Optimizing livestock production efficiency through maternal nutritional management and fetal developmental programming

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Implications

• All major milestones of animal development are accomplished inside the uterus, which is profoundly affected by maternal nutrition.
• The fetal stage sets the trajectory for long-term growth of farm animals.
• Skeletal muscle and adipose tissue are the most susceptible to maternal nutritional and physiological fluctuations.
• All muscle fibers are formed during the fetal stage and insufficient maternal influence corresponding to major stages of fetal muscle development reduces muscle fiber number and muscle mass in offspring.
• Nutrient deficiency during late gestation and neonatal stages, when adipose tissues are actively developing, reduces overall fat cell formation including intramuscular fat (marbling).
• Enhancing adipogenic differentiation of intramuscular progenitor cells increases intramuscular adipocytes and thus marbling while reducing the population of fibroblasts as well as connective tissue deposition.
• The long duration of pregnancy and lactation stage of beef cattle provides unique advantages for stage-specific nutrient supplementation, achieving precision animal production management to improve animal production efficiency and quality.

Key words: adipose tissue, fetal programming, fetus, meat quality, production efficiency, skeletal muscle
Introduction

Frequent droughts and unfavorable geological locations result in forage deficiency. Because of the seasonal nature of reproduction in cows and ewes, they frequently experience nutrient shortage during pregnancy. Maternal nutrient deficiency negatively affects fetal development, which has long-term negative impacts on offspring growth performance (Funston et al., 2010a; Du et al., 2013; Robinson et al., 2013). On the other hand, nutrient supplementation promotes fetal development, especially muscle development due to its low priority in nutrient partitioning, improving lean:fat ratio and overall production efficiency of offspring (Du et al., 2010).

Meat animals are raised primarily for their lean meat, which is mainly composed from muscle fibers, intramuscular fat (marbling fat), and connective tissues. Muscle fiber development can be roughly separated into prenatal and postnatal stages. For livestock, all muscle fibers are formed before birth, and postnatal muscle growth is due to increase in the diameter and length of existing muscle fibers. Thus, increasing muscle fiber formation during fetal development enhances muscle mass in offspring. Sufficient maternal nutrition during mid-gestation, especially proteins, promotes formation of muscle fibers.

Beside muscle, adipose tissue development is also profoundly affected by maternal nutrition. There are four fat depots in animals: visceral, subcutaneous, intermuscular, and intramuscular fat, of which, only intramuscular fat (marbling fat) is critical for meat palatability. In beef cattle, visceral and subcutaneous fat mainly develop during the mid-gestation to neonatal stage, which is slightly before the development of intramuscular adipocytes (fat cells). Enhancing intramuscular adipocyte formation during the late gestation and neonatal stages provides sites for intramuscular lipid accumulation during fattening, increasing marbling.

Adipose tissue is considered a specialized connective tissue, and adipocytes are imbedded inside a connective tissue network. Fibroblasts are mainly responsible for synthesis of connective tissue, which is a major contributor to background toughness of meat. Recent studies show that adipocytes and fibroblasts are developed from the same pool of progenitor cells. Enhancing intramuscular adipogenesis while inhibiting fibrogenesis increases marbling and reduces connective tissue content, improving tenderness of meat.

Maternal Nutrition and Skeletal Muscle Development

Early embryonic development and primary and secondary muscle fiber formation

During early embryonic development, a fertilized cell first develops into a blastocyst, and the inner cell mass of the blastocyst then separates into hypoblast and epiblast layers (Johnson and McConnell, 2004). Epiblast cells then give rise to three primary germ layers, including ectoderm, mesoderm, and endoderm (Wells and Melton, 2000). The mesoderm layer further develops into somites, which then split into the dorsal and ventral portions to become dermomyotomes and sclerotomes, respectively (Aoyama and Asamoto, 1988). The sclerotome cells give rise to skeleton and ribs while dermomyotome cells further develop into muscle cells and fat cells (Bailey et al., 2001). Cells in myotome further proliferate and migrate to limb buds to form limb muscle (Sassoon et al., 1989), committing to the muscle lineage through the expression of transcription factors including Pax3 (Griffone et al., 2007) and subsequent expression of Myf5 and MyoD (Tajbakhsh et al., 1997). During embryonic development, these myoblasts proliferate and fuse into primary muscle fibers. The number of primary muscle fibers formed is very limited. However, primary fibers function as templates for the formation of secondary muscle fibers during the fetal stage (Du et al., 2010).

