TRIENNIAL GROWTH SYMPOSIUM: THE NUTRITION OF MUSCLE GROWTH: Impacts of nutrition on the proliferation and differentiation of satellite cells in livestock species[1,2]

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ABSTRACT: Nutrition and other external factors are known to have a marked effect on growth of skeletal muscle, modulated, at least in part, through effects on satellite cells. Satellite cells and their embryonic precursors play an integral role in both prenatal and postnatal skeletal muscle growth of mammals. Changes in maternal nutrition can impact embryonic muscle progenitor cells which ultimately impacts both prenatal and postnatal skeletal muscle development. Satellite cells are important in postnatal skeletal muscle growth as they support the hypertrophy of existing myofibers. Hypertrophy of existing fibers is the only mechanism of postnatal muscle growth because muscle fiber number is fixed at birth and fiber nuclei have exited the cell cycle. Because fiber nuclei do not divide, additional nuclei required for hypertrophy must be acquired from satellite cells. To date, little research has aimed at determining whether nutrition directly impacts satellite cell populations within skeletal muscle of livestock species. However, it is well established that nutrition alters circulating concentrations of various growth factors such as insulin-like growth factor 1, epidermal growth factor, hepatocyte growth factor, and fibroblast growth factor. Each of these different growth factors impacts satellite cell proliferation and/or activation, indicating that nutrition likely plays a large role in skeletal muscle growth through impacting the satellite cell pool in both prenatal and postnatal growth. The relationship among nutrition, growth factors, and satellite cells relative to skeletal muscle growth is an important area of research that warrants further consideration.

Key words: growth, livestock, nutrition, satellite cells, skeletal muscle

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INTRODUCTION

Skeletal muscle growth is of the utmost importance in the livestock industry as it becomes the marketable product, meat. It is especially important to producers that their livestock grow to produce more lean product and less fat to increase overall production efficiency, while still maintaining a certain level of fat to ensure production of a high-quality product. Many different factors, including nutrition, genetics, and management practices, are known to impact growth of skeletal muscle in livestock species. It is well established that skeletal muscle growth occurs through a combination of protein accretion, myonuclear accretion,

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and accrual of extracellular matrix (Allen et al., 1979; Laurent et al., 1985; Mozdziak et al., 1997). Skeletal muscle is a heterogeneous tissue and grows through several different mechanisms, making determination of how these different factors impact skeletal muscle growth complicated. Satellite cells (**SC**) and their embryonic precursor cells play an integral role in skeletal muscle growth both prenatally and postnatally as they are required for myonuclear accretion. Prenatally skeletal muscle forms from embryonic muscle progenitor cells that are believed to be distinct from SC, but share a common origin (Gros et al., 2005; Lepper et al., 2009). These cells fuse to form the primary and secondary myofibers during prenatal skeletal muscle development. Postnatally, it is generally accepted that SC are important for providing the nuclei necessary for hypertrophy of existing myofibers. However, recent data suggest that skeletal muscle hypertrophy occurs in the absence of SC in a rodent model, but whether or not this occurs in livestock is unknown (Murach et al., 2018). It is well established that nutrient status of an animal impacts overall skeletal muscle growth. Nutrient status also alters concentration of circulating growth factors, which in turn, impact SC and skeletal muscle growth. However, the specific effect that nutrition has on the SC pool within skeletal muscle of livestock species is unknown, indicating that additional research is warranted in this area. Consequently, the purpose of this review is to focus on how nutrient status impacts SC and ultimately, skeletal muscle growth of livestock species.

EFFECTS OF NUTRITION ON SKELETAL MUSCLE GROWTH

Skeletal muscle hypertrophy occurs through protein accretion when the fractional synthesis rate of protein exceeds the fractional breakdown rate (Goll et al., 2008; Kumar et al., 2009). As such, the nutrient that is most discussed in the literature relative to skeletal muscle growth is protein. However, it is important to remember that energy density of the diet is also an important factor as energy is required for growth. Past research demonstrates that plane of nutrition affects body composition of cattle differently depending on what stage of growth that specific animal is in (Owens et al., 1995). In growing beef cattle, BW is closely paralleled by protein accretion, and fat accretion is more closely related to energy density of the diet the animals are consuming (Gill et al., 1993). It is well documented that provision of an adequate amount of dietary protein or essential AA results in increased muscle protein synthesis in healthy human adults (Hulmi et al., 2010). This is less understood in ruminant species as the AA composition of the diet is not the same as the AA that are absorbed in the small intestine due to ruminal modification of the AA composition via microbial protein synthesis. Various nutritional approaches aimed at increasing the amount of lean growth in livestock species have studied the amounts of dietary protein and(or) the protein:energy ratio in the diet (Mitchell, 2007). Previous research in growing pigs demonstrates that as protein content of the diet increased from 15.5% to 17.4% CP, growth rate also increased (Cooke et al., 1972). This same research group demonstrated that dietary a CP level of 22.3% resulted in maximum growth performance and lean content, whereas CP levels of 25.3% and 27.3% resulted in decreased rates of growth (Cooke et al., 1972). Concurrently, as dietary protein concentration and protein deposition are increased, the rate of fat deposition decreases resulting in accrual of an increased amount of lean in the body (Cooke et al., 1972). More recently, it was reported that as dietary protein concentrations increased (100, 120, or 140 g/kg DM), there was also a linear increase in ADG of backgrounding cattle (Amaral et al., 2018). Additionally, feeding metabolizable protein at 85%, 100%, or 115% of the daily requirements also resulted in a linear increase in the weight gain of preconditioning beef steers (Moriel et al., 2015). These reports highlight the importance of protein concentration in the diet of growing animals to achieve optimal levels of skeletal muscle growth.

AA are known to play a role in skeletal muscle growth as they function as cell signaling molecules, regulators of gene expression, and the protein phosphorylation cascade (Wu, 2009). Studies conducted in both human and rodents demonstrate that leucine is believed to regulate protein synthesis by releasing inhibition of the initiation factor 4 complex, which ultimately activates the protein kinase mammalian target of rapamycin resulting in increased protein synthesis rates in skeletal muscle (Gautsch et al., 1998; Norton and Layman, 2006).

A study conducted in neonatal piglets showed that skeletal muscle growth is controlled through both AA and insulin concentrations (Davis et al., 2002). Specifically, arginine supplementation in neonatal piglets increases skeletal muscle through modulation of the mTOR signaling pathway (Wu et al., 2004; Yao et al., 2008). This research aligns with that of others demonstrating that non-essential

AAs (**NEAA**) play an integral role in growth and development of both humans and animals (Wu et al., 2013). The NEAA glutamine, glutamate, proline, glycine, and arginine are known to be important for cell growth and differentiation, protein degradation, cell signaling, gene expression, and nutrient metabolism, as well as many other physiological functions (Wu et al., 2013). Glutamine has also been shown to increase both mTOR signaling and protein synthesis of skeletal muscle in humans and animals (Xi et al., 2011).

Although a complete review of dietary protein and AA concentrations is beyond the scope of this review, it is important to understand that adequate dietary protein nutrition is important in ensuring that optimal skeletal muscle growth occurs in livestock species. Most of the research analyzing specific AAs has been completed using human, rodent, and porcine models, but recent research does indicate that dietary protein concentration is related to gross, phenotypic changes relating to growth of skeletal muscle in cattle (Moriel et al., 2015; Hales et al., 2016; Amaral et al., 2018). To date, most of the research analyzing the impacts of nutrient status on growth of skeletal muscle in livestock has not focused on SC specifically, but rather on phenotypic changes relating to overall skeletal muscle growth.

