *et al.*, 2002). Muscle fibers are produced from the accumulation of

mature myotube derived myofibrils. The increase in muscle fiber number is referred to hyperplastic growth of muscle while enlargement of existing fibers called hypertrophy. Rowlerson and Vegetti (2001) documented that muscle growth in fish occurs throughout their life time until muscle fibers reach to 100-300 µm diameters for most of the species by consequent hyperplastic including a distinct population of myogenic cells for mosaic growth and hypertrophy.

4.1. Prenatal myogenesis

Myoblasts are originated from the segmented structural part of dermomyotome called somites during mesoderm of embryonic stage. The formation of dermomyotome and myotome is affected by the members of Wnt family of proteins (Geetha-Loganathan *et al.*, 2008) and consequently enhance the developmental of myogenesis. Myoblasts are named as somatic, embryonic, fetal and adult (or satellite) on the basis of different development stages of organisms.

Myoblasts which are developed from mesodermal myogenic precursor cells exit the cell cycle to stop dividing and to differentiate by special signal (Rehfeldt *et al.*, 2000). This process starts with the commitment of an embryonic precursor to the myogenic lineage followed by proliferation of those committed myoblasts which are differentiated into post-mitotic myocytes (Knight and Kothary, 2011) and lastly fuse to form a multinucleated myotube. It was proved from several studies that there is no net increase in the number of muscle fibers after hatch or birth (Zhu *et al.*, 2004 and Velleman, 2007), in addition, development of skeletal muscle in this stage has long term effects on postnatal growth and physiological function (Rehfeldt *et al.*, 2011b) both in animals and in human beings.

Muscle fibers are produced from two distinct phases which include myoblast fusion to primary myoblast as initial phase and later primary myoblast to secondary myoblast by the second web of differentiation (Beermann *et al.*, 1978; Miller *et al.*, 1993 and Rehfeldt *et al.*, 2000). During limb muscle formation of chicken, the ectodermal signals ensure the growth in trunk (Amthor *et al.*, 1999) by limiting differentiation in myogenic cells. Indeed cell proliferation is a fundamental growth process for muscle growth and maintenance in vertebrate, high cell density could decrease or stop the rate of proliferation. This proliferation process is able to synthesize contractile proteins, acetylcholine receptors and other proteins (McFarland, 1992) for the development of muscle fibers. For any deficiencies, the appropriate number of embryonic muscle cells is produced that unlikely to be compensated during later development of muscle. In this period, proliferative and *PAX-3* promoting signals inhibit differentiation (Amthor *et al.*, 1999) for continuous embryonic muscle growth. The study on double-muscled cattle in prenatal period suggested that delaying differentiation and extending myoblast proliferation result higher number of muscle fibers (Picard *et al.*, 1995). The differentiation process executes withdrawal of myoblast from cell cycle (Stockdale and Holtzer, 1961) and also expression of muscle-specific genes (Du *et al.*, 2010).

4.2. Factors affecting muscle growth in prenatal stage

The malnutrition and over-nutrition negatively affect the growth performance of offspring by reducing secondary muscle fibers and muscle mass, especially early to mid-gestation in ruminant animals (Du *et al.*, 2010). But maternal nutrient deficiency at late gestation reduces muscle fiber size in fetal sheep (Greenwood *et al.*, 1999) rather than number of fibers. Similarly nutrient availability between single and twin littermates or competition within littermates affects the muscle fiber hypertrophy (McCoard *et al.*, 2000) but no change occurs during hyperplasia. Both the size and number of muscle fibers observed lower as a result of lower body weight in last two-thirds of pregnancy by a low plane of maternal nutrition in neonatal calf (Freetly *et al.*, 2000). Therefore proper nutritional plan during gestation period could enhance fetal skeletal muscle development by altering muscle fiber composition, lowering excessive fat and proper marbling in the offspring.