During the fetal stage, precursor cells surrounding primary muscle fibers continue to proliferate, which profoundly increases their numbers. These precursor cells then fuse to form secondary muscle fibers, accounting for the majority of skeletal muscle fibers (Beermann et al., 1978). The population of myogenic precursor cells not only determines the number of muscle fibers formed, it also affects the density of satellite cells in postnatal muscle, which is very important for postnatal muscle growth. In short, to enhance lean growth, it is critically important to promote proliferation of myogenic precursor cells during the fetal stage.

Fetal developmental programming of lean growth

Because all fetal nutrients are derived from dams, maternal nutrition has profound impacts on fetal muscle development. Proper maternal nutrition provides nutrients needed for myogenic cell proliferation and thus muscle fiber formation. Because skeletal muscle has a lower priority in nutrient partitioning compared with the brain, heart, and liver, muscle development is particularly vulnerable to the variation of nutrients (Zhu et al., 2006). Furthermore, insufficient maternal nutrition reduces concentration of growth factors such as IGF-1 and IGF-2 in fetal circulation, which decreases myogenic cell proliferation and formation of muscle fibers (Gonzalez et al., 2013).

During bovine fetal development, primary muscle fibers form within the first 2 mo after conception (Russell and Oteruelo, 1981). The secondary myogenesis, which forms the majority of muscle fibers, occurs during 2 and 7 mo of gestation in cattle (Du et al., 2010). Afterward, there is a very limited formation of new muscle fibers, and muscle growth is due to the increase in muscle fiber sizes and lengths (Figure 1). Therefore, reduction in the proliferation of myogenic precursor cells decreases the formation of muscle fiber numbers, which has long-lasting, irreversible negative effects on offspring muscle growth (Stannard and Johnson, 2004; Zambrano et al., 2005; Zhu et al., 2006). Consistently, 50% nutrient deficiency of ewes during Day 28 to Day 78 of gestation (Roughly equivalent to 2 to 5 mo of gestation in cattle) reduces the total formation of secondary muscle fibers (Zhu et al., 2004), which correlates with long-term reduction in muscle fiber number and muscle mass in offspring lambs (Zhu et al., 2006). Intrauterine nutrient deficiency in pigs reduced birth weight, which was correlated with less muscle fibers in offspring (Dwyer et al., 1994). In beef cattle, maternal diets at 60% of NRC nutrient requirement up to 85 or 140 d of gestation reduced fetal IGF-1 expression and myogenic progenitor cell density, resulting in reduced muscle fiber numbers at 254 d of gestation (Gonzalez et al., 2013). These data clearly show that nutrient deficiency between early- to mid-gestation reduces muscle fiber number and muscle mass, negatively affecting growth performance of offspring.

