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Zoey Weisman
zoey.weisman@uconn.edu

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Zoey Weisman
University of Connecticut
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Dr. Sarah Reed, Thesis Advisor

Dr. Steven Zinn, Honors Advisor
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INTRODUCTION

Maternal nutrition is the diet that a mother intakes from before pregnancy through the end of lactation (Martin, 2018). Pregnant ruminants that are raised in pastures are frequently subject to undernutrition due to changes in the quantity and quality of available forage, which results in detrimental effects on the development of muscle and adipose tissues of offspring that can persist into adulthood (Hoffman, 2014). The quality of animal feed varies due to weather changes and climate fluctuations. Sheep usually gestate during the winter, as they are short-day breeders, and during this time, the forage consumed is limited and usually of poor quality (Martin, 2018).

Ruminants are also commonly overfed during gestation due to differing livestock management practices, all of which are implemented to improve production efficiency. Some management systems feed excess nutrients during the winter months in an attempt to combat the decrease in forage quality, which can lead to over-conditioning of ewes. One common method in the sheep industry, flushing, is when mothers are fed increased total dietary nutrients for a period of time before and after breeding to increase ovulation (Hoffman, 2014). Undernutrition and overnutrition during gestation can alter the body condition of ewes, which consequently alters their offspring. Poor maternal nutrition during gestation in ewes has significant effects on the processes of fetal growth and development, specifically adipogenesis and myogenesis in offspring. The severity of these effects on offspring depends on when changes in diet occur during gestation, as well as the duration that the mother consumes the diet (Smith et al., 2021).

MATERNAL PROGRAMMING & OBESITY
The health of a mother during pregnancy can alter both the prenatal and postnatal health of the fetus. Maternal programming is a result of exposure of the mother during gestation to environments that can be harmful to the fetus, which prepares the offspring for a similar environment after birth, potentially affecting future growth and development (Hoffman et al., 2014). Fetal programming in response to maternal malnutrition is crucial to preserve vital organs and allow the offspring to develop advantageous metabolic adaptations for postnatal survival (Hales and Barker, 2001). If the offspring is born into an environment with conditions different from those experienced in utero, the changes from maternal programming could result in increased susceptibility of the offspring to health issues and a poor body condition.

One case of maternal programming is a result of overfeeding or underfeeding of the mother during gestation. This can be in the form of the excess or restriction of total dietary energy or by restricting specific nutrients, most commonly protein or fat. Poor maternal nutrition during gestation can alter the postnatal growth of offspring by programming stem cell populations and negatively impacting satellite cell function (Hoffman et al., 2017). These alterations in gene expression and stem cell functions are the mechanisms that affect the growth and development of organs, adipose, and muscle in offspring (Pillai et al., 2017). Changes in stem cell functions due to programming during intrauterine life can result in diseases such as obesity. If a ewe is obese during pregnancy, the offspring undergo adaptations in utero to survive in a postnatal environment with an abundance of nutrients (Hoffman, 2014). However, not every obese mother will give birth to offspring that will become obese as an adult. The effects of maternal programming are dependent on if the environment the offspring is born into is similar to the conditions the fetus experienced during gestation. The Thrifty Phenotype Hypothesis states that the adaptations which occur during gestation will only be detrimental if the offspring is born
into conditions different than those the mother forecasts to the fetus in utero (Hales and Barker, 2001). Therefore, if a ewe is fed a restricted diet while pregnant, but the fetus is fed adequate nutrients or is overfed postnatally, the offspring will likely become obese. When a developing fetus is subject to suboptimal intrauterine conditions during gestation, negative alterations to the structure and function of cells, tissues, and organs can occur (Entringer et al., 2012 and Tong, 2010). Given the appropriate conditions, maternal programming can have adverse consequences on the health of offspring.