EFFECTS OF NUTRITION ON CIRCULATING GROWTH FACTORS

GH and IGF-1 are directly responsible for controlling growth in both cattle and swine (Lucy, 2008). GH regulates key metabolic pathways of intermediary metabolism and is known to be lipolytic, decreases protein catabolism and increases both plasma insulin and glucose concentrations (Brumby, 1959; Elsasser et al., 1989). Circulating GH acts on the liver to increase circulating concentrations of IGF-1 demonstrating that a strong relationship exists between these two growth factors (Boyd and Bauman, 1989). Circulating IGF-1 from the liver is known to act in an endocrine manner to influence growth and differentiation of tissues such as skeletal muscle and bone (Boyd and Bauman, 1989). However, many other tissues, including skeletal muscle, are known to synthesize IGF-1. Locally produced IGF-1 is thought to function via autocrine and(or) paracrine mechanisms to modulate skeletal muscle growth (Hannon et al., 1991). In addition, other growth factors such as IGF-2, epidermal growth factor (**EGF**), fibroblast growth factor (**FGF**), and transforming growth factor beta (**TGFβ**) are known to impact both skeletal muscle growth and differentiation and proliferation of SC. Nutritional status of an animal can impact circulating concentrations of each of these different growth factors, which in turn, can impact skeletal muscle growth and/or proliferation and differentiation of SC (Thissen et al., 1994). Both the protein and energy constituents of a diet have direct effects on the GH-IGF axis in ruminant and other livestock animals (Brameld et al., 1998). A study conducted in cattle demonstrates that when cattle receive a low level of metabolizable energy, plasma IGF-1 concentrations were similar in steers feed 11% or 14% CP, but were greater in animals at these levels vs. those fed 8% CP (Elsasser et al., 1989). In this same study, cattle that were fed a high level of metabolizable energy showed a linear increase in plasma IGF-1 concentrations as protein concentration in the diet increased (Elsasser et al., 1989). Additional research shows that energy source and concentration in the diet impact circulating GH concentrations in beef heifers (Houseknecht et al., 1988). Heifers fed a diet with low energy and fiber have increased circulating concentrations of GH compared with heifers fed a high-energy starch diet or a high-energy fiber diet (Houseknecht et al., 1988). This research demonstrates that dietary energy and protein concentrations, as well as the types of feed the energy and protein come from, have an impact on circulating concentrations of GH and IGF-1 in ruminant species.

Insulin-like growth factors have long been implicated as being involved in pre- and postnatal growth, lactation, reproduction, and immune function (McGuire et al., 1992). Nutrition impacts circulating concentrations of IGF-1 in humans, rats, and livestock species (Houseknecht et al., 1988; Elsasser et al., 1989; McGuire et al., 1992). In most species, fasting decreases circulating concentrations of IGF-1, and those concentrations can be returned to normal concentrations following realimentation (Clemmons and Underwood, 1991). Circulating concentrations of IGF-1 are decreased in growing cattle that are severely underfed (Breier et al., 1986; Ellenberger et al., 1989; Ronge and Blum, 1989). The role that nutritional status has on circulating concentrations of IGF-2 is less understood. Research demonstrates that steers fed 1% dry matter of the their BW had similar circulating IGF-2 concentrations when compared to steers that were fed 3% dry matter of their BW per day (Breier et al., 1988). Anytime that IGF is discussed relative to growth, you must also discuss the IGFBP, as they are directly related to concentration and action of the IGF. Circulating concentrations of IGFBP are

dependent on circulating concentrations of IGF, thus making concentrations of the IGFBP also dependent on nutrient status of an animal (McGuire et al., 1992). To date, little research has been conducted relative to the specific impact of nutrition on IGFBP concentrations and the resultant effects of those IGFB on SC activity. Furthermore, as some IGFBP promote the actions of IGF-1 and others antagonize IGF-1 action, it could be postulated that IGFBP concentrations are going to fluctuate with those of IGF-1 to best maintain the actions of IGF-1 in accordance with the nutrient status of the animal. Although conflicting results have been reported, circulating concentrations of GH typically decrease with a decreased plane of nutrition (Breier et al., 1986; Hayden et al., 1993). More complete reviews of the impact of nutrition on the GH-IGF axis in livestock animals can be found elsewhere (Clemmons and Underwood, 1991; Brameld, 1997; Brameld et al., 1998). How plane of nutrition impacts circulating concentrations of EGF, FGF, and TGFβ is not currently understood in in vivo livestock animals. Epidermal growth factor, FGF, IGF-1, and TGFβ are each known to impact SC, myoblast, and myofiber growth in primary cultured cells of livestock species (Allen and Boxhorn, 1989; Brameld et al., 1998).

Research conducted in primary porcine SC demonstrates that EGF stimulates proliferation in 2% FBS, but not in serum-free media (Doumit et al., 1993). This research group also found that treatment with a combination of FGF and IGF-1 or a combination of EGF and IGF-1 increased proliferation rates (Doumit et al., 1993). Insulin-like growth factor-1 is known to activate porcine SC through the mTOR pathway (Han et al., 2008). Treatment with IGF-1, FGF, EGF, or TGFβ increase proliferation rates in bovine satellite cells (Greene and Allen, 1991; Kamanga-Sollo et al., 2008, 2014; Reiter et al., 2014). Furthermore, treatment of fused bovine SC cultures with IGF-1, FGF, EGF, or TGFβ results in increased protein synthesis rates (Kamanga-Sollo et al., 2004; Reiter et al., 2014). However, treatment of bovine SC cultures with IGF-1 stimulates differentiation, whereas treatment with EGF, FGF, or TGFβ inhibits differentiation (Allen and Boxhorn, 1987; Greene and Allen, 1991). GH has been shown to increase protein synthesis rate in bovine skeletal muscle cells without altering mRNA expression of IGF-1 mRNA (Ge et al., 2012). Although this is by no means a complete review of how growth factors impacts SC proliferation, activation and subsequent skeletal muscle growth, the results reported above show that growth factors have a strong ability to modulate proliferation and differentiation of SC and the resultant skeletal muscle. Additional research is needed to determine specifically how nutrition impacts concentration of both circulating and locally produced growth factors and the effects that these growth factors have on SC growth.

IMPACT OF NUTRITION ON SKELETAL MUSCLE GROWTH PRENATALLY

Skeletal muscle mass increases through two different mechanisms, hyperplasia and hypertrophy. In mammals, hyperplasia of skeletal muscle only occurs prenatally and ceases shortly after birth, whereas hypertrophy of skeletal muscle occurs both prenatally and postnatally (Zhu et al., 2004; Greenwood et al., 2005; Du et al., 2013). Skeletal muscle growth requires the proliferation and differentiation of muscle progenitor cells in both fetal and adult tissues (Gros et al., 2005). It is important to note that in fetal tissues embryonic muscle progenitor cells are believed to originate from the same population of cells as SC, but are phenotypically distinct from SC (Gros et al., 2005; Lepper et al., 2009). The three main cell types which comprise skeletal muscle are myocytes, adipocytes, and fibroblasts. During prenatal development, each of these cell types develops from common progenitor cells in the mesoderm. Development of these different fetal tissues occurs through competition for progenitor cells during gestation (Du et al., 2010). When maternal nutrition is varied, the muscle composition of the offspring is also altered and can have impacts on meat quality and yield. Specifically, mid-gestation is a critical time period in which fat and muscle develop simultaneously in the fetus (Du et al., 2010). Furthermore, skeletal muscle is especially sensitive to the effects of fetal programming as it has lower priority than some of the other organs such as the brain, liver, and heart (Zhu et al., 2006). Skeletal muscle development of the fetus during gestation is especially important because there is no increase in the number of muscle fibers after birth.