Muscle development after late gestation

After muscle fibers are formed by the end of second trimester, myogenic cells continue to proliferate and fuse with existing muscle fibers, which increase muscle fiber diameters (Figure 1). In addition, muscle fibers increase in length through synthesizing new myofibrillar proteins. During this process, a portion of proliferating myogenic cells become quiescent and develop into reserved myogenic cells in postnatal muscle (myogenic stem cells), termed satellite cells, which are located below a thin layer of connective tissue.
tissue surrounding mature muscle fibers. The proliferation and fusion of satellite cells with existing muscle fibers increase the nuclear density in muscle fibers, which promote myofibrillar protein synthesis and increase muscle fiber sizes (Kuang et al., 2007). However, if nutrients are not optimally available, myogenic cell proliferation is impaired. As a result, maternal nutrient insufficiency during late gestation reduces fetal fiber sizes though the muscle fiber number is not affected (Greenwood et al., 1999; Zhu et al., 2004). In addition, the density of satellite cells may also be reduced, which negatively affects postnatal muscle growth. These notions are supported by experimental data. Due to rapid growth at late gestation, fetuses of ewes with twin pregnancy commonly experience nutrient deficiency. Consistently, when comparing the fetal muscle growth of sheep with single and twin pregnancies, the competition between littermates for nutrients at late gestation impacts fetal skeletal muscle mass, but only reduced muscle fiber size (hypertrophy) not number (hyperplasia; McCoard et al., 2000). Similar impairment in growth performance of offspring from twin pregnancy was also observed in beef cattle (Gregory et al., 1996). In addition, nutrient supplementation during late gestation in a winter grazing system improved calf birth weight and growth performance of offspring steers (Larson et al., 2009), partially through providing additional nutrients to optimize muscle development during late gestation. A low plane of maternal nutrition during the last two thirds of pregnancy reduces neonatal calf weight (Freetly et al., 2000), likely as a result of reduction in muscle fiber size.

**Maternal Nutrition and Adipose and Connective Tissue Development**

**Adipose tissue and marbling fat development**

Intramuscular fat, which is commonly referred to as marbling, contributes to the flavor and juiciness of meat; consequently, it is valuable for the palatability of meat. However, visceral, subcutaneous, and intermuscular

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Figure 1. Timelines for skeletal muscle and adipose tissue development in beef cattle and pigs. The times for muscle fiber hyperplasia and hypertrophy and adipocyte hyperplasia and hypertrophy are approximate.
fat account for the majority of body fats, which have low commercial value. To accumulate these fats, a large amount of nutrients are required, reducing feed efficiency. As a result, meat animals are selected for high lean:fat ratio, which also reduces marbling fat. To solve this problem, animal scientists strive to enhance marbling while reducing fat accumulation in other depots.

The amount of fat mass is determined by the number and size of adipocytes. The number of adipocytes is primarily determined during the fetal and early postnatal development and the total number of adipocytes becomes fixed at adolescence (Spalding et al., 2008). Therefore, the early developmental stage is ideal for altering adipocyte formation (Du et al., 2015); the desirable outcome is to have more intramuscular adipocytes while reducing adipocyte formation in other depots.

The initial events of adipose tissue development, including the formation of a vascular network in future adipose tissue, initiate at a slightly later time than primary myogenesis (Fève, 2005; Gnanalingham et al., 2005; Muhlhauser et al., 2006). The early adipogenic commitment overlaps with the period of secondary myogenesis (initiates around the end of first trimester in ruminant animals). The major formation of adipocytes occurs during late gestation to early weaning stages. Thus, maternal nutritional management during this period affects adipocyte hyperplasia, which alters overall adipose tissue development as well as intramuscular adipocyte density and thus marbling fat (Du et al., 2013).

Adipocytes are first detectable in the visceral fat in cattle and followed by subcutaneous and then intermuscular fat. The formation of intramuscular adipocytes occurs later, primarily after late gestation and around 250 d of age. The chronological difference in adipocyte formation provides an opportunity to enhance marbling adipocyte formation without increasing overall adiposity of beef cattle, which is called the “marbling window” (Corah and McCully, 2007; Du et al., 2013). Providing high-energy supplementation to early weaning calves before 250 d of age will promote intramuscular formation of adipocytes, which then accumulate lipids during the fattening stage, forming abundant marbling fat (Figure 1).