**EFFECTS OF POOR MATERNAL NUTRITION ON OFFSPRING BIRTH WEIGHT AND BODY COMPOSITION**

The prenatal developmental period in sheep lasts approximately 147 days. During this time, the nutrition of mother ewes can have varying effects on the fetal birth weight and body composition of offspring. In the last two months of gestation, the sheep fetus rapidly gains weight. Offspring of ewes fed excess energy in their diets have greater live weights at the time of weaning and also have greater average daily growth from the time of birth to weaning, compared to ewes fed their total energy requirements. This increase in weight in the offspring of overfed ewes is likely due to an increase in adiposity of organs including the liver, heart, omasum, and kidneys (Pillai et al., 2017). Furthermore, underfeeding a mother during gestation can cause intrauterine growth restriction, resulting in a lower birth weight of the fetus (Pillai et al., 2016). However, poor maternal nutrition does not always alter fetal birth weight. One study revealed that the offspring of underfed ewes had birth weights that were similar to ewes fed sufficient nutrients, but had less total muscle mass (Woo et al., 2011). Overnutrition in ewes causes similar phenotypic changes in the offspring to those from restricted nutrition, including increased adipose deposition and decreased muscle mass. Although these alterations to tissue development
appear to influence body compositions similarly in offspring of both overfed and underfed ewes, the mechanisms by which they occur are different (Hoffman et al., 2016 and Martin, 2018). It has also been shown that poor maternal nutrition can alter collagen content in the musculature of offspring. Collagen is associated with fibrosis which reduces the efficiency of muscles and can cause metabolic changes. In one study, offspring of obese ewes had increased collagen content and upregulation of genes promoting collagen, but also lessened ability for collagen remodeling (Hoffman et al., 2017). Feeding excess-nutrient or nutrient-deficient diets to a mother sheep can have adverse effects on offspring body composition.

**MESENCHYMAL STEM CELLS & WNT SIGNALING**

Mesenchymal stem cells are multipotent stem cells that contribute to bone, muscle, and adipose tissue development that are responsive to changes in the maternal environment. Mesenchymal stem cells are needed for tissue growth, maintenance, and repair from the beginning of fetal development through an animal's entire life (Pillai et al., 2016). These cells are crucial to the development of fetal skeletal muscle because they are involved in the complex processes of myogenesis, adipogenesis, and fibrogenesis. Mesenchymal stem cells are also present postnatally, however, they only impact myogenesis during the fetal stage. There are multiple mechanisms that result in the shift in the commitment of mesenchymal stem cells. Wnt signaling plays a key role in the formation of myocytes and adipocytes in fetal muscle. In ruminants, myogenesis occurs during early to midgestation, and adipogenesis occurs in late gestation. Through autocrine and paracrine actions, Wnt growth factors can regulate the distribution of adipose throughout the body (Christodoulides et al., 2008). Therefore, myogenesis can be enhanced by increasing Wnt signaling, and adipogenesis can be enhanced by inhibiting Wnt signaling as gestation progresses (Du et al., 2010). Further, inflammation reduces
adipogenesis by inhibiting AMP-activated protein kinase (AMPK) and also reduces myogenesis by downregulating Wnt signaling (Du et al., 2009). Mesenchymal stem cells and Wnt signaling play key roles in the fetal development of adipose and muscle tissue, and can be altered as a result of poor maternal nutrition.