Early skeletal muscle development can be separated into primary myogenesis and secondary myogenesis. Primary myofibers form during the first trimester of pregnancy and are followed by formation of secondary myofibers. Secondary myofibers account for the majority of muscle that is formed within an adult mammal. Because mammals are born with a set number of muscle fibers that are not capable of hyperplasia, it is likely that

in utero alterations to muscle fiber development impact muscle fiber characteristics. At least three different muscle fiber types (type I, type IIa, and type IIb) are found within the skeletal muscle of cattle, each differing in their metabolic properties, including anabolic and catabolic attributes, which have an effect on end-product quality (Thornton et al., 2012). Lambs resulting from dams which experiences gestational nutrient restriction were shown to have altered muscle profiles including fewer and larger muscle fibers and more slow-twitch muscle fibers in some muscle groups (Fahey et al., 2005; Zhu et al., 2006). Recent work revealed that bovine fetal primary myofibers were larger with maternal caloric restriction (Gonzalez et al., 2013). These data provide evidence that maternal plane of nutrition during gestation alters skeletal muscle development and composition in utero. Both maternal undernutrition and overnutrition during gestation have been shown to alter skeletal muscle growth in the resultant offspring (Larson et al., 2009; Tong et al., 2009; Yan et al., 2011; Long et al., 2012).

When the maternal plane of nutrition is below maintenance levels, it is hypothesized that the offspring develop a "thrifty" phenotype, allowing for the animal to survive during instances of low nutrition (Neel, 1962, 1999; Hales and Barker, 1992). The "thrifty" phenotype hypothesis suggests that when the fetal environment is poor, there is an adaptive response, which optimizes the growth of key body organs at the detriment of others which leads to altered postnatal development (McMillen and Robinson, 2005). Maternal nutrient restriction during gestation is generally believed to inhibit the expression of growth factors in the developing fetus, which ultimately negatively affects fetal skeletal muscle growth (Long et al., 2012). Previous research provides evidence that maternal plane of nutrition during the second trimester of gestation is the most critical period for skeletal muscle development (Zhu et al., 2004; Du et al., 2010, 2015). However, other research suggests that any effects of inadequate nutrition during the first or second trimesters of gestation can be alleviated as long as adequate levels of nutrition are provided during the third trimester (Greenwood and Thompson, 2007).

Recent data demonstrate that restricting maternal nutrition in either the second or third trimester of gestation results in offspring that have less and smaller myofibers at birth than those offspring that come from dams that did not experience a nutritional restriction (Underwood et al., 2010; Micke et al., 2011; Long et al., 2012; Mohrhauser et al., 2015). Currently, the molecular mechanism(s) through which these changes occur remains unknown. The main pathway that is thought to regulate differentiation of mesenchymal stem cells in utero is the Wnt/β-catenin pathway (Bonnet et al., 2010; Du et al., 2013). Activation of the Wnt/β-catenin pathway causes an increase in β-catenin which results in the mesenchymal stem cells to differentiate along the myogenic lineage and subsequent formation of skeletal muscle (Du et al., 2011, 2013). When the Wnt/β-catenin pathway is downregulated, formation of adipogenesis is favored and myogenesis is inhibited (Du et al., 2010). Bovine fetuses from dams that experienced a nutrient restriction of 60% of NRC recommended nutrients for the first 85 d of gestation had a reduction in paired box transcription factor 7 expression, required for pre- and postnatal muscle formation (Gonzalez et al., 2013). Furthermore, IGF-1 mRNA expression was also shown to be lower in nutrient restricted fetuses from dams that revived a 60% decrease in NRC recommendations (Gonzalez et al., 2013). In an additional study, mRNA expression was measured in the offspring of dams that received a decreased plane of nutrition (80% of NRC recommendation; Mohrhauser et al., 2015). Myosin heavy chain IIa and tissue inhibitor of metalloproteinase 3 were both expressed significantly lower in offspring from restricted dams in the semitendinosus muscle sampled at weaning (Mohrhauser et al., 2015). In cattle, providing dams with either a low starch or high starch diet during late gestation resulted in calves that had differential expression of imprinted genes in the longissimus lumborum muscle (Wang et al., 2015). Given that a high starch diet provides more energy, the mechanism through which energy is involved in the development of skeletal muscle prenatally needs to be further explored. Additionally, maternal plane of nutrition and time of weaning have been shown to alter the transcriptome of the longissimus lumborum of the offspring (Moisá et al., 2015). Another research group analyzed high, intermediate, and low planes of maternal nutrition (146%, 87%, and 72% of NRC recommendations, respectively) during mid-gestation and showed no differences in fetal growth characteristics of the skeletal muscle despite finding a difference in mRNA expression of genes related to adipogenesis and myogenesis (Jennings et al., 2016). Although there are several studies that have aimed to identify the differences in molecular mechanisms that occur within skeletal muscle following an alteration of maternal plane of nutrition, the mechanism is still not fully understood. As such, additional research needs to be completed to determine the specific

pathways through which maternal plane of nutrition is capable of altering skeletal muscle growth characteristics of the fetus that are capable of persisting throughout the life of the animal. It can be postulated that different mechanisms, such as the involvement of microRNAs, need to be investigated as there have been very studies that have shown a difference in mRNA expression in skeletal muscle of offspring following a restriction in maternal plane of nutrition.

Research analyzing the effects of maternal overnutrition during gestation on skeletal muscle development of the offspring provides conflicting results. Feeding ewes at either 20% or 80% above maintenance levels during gestation had no effect on birth or weaning weights of the offspring (Quigley et al., 2008). In contrast, other studies demonstrated that when the dam is fed at a level that causes an overweight phenotype, production of both the dams and offspring was decreased. This decrease was characterized by increased rates of dystocia and mortality as well as decreased milk production and reproductive soundness in the dam, and decreased weights and growth of the offspring (Han et al., 2000; Wallace et al., 2004; Larson et al., 2009; Long et al., 2012). Additional studies analyzing overnutrition of dams during gestation indicate a positive effect on production of offspring measured by increased weight and muscle mass as well as improved feedlot performance (Samuelsson et al., 2008; Zhu et al., 2008; Du et al., 2010, 2015; Yan et al., 2010, 2011). Research analyzing maternal overnutrition likely has conflicting results due to differences in the level of overnutrition, timing of the overnutrition, and differences among species. In a recent study, maternal nutrition was increased to 190% of NRC recommendation in cattle and showed that there was no difference in skeletal muscle development, but differential mRNA expression in fetal skeletal muscle tissues in late gestation was observed and there were also differences relative to the sex of the fetus (Gionbelli et al., 2018). As feed is the largest cost associated with any production operation and the current research results relating to offspring of performance following maternal overnutrition are conflicting, one can extrapolate that potential benefits that might come from this type of a management system are not great enough to outweigh the economic burden that this might place on producers.

As stated previously, dietary protein concentration is thought to be the most important nutrient associated with skeletal muscle growth in growing animals, as such, several research studies have analyzed the effects of restricting maternal dietary protein concentration during gestation on development of the offspring. Restricting maternal protein intake to 70% of NRC recommended amounts in the diet of the dam during the second trimester had no effect on birth or weaning weights of the offspring (Micke et al., 2010). Decreasing protein concentration to 70% of NRC recommended amounts in the diet of the dam in either the first or second trimester of gestation alters mRNA expression of IGF-1, IGF-2 and their receptors, IGF-1 receptor and IGF-2 receptor, in the skeletal muscle of the offspring (Micke et al., 2011). Additionally, when maternal nutrition is restricted to 70% of the NRC recommendation, but protein is supplemented at 100% of the NRC recommendation, there is no difference in skeletal muscle growth of the offspring relative to growth and gene expression of the offspring (Long et al., 2012). This research demonstrates that dietary protein intake of the dam is very important to proper development of skeletal muscle in the offspring; however, more research is needed to determine which concentration of dietary protein is optimal, the period of gestation at which maternal protein nutrition is most important, and how protein concentration in the maternal diet affects skeletal muscle growth at a molecular level.

