INTRODUCTION

Facts and science of the growth and development of skeletal muscle have a long history of interest to animal scientists. Technological advances and scientific insights have had profound effects on our understanding of skeletal muscle growth and development. Not only will a better understanding of this process lead to improved strategies to increase the efficiency of lean tissue deposition in domestic animals, but it also has human health implications (Reecy et al., 2003). Thus, there is an ever-growing need to define the molecular mechanisms controlling embryonic and postnatal skeletal muscle growth and development. In the history of skeletal muscle growth and development research, there have been a number of landmark discoveries like the ability to grow myoblast myotubes in cell culture (Rinaldini, 1959), the discovery of the satellite cell (Mauro, 1961), and the discovery of myoD (Lassar et al., 1986) to name a few. Muscle tissue consists of fibers (cells) that are highly specialized for the active generation of force for contraction. Because of this characteristic, muscle tissue provides motion, maintenance of posture, and heat production. Based on certain structural and functional characteristics, muscle tissue is classified into three types: cardiac, smooth and skeletal.

Characteristics of Muscle Fiber Types:

Skeletal muscle tissue is named for its location - attached to bones. It is striated; that is, the fibers (cells) contain alternating light and dark bands (striations) that are perpendicular to the long axes of the fibers. Skeletal muscle tissue can be made to contract or relax by conscious control (voluntary). All skeletal muscle fibers are not alike in structure or function (Kundu et al., 1991a). For example, skeletal muscle fibers vary in colour depending on their content of myoglobin (myoglobin stores oxygen until needed by the mitochondria). Skeletal muscle fibers contract with different velocities, depending on their ability to split Adenosine Triphosphate (ATP). Faster contracting fibers have greater ability to split ATP (Pandya et al., 2003). In addition, skeletal muscle fibers vary with respect to the metabolic processes they use to generate ATP. They also differ in terms of the onset of fatigue. Based on various structural and functional characteristics, skeletal muscle fibers are classified into three types: Type I
fibers, Type II B fibers and type II A fibers. The characteristics of individual fiber types are summarized in (Table 1).

**Type I Fibers:**

These fibers, also called slow twitch or slow oxidative fibers, contain large amounts of myoglobin, many mitochondria and many blood capillaries. Type I fibers are red, split ATP at a slow rate, have a slow contraction velocity, very resistant to fatigue and have a high capacity to generate ATP by oxidative metabolic processes. Such fibers are found in large numbers in the postural muscles of the neck.

**Type II A Fibers:**

These fibers, also called fast twitch or fast oxidative fibers, contain very large amounts of myoglobin, very many mitochondria and very many blood capillaries. Type II A fibers are red, have a very high capacity for generating ATP by oxidative metabolic processes, split ATP at a very rapid rate, have a fast contraction velocity and are resistant to fatigue. Such fibers are infrequently found in humans.

**Type II B Fibers:**

These fibers, also called fast twitch or fast glycolytic fibers, contain a low content of myoglobin, relatively few mitochondria, relatively few blood capillaries and large amounts glycogen. Type II B fibers are white, geared to generate ATP by anaerobic metabolic processes, not able to supply skeletal muscle fibers continuously with sufficient ATP, fatigue easily, split ATP at a fast rate and have a fast contraction velocity. Such fibers are found in large numbers in the muscles of the arms.

**Structure of individual muscle fiber:**

The individual muscle fiber is surrounded by a cell membrane, which allows the contents of the fibers to be quite different from that of the body fluids outside them. Inside the fiber are the myofibrils, which constitute the contractile apparatus, and a system for controlling the myofibrils through changes in calcium concentration. This
system, the *sarcoplasmic reticulum* (SR), is a closed set of tubes containing a high concentration of calcium. Each myofibril runs the whole length of the muscle fiber with a variable number of segments, the *sarcomeres*; it is only one or two micrometres in diameter, and is surrounded by the SR network. The myofibril consists of many much thinner and shorter protein rods, which are the *myofilaments*. These are of two kinds: thick filaments, which are made predominantly from a single protein, *myosin*, and thin filaments, which contain the protein *actin* (Greunstein and Rich, 1975). The actual contraction takes place by an interaction of the actin with projections on the myosin molecules (*crossbridges*) The net effect of many of these small movements and small forces is to shorten the myofibrils, and thus the whole muscle; hence some part of the skeleton is moved, by means of the attachment of the muscle at each end to bone, directly or via tendons (Fig. 1).