When mesenchymal stem cells are committed to fetal adipogenesis and fibrogenesis instead of myogenesis, the outcome presents as long-lasting negative effects on the muscles of sheep offspring. These effects include an increase in intramuscular fat and connective tissue, as well as a reduction in the number and diameter of muscle fibers (Du et al., 2009). Maternal diet can determine the differentiation of mesenchymal stem cells into either muscle or adipose cells. Therefore, poor nutrition during gestation can contribute to alterations of offspring body composition, which typically includes increased adipose and decreased muscle. During the differentiation of mesenchymal stem cells, the metabolism changes from glycolytic to aerobic, which requires greater mitochondrial activity (Pillai et al., 2016). One study of maternal nutrition during gestation in sheep investigated the effects of maternal over and underfeeding on mesenchymal stem cell function in offspring. When proliferation of mesenchymal stem cells is restricted, the metabolic activity decreases, which extends the life of the cells by lowering oxidative stress. Poor maternal diet of ewes negatively affected offspring mesenchymal stem cells by reducing proliferation by 50% and reducing mitochondrial metabolic activity in the offspring of both restricted-fed and overfed animals. This reduced proliferation of mesenchymal stem cells reduces the stem cell pool which can affect the development of fat and muscle tissues (Pillai et al., 2016). In the offspring of ewes fed restricted and excess-nutrient diets, the cells had reduced expression of Peroxisome proliferator-activated receptor-gamma coactivator (PGC)-1alpha in their muscle at birth which regulates cellular energy metabolism, as well as
reduced oxidative phosphorylation compared to the offspring of ewes fed 100% of their nutrition requirements (Pillai et al., 2016). This evidence suggests that the mesenchymal stem cells in these offspring were programmed to limit the production of reactive oxygen species, which extends the lifespan of stem cells because the fetus is preparing to be birthed in a potentially unfavorable environment. The mesenchymal stem cells in offspring of restricted-fed and overfed ewes also displayed reduced basal respiration and ATP production, which indicates that they had a lesser ability to upregulate the production of ATP during an energy deficit. The changes in mesenchymal stem cells seen in this study indicate that poor maternal nutrition can cause metabolic dysfunction and diseases including obesity and oxidative stress, and also impair the development of muscle (Pillai et al., 2016).

**MYOGENESIS**

In an adult sheep, skeletal muscle makes up approximately 40% of the body weight, and is the primary location where glucose and fatty acids are utilized. When skeletal muscle is impaired during fetal development, it can result in lasting effects on the offspring (Du et al., 2009). Myogenesis is the process of the formation of muscle fibers, also called myofibers. The net number of myofibers is determined before parturition, so alterations to prenatal myogenesis will consequently alter postnatal myogenesis and result in phenotypic changes (Wynn, 2018). Early to mid-gestation is the most important time during the prenatal period for the number of muscle fibers to increase, which directly determines the potential for postnatal muscle growth. The total mass of a muscle is determined by both hyperplasia and hypertrophy. Hyperplasia is the increase in the number of muscle fibers, which occurs throughout the prenatal period. The number of muscle fibers after birth will either decrease or remain the same, but will never increase. Primary and secondary muscle fibers are formed during differentiation, which supports
the hypertrophy of muscles (Martin, 2018). Hypertrophy is the increase in the size of myofibers, which also occurs prenatally and continues through the prepubertal stage and sometimes after, given the correct stimuli (Costa et al., 2021). Muscle fibers undergo hypertrophy when protein accumulation is occurring at a greater rate than protein degradation. A majority of protein accumulation occurs during embryonic development. As an animal ages, the rate of protein accumulation slows down until it is equal to the rate of protein degradation (Costa et al., 2021).

Myogenesis is regulated by paired box factors and myogenic regulatory factors which work together to ensure the process is tightly controlled (Costa et al., 2021). During myogenesis, after myoblasts are recruited from the mesenchyme, they differentiate into muscle progenitor cells. When the muscle progenitor cells proliferate, they co-express the paired box transcription factors Pax3 and Pax 7 (Martin, 2018). Pax7 is responsible for maintaining myogenic progenitor cells in a proliferative state before they differentiate into the myogenic lineage and become myoblasts through the expression of myogenic regulatory factors (MRFs). Pax7 regulates the expression of four MRFs, including myogenic differentiation factor 1 (MyoD), myogenic factor 5 (MYF5), MRF4, and myogenin. These factors are expressed in a designated order to properly regulate the formation of myotubes, which later become myofibers (Martin, 2018). MYF5 and MyoD share the role of committing progenitor cells into muscle cells during primary myogenesis. MyoD binds to myostatin to remove myoblasts from the cell cycle, and also upregulates myogenin expression. The role of myogenin and MRF4 is to assist in the differentiation of myoblasts as they fuse together during secondary myogenesis, which is accomplished by targeting the proteins that determine whether cells proliferate or differentiate (Asfour et al., 2018). The paired box transcription factors Pax3 and Pax7 are vital for regulating the development of muscles in offspring during gestation, as well as muscle maintenance and
repair after parturition (Wynn, 2018). The regulation of processes involved in the formation of muscle cells is extremely important in fetal development.