Some recent studies suggest that maternal plane of nutrition is able to alter SC of the resultant offspring. A recent report in sheep shows that when ewes are underfed (60% of NRC requirements) during gestation, the offspring from restricted ewes had altered expression of myogenic regulatory factors at birth and at 3 mo of age (Raja et al., 2016). In addition, at 3 mo of age the offspring from restricted ewes produced SC cultures that had a decreased fusion index (Raja et al., 2016). When gilts are supplemented with 25-hydroxycholecalciferol to improve vitamin D status before breeding and during early gestation, fetuses from supplemented gilts had an increased number of muscle fibers, more myoblasts that expressed Pax7 and an increased proliferative phase of their myoblasts, indicating that maternal vitamin D nutrition is important to the development of fetal skeletal muscle (Hines et al., 2013). In another study, supplementation of gilts with vitamin D from breeding through weaning resulted in offspring with improved pre- and postnatal skeletal muscle development as characterized by increased muscle fiber numbers as well as increased cross-sectional areas of the muscle at both birth and weaning in the piglets (Zhou et al., 2016). The authors postulated that this improved growth was modulated through increased IGF-2, IGF-2 receptor, MyoD,

and myogenin mRNA expression in the skeletal muscle of piglets from gilts that received supplemental vitamin D throughout gestation (Zhou et al., 2016). These results demonstrate that maternal plane of nutrition is capable of altering SC activity of the offspring both pre- and postnatally.

IMPACT OF NUTRITION ON SATELLITE CELLS POSTNATALLY

Postnatal increases in muscle size are due to hypertrophy rather than hyperplasia as muscle fiber number does not significantly increase after birth in livestock species. Although muscle fiber number does not increase postnatally, muscle DNA content continues to increase throughout growth due to SC proliferation, differentiation, and fusion with existing myofibers. As such, SC play an integral role in postnatal growth of skeletal muscle. Recent research in rats suggests that hypertrophy of skeletal muscle can occur independently of SC, but it is unknown whether this occurs in livestock species (Murach et al., 2018). As stated previously in this review, plane of nutrition during growth has long been demonstrated to impact growth of skeletal muscle, especially in growing animals. However, little research has been completed to determine the role that nutrition has on proliferation and differentiation of SC in postnatal skeletal muscle of livestock animals.

Older research conducted in chickens showed that removing feed from chicks for 48 h between 7 and 9 d post hatch, and then refeeding them normally until 27 d of age resulted in a decrease in total muscle weight, as well as decrease in muscle nuclei number (Moss, 1968). Another study in poultry showed that altering the methionine:cysteine ratio in the diet affected SC and subsequent muscle growth in the pectoralis major (Powell et al., 2013). A recent study showed that providing either a low or high plane of nutrition (1.9 vs. 3.8 Mcal of gross energy/d) to dairy bull calves altered SC activity and skeletal muscle growth (MacGhee et al., 2017). When calves are provided with a high plane of nutrition, the cross-sectional area of their muscle fibers was significantly increased after 8 wk when compared with those animals receiving a low plane of nutrition (MacGhee et al., 2017). Additionally, providing a high plane of nutrition results in SC with a significantly increased mitotic index after 2 wk, but a significantly decreased mitotic index after 4 wk when compared with calves receiving a low plane of nutrition (MacGhee et al., 2017). This research demonstrates the first report in cattle of how nutrition specifically impacts proliferation and differentiation of SC in postnatal life. There are also several reports of how nutrition impacts proliferation and differentiation of SC in humans. Short-term calorie restriction has been shown to enhance skeletal muscle stem cell function in humans (Cerletti et al., 2012). Creatine supplementation increases SC and myonuclei number in human skeletal muscle, demonstrating that in humans, dietary protein concentrations impacts overall SC number in the skeletal muscle (Olsen et al., 2006). Protein supplementation has also been shown to increase myonuclear accretion in type II muscle fibers of humans (Farup et al., 2014). A recent study in rats showed that when pups suckled mothers that were fed either 17% or 8% protein, the pups that suckled from the protein restricted mothers had decreased weight gain and a decreased number of myogenic cells and myotube expansion when muscle cells were cultured, but there was no difference in expression of myogenic marker proteins (de Melo et al., 2011). However, more research needs to be conducted in this area to determine how nutrition might impact SC during postnatal skeletal muscle growth of livestock species.

Although not much research has been conducted in vivo relating nutrition to proliferation and differentiation of SC, a number of in vitro studies have analyzed the effects of how different nutrients may impact SC and myoblast proliferation in culture. A classic example of how nutrition impacts growth of muscle cells in culture can be observed through the practice of reducing serum concentrations in culture media to induce differentiation. This indicates that in vitro a lower plane of nutrition promotes differentiation, whereas a higher plane of nutrition promotes proliferation and growth of existing myofibers. When myoblasts are provided with low glucose medium, when compared with high glucose medium, differentiation is increased (Dodson et al., 1990). However, a conflicting report showed no difference in differentiation of primary sheep myoblasts when low and high concentrations of glucose were provided (Brameld et al., 1998). More recently, it was found that high glucose induces differentiation and adipogenesis in porcine satellite cells through the mTOR pathway (Yue et al., 2010). These results demonstrate that available glucose has an impact on proliferation and differentiation of cultured SC.

Another nutrient that has been shown to alter proliferation and differentiation of SC are fatty acids. In an early report, linoleic acid stimulated differentiation of rat satellite cells (Allen et al.,

1985). However, more recent research demonstrates that provision of fatty acids to SC promotes adipogenic gene expression (Choi et al., 2015). A lack of available zinc has also been shown to inhibit C2C12 myoblast differentiation and decrease expression of myogenic regulatory factors (Petrie et al., 1996). Zinc also promotes proliferation and activation of C2C12 cells through the PI3K/Akt and ERK signaling cascades (Ohashi et al., 2015). A lack of calcium results in inhibition of muscle cell fusion (Merlie and Gros, 1976; Morris et al., 1976). Provision of calcium has also been found to increase satellite cell activation in rats (Hara et al., 2012). Several different nutrients have been shown to impact SC proliferation, activation, and subsequent fusion in in vitro systems, but more research is needed to determine how these different nutrients impact SC using both in vivo and in vitro livestock models.

SUMMARY AND CONCLUSIONS

Satellite cells are crucial to skeletal muscle growth during both prenatal and postnatal periods of life. Prenatally, SC are important as they are responsible for initial formation of the myofibers. In mammals, the number of myofibers an animal is born with does not change following birth, demonstrating that prenatal development of myofibers is integral in determining postnatal growth potential. During postnatal growth, SC are required for myonuclear accretion, a necessity for skeletal muscle hypertrophy. Nutrient status of growing animals impacts growth of skeletal muscle, which is especially important in livestock animals as it eventually becomes the marketable product, meat. Recently, many studies analyzing the effects of maternal nutrition on growth of skeletal muscle in livestock have been completed. From these studies, it is apparent that nutrient status of the dam, especially during the second and third trimesters, affects skeletal muscle development of the offspring in utero which can impact lifelong production of those offspring. More research is needed to determine exactly how and when maternal plane of nutrition should be altered during gestation in order to produce offspring with the highest growth potential. In postnatal growth, the impacts of nutrition on SC specifically are currently understudied in livestock species. Nutrient status of an animal is known to impact concentrations of circulating growth factors, which in turn are known to impacts skeletal muscle growth and SC. The exact mechanism through which this occurs has not been studied. Several early studies in chickens show that nutrient status affects SC and muscle growth, indicating that nutrition plays a large role in this process. A more recent study in dairy calves shows that plane of nutrition impacts mitotic index of SC. Furthermore, several studies have been completed using cultured SC models that show different nutrients are able to affect SC proliferation and differentiation. However, many of these studies have not been completed in cells from livestock animals. Consequently, additional in vivo and in vitro studies are needed to understand how nutrition alters proliferation and differentiation of SC in livestock animals. The information gained from completion of these studies will help in developing diets for livestock animals that optimize growth of skeletal muscle and, ultimately, production of more meat.