**Body muscle make up:**

Most skeletal muscles of the body are a mixture of all three types of skeletal muscle fibers, but their proportion varies depending on the usual action of the muscle. For example, postural muscles of the neck, back, and leg have a higher proportion of type I fibers. Muscles of the shoulders and arms are not constantly active but are used intermittently, usually for short periods, to produce large amounts of tension such as in lifting and throwing. These muscles have a higher proportion of type I and type II B fibers.

Even though most skeletal muscle is a mixture of all three types of skeletal, all the skeletal muscle fibers of any one motor unit are all the same (Kujelberg, 1973a; Larsson and Tesch 1986). In addition, the different skeletal muscle fibers in a muscle may be used in various ways, depending on need. For example, if only a weak contraction is needed to perform a task, only type I fibers are activated by their motor units. If a stronger contraction is needed, the motor units of type II A fibers are activated. If a maximal contraction is required, motor units of type II B fibers are activated as well. Activation of various motor units is determined in the brain and spinal cord (Kujelberg, 1973a). Although the number of the different skeletal muscle fibers does not change, the characteristics of those present can be altered.
Fiber type modifications:

Various types of exercises can bring about changes in the fibers in a skeletal muscle. Endurance type exercises, such as running or swimming, cause a gradual transformation of type II B fibers into type II A fibers. The transformed muscle fibers show a slight increase in diameter, mitochondria, blood capillaries, and strength. Endurance exercises result in cardiovascular and respiratory changes that cause skeletal muscles to receive better supplies of oxygen and carbohydrates but do not contribute to muscle mass. On the other hand, exercises that require great strength for short periods, such as weight lifting, produce an increase in the size and strength of type II B fibers. The increase in size is due to increased synthesis of thin and thick myofilaments.

Skeletal muscle is both a highly ordered and a heterogeneous tissue in the sense that it contains intracellular structures, such as complex sarcoplasmic and transverse tubular membrane systems and mitochondria, surrounding a highly structured myofilament array. Skeletal muscle is an extremely adaptive tissue, with even the most intensive training modules being unable to inflict permanent damage upon the fibers (Linge 1962). Sometime after birth, myofibers are terminally differentiated, i.e. they become specialized cells whose properties arise from tissue specific gene expression (Lluis et. al., 2006). Additionally, it means that skeletal muscle fibers are maintained through a system that is non-mitotic (meaning the fibers themselves are unable divide and replicate). They must survive the length of the organism’s lifetime and possess distinct methods of cellular remodeling and repair to ensure this survival (Latella & Crescenzi 2000, Hawke 2001). Carrying out an essential role in this process are intriguing molecules referred to as satellite cells which can be thought of as stem cells of the muscle. These mononucleated myoblasts (precursors to muscle cells) lie dormant outside the sarcolemma sandwiched in small pockets between adjacent muscle fibers upon normal situations, but can be triggered by muscular trauma (Hawke 2001). Upon activation they fuse with the damaged tissue and donate their nuclei to initiate the repair process.