Fibro/adipogenic cell, fibroblast, and connective tissue development

Connective tissue and its cross-linking are responsible for the background toughness of meat. Fibroblasts synthesize collagens and also secrete enzymes catalyzing collagen cross-linking. Thus, reducing fibroblast density in muscle decreases collagen accumulation and thus improves meat tenderness (Du et al., 2015).

Recent studies show that fibroblasts and adipocytes share a common source of progenitor cells. Due to their dual capacity to differentiate into both fibrogenic cells and adipocytes, these progenitor cells are named fibro/adipogenic progenitors (FAPs) (Joe et al., 2010; Uezumi et al., 2010). These progenitor cells in adipose tissue are frequently associated with blood vessels and are platelet-derived growth factor receptor α (PDGFRα) positive (Wang et al., 2017). A large portion of FAPs are derived from the dermomyotome during prenatal development (Du et al., 2015; Yang et al., 2013), which frequently associates with capillary vessels (Wang et al., 2017). Indeed, adipogenesis and neovascularization are temporally and spatially associated processes (Cao, 2007). These FAPs also exist in adipose tissue postnatally, but their density decreases as animals become older (Du et al., 2013; Wang et al., 2017).

Maternal food restriction inhibits the expression of vascular epithelial growth factor in microvascular and aortic endothelial cells during fetal development, which decreases angiogenesis (Khorram et al., 2007) and the formation of PDGFRα+ progenitor cells. Adipose tissue development is closely associated with capillary network development, and impaired neovascularization negatively affects fetal adipose development (Wang et al., 2017). However, because adipose tissue has high plasticity, compensatory growth of adipose tissue often results in fatter offspring born to nutrient-restricted mothers. Such growth is frequently associated with increased feed intake and low muscle mass, which reduces energy consumption, resulting in excessive energy accumulation in adipose tissue through adipocyte hypertrophy (Gonzalez-Bulnes et al., 2012; Zhu et al., 2006).

Adipogenesis and fibrogenesis are usually ongoing simultaneously, together forming the basic structure of adipose tissue, where adipocytes are imbedded in a network of connective tissue. Due to their shared pool of PDGFRα+ progenitor cells, a positive correlation between adipogenesis and fibrogenesis is commonly observed. Indeed, both marbling and collagen accumulation are elevated in genetically high-marbling Wagyu cattle (Duarte et al., 2013). In addition, maternal overnutrition in beef cattle enhances both adipose and collagen deposition in offspring skeletal muscle (Duarte et al., 2014). On the other hand, the shared progenitor cells also suggest a competitive relationship between adipogenesis and fibrogenesis when the total density of progenitor cells is unaltered. Thus, during the stages when PDGFRα+ progenitor cells are abundant, promoting intramuscular adipogenesis through providing a high-energy diet will correspondingly reduce intramuscular fibrogenesis, which will lead to a meat with increased marbling but reduced connective tissue content, thus improving tenderness. Consistently, 40% nutrient restriction of beef cows, which reduced adipogenesis, increased the expression of fibrogenic markers, suggesting enhanced connective tissue formation when nutrients are deficient (Gonzalez et al., 2013).

Maternal Nutrient Supplementation and Beef Cattle Production

Due to the seasonal nature of pastures and cow reproduction, cows on forage-based production systems frequently experience nutrient deficiency during pregnancy. Periods of maternal nutrient deficiency during pregnancy, depending on the duration and timing, program fetal skeletal muscle and adipose tissue development.
Nutrient supplementation during the early- to mid-gestation of bovine pregnancy has the potential to improve muscle development and lean:fat ratio of offspring. We demonstrated that protein supplementation during the period of 60 to 180 d of gestation enhanced lean growth and lean:fat ratio in offspring (Underwood et al., 2008). We also observed that offspring calves born to cows assigned to improved pasture during mid-gestation had better growth performance and meat quality compared with those of cows assigned to native range pasture in Miles City, MT (Underwood et al., 2010). These data strongly support that a higher gestational plane of nutrition, especially proteins, improves lean growth in subsequent calves, which might also be associated with enhanced intramuscular adipogenesis and marbling. However, in a recent study of young heifers, alteration of maternal dietary energy between 85 and 180 d of gestation does not affect the myogenesis and fetal weight right after treatment (Jennings et al., 2016). In this study, the adipose tissue development was enhanced, which increased overall fat deposition in offspring (Mohrhauser et al., 2015). These studies suggest that dietary energy primarily affects adipose rather than muscle development. In addition, because placenta mediates nutrient delivery to fetuses, changes in placental function due to maternal nutrition also affect fetal development (Vonnahme et al., 2006).