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LITERATURE CITED

Allen, R. E., and L. K. Boxhorn. 1987. Inhibition of skeletal muscle satellite cell differentiation by transforming growth factor-beta. J. Cell. Physiol. 133:567–572. doi:10.1002/jcp.1041330319

Allen, R. E., and L. K. Boxhorn. 1989. Regulation of skeletal muscle satellite cell proliferation and differentiation by transforming growth factor-beta, insulin-like growth factor I, and fibroblast growth factor. J. Cell. Physiol. 138:311–315. doi:10.1002/jcp.1041380213

Allen, R. E., L. S. Luiten, and M. V. Dodson. 1985. Effect of insulin and linoleic acid on satellite cell differentiation. J. Anim. Sci. 60:1571–1579. doi:10.2527/JAS1985.6061571x

Allen, R. E., R. A. Merkel, and R. B. Young. 1979. Cellular aspects of muscle growth: myogenic cell proliferation. J. Anim. Sci. 49:115–127. doi: 10.2527/jas1979.491115x

Amaral, P. M., L. D. S. Mariz, D. Zanetti, L. F. Pradoes, M. I. Marcondes, S. A. Santos, E. Detmann, A. P. Faciola, and S. C. Valadares Filho. 2018. Effect of dietary protein content on performance, feed efficiency and carcass traits of feedlot Nellore and Angus× Nellore cross cattle at different growth stages. J. Agric. Sci. 156:110–117. doi:10.1017/S0021859617000958

Bonnet, M., I. Cassar-Malek, Y. Chilliard, and B. Picard. 2010. Ontogenesis of muscle and adipose tissues and their interactions in ruminants and other species. Animal. 4:1093–1109. doi:10.1017/S1751731110000601

Boyd, R. D., and D. E. Bauman. 1989. Mechanisms of action for somatotropin in growth. In: D. R. Campion, G. J. Hausman, and R. J. Martin, editor, Animal growth regulation. Springer, Boston, MA; p. 257–293. doi:10.1007/978-1-4684-8872-2_12

Brameld, J. M. 1997. Molecular mechanisms involved in the nutritional and hormonal regulation of growth in pigs. Proc, Nutr. Soc. 56:607–619. doi:10.179.PSN19970061

Brameld, J. M., P. J. Buttery, J. M. Dawson, and J. M. Harper. 1998. Nutritional and hormonal control of skeletal-muscle cell growth and differentiation. Proc. Nutr. Soc. 57:207–217. doi:10.1079.PSN19980033

Breier, B. H., J. J. Bass, J. H. Butler, and P. D. Gluckman. 1986. The somatotrophic axis in young steers: influence of nutritional status on pulsatile release of growth hormone and circulating concentrations of insulin-like growth factor 1. J. Endocrinol. 111:209–215. doi:10.1677/joe.0.1110209

Breier, B. H., P. D. Gluckman, and J. J. Bass. 1988. Influence of nutritional status and oestradiol-17 beta on plasma growth hormone, insulin-like growth factors-I and -II and the response to exogenous growth hormone in young steers. J. Endocrinol. 118:243–250. doi:10.1677/joe.0.1160169

Brumby, P. 1959. The influence of growth hormone on growth in young cattle. N. Z. J. Agric. Res. 2:683–689. doi:10.1080/00288233.1959.10422827

Cerletti, M., Y. C. Jang, L. W. Finley, M. C. Haigis, and A. J. Wagers. 2012. Short-term calorie restriction enhances skeletal muscle stem cell function. Cell Stem Cell. 10:515–519. doi:10.1016/j.stem.2012.04.002

Choi, S. H., S. K. Park, B. J. Johnson, K. Y. Chung, C. W. Choi, K. H. Kim, W. Y. Kim, and B. Smith. 2015. Ampkα, C/ebpβ, CPT1β, GPR43, pparγ, and SCD gene expression in single- and co-cultured bovine satellite cells and intramuscular preadipocytes treated with palmitic, stearic, oleic, and linoleic acid. Asian-Australas. J. Anim. Sci. 28:411–419. doi:10.5713/ajas.14.0598

Clemmons, D. R., and L. E. Underwood. 1991. Nutritional regulation of IGF-I and IGF binding proteins. Annu. Rev. Nutr. 11:393–412. doi:10.1146/annurev.nu.11.070191.002141

Cooke, R., G. Lodge, and D. Lewis. 1972. Influence of energy and protein concentration in the diet on the performance of growing pigs 1. Response to protein intake on a high-energy diet. Anim. Sci.. 14:35–46. doi:10.1017/S0003356100000258

Davis, T. A., M. L. Fiorotto, D. G. Burrin, P. J. Reeds, H. V. Nguyen, P. R. Beckett, R. C. Vann, and P. M. O'Connor. 2002. Stimulation of protein synthesis by both insulin and amino acids is unique to skeletal muscle in neonatal pigs. Am. J. Physiol. Endocrinol. Metab. 282:E880–E890. doi:10.1152/ajpendo.00517.2001

de Melo, J. F., N. Aloulou, J. L. Duval, P. Vigneron, L. Bourgoin, C. G. Leandro, C. M. de Castro, and M. D. Nagel. 2011. Effect of a neonatal low-protein diet on the morphology of myotubes in culture and the expression of key proteins that regulate myogenesis in young and adult rats. Eur. J. Nutr. 50:243–250. doi:10.1007/s00394-010-0132-9

Dodson, M. V., B. A. Mathison, and B. D. Mathison. 1990. Effects of medium and substratum on ovine satellite cell attachment, proliferation and differentiation in vitro. Cell Differ. Dev. 29:59–66. doi:10.1019/0922-3371(90)90024-Q

Doumit, M. E., D. R. Cook, and R. A. Merkel. 1993. Fibroblast growth factor, epidermal growth factor, insulin-like growth factors, and platelet-derived growth factor-BB stimulate proliferation of clonally derived porcine myogenic satellite cells. J. Cell. Physiol. 157:326–332. doi:10.1002/jcp.1041570216

Du, M., Y. Huang, A. K. Das, Q. Yang, M. S. Duarte, M. V. Dodson, and M. J. Zhu. 2013. Meat science and muscle biology symposium: manipulating mesenchymal progenitor cell differentiation to optimize performance and carcass value of beef cattle. J. Anim. Sci. 91:1419–1427. doi:10.2527/jas.2012-5670

Du, M., J. Tong, J. Zhao, K. R. Underwood, M. Zhu, S. P. Ford, and P. W. Nathanielsz. 2010. Fetal programming of skeletal muscle development in ruminant animals. J. Anim. Sci. 88(13 Suppl.):E51–E60. doi:10.2527/jas.2009-2311

Du, M., B. Wang, X. Fu, Q. Yang, and M. J. Zhu. 2015. Fetal programming in meat production. Meat Sci. 109:40–47. doi:10.1016/j.meatsci.2015.04.010

Du, M., J. X. Zhao, X. Yan, Y. Huang, L. V. Nicodemus, W. Yue, R. J. McCormick, and M. J. Zhu. 2011. Fetal muscle development, mesenchymal multipotent cell differentiation, and associated signaling pathways. J. Anim. Sci. 89:583–590. doi:10.2527/jas.2010-3386

Ellenberger, M. A., D. E. Johnson, G. E. Carstens, K. L. Hossner, M. D. Holland, T. M. Nett, and C. F. Nockels. 1989. Endocrine and metabolic changes during altered growth rates in beef cattle. J. Anim. Sci. 67:1446–1454. doi:10.2527/jas1989.6761446x