Without satellite cells we would be unable to adapt and repair damaged muscle tissue. This has been displayed in rats that completely lost the hypertrophic response to mechanical overload after irradiation to destroy satellite cell populations (Hawke 2001)
Interestingly, it is also illustrated in the fact the cardiac muscle appears to be absent of them, explaining why after trauma such as myocardial infarction the damaged tissue is unable to restore function and becomes necrotic (Tsika 2006). Satellite cells represent an excellent starting point when addressing the development of skeletal muscle hypertrophy as they initiate the process of repair and adaptation. At this point, two important concepts should be clear to the reader: That increases in muscle size occur primarily from the repair of existing muscle fibers, not division of the muscle cells themselves (hyperplasia), and a certain degree of muscle fiber damage must take place to trigger the process (Hawke 2001, Goldspink 2003). Though many individuals tend to relate a high level of delayed onset muscle soreness (DOMS) to a productive workout, it should be noted that muscle damage may occur without extreme levels of soreness and that DOMS may actually signify remodeling rather than damage (Yu J-G et. al., 2004). Additionally, recent research has suggested that the muscle fascia plays a major role in the sensation of DOMS rather than the fibers themselves (Gibson 2009).

**Somatic growth vs. Muscle development:**

Somatic growth is generally viewed as an increase in body size resulting from growth of several if not all of the tissues comprising the organism. Skeletal muscle tissue represents a significant portion of the mass of most fish, and may compose between 30–80% of their live weight (Weatherley and Gill, 1987). Because skeletal muscle may be the main contributor to body mass it is probable that the size of an organism is limited by the growth of this tissue. Investigating muscle growth is an important focus of study from a biological standpoint as the growth of this tissue may underlie the overall somatic growth in any organism.

Although somatic growth can be easily measured in the form of body weight, it only gives an indirect estimation of the muscle growth. A long-established method to measure muscle growth, which provides useful quantitative data, is the measurement of muscle morphological parameters in a representative area of lateral muscle in fish of different ages, sizes or conditions (Johnston et. al., 1998; Valente et. al., 1999). Commonly, a whole cross-section of the body is used to measure the total transversal myotomal area (Abdel et. al., 2005). The size and number of muscle fibers in restricted areas of the cross-section is used to characterize muscle cellularity and to obtain an
estimate of the size and number of muscle fibers (Weatherley and Gill, 1985; Rawlinson et al., 1995; Abdel et al., 2005). Some previous studies have shown that the diameter and number of muscle fibers varies along the length of the body and in different regions of the same cross-section (Strickland, 1983; Kissing et al., 1991; Rawlinson et al., 1995; Johnston, 2001a). For example, Kiessling et al., (1991) found marked differences between the ventral and dorsal region of the white epiaxial muscle indicating different growth areas within the muscle. However, previous works did not particularly focus on comparing muscle cellularity between distant zones of the trunk musculature that display different gross morphology (Abdel et al., 2005).

To improve the productivity of economically important animals, generally two important traits like overall growth and body weight are targeted. However, these traits are dependent on the growth of the skeletal muscle, which constitutes most of the somatic tissue. It is therefore, imperative to understand the relationship between the skeletal muscle development and growth. Indeed, skeletal muscle dynamics has become an important theme in meat science and aquaculture as well. Advances in understanding muscle development have altered the yield of Atlantic salmon (Johnston et al., 1997), rainbow trout (Weatherley et al., 1980; Stickland 1983; Kiessling et al., 1991) and pigs (Dwyer et al., 1993). Moreover, it has been demonstrated that in pig breeding programs, selection for muscle characteristics is a promising way to increase overall body mass (Lefaucheur, 2010).

The basic functional unit of muscle tissues is the muscle fibre or fasciculus. In several fish and shellfish, skeletal muscle growth involves hyperplasia of fasciculi until a certain age, coupled with hypertrophy of individual fasciculi throughout the lifetime of the fish. Hence, muscle mass is determined by the number and size distribution of fasciculi, a trait collectively referred to as muscle cellularity (Rehfeldt et al., 1993). Not surprisingly, muscle cellularity is considered to be an important measure of muscle growth (Fauconneau et al., 1993; Hurling et al., 1996). It is of particular interest in aquaculture owing to the large variation observed at the genotypic and phenotypic levels (Johnston and Moon, 1980; Weatherley et al., 1980; Kiessling et al., 1991). Studies have shown that the number of fasciculi constituting a given body size varies between fish species as well as between different strains of the same species (Weatherley et al., 1980). Furthermore, fasciculi size and number are affected by
environmental factors like diet (Kiessling et al., 1991) and exercise (Johnston and Moon, 1980).