Several studies in poultry (turkey, chicken) identified that a little rising of incubation temperature (38.58˚C) could influence post-hatch muscle growth (Maltby *et al.*, 2004 and Hammond *et al.*, 2007), specifically 7-10 days of incubation period in cock rather than hen (Werner and Wicke, 2008). The primary muscle fibers forms within two months of post-conception in bovine fetus, but most of the fibers forms between 2-8 months of gestation (Russell and Oteruelo, 1981). The reduction of muscle fiber formation during gestation period has long-lasting negative effects on physiological consequences in progenies (Du *et al.*, 2010). In high growing chicken lines, muscle fibers observed comparatively bigger than slow growing lines including lower glycogen contents (Berri *et al.*, 2006 and Duclos *et al.*, 2007).

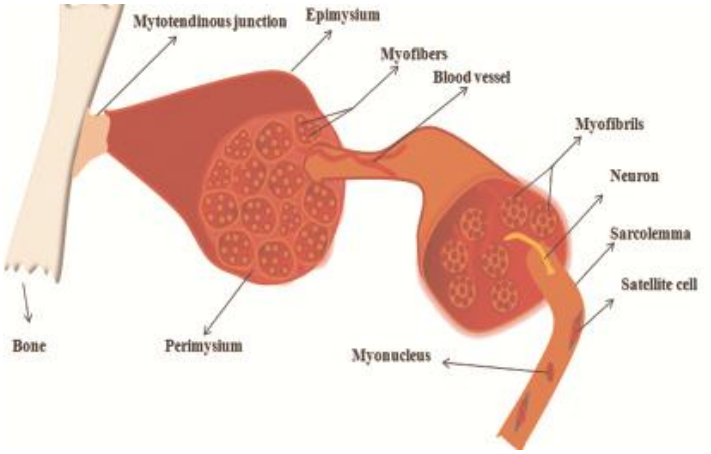

Figure 1. The basic structure of skeletal muscle. Skeletal muscle is made up of a number of bundled muscle fibers which are derived from myofibrils (fusion of myoblast) and wrapped in a connective tissue covering (endomysium). The myofibrils consist of thick and thin filaments organized into a contractile unit called a sarcomere and also surrounded by a basal lamina under which satellite cells are located. A bundle of muscle fiber is covered by perimysium and several bundles are surrounded by epimysium that connects with bone by myotendinous junction.

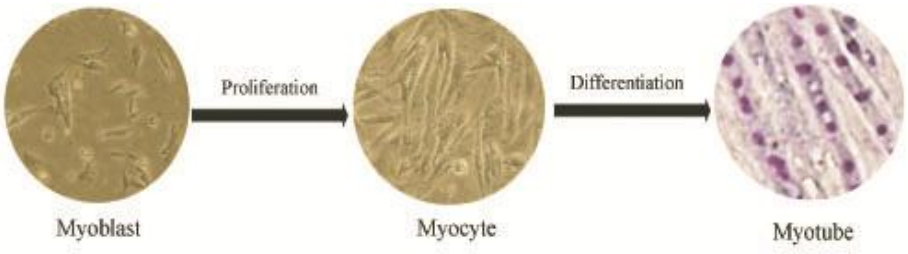

Figure 2. Primary chicken myoblast fusion into mature myotubes. Myoblasts (12 h) are proliferated into Myocyte (72 h) in growth medium and finally fused to myotube while cell nuclei are visible in a line by Giemsa staining at 144 h of differentiation (Shahjahan *et al.*, 2015) in differentiated medium.