Developmental programming can also be extended to the postnatal management of cattle, for example, through enhancing milk production of cows or supplementation of high-energy starters to early weaning calves to enhance marbling fat development. Maternal milk composition is altered by maternal diet, providing another opportunity to alter neonatal nutrition, and thus, offspring development and subsequent meat production. Indeed, maternal high-energy intake during lactation affects milk composition, which has long-term effects on the metabolic health in offspring mice (Vogt et al., 2014). We found that maternal over-nutrition during lactation promotes adipose development in mice (Liang et al., 2016).

Marbling fat accumulation is due to both formation of intramuscular adipocytes (hyperplasia) and accumulation of lipids in existing adipocytes (hypertrophy). In conventional beef production, cattle are supplemented with a high-grain diet during the finishing stage to induce intramuscular adipocyte hypertrophy, thereby enhancing marbling fat deposition. To be effective, however, there must be a sufficient number of intramuscular adipocytes, which allow intramuscular fat accumulation. The effectiveness of nutritional management to increase intramuscular adipocyte density is age dependent because the density of adipogenic progenitor cells decline as the animal age increases. Therefore, fetal and neonatal stages are the most effective stages to alter intramuscular adipocyte formation, followed by the weaning to 250 d of age, the so called marbling window (Corah and McCully, 2007). After 250 d of age, nutritional supplementation becomes far less effective in inducing intramuscular adipocyte hyperplasia due to the reduction of progenitor density (Du et al., 2015). However, fetal and neonatal stages also correspond to the development of visceral and subcutaneous fat, and high maternal nutrition promotes adipogenesis in all major depots, resulting in an overall increase in calf adiposity. On the other hand, during the marbling window stage, mainly intramuscular adipogenesis is active and high-grain supplementation can enhance intramuscular fat specifically, resulting in better formation of intramuscular adipocytes and better marbling fat accumulation (Du et al., 2015).

Consistently, early weaning improved intramuscular fat content in beef cattle (Moisâ et al., 2015). An additional advantage of early weaning is reduction of the nutritional demand to cows due to lactation, which will provide better nutrients for fetal muscle development (Figure 2).

These concepts of nutritional management during early calf development to enhance marbling can be important for grass-fed beef production. Grass-fed beef is produced naturally, and with greater contents of unsaturated fatty acids, especially n-3 fatty acids (Mann et al., 2003; Ponnampalam et al., 2006), which are good for health and have an increasing demand from consumers. However, due to the absence of a fattening stage because of an inability to feed a high-grain diet, grass-fed beef has low marbling (Mandell et al., 1998; Sitz et al., 2005). Increasing intramuscular adipocyte density through nutritional management during the early stage of cattle development is an alternative way to enhance marbling in grass-fed beef.

Finally, better maternal nutrition not only improves fetal development, but also improves the body condition score of cows, which is known to shorten postpartum estrus and increase pregnancy rate (Lents et al., 2008). In addition, better gestational nutrition also enhances the reproduction efficiency of offspring heifers (Funston et al., 2010b; Robinson et al., 2013; Sullivan et al., 2009), thus improving overall production efficiency of cow-calf herd (Figure 2).