**Cellular mechanisms of post-embryonic growth:**

Muscle is a post-mitotic tissue and post-embryonic growth involves populations of undifferentiated myoblasts. As fibers expand they absorb myoblast nuclei in order to maintain a relatively constant nuclear to cytoplasmic ratio (Koumans et. al., 1994). New fibers form on the surface of existing fibers by the fusion of myoblasts to form multinucleated myotubes. The decision of a myoblast to leave the cell cycle and differentiate is determined by antagonistic signals for proliferation and differentiation that are incompletely understood in mammals and hardly studied at all in fish. In mammals, members of the MyoD gene family are thought to activate the muscle differentiation program and inhibit proliferation of the myoblast producer cells (Lluis et. al., 2006).

The MyoD families are components of a highly redundant and poorly understood regulatory system modulating muscle growth which includes numerous growth factors including insulin-like growth factor-1 (IGF-1) (Olson, 1992; Florini et. al., 1991), and in mammals, a newly discovered member of the transforming growth factor (TGF-b) family called myostatin (McPherron et. al., 1997). Null mutations of the myostatin gene result in mice with skeletal muscles three times heavier than wild-type animals, but which are otherwise normal and healthy (McPherron et. al., 1997; Grobet et. al., 1998). Null mutations for IGF-1 in mice result in muscles with a reduced number of fibers (Liu et. al., 1993). For a discussion of some of the numerous other factors thought to have a role in controlling the transcription of muscle genes see the work of Olson (1992).

**Types of Muscle Hypertrophy:**

Muscle hypertrophy can simply be defined as an increase in size of the myofibril(s). Though easily defined, it is an intriguing and complex adaptive process carried out in response to external stimuli. Causing this adaptation leads to a greater cross sectional area thus a greater ability to produce force. A more in depth examination shows us that
there exist two major forms of hypertrophy that occur in skeletal muscle; myofibrillar and sarcoplasmic. Myofibrillar hypertrophy is characterized by an increase in number of the actual contractile components of the sarcolemma, mainly being the myosin heavy chain proteins and actin filaments (Greunstein and Rich, 1975). This type of hypertrophy is responsible for the concomitant increases in muscle force production with fiber growth. Sarcoplasmic hypertrophy occurs when the non-contractile components of the muscle fiber increase, examples of this include enlargement of the sarcoplasmic reticulum, increased intracellular fluid retention, and increased storage of metabolic fuels such as glycogen and creatine phosphate.

**Muscle growth in fishes:**

Muscle growth is a dynamic process in fish that begins early in their development and continues throughout much if not all of their life span (Koumans *et. al.*, 1993a). During initial myogenesis several mitotically active stem cells, referred to as myosatellite cells, fuse together to form individual multinucleated muscle fiber cells (Nag and Nursall, 1972; Campion, 1984; Schultz, 1989; Matschak and Stickland, 1995; Koumans *et. al.*, 1990). Resulting muscle fibers are the fundamental unit of skeletal muscle tissue, as hundreds to several thousand fibers collectively form a single muscle mass. Further recruitment of new fibers (hyperplasia) and enlargement of existing fibers (hypertrophy) within of sexual maturity in fish. Individual fibers are often classified as being red, pink, or white based on metabolic and structural properties. In fish musculature different fiber types are not intermingled but are separated into either superficial muscle masses comprised exclusively of red fibers or deep muscle masses composed entirely of white fibers (Weatherley *et. al.*, 1980b). Because deep muscle masses may comprise more than 90% of the total volume of muscle tissue in fish (Weatherley and Gill, 1989) it is probable that increasing body size is due in large part to growth of white muscle fibers.