Elsasser, T. H., T. S. Rumsey, and A. C. Hammond. 1989. Influence of diet on basal and growth hormone-stimulated plasma concentrations of IGF-I in beef cattle. J. Anim. Sci. 67:128–141. doi:10.2527/jas1989.671128x

Fahey, A. J., J. M. Brameld, T. Parr, and P. J. Buttery. 2005. The effect of maternal undernutrition before muscle differentiation on the muscle fiber development of the newborn lamb. J. Anim. Sci. 83:2564–2571. doi:10.2527/2005.83112564x

Farup, J., S. K. Rahbek, S. Riis, M. H. Vendelbo, F. d. Paoli, and K. Vissing. 2014. Influence of exercise contraction mode and protein supplementation on human skeletal muscle satellite cell content and muscle fiber growth. J. Appl. Physiol. (1985). 117:898–909. doi:10.1152/japplphysiol.00261.2014

Gautsch, T. A., J. C. Anthony, S. R. Kimball, G. L. Paul, D. K. Layman, and L. S. Jefferson. 1998. Availability of eif4e regulates skeletal muscle protein synthesis during recovery from exercise. Am. J. Physiol. 274(2 Pt 1):C406–C414. doi:10.1152/ajpcell.1998.274.2.C406

Ge, X., J. Yu, and H. Jiang. 2012. Growth hormone stimulates protein synthesis in bovine skeletal muscle cells without altering insulin-like growth factor-I mRNA expression. J. Anim. Sci. 90:1126–1133. doi:10.2527/jas.2011-4358

Gill, D. R., F. N. Owens, M. C. King, and H. G. Dolezal. 1993. Body composition of grazing or feedlot steers differing in age and background. In Anim. Sci. Res. Rep., Oklahoma State Univ., Stillwater. p. 185–190.

Gionbelli, T. R. S., C. M. Veloso, P. P. Rotta, S. C. Valadares Filho, B. C Carvalho, M. I. Marcondes, C. S Cunha, M. A. S. Novaes, L. D. Prezotto, M. S. Duarte, et al. 2018. Foetal development of skeletal muscle in bovines as a function of maternal nutrition, foetal sex and gestational age. J. Anim. Physiol. Anim. Nutr. (Berl.). 102:545–556. doi:10.1111/jpn.12786

Goll, D. E., G. Neti, S. W. Mares, and V. F. Thompson. 2008. Myofibrillar protein turnover: the proteasome and the calpains. J. Anim. Sci. 86(14 Suppl.):E19–E35. doi:10.2527/jas.2007-0395

Gonzalez, J. M., L. E. Camacho, S. M. Ebarb, K. C. Swanson, K. A. Vonnahme, A. M. Stelzleni, and S. E. Johnson. 2013. Realimentation of nutrient restricted pregnant beef cows supports compensatory fetal muscle growth. J. Anim. Sci. 91:4797–4806. doi:10.2527/jas.2013-6704

Greene, E. A., and R. E. Allen. 1991. Growth factor regulation of bovine satellite cell growth in vitro. J. Anim. Sci. 69:146–152. doi:10.2527/1991.691146x

Greenwood, P., L. Cafe, H. Hearnshaw, and D. Hennessy. 2005. Consequences of nutrition and growth retardation early

in life for growth and composition of cattle and eating quality of beef. Recent Adv. Anim. Nutr. Aust. 15:183–195. doi:1aa5b403-78c5-417b-8452-df30f312bd27

Greenwood, P., and A. Thompson. 2007. Consequences of maternal nutrition during pregnancy and of foetal growth for productivity of sheep. Recent Adv. Anim. Nutr. Aust. 16:185–196. doi:10.4135/9781412984614.n12

Gros, J., M. Manceau, V. Thomé, and C. Marcelle. 2005. A common somitic origin for embryonic muscle progenitors and satellite cells. Nature. 435:954–958. doi:10.1038/nature03572

Hales, C. N., and D. J. Barker. 1992. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia. 35:595–601. doi:10.1007/BF00400248

Hales, K. E., S. D. Shackelford, J. E. Wells, D. A. King, N. A. Pyatt, H. C. Freetly, and T. L. Wheeler. 2016. Effects of dietary protein concentration and ractopamine hydrochloride on performance and carcass characteristics of finishing beef steers. J. Anim. Sci. 94:2097–2102. doi:10.2527/jas.2015-0225

Han, B., J. Tong, M. J. Zhu, C. Ma, and M. Du. 2008. Insulin-like growth factor-1 (IGF-1) and leucine activate pig myogenic satellite cells through mammalian target of rapamycin (MTOR) pathway. Mol. Reprod. Dev. 75:810–817. doi:10.1002/mrd.20832

Han, I. K., P. Bosi, Y. Hyun, J. D. Kim, K. S. Sohn, and S. W. Kim. 2000. Recent advances in sow nutrition to improve reproductive performance. Asian-Aust. J. Anim. Sci. 13:335–355.

Hannon, K., A. Gronowski, and A. Trenkle. 1991. Relationship of liver and skeletal muscle IGF-1 mRNA to plasma GH profile, production of IGF-1 by liver, plasma IGF-1 concentrations, and growth rates of cattle. Proc. Soc. Exp. Biol. Med. 196:155–163. doi:10.3181/00379727-196-43172

Hara, M., K. Tabata, T. Suzuki, M. K. Do, W. Mizunoya, M. Nakamura, S. Nishimura, S. Tabata, Y. Ikeuchi, K. Sunagawa, et al. 2012. Calcium influx through a possible coupling of cation channels impacts skeletal muscle satellite cell activation in response to mechanical stretch. Am. J. Physiol. Cell Physiol. 302:C1741–C1750. doi:10.1152/ajpcell.00068.2012

Hayden, J. M., J. E. Williams, and R. J. Collier. 1993. Plasma growth hormone, insulin-like growth factor, insulin, and thyroid hormone association with body protein and fat accretion in steers undergoing compensatory gain after dietary energy restriction. J. Anim. Sci. 71:3327–3338. doi:10.2527/1993.71123327x

Hines, E. A., J. D. Coffey, C. W. Starkey, T. K. Chung, and J. D. Starkey. 2013. Improvement of maternal vitamin D status with 25-hydroxycholecalciferol positively impacts porcine fetal skeletal muscle development and myoblast activity. J. Anim. Sci. 91:4116–4122. doi:10.2527/jas.2013-6565

Houseknecht, K. L., D. L. Boggs, D. R. Campion, J. L. Sartin, T. E. Kiser, G. B. Rampacek, and H. E. Amos. 1988. Effect of dietary energy source and level on serum growth hormone, insulin-like growth factor 1, growth and body composition in beef heifers. J. Anim. Sci. 66:2916–2923. doi:10.2527/jas1988.66112916x

Hulmi, J. J., C. M. Lockwood, and J. R. Stout. 2010. Effect of protein/essential amino acids and resistance training on skeletal muscle hypertrophy: a case for whey protein. Nutr. Metab. (Lond.) 7:51. doi:10.1186/1743-7075-7-51

Jennings, T. D., M. G. Gonda, K. R. Underwood, A. E. Wertz-Lutz, and A. D. Blair. 2016. The influence of maternal nutrition on expression of genes responsible for adipogenesis and myogenesis in the bovine fetus. Animal. 10:1697–1705. doi:10.1017/S1751731116000665

Kamanga-Sollo, E., K. J. Thornton, M. E. White, and W. R. Dayton. 2014. Role of G protein-coupled estrogen receptor-1, matrix metalloproteinases 2 and 9, and heparin binding epidermal growth factor-like growth factor in estradiol-17β-stimulated bovine satellite cell proliferation. Domest. Anim. Endocrinol. 49:20–26. doi:10.1016/j.domaniend.2014.04.004