EFFECTS OF POOR MATERNAL NUTRITION ON MUSCLE TISSUE OF OFFSPRING

Feeding ewes nutrient-restricted diets or diets over the total dietary nutrients had negative effects on the prenatal muscle development in offspring throughout gestation, and these effects are localized to specific areas in the muscle. Furthermore, offspring of ruminants fed nutrient-deficient diets during early to mid-gestation present a decrease in the rate of formation of secondary muscle fibers and also have a lower number of adipocytes within the muscles. Alternatively, offspring of overfed ewes also had a higher number of adipocytes in their muscles (Du et al., 2010).

Satellite cells are crucial for muscle repair and hypertrophy (Hoffman et al., 2017). In one study, researchers strategically chose time points to euthanize offspring in this experiment to represent the different stages of muscle development. Day 45 represents primary myogenesis, day 90 represents secondary myogenesis, and day 135 represents hypertrophy later in gestation. The semitendinosus and triceps brachii muscles displayed alterations due to maternal diet at various time points. At day 45, 90, and 135 of gestation, Pax7-positive cells were decreased in the semitendinosus of restricted offspring, and also at day 90 in the triceps brachii. Pax7-positive cells were decreased in the over offspring at day 90 in the semitendinosus, and at day 45 and 90 in the triceps brachii. These decreases indicate that the formation of muscle fibers during both primary and secondary myogenensis may have been inhibited, and also that satellite cell incorporation in muscles was reduced towards the end of gestation (Wynn, 2018). One study revealed that restricted maternal nutrition reduced the number of satellite cells in offspring by
33%. When the lambs in this study reached six weeks of age, the satellite cell function was observed again and showed a 32% reduction of the number of regenerating muscle fibers, indicating that restricted maternal nutrition continues to have adverse postnatal effects on satellite cell quantity and function, as well as muscle growth (Woo et al., 2011).

Offspring of malnourished mothers have altered expression of myogenic factors in muscle satellite cells. For example, offspring of ewes fed excess nutrients during mid-gestation had lower levels of MyoD and myogenin in their semitendinosus muscle, which resulted in decreased muscle hypertrophy. These results indicate that fetal myogenesis and satellite cell function during this time is impaired in offspring of underfed ewes (Hoffman et al., 2017). Lambs born to ewes that were overfed during gestation also had increased gene expression of myostatin and follistatin in the semitendinosus muscle tissue (Hoffman, 2014). Myostatin is a member of the transforming growth factor (TGF)-β family and works in conjunction with follistatin to regulate muscle mass. Myostatin negatively regulates muscle mass, and follistatin can bind to myostatin to directly inhibit its activity and in turn, increase muscle growth and increase fat accumulation (Lee et al., 2010) The mechanism through which this occurs is either the inhibition or attenuation of primary and secondary myogenesis (Wynn, 2018). Poor maternal nutrition causes alterations in gene expression related to epigenetic regulation and cell signaling in muscles (Pillai et al., 2017). Glycoproteins in the Wnt family are secreted through autocrine and paracrine signaling which can maintain precursor cells or cause them to proliferate and differentiate. The Wnt signaling pathway is dependent on β-catenin, which is required for the growth of various tissues, one of which is muscle. Offspring born from both overfed and restricted-fed ewes had increased expression of β-Catenin in their muscle tissue. β-catenin controls the expression of Pax3 and Gli, which are transcription factors that are crucial for the
development of skeletal muscle. Therefore, when the β-catenin pathway is blocked, the number of myocytes is reduced (Du et al., 2010).