Growth in fish involves the recruitment and hypertrophy of muscle fibers (Koumans *et. al.*, 1993a). Muscle recruitment is particularly important in species that reach a large ultimate body size. The number of muscle fibers recruited to reach a particular girth varies between families and strains and is influenced by environmental factors including diet, exercise, light and temperature regimes. The resulting variation in
muscle cellularity and associated changes in connective tissue matrix are thought to be important determinants of texture and other flesh quality characteristics (Abdel et. al., 2005).

Several studies have demonstrated that recently recruited fibers in fish are relatively small in size and that these fibers increase in cross-sectional area through hypertrophic growth (Moss and LeBlond 1971; Stickland 1983; Ennion et. al., 1995). Growth of skeletal muscle occurs by both hyperplasia and hypertrophy of muscle fibers (Zimmerman and Lowery, 1999). In mammalian muscle, hyperplasia is restricted largely to the pre- and perinatal periods (Chiakulas and Pauly 1965; Moss and Leblond 1971; Goldspink 1972; Schultz 1974). In contrast to that, in teleost hyperplasia continues, together with hypertrophic growth, during the post-larval period. These processes give rise to a typical mosaic appearance of muscle fibers in transversal sections, with fibers of different diameter intermingled (Zimmerman and Lowery, 1999). The relative contribution of muscle fiber hypertrophy and hyperplasia to the total muscle growth varies markedly among different species (Greer-Walker 1970; Weatherley and Gill 1985; Weatherley et. al., 1980a & 1988) and in different muscle fiber types (Stickland 1983; Kiessling et. al., 1991). It also seems to depend on external factors like temperature (Ayala et. al., 2000, 2001a, b, 2003; Lopez Albors et. al., 2003), photoperiod (Johnston et. al., 2003b), exercise training (Johnston and Moon 1980), and diets (Weatherley et. al., 1980b; Fauconneau et. al., 1997). The plasticity of muscle growth under different production conditions determines different muscle cellularity, which is a major factor in determining quality, in particular the texture and processing characteristics of the flesh (Johnston, 1999; Kundu 1990, Ph D thesis).

**Amphibians:**

Muscle fibers of *Xenopus laevis*, a frog formerly classified as a toad, were the first to be typed based on a combination of physiological, morphological, histochemical and biochemical characteristics. Currently the most widely accepted criterion for muscle fiber typing is the myosin heavy chain (MHC) isoform composition because it is assumed that variations of this protein are the most important contributors to functional diversity. MHC isoforms expressed in *Xenopus* muscle are functionally different thereby
validating the idea that MHC isoform composition is the most reliable criterion for vertebrate skeletal muscle fiber type classification (Olena et. al., 2006).

**Reptiles:**

Lizard skeletal muscle fiber types were investigated in the ileiophibularis (IF) muscle of the desert iguana (*Dipsosaurus dorsalis*). Three fiber types were identified based on histochemical staining for myosin ATPase (mATPase), succinic dehydrogenase (SDH), and glycerophosphate dehydrogenase (GPDH) activity (Bonine et. al., 2005). The pale region of the IF contains exclusively fast-twitch-glycolytic (FG) fibers, which stain dark for mATPase and GPDH, light for SDH. The red region of the IF contains fast-twitch-oxidative-glycolytic (FOG) fibers, which stain dark for all three enzymes, and tonic fibers, which stain light for mATPase, dark for SDH, and moderate for GPDH confirm these histochemical interpretations (Gleeson & Putnam, 1980). Lizard FG and FOG fibers possess twitch contraction times and resistance to fatigue comparable to analogous fibers in mammals, but are one-half as oxidative and several times as glycolytic as analogous fibers in rats. Lizard tonic fibers demonstrate the acetylcholine sensitivity common to other vertebrate tonic fibers (Gleeson & Putnam, 1980 & Bonine et. al., 2005).