Kamanga-Sollo, E., M. E. White, M. R. Hathaway, K. Y. Chung, B. J. Johnson, and W. R. Dayton. 2008. Roles of IGF-I and the estrogen, androgen and IGF-I receptors in estradiol-17beta- and trenbolone acetate-stimulated proliferation of cultured bovine satellite cells. Domest. Anim. Endocrinol. 35:88–97. doi:10.1016/j.domaniend.2008.02.003

Kamanga-Sollo, E., M. S. Pampusch, G. Xi, M. E. White, M. R. Hathway, and W. R. Dayton. 2004. IGF-I mRNA levels in bovine satellite cell cultures: effects of fusion and anabolic steroid treatment. J. Cell. Physiol. 201:181–189. doi:10.1002/jcp.20000

Kumar, V., P. Atherton, K. Smith, and M. J. Rennie. 2009. Human muscle protein synthesis and breakdown during and after exercise. J. Appl. Physiol. (1985). 106:2026–2039. doi:10.1152/japplphysiol.91481.2008

Larson, D. M., J. L. Martin, D. C. Adams, and R. N. Funston. 2009. Winter grazing system and supplementation during late gestation influence performance of beef cows and steer progeny. J. Anim. Sci. 87:1147–1155. doi:10.2527/jas.2008-1323

Laurent, G. J., R. J. McAnulty, and J. Gibson. 1985. Changes in collagen synthesis and degradation during skeletal muscle growth. Am. J. Physiol. 249(3 Pt 1):C352–C355. doi:10.1152/ajpcell.1985.249.3.C352

Lepper, C., S. J. Conway, and C. M. Fan. 2009. Adult satellite cells and embryonic muscle progenitors have distinct genetic requirements. Nature. 460:627–631. doi:10.1038/nature08209

Long, N. M., C. B. Tousley, K. R. Underwood, S. I. Paisley, W. J. Means, B. W. Hess, M. Du, and S. P. Ford. 2012. Effects of early- to mid-gestational undernutrition with or without protein supplementation on offspring growth, carcass characteristics, and adipocyte size in beef cattle. J. Anim. Sci. 90:197–206. doi:10.2527/jas.2011-4237

Lucy, M. C. 2008. Functional differences in the growth hormone and insulin-like growth factor axis in cattle and pigs: implications for post-partum nutrition and reproduction. Reprod. Domest. Anim. 43(Suppl. 2):31–39. doi:10.1111/j.1439-0531.2008.01140.x

MacGhee, M. E., J. S. Bradley, S. R. McCoski, A. M. Reeg, A. D. Ealy, and S. E. Johnson. 2017. Plane of nutrition affects growth rate, organ size and skeletal muscle satellite cell activity in newborn calves. J. Anim. Physiol. Anim. Nutr. (Berl.). 101:475–483. doi:10.1111/jpn.12568

McGuire, M. A., J. L. Vicini, D. E. Bauman, and J. J. Veenhuizen. 1992. Insulin-like growth factors and binding proteins in ruminants and their nutritional regulation. J. Anim. Sci. 70:2901–2910. doi:10.2527.1992.7092901x

McMillen, I. C., and J. S. Robinson. 2005. Developmental origins of the metabolic syndrome: prediction, plasticity, and programming. Physiol. Rev. 85:571–633. doi:10.1152/physrev.00053.2003

Merlie, J. P., and F. Gros. 1976. In vitro myogenesis. Expression of muscle specific function in the absence of cell fusion. Exp. Cell Res. 97:406–412. doi:10.1016/0014-4827(76)90632-7

Micke, G. C., T. M. Sullivan, K. L. Gatford, J. A. Owens, and V. E. Perry. 2010. Nutrient intake in the bovine during early and mid-gestation causes sex-specific changes in progeny plasma IGF-I, liveweight, height and carcass traits. Anim. Reprod. Sci. 121:208–217. doi:10.1016/j.anireprosci.2010.05.017

Micke, G. C., T. M. Sullivan, I. C. McMillen, S. Gentili, and V. E. Perry. 2011. Protein intake during gestation affects postnatal bovine skeletal muscle growth and relative expression of IGF1, IGF1R, IGF2 and IGF2R. Mol. Cell. Endocrinol. 332:234–241. doi:10.1016/j.mce.2010.10.018

Mitchell, A. D. 2007. Impact of research with cattle, pigs, and sheep on nutritional concepts: body composition and growth. J. Nutr. 137:711–714. doi:10.1093/jn/137.3.711

Mohrhauser, D. A., A. R. Taylor, M. G. Gonda, K. R. Underwood, R. H. Pritchard, A. E. Wertz-Lutz, and A. D. Blair. 2015. The influence of maternal energy status during mid-gestation on beef offspring tenderness, muscle characteristics, and gene expression. Meat Sci. 110:201–211. doi:10.1016/j.meatsci.2015.07.017

Moisá, S. J., D. W. Shike, L. Shoup, S. L. Rodriguez-Zas, and J. J. Loor. 2015. Maternal plane of nutrition during late gestation and weaning age alter Angus × Simmental offspring longissimus muscle transcriptome and intramuscular fat. PLoS One. 10:e0131478. doi:10.1371/journal.pone.0131478

Moriel, P., L. F. Artioli, M. H. Poore, A. W. Confer, R. S. Marques, and R. F. Cooke. 2015. Increasing the metabolizable protein supply enhanced growth performance and led to variable results on innate and humoral immune response of preconditioning beef steers. J. Anim. Sci. 93:4473–4485. doi:10.2527/jas.2015-9238

Morris, G. E., M. Piper, and R. Cole. 1976. Differential effects of calcium ion concentration on cell fusion, cell division and creatine kinase activity in mucle cell cultures. Exp. Cell Res. 99:106–114. doi:10.1016/0014-4827(76)90685-6

Moss, F. P. 1968. The relationship between the dimensions of the fibres and the number of nuclei during normal growth of skeletal muscle in the domestic fowl. Am. J. Anat. 122:555–563. doi:10.1002/aja.1001220308

Mozdziak, P. E., E. Schultz, and R. G. Cassens. 1997. Myonuclear accretion is a major determinant of avian skeletal muscle growth. Am. J. Physiol. 272(2 Pt 1):C565–C571. doi:10.1152/ajpcell.1997.272.2.C565

Murach, K. A., C. S. Fry, T. J. Kirby, J. R. Jackson, J. D. Lee, S. H. White, E. E. Dupont-Versteegden, J. J. McCarthy, and C. A. Peterson. 2018. Starring or supporting role? Satellite cells and skeletal muscle fiber size regulation. Physiology (Bethesda). 33:26–38. doi:10.1152/physiol.00019.2017

Neel, J. V. 1962. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? Am. J. Hum. Genet. 14:353–362. PMCID: PMC1932342

Neel, J. V. 1999. The "thrifty genotype" in 1998. Nutr. Rev. 57(5 Pt 2):S2–S9. doi:10.1111/j.1753-4887.1999.tb01783.x

Norton, L. E., and D. K. Layman. 2006. Leucine regulates translation initiation of protein synthesis in skeletal muscle after exercise. J. Nutr. 136:533S–537S. doi:10.1093/jn/136.2.533S

Ohashi, K., Y. Nagata, E. Wada, P. S. Zammit, M. Shiozuka, and R. Matsuda. 2015. Zinc promotes proliferation and activation of myogenic cells via the PI3K/akt and ERK signaling cascade. Exp. Cell Res. 333:228–237. doi:10.1016/j.yexcr.2015.03.003

Olsen, S., P. Aagaard, F. Kadi, G. Tufekovic, J. Verney, J. L. Olesen, C. Suetta, and M. Kjaer. 2006. Creatine supplementation augments the increase in satellite cell and myonuclei number in human skeletal muscle induced by strength training. J. Physiol. 573(Pt 2):525–534. doi:10.1113/jphysiol.2006.107359