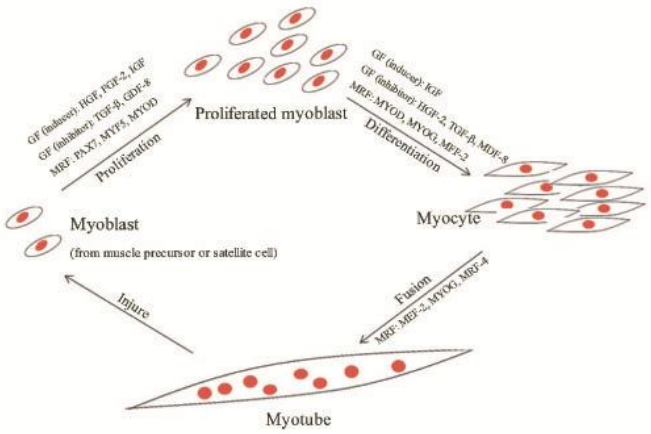

Figure 3. A graphical representation is showing the involvement of growth factors (GFs) and myogenic regulation factors (MRFs) in different stages of myogenesis. Embryonic precursors or quiescent satellite cells are become activated to form proliferating myoblasts and then differentiate into myocytes. Those are fused to form multinucleated myotubes and their accumulations finally produce muscle fiber containing satellite cells beneath the basal lamina which activate during any injury of muscle to produce myoblast for further repairing.

4.3. Post-natal muscle growth

Postnatal muscle growth occurs mainly for the increase of muscle fiber size rather than forming new fiber except little increase of muscle fibers in neonatal rats, mice and human infants (Picard, 2002 and Karunaratne *et al.*, 2005). Muscle growth in this time is considered due to muscle cell hypertrophy as muscle fiber hyperplasis executes during prenatal period. In post-natal muscle development there is negative correlation between muscle fiber number and its thickness, although studies on cattle did not reveal such evidences (Wegner *et al.*, 2000). However, an exception is double-muscled cattle which exhibits almost double the number of muscle fibers than other cattle breeds without increasing fiber size, although strong hypertrophy could reduce the capacity of fibers to adapt activity-induced demands associating inferior meat quality and stress susceptibility of pig breeds (Rehfeldt *et al.*, 2000). This study also explained that the total number of muscle fibers does not alter after hatch of chicken, quail or born of mice, rat, cattle and pig because the growth is mainly occurred by hypertrophy, satellite cells and myogenic precursors of fibers.

Muscle regeneration requires the recruitment of an undifferentiated progenitor from the injured site to execute this process by satellite cells (Bentzinger *et al.*, 2012). These cells are located between the basal lamina and the sarcolemma (Kuang *et al.*, 2007) of the mature muscle fibers. The review of Shi and Garry (2006) documented that satellite cells are originated from central region of the dermomyotome of the somite. In addition, the bone marrow or vascular components also originate satellite cells which are derived from somite and non-somite and associated with growth or tissue repair and disease, respectively. The higher vertebrates need extracellular matrix from injured tissue as a template to regenerate new muscle fibers (Ciciliot and Schiaffino, 2010) but amphibians, fish and some lower organisms can regenerate muscle without such template (Poss, 2010). The satellite cells appear between 13 and 16 days in chicken embryogesis which are able to multiply and fuse with fibers to provide new nuclei for protein anabolism (Rehfeldt *et al.*, 2011a).

4.4. Factors affecting muscle growth in post-natal stage

The study of Berri *et al.*, (2006) pointed out deprivation of nutrients after hatch of chick could reduce satellite cell proliferation in pectoralis major muscle and also the expression of neonatal isoform related to embryonic isoform of myosin heavy chain. Scheuermann *et al.*, (2003) stated that sex is an important issue for the variation of myofiber numbers in commercial strain crosses where males showed higher myofiber density in pectoralis muscle than females chicken. The number of secondary muscle fibers of pigs was negatively and positively affected by multiple litters and over nutrition or injection of growth hormone (10 and 24 days of pregnancy), respectively, in addition, a close relation showed between muscle fiber diameter and fiber nuclei in chicken (Picard *et al.*, 2002). McFarland (1992) explained that in postnatal period the DNA content of cells increases subsequently where size of muscle nuclei relates to fiber size. Therefore the rate of DNA accretion in this stage correlates with muscle growth of organisms and the amount of DNA content during postnatal period is not the results of nuclear replication of myotubes but the increase of satellite cell derives myonuclei and accumulation of muscle protein by the growth of muscle. Scheuermann *et al.*, (2003) reported that the main factor of sex differences on body weight and muscularity might be predetermined during embryonic development when myofibers numbers are established. The muscle growth of fish is highly variable depending on water temperature, dissolved O_2 and NH_3 levels, photoperiod, salinity, ingested food quality, age and maturity of fish (Kiessling *et al.*, 2006).