**Muscle development in birds and mammals:**

In bird species, muscle development starts with the emergence of the first somites 24 h after the beginning of incubation. Myogenic precursor cells arise from the somites to give myoblasts, which after multiplication, migration to their final location and fusion into multinucleate myotubes, eventually differentiate into muscle fibers. The final number of fibers is reached at the end of embryogenesis in most muscles under normal conditions. Post hatch muscle growth is achieved by an increase in fiber size, which is associated with an increase in the number of nuclei per fiber (so called myonuclei, Moss, 1968). Myonuclei have lost the ability to divide so that this increase is due to the mitotic activity of a residual population of myogenic precursor cells present between the muscle fiber and its surrounding basement membrane, the so-called muscle satellite cells. These cells have the capability to multiply and fuse with the adjacent fibers, bringing in more nuclei and a greater capacity for protein synthesis. This phenomenon
accounts for more than 98% of the final DNA content of muscle (Moss, 1968). Several
growth factors have been identified as candidate to modulate muscle growth at each
stage of development. Insulin-like Growth Factors (IGF-1 and IGF-2) exert a general
effect on overall body growth (Philippou et. al., 2007) and both genes are expressed in
the muscle tissue together with specific receptors, suggesting a paracrine mode of
action. Indeed, overexpression of the IGF-1 gene in the muscle tissue of transgenic
mice leads to selective muscle hypertrophy. The IGFs have been shown to stimulate the
proliferation, the differentiation and the metabolism of a number of myogenic cell lines
from different species as well as the anabolism of differentiated myotubes or muscle
fibers (Florini et. al., 1996).

Maximal running exercise, without the eccentric components, affects the activities of
lysosomal enzymes in all types of rat muscular fibers. The lack of uniform activity
profile for the lysosomal enzymes studied probably reflects the variety of their cellular
functions. It is widely known that physical exercise reflects in the imbalance of
homeostasis. The resulting changes depend on the intensity of stimulation and its
duration. One of possible exercise-associated consequences is the damage of muscle
fibers (Armstrong et. al., 1983) which might consider either sarcolemma, or myofibrils
and cell organelle (Takekura et. al., 2001). Free radicals (Neiss et. al., 1999) increased
acidification, hypoxia, and some metabolites and elevated intracellular Ca\(^{2+}\)
concentration are mentioned as the factors responsible for those injuries (Ebbing &
Clarkson 1989). Exercise stress reflects in the changes of protein composition either in
cytosol or in plasma membranes and extracellular matrix (Farges et. al., 2002, Yu JG
et. al., 2002).

Stretch, or increased tension, is a major component contributing to postnatal increases
in skeletal muscle mass (Goldberg , 1967; Goldspink, 1974 & 1977). In the avian wing-
weighting model of stretch hypertrophy, the anterior latissimus dorsi (ALD) muscle is
chronically stretched and loaded. This results in significant activation of skeletal
muscle satellite cells, stimulating their reentry into the cell cycle (Winchester et. al.,
1991). These activated satellite cells subsequently fuse with existing fibers, causing
myofiber enlargement (hypertrophy) (Alway et. al., 1989 & 1990, Mc Cormick and
Schultz 1992, Sola et. al., 1973), and generate nascent myofibers, thereby increasing
myofiber number i.e. hyperplasia (Zimmerman and Lowery, 1999; Alway et. al., 1989
& 1990, Sola et. al., 1973). It appears that stretch converts mature muscle fibers into a more immature form, as embryonic isoforms of myosin are stimulated by chronic load (Kennedy et. al., 1988, McCormick and Schultz 1992).