Owens, F. N., D. R. Gill, D. S. Secrist, and S. W. Coleman. 1995. Review of some aspects of growth and development of feedlot cattle. J. Anim. Sci. 73:3152–3172. doi:10.2527/1995.73103152x

Petrie, L., J. N. Buskin, and J. K. Chesters. 1996. Zinc and the initiation of myoblast differentiation. J. Nutr. Biochem. 7:670–676. doi:10.1016/S0955-2863(96)00129-5

Powell, D. J., D. C. McFarland, A. J. Cowieson, W. I. Muir, and S. G. Velleman. 2013. The effect of nutritional status on myogenic satellite cell proliferation and differentiation. Poult. Sci. 92:2163–2173. doi:10.3382/ps.2013-03107

Quigley, S. P., D. O. Kleemann, S. K. Walker, P. A. Speck, S. R. Rudiger, G. S. Nattrass, M. J. DeBlasio, and J. A. Owens. 2008. Effect of variable long-term maternal feed allowance on the development of the ovine placenta and fetus. Placenta. 29:539–548. doi:10.1016/j.placenta.2008.02.014

Raja, J. S., M. L. Hoffman, K. E. Govoni, S. A. Zinn, and S. A. Reed. 2016. Restricted maternal nutrition alters myogenic regulatory factor expression in satellite cells of ovine offspring. Animal. 10:1200–1203. doi:10.1017/S1751731116000070

Reiter, B. C., E. Kamanga-Sollo, M. S. Pampusch, M. E. White, and W. R. Dayton. 2014. Epidermal growth factor receptor is required for estradiol-stimulated bovine satellite cell proliferation. Domest. Anim. Endocrinol. 48:48–55. doi:10.1016/j.domaniend.2014.01.001

Ronge, H., and J. Blum. 1989. Insulinlike growthfactor I responses to growth hormone in dry and lactating dairy cows. J. Anim. Physiol. Anim. Nutr. 62: 280–288. doi:10.1111/j1439-0396.tb00845.x

Samuelsson, A. M., P. A. Matthews, M. Argenton, M. R. Christie, J. M. McConnell, E. H. Jansen, A. H. Piersma, S. E. Ozanne, D. F. Twinn, C. Remacle, et al. 2008. Diet-induced obesity in female mice leads to offspring hyperphagia, adiposity, hypertension, and insulin resistance: a novel murine model of developmental programming. Hypertension. 51:383–392. doi:10.1161/HYPERTENSIONAHA.107.101477

Thissen, J. P., J. M. Ketelslegers, and L. E. Underwood. 1994. Nutritional regulation of the insulin-like growth factors. Endocr. Rev. 15:80–101. doi:10.1210/edrv-15-1-80

Thornton, K. J., C. M. Welch, L. C. Davis, M. E. Doumit, R. A. Hill, and G. K. Murdoch. 2012. Bovine sire selection based on maintenance energy affects muscle fiber type and meat color of F1 progeny. J. Anim. Sci. 90:1617–1627. doi:10.2527/jas.2011-4678

Tong, J. F., X. Yan, M. J. Zhu, S. P. Ford, P. W. Nathanielsz, and M. Du. 2009. Maternal obesity downregulates myogenesis and beta-catenin signaling in fetal skeletal muscle. Am. J.

Physiol. Endocrinol. Metab. 296:E917–E924. doi:10.1152/ajpendo.90924.2008

Underwood, K. R., J. F. Tong, P. L. Price, A. J. Roberts, E. E. Grings, B. W. Hess, W. J. Means, and M. Du. 2010. Nutrition during mid to late gestation affects growth, adipose tissue deposition, and tenderness in crossbred beef steers. Meat Sci. 86:588–593. doi:10.1016/j.meatsci.2010.04.008

Wallace, J. M., J. S. Milne, and R. P. Aitken. 2004. Maternal growth hormone treatment from day 35 to 80 of gestation alters nutrient partitioning in favor of uteroplacental growth in the overnourished adolescent sheep. Biol. Reprod. 70:1277–1285. doi:10.1095/biolreprod.103.023853

Wang, X., X. Lan, A. E. Radunz, and H. Khatib. 2015. Maternal nutrition during pregnancy is associated with differential expression of imprinted genes and DNA methyltranfereases in muscle of beef cattle offspring. J. Anim. Sci. 93:35–40. doi:10.2527/jas.2014-8148

Wu, G. 2009. Amino acids: metabolism, functions, and nutrition. Amino Acids. 37:1–17. doi:10.1007/s00726-009-0269-0

Wu, G., D. A. Knabe, and S. W. Kim. 2004. Arginine nutrition in neonatal pigs. J. Nutr. 134(10 Suppl.):2783S–2790S; discussion 2796S. doi:10.1093/jn/134.10.2783S

Wu, G., Z. Wu, Z. Dai, Y. Yang, W. Wang, C. Liu, B. Wang, J. Wang, and Y. Yin. 2013. Dietary requirements of "nutritionally non-essential amino acids" by animals and humans. Amino Acids. 44:1107–1113. doi:10.1007/s00726-012-1444-2

Xi, P., Z. Jiang, C. Zheng, Y. Lin, and G. Wu. 2011. Regulation of protein metabolism by glutamine: implications for nutrition and health. Front. Biosci. (Landmark Ed.). 16:578–597. doi:10.2741/3707

Yan, X., Y. Huang, J. X. Zhao, N. M. Long, A. B. Uthlaut, M. J. Zhu, S. P. Ford, P. W. Nathanielsz, and M. Du. 2011. Maternal obesity-impaired insulin signaling in sheep and induced lipid accumulation and fibrosis in skeletal muscle of offspring. Biol. Reprod. 85:172–178. doi:10.1095/biolreprod.110.089649

Yan, X., M. J. Zhu, W. Xu, J. F. Tong, S. P. Ford, P. W. Nathanielsz, and M. Du. 2010. Up-regulation of toll-like receptor 4/nuclear factor-kappab signaling is associated with enhanced adipogenesis and insulin resistance in fetal skeletal muscle of obese sheep at late gestation. Endocrinology. 151:380–387. doi:10.1210/en.2009-0849

Yao, K., Y. L. Yin, W. Chu, Z. Liu, D. Deng, T. Li, R. Huang, J. Zhang, B. Tan, W. Wang, et al. 2008. Dietary arginine supplementation increases mtor signaling activity in skeletal muscle of neonatal pigs. J. Nutr. 138:867–872. doi:10.1093/jn/138.5.867

Yue, T., J. Yin, F. Li, D. Li, and M. Du. 2010. High glucose induces differentiation and adipogenesis in porcine muscle satellite cells via mtor. BMB Rep. 43:140–145. doi:10.5483/BMBRep.2010.43.2.140

Zhou, H., Y. Chen, G. Lv, Y. Zhuo, Y. Lin, B. Feng, Z. Fang, L. Che, J. Li, S. Xu, et al. 2016. Improving maternal vitamin D status promotes prenatal and postnatal skeletal muscle development of pig offspring. Nutrition, 32:1144–1152. doi:10.1016/j.nut.2016.03.004

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. doi:10.1095/biolreprod.104.034561

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(Pt 1):241–250. doi:10.1113/jphysiol.2006.112110

Zhu, M. J., B. Han, J. Tong, C. Ma, J. M. Kimzey, K. R. Underwood, Y. Xiao, B. W. Hess, S. P. Ford, P. W. Nathanielsz, et al. 2008. AMP-activated protein kinase signalling pathways are down regulated and skeletal muscle development impaired in fetuses of obese, over-nourished sheep. J. Physiol. 586:2651–2664. doi:10.1113/jphysiol.2007.149633