In addition to Wnt signaling, poor maternal nutrition can locally alter muscle development through the somatotrophic axis. One study revealed that insulin-like growth factor (IGF)-1 gene expression, which is involved in the somatotropic axis, is increased in the quadriceps muscle in offspring of restricted-fed ewes (Hoffman, 2014). Offspring of obese ewes have increased collagen accumulation and cross-linking in their longissimus dorsi and semitendinosus muscles as well as increased skeletal muscle fibrogenesis. The offspring of obese ewes displayed increased signaling of TGF-β and expression of related enzymes, which promoted collagen synthesis and collagen content in the skeletal muscle of offspring. This upregulation of TGF-β also increased fetal myocardium fibrogenesis and turnover of myocardial tissue in the fetus. (Huang, 2012).

The components of a maternal diet fed during gestation are also a factor in offspring muscle development and can determine the offspring’s phenotype. Corn-based diets are high in starch. When a ewe is fed a corn-based diet during gestation, the muscles of her offspring are affected, resulting in a higher birth weight than the offspring of mothers fed high fiber diets. A maternal starch-based corn diet presented a greater impact on the fetal muscle development of offspring compared to those born from maternal diets high in protein, fiber, or fat (Peñagaricano et al., 2014). A large number of the genes in the longissimus dorsi muscle showed changes in expression from this diet, which had effects on the processes of muscle differentiation and myogenesis of ewes in the study. Additionally, there was a decrease in the expression of several genes associated with skeletal muscle development in the offspring of sheep fed the corn diet, including myogenic differentiation 1 (MYOD1), ankyrin repeat domain 1 (ANKRD1), and B-cell
lymphoma 9 (BCL9). MYOD1 plays an important role in the specification and differentiation of skeletal muscle, and it was significantly altered in the longissimus dorsi muscle. The transcription factor ANKRDI is part of the signaling cascade of muscle remodeling, myofibrillar assembly, and myogenic differentiation, which were all reduced in the fetal longissimus dorsi muscle as a result. BCL9 is involved in the Wnt signaling pathway, which controls differentiation during muscle regeneration and development. Offspring of sheep on the starch-based corn maternal diet also displayed down-regulation of genes associated with sarcomere organization and the muscle myosin complex which are involved in muscle contraction (Peñagaricano et al., 2014). The development of muscle is hindered in offspring of overfed and underfed ewes.

**ADIPOGENESIS**

Adipogenesis is the process in which mesenchymal stem cell precursors develop into adipocytes. In a sheep fetus, adipogenesis begins during mid gestation and continues through late gestation. Adipocytes continue to form throughout an animal’s life, but a majority of these cells are formed during embryonic development (Costa et al., 2021). Adipocytes form from stem cells in two main phases. The first phase involves the recruitment of mesenchymal stem cells to form preadipocytes which then proliferate, and the second phase involves the differentiation of those preadipocytes into mature fat cells (Pillai et al., 2016). The intrauterine environment can largely impact fetal adipogenesis, as it is sensitive to the concentrations of insulin-like growth factors, insulin, glucose, and glucocorticoids. In the fetus, cytokines regulate adipose tissue metabolism and control the growth of fat cells. Offspring of animals that are exposed to pro-inflammatory cytokines during gestation have been shown to have larger fat deposits (Entringer et al., 2012). Additionally, adipocytes have a higher lipogenic capacity postnatally when they are exposed to
an excess substrate supply during fetal development. This results in the fetus storing excess energy as fat, which in turn, makes them more susceptible to obesity (Entringer et al., 2012).