5. Pathway associated with myogenesis

The skeletal muscle formation is critically modulated by Wnt signalling pathway that controls the formation of the dermomyotome, expression of myogenic regulatory factors (MRFs) for myogenic lineage progression and satellite cell differentiation as well as in satellite cell self-renewal (von Maltzahn *et al.*, 2012). The Wnt family consisting of several lipid modified secreted glycoproteins released by tissues surrounding the somites is regulated by autocrine or paracrine mechanisms which influence the development of various cellular types, and its signal causes cell proliferation and differentiation or maintenance of precursor cells (Hollway and Currie, 2005 and Johnson and Rajamannan, 2006). According to Wnt pathway (Huelsken and Birchmeier, 2001), cytoplasmic β-catenin stabilizes, and some β-catenin enter the nucleus and interact with the members of T-cell factor or lymphoid enhancer factor family of transcription factors to activate specific target genes (Dierick and Bejsovec, 1999 and Hecht and Kemler, 2000). The expression of *β-catenin* is essential for overload-induced muscle hypertrophy (Armstrong *et al.*, 2006), determination of types of muscle fiber and number of myofiber in vertebrate embryos (Hutcheson *et al.*, 2009), and formation of higher amount of slow myosin positive fibers

(von Maltzahn *et al.*, 2012). Therefore the role of *β-catenin* is very essential to regulate embryonic, postnatal and oncogenic growth of tissues.

6. Effect of growth factors (GFs) in myogenesis

Different types of GFs associating skeletal muscle development are presented in Figure 3. Hepatocyte growth factor (HGF) stimulates the migration (Bandow *et al.*, 2004) and proliferation (Amano *et al.*, 2002) of myogenic cells during the development of skeletal muscles. The supplementation of HGF enhanced the surface elasticity of bovine satellite cells in culture medium (Lapin *et al.*, 2013). Bandow *et al.*, (2004) examined that inactivation of HGF caused hypoplasia of mice tongue muscles. *Fibroblast growth factor 2* (*FGF2*) regulates muscle growth by stimulating myoblast and satellite cells proliferation and acts as strong inhibitor of differentiation (Velleman, 2007). Thus expression of *FGF2* is required in hyperplasia stage of embryonic period for the formation of more muscle fibers. However, Brunetti and Goldfine (1990) documented that *FGF2* inhibits the transcription of myogenin during myogenesis which is needed for the initiation of myotube formation. But *FGF2* expression is more responsive to fast growing turkey satellite cells containing higher level of heparan sulfate proteoglycans than slower growing satellite cells (McFarland *et al.*, 2003). Insulin-like growth factors (IGFs) stimulate both proliferation and differentiation of myogenic cells (Kamanga-Sollo *et al.*, 2003) where reverse role identified in differentiation for both *transforming growth factor* (*TGF-β*) and *myostatin* (*GDF-8*). Thus inhibiting the expression of *TGF-β* and *GDF-8* could be a key target for meat producing animals. The transcript abundance of *IGF-I* in pectoralis major muscle of chicken was identified higher in low weight select than high weight select line by 28 days of post hatch, although its expression was lower during embryogenesis (Wu *et al.*, 2011). The diverse mRNA expression of growth hormone (GH) and *IGF-I* in breast muscle of 35-day-old Japanese quails identified in different glycerol levels (0, 4, and 8% dietary glycerol instead of corn) (Gasparino *et al.*, 2012). Similarly the expression of mRNA *IGF-I, IGF-II,* and type I and II IGF receptors (*IGF-IR* and *IGF-IIR*) in breast, leg and myocardium during an early postnatal development growth stage of duck at 1-8 weeks of post hatch period (Song *et al.*, (2013) which indicating the expression of these 4 genes differed in proliferation and differentiation of muscle tissues. *IGFs* in fish promote myogenic cell proliferation, differentiation and synthesis of protein (Fuentes *et al.*, 2013). *Myostatin* (*MSTN*) negatively regulated muscle growth and develops during embryogenesis in vertebrates: sole gene in mammals, gene duplication in fish and alternative splicing in birds (Huang *et al.*, 2011). Therefore splicing of *MSTN* gene could affect embryonic muscle development of animals.