Although the structural phenotype associated with stretch hypertrophy has been nicely characterized, very little is known concerning the mechanism of how stretch is translated into hypertrophy. One group of factors intimately associated with regulation of skeletal muscle development, and, therefore, potential activators and mediators of satellite cells in vivo during postnatal hypertrophy, is the fibroblast growth factors (FGFs). In clonal skeletal muscle cell lines, FGFs stimulate proliferation and inhibit differentiation (Gospodarowicz et. al., 1975; Linkhart et. al., 1980 & 1981, Olwin and Hauschka 1986, Rando and Blau 1994). Although FGFs are powerful regulators of skeletal muscle development in vitro and are localized to developing embryonic skeletal muscle in vivo, it is not known which members of the FGF family are expressed in adult skeletal muscle. It is also not clear what biological importance, if any, these expressed FGFs might have during postnatal skeletal muscle hypertrophy (Mitchell et. al., 1999). It has been found that several members of the FGF family are expressed in adult skeletal muscle and that they exhibit differential mRNA expression and protein localization patterns in response to stretch-induced hypertrophy. These results implicate certain FGFs as potential activators and mediators of postnatal skeletal muscle hypertrophy and also suggest distinct roles for individual FGFs during postnatal skeletal muscle development (Mitchell et. al., 1999).

Most muscles in mammalian and avian species studied to date contain a mixture of different types of skeletal muscle fibers. This reflects the fact the muscle fibers are adapted morphologically and biochemically to support a specific functional requirement. Adaptive changes in muscles which occur during development or in response to disease, exercise or experimental manipulation are often reflected in observable changes within the populations of specific types of muscle fibers. Cytoenzymatic techniques can be used to visualize a variety of biochemical parameters at the fiber level and have been used as a basis for a number of systems for classifying types of muscle fibers. Since fibers are dynamic, a workable system must be one that can be applied across all physiologic and developmental states, and preferably across species lines.
From developmental studies of chicks (Ashmore et. al., 1970 & 1974), lambs (Ashmore et. al., in press), pigs and cattle (unpublished) it has been concluded that a dual system of nomenclature is required to identify fiber types accurately. The myofibrillar adenosine triphosphatase (ATPase) reaction can be used to differentiate two fiber types which in adult muscles are physiologically “fast twitch” or “slow twitch” (Johnston & Tota, 1974; Bernard et. al., 1971, Estrom et. al., 1968). In fetal muscles and in muscles of some neonates this reaction must be done after acid preincubation (Guth et. al., 1970) in order to identify these two fiber types accurately, since the histochemical reaction after alkaline preincubation oftentimes does not correlate well with specific myofibrillar ATPase activity as assayed biochemically or with physiologic measurements of contraction (Johnston & Tota, 1974; Guth et. al., 1972). Second, since the speed of contraction of a fiber may not be related to its capacity for repetitive contraction, an estimate of dependence upon aerobic metabolism for energy generation is required. So a system of nomenclature was used which describes a fiber as α if it is, or is to be, a fast twitch fiber and β if it is, or is to be, a slow twitch fiber.

Birds and mammals maintain their body temperature in a cold environment by thermogenesis. Shivering is an acute thermogenic response to protect organs, whereas nonshivering responses allow long-term adaptation to a cold environment. The brown adipose tissue (BAT) is a thermogenic organ, present in mammals, that mediates a nonshivering adaptive response. This organ is highly developed in small mammals, such as rodents, as well as in neonatal humans (Himms Hagen, 1990; Jansky, 1973; Nicholls & Locke 1984). BAT does not appear to be a major organ for thermogenesis in humans and other large mammals because it accounts for only 0.3% of the total body weight (Block, 1994). Instead, skeletal muscle is thought to be involved in thermogenesis in humans and other large mammals because it is the largest organ in the human body, accounting for 40% of the total body weight (Zurlo et. al., 1990). Analysis of gene expression patterns combined with histological analysis showed that cold exposure caused a transformation of skeletal muscle fibers from fast-twitch to slow-twitch in chicks acquiring thermogenesis.