EFFECTS OF POOR MATERNAL NUTRITION ON ADIPOSE TISSUE OF OFFSPRING

Poor maternal nutrition in ewes can alter adiposity in offspring and have consequences that persist throughout adulthood (Hoffman, 2014). Studies have shown that ewes who were overfed during gestation gave birth to offspring with increased lipid oxidation and deposition in their muscle (Martin, 2018). This information suggests that maternal overfeeding predisposes offspring to altered lipid utilization and storage after they are born (Martin, 2018). A study of various isoenergetic diets fed to ewes during gestation revealed that the offspring of sheep on a dried corn distillers grains diet high in protein, fiber, and fat, were mostly impacted in the development of fetal adipose deposits in subcutaneous and perirenal tissues (Peñagaricano et al., 2014). In the subcutaneous adipose tissue of offspring on the dried corn distillers grains diet, there was higher expression of genes involved in adipose tissue and organ development, metabolism of lipids and lipoproteins, and adipose tissue development, overall resulting in increased adiposity of offspring. These genes include ANKRD1, kringle containing transmembrane protein 1 (KREMEN1), muscle-related coiled-coil protein (MURC), and synaptopodin 2-like (SYPO2L) (Peñagaricano et al. 2014). Seven genes that are part of the Wnt receptor signaling pathway showed high expression in the perirenal fat tissue of fetuses from ewes fed the dried corn distillers grains diet. These genes are associated with embryonic and fetal development, adipogenesis, and lipid metabolism (Peñagaricano et al., 2014).

When animals are fed restricted diets, adipogenesis in offspring occurs at a lower rate during fetal development to favor the vital organs (Entringer et al., 2012). If the offspring is born
into an environment where nutrition is no longer restricted, fat deposition will occur, mainly in the visceral adipose, in order to make up for the lack of deposition during gestation (Entringer et al., 2012). Studies have shown that when ewes are fed restricted diets during early to mid-gestation, the time when maximal growth rate of the placenta occurs, the offspring will be born with greater perirenal adipose tissue contents (Gnanalingham et al., 2005). Offspring are at risk of obesity when they are born from both overnourished and undernourished ewes.

ENDOCRINE SYSTEM EFFECTS ON ADIPOGENESIS AND MYOGENESIS

As gestation progresses, the fetus becomes increasingly sensitive to endocrine effects and less sensitive to autocrine and paracrine influences (Greenwood, 1997). The intrauterine environment is subject to changes from poor maternal nutrition, such as changes in concentrations of hormones and growth factors which affect the offspring’s metabolism. Lambs with lower birth weights have a less mature endocrine system and metabolism, which has adverse effects on myogenic regulatory factors and the levels of circulating glucose, insulin, and IGF-1 in the plasma (Greenwood, 1997). Insulin and glucose are crucial for maintaining energy balance and regulating metabolism in the body. Together, these hormones facilitate the accumulation of adipose tissue and inhibit myogenic differentiation. As a result, lambs born from malnourished ewes can exhibit reduced deposition of muscle, increased adipose content, and metabolic dysregulation (Hoffman et al., 2017 and Hoffman, 2014). The release of growth hormone (GH) stimulates the production of IGF-1 to increase postnatal muscle mass and decrease adiposity in the fetus. When IGF-1 is activated by GH, the muscle progenitor cells proliferate and myoblasts fuse together. Therefore, the growth of muscle is dependent upon the regulation of the GH/IGF axis; if the axis is impaired, muscle growth is restricted (Martin, 2018). IGF-1 plays a key role in prenatal muscle development, which involves the regulation of
proliferation and differentiation of precursor cells. When IGF-1 inhibits the expression of myogenin, proliferation increases. Conversely, when IGF-1 is present in greater concentrations, it can increase the expression of myogenin to favor differentiation instead (Hoffman, 2014). In one study, offspring of ewes, both underfed and overfed during gestation, exhibited decreased concentrations of IGF-1 and increased concentrations of insulin in the blood. Furthermore, adult offspring of restricted ewes in this study displayed increased secretion of insulin and glucose, and reduced sensitivity to insulin. As a result, these offspring displayed reduced muscle mass and increased accumulation of adipose (Martin, 2018). The development of both adipose and muscle is under hormonal control. Furthermore, the concentrations of hormones in the fetus are altered by poor maternal nutrition, which consequently alters myogenesis and adipogenesis.