**Conclusion**

The profound impact of maternal nutrition on fetal development has been well established, of which fetal skeletal muscle and adipose tissue development...
development are particularly vulnerable due to their low nutrient partitioning priority compared with vital organs and tissues. Because meat animals are raised primarily for their meat production, improving maternal nutrition during gestation, especially during the periods corresponding to major muscle and intramuscular fat development, will improve fetal muscle and adipose tissue development, which has long-term impacts on the production efficiency in offspring animals. The long duration of pregnancy and lactation stage of beef cattle provides unique advantages for stage-specific nutrient supplementation, achieving precision animal production management to improve animal production efficiency and quality (Figure 2).

**Literature Cited**

Aoyama, H., and K. Asamoto. 1988. Determination of somite cells: Independence of cell differentiation and morphogenesis. Development 104:15–28.

Bailey, P., T. Holowacz, and A.B. Lassar. 2001. The origin of skeletal muscle stem cells in the embryo and the adult. Curr. Opin. Cell Biol. 13:679–689. doi:10.1016/S0955-0674(00)00271-4

Beermann, D.H., R.G. Cassens, and G.J. Hausman. 1978. A second look at fiber type differentiation in porcine skeletal muscle. J. Anim. Sci. 46:125–132. doi:10.2527/jas1978.461125x

Cao, Y. 2007. Angiogenesis modulates adipogenesis and obesity. J. Clin. Invest. 117:2362–2368. doi:10.1172/JCI32239

Corah, L., and M. McCully. 2007. Declining quality grades: A review of factors reducing marbling deposition in beef cattle. Certified Angus Beef LLC. http://www.cabpartners.com/news/research/declining_quality_grades.pdf

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, D.M. Larson, and S.P. Ford. 2008. Natriuretic overnutrition enhances mRNA expression of adipogenic markers and collagen deposition in skeletal muscle of beef cattle fetuses. J. Anim. Sci. 92:3846–3854. doi:10.2527/jas.2014-7568

Duarte, M.S., P.M. Gionbelli, P.V. Paulino, N.V. Serao, C.S. Nascimento, M.E. Botelho, T.S. Martins, S.C. Filho, M.V. Dodson, S.E. Guimarães, and M. Du. 2014. Maternal overnutrition enhances mRNA expression of adipogenic markers and collagen deposition in skeletal muscle of beef cattle fetuses. J. Anim. Sci. 92:3846–3854. doi:10.2527/jas.2014-7568

Dwyer, C.M., N.C. Stickland, and J.M. Fletcher. 1994. The influence of maternal nutrition on muscle fiber number development in the porcine fetus and on subsequent postnatal growth. J. Anim. Sci. 72:911–917.

Fève, B. 2005. Adipogenesis: Cellular and molecular aspects. Best Pract. Res. Clin. Endocrinol. Metab. 19:483–499. doi:10.1016/j.beem.2005.07.007

Freathy, H.C., C.L. Ferrell, and T.G. Jenkins. 2000. Timing of reactivation of mature cows that were feed-restricted during pregnancy influences calf birth weights and growth rates. J. Anim. Sci. 78:2790–2796. doi:10.2527/2000.78112790x

Funston, R.N., D.M. Larson, and K.A. Vonahmme. 2010a. Effects of maternal nutrition on conceptus growth and offspring performance: Implications for beef cattle production. J. Anim. Sci. 88:E205–E215. doi:10.2527/jas.2009-2351

Funston, R.N., J.L. Martin, D.C. Adams, and D.M. Larson. 2010b. Winter grazing system and supplementation of beef cattle during late gestation influence heifer progeny. J. Anim. Sci. 88:4094–4101. doi:10.2527/jas.2010-3039

Gnanalingham, M.G., A. Mostyn, M.E. Symonds, and T. Stephenson. 2005. Ontogeny and nutritional programming of adiposity in sheep: Potential role of glucocorticoid stimulation. Peroxisome proliferator-activated receptor-gamma, adiponectin and leptin mRNA expression in adipose tissue before birth. Endocrinology 148(2):878–885.
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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