Postnatal skeletal muscle growth is classically attributed to fiber hypertrophy and myogenic differentiation, but these processes do not account for the size-independent increase of muscle mechanical performance that occurs during postnatal growth. There
is also little knowledge about the precise time-course of contractile function or the underlying factors that affect it. Expression of four myosin heavy chain isoforms (embryonic, neonatal, IIX and IIB), as well as desmin, correlated significantly with muscle mechanical function (Greunstein and Rich, 1975). Stepwise multiple regressions showed that, of the variables measured, percentage content of neonatal myosin heavy chain was the best predictor of mechanical function during the postnatal time-course (Greunstein and Rich, 1975). These data provide the first specific structural basis for increases in muscle tension development during growth. Therefore, models of muscle growth must be modified to include an intrinsic quality enhancement component.

Recently, a narrative review has suggested that increase in muscle fiber number (hyperplasia) in animals occur as a result of stretch overload, whereas compensatory hypertrophy (ablation, tenotomy) does not generally change fiber number (Zimmerman and Lowery, 1999; Antonio & Gonyea, 1993). In addition, it was also reported that exercise models in animals have led to mixed results with regard to increases in muscle fiber number (Antonio & Gonyea, 1993). Although the above-mentioned review provided valuable information, it relied on the traditional narrative approach that is, chronologically arranging and then describing studies. A need exists for the quantification of the magnitude and direction of changes in skeletal muscle fiber number as a result of different types of mechanical overload in animals (George, 1966). The effect of propranolol on beta-adrenergic agonist clenbuterol-induced changes in muscle fiber size and protein content were studied by Jones et. al., (1985). Propranolol did not inhibit the ability of clenbuterol to stimulate protein accretion but reduced the increase in muscle fiber size. The compositional and physical characteristics of clenbuterol-induced muscle growth thus appeared to be separated by propranolol (Maltin et. al., 1987).

Current knowledge about the significance of muscle fiber type in modulating growth performance and meat quality is presented for various species. Fiber type can be modified by numerous intrinsic and extrinsic factors, such as muscle type, species, breed, major genes, individual, sex, fetal and postnatal nutrition, ambient temperature, exercise, growth-promoting agents, and transgenes. The relatively high heritability of some histochemical characteristics suggests that selection can be efficiently used to
manipulate muscle fiber type composition by including fiber attributes in selection indexes. The correlated responses of growth and meat quality traits to this selection may be useful studies for better understanding the significance of muscle fiber type in determining growth performance and meat quality (Lefaucheur and Gerrard, 2010). Finally, increasing the total number of fibers, a characteristic established before birth in most farm mammals (Lefaucheur and Gerrard, 1998), is a promising way to increase muscle mass without increasing fiber size, which is sometimes speculated to alter meat quality. However, there still is not enough research that definitely demonstrates the deleterious effect of increasing fiber size on meat quality, and more research is also needed to better understand the mechanisms that regulate the total number of fibers (Grobet et. al., 1997; Lefaucheur and Gerrard, 2000).

Great strides have been made in our understanding of skeletal muscle growth and development. These advancements have come about because of technological innovations and scientific discoveries. It can be believed that these technological innovations are advances that will have profound influences on our understanding of skeletal muscle growth and development. However, future advances will be necessary if we are to successfully develop new strategies to enhance lean tissue deposition in livestock and/or prevent muscle loss in at risk individuals (Reecy et. al., 2003).

The purpose of this study was to investigate several aspects of somatic and muscle fiber growth in the white leghorn chick (Gallus gallus domesticus) and Swiss albino mouse (Mus musculus domesticus). Somatic growth was evaluated through weight and age relationships and muscle growth was investigated by measuring the mean diameter of fibers representative of recently hatched to sexually mature specimens. These measurements enabled us to assess the contributions of hyperplasia and hypertrophy throughout the entire life span of these species of chick and mice. We believe our findings provide considerable insight into the processes that enable these species to grow to their impressive ultimate adult size. This understanding, in addition to its biological interest, has potential value for enhancing applied biomedical techniques and furthers the knowledge of hyperplastic and hypertrophic growth patterns in chick and mice. These animals are currently the very commonly used species for which the dynamics of muscle fiber growth have been discerned.