REALIMENTATION

Realimentation is the process of restoring an animal back to full nutrition after it has been fed a restricted diet for a period of time. This process allows for the possibility of reducing the negative effects that a restrictive maternal diet has on fetal metabolism. Nutrient restriction during gestation of ewes causes alterations in amino acid and lipid metabolism in offspring (Smith et al., 2021). One of these alterations is an excess amount of the proteins required for glucose and energy metabolism. The offspring of underfed ewes showed increased gene expression of IGF binding protein-w, glycogen synthase 2, and pyruvate dehydrogenase kinase 1 in the liver, as well as the IGF-1 receptor in the longissimus dorsi muscle. Following realimentation, the expression of these genes returned back to normal (Smith et al., 2021). In one study, ewes that were severely malnourished were re-alimented with a nutritionally complete diet for either nine or sixteen days, and their offsprings’ fetal growth rate was immediately increased as a result (Greenwood, 1997). Furthermore, offspring of underfed ewes displayed decreased
liver weights, which returned back to normal following realimentation (Smith et al., 2021). The intrauterine growth restriction from underfeeding was also overcome through intravenous nutritional supplementation during late gestation (Greenwood, 1997). In another study, one group of ewes was fed 50% of their nutrient requirements from day 28 to day 78 of gestation then gradually realimented back to 100% of nutrient requirements until parturition. The offspring of these sheep were born with more fat than the group fed 100% of their nutrient requirements and the group fed 50% of their nutrient requirements (Brenman, 2012). Therefore, the results of this study indicate that realimentation following undernutrition of ewes can lead to increased adipose content in offspring. Realimentation during gestation has been shown to reduce the effects of, or allow full recovery from, the impairment that restrictive maternal diets inflict on fetal growth and development of sheep.

**CONCLUSION**

Maternal programming is a result of a challenge that the animal faces during development which causes persistent effects throughout its lifespan. Maternal undernutrition and overnutrition during gestation have been shown to cause fetal programming in animals that impairs the potential for growth and development of muscle and adipose tissue (Du et al., 2010). Poor maternal nutrition during gestation of ewes can also cause phenotypic changes in offspring by reducing hyperplasia and hypertrophy of muscles and increasing adiposity of the fetus. Mechanisms by which this occurs include altering gene expression, concentrations of circulating growth factors, secretion of metabolic hormones, and stem cell function (Hoffman et al., 2017). Feeding restricted or excess nutrients to ewes alters the expression of several key genes and pathways involved in primary and secondary myogenesis as well as adipogenesis. Development
of muscle and adipose tissue are also influenced by changes in hormone concentrations and altered sensitivity to hormones caused by poor maternal nutrition.

Body condition scoring is an effective method used in livestock management to assess the adiposity of a live animal. Regular body condition scoring of ewes during gestation using a subjective measurement on a 1 to 5 point scale can help farmers determine if a ewe is being fed an adequate diet (Kenyon et al., 2014). From there, the diet can be altered if necessary to better meet the needs of the ewe which in turn benefits the fetus. Changes in maternal intake can include different isoenergetic diets which have varying proportions of protein, fat, and carbohydrates. If a ewe is undernourished, which is a common problem during gestation, realimentation can be a helpful technique using a diet high in these nutrients, such as the previously mentioned dried corn distillers grains. Practicing proper management of livestock is a crucial aspect of maintaining the health and profitability of the animals.
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