7. Effect of myogenic regulatory factors (MRFs) in myogenesis

Myogenesis is regulated by a group of muscle-specific transcription factors named *myogenic differentiation* (*MYOD*), *myogenic factor* (*MYF*) *4/5* and *myogenin* which are collectively referred to MRFs (Figure 3). Those are highly conserved and expressed in the skeletal muscle lineage including *PAX3* and *PAX7*. Buckingham (2001) stated that *paired box* transcription factors were firstly expressed in a portion of cells of mesoderm followed by *MYF5* and *MYOD*. *PAX3* is known to be essential for skeletal myogenesis that also upregulated *MYOD* during skeletal muscle development, in addition, *PAX7* maintains a population of quiescent satellite cells to play a role in the expansion for the activation of myoblasts combined with *MYF5* (Ridgeway and Skerjanc, 2001 and Knight and Kothary, 2011). *MYOD* and *MYF5* are required for the commitment to the muscle cell type, whereas *myogenin* and *MRF4* are required for inducing differentiation and formation of muscle fibers (Rehfeldt *et al.*, 2011a). The study of Meadows *et al.*, (2008) pointed out that *MYF5*, *MYOD* and *MRF4* are functionally activated muscle stem cells from the quiescent state, and induce the expression of target genes essential for muscle stem cell proliferation, in addition, their involvement observed during differentiation and fusion of embryonic myoblasts into multinucleated fibers. *MYOD* is believed to determine the differentiation potential for myoblasts that is activated and combined with *myogenin* and *myocyte enhancer factor 2* (*MEF2*) to induce the process of differentiation (Knight and Kothary, 2011) but the study of Pownall and Emerson (1992) stated that in avian species *MYOD* could induce other MRFs and eventually result in the expression of muscle-specific proteins.

The myoblasts of Angus cattle were assessed for rate of proliferation, and gene expression of *PAX7*, *MYOD*, *MYF5* and *MYOG*, in which proliferation rate found greater than Hereford and Wagyu X Angus at 5–20 hours of *in vitro* culture (Coles *et al.*, 2015). The study of Yin *et al.*, (2014) on *PAX3*, *MYOD1* and *MRF4* genes showed greater expression in pectoralis major muscle of chicken for low weight select than high weight select at day old but the expression of these genes showed higher for high muscle weight than low muscle weight at

28 days. The expression levels of *MYF5*, *MYF6*, *MYOD* and *myogenin* genes were identified by Zhu *et al.*, (2014) in the breast muscle (BM) and leg muscles (LM) of Jinding ducks (*Anas platyrhynchos domestica*) at 13, 17, 21, 25 and 27 d of embryonic periods. In this study, *MYOD* gene showed lower expression level in BM at 21 d of embryonic period than LM. Therefore higher expression patterns of *MYOD* in BM suggesting its involvement to maintain the development of different muscles. These findings also agreed with the study of Liu *et al.*, (2011). In the pectoral muscle of Pekin duck, fastest muscle development identified on 19 or 20 d in embryonic period (Gu *et al*., 2013) by the expression of *MRF4* and *MYOG* genes. During somite formation for carp fish, *MYF-5* expressed firstly followed by *MEF2C* and *MYOD*, then *MYOG* and *MEF2A*, and lastly skeletal *myosin heavy chain* and *alpha-actin* (Watabe, 1999).

8. Conclusions

Muscle development is an integrated and continuous process which could be improved by selection procedure along with the identification of quantitative trait loci and candidate genes by genome wide association studies for respective traits. Functional studies on specific genes associating muscle growth, discovering mutations with association studies on those genes could enhance the marker assisted selection in farm animals, in addition, the mechanism of growth and regulatory factors for individual muscle growth are also needed to explore.

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Conflict of interest

None to declare.

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