

Maternal Plane of Nutrition: Impact on the Offspring^{1,2}

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INTRODUCTION

Maternal nutritional plane (Barker, 2004; Wu et al., 2006; Caton and Hess, 2010) has been implicated in developmental programming and resulting pre and postnatal changes that affect long-term offspring health and performance. Developmental Programming is the concept that perturbations during critical prenatal or postnatal developmental stages can have lasting impacts on growth and adult function. Maternal nutritional status (Wallace, 1948; Wallace et al., 1999; Godfrey and Barker, 2000; Wu et al., 2006; Caton and Hess, 2010) is a major factor implicated in development and function of the fetal organ systems. Effective nutritional (Bull and Carroll, 1937; Barcroft, 1946; Morrison, 1949; Maynard and Loosli, 1956; Crampton and Lloyd, 1959; NRC, 1970) management during gestation has long been recognized as an important component of sound livestock production practices. Recent observations and reviews in the area of developmental programming (Barker et al., 1992; Godfrey and Barker, 2000; Armitage et al 2004; Barker, 2004; Wu et al., 2006) have elevated the concept of maternal onset of adult disease and lifelong performance to the forefront of investigation in numerous national and international laboratories. In addition, underlying concepts of developmental programming are driving the development of research programs in multiple laboratories and shaping thought in funding arenas in both biomedical and animal agriculture,. This review will focus on the impacts of maternal nutritional plane on resulting offspring outcomes in ruminant livestock species..

MATERNAL NUTRITION AND DEVELOPMENTAL PROGRAMMING

Developmental Programming

Developmental programming, also termed “Fetal Programming,” “The Barker Hypothesis,” or, “developmental origins of health and disease,” is a major concept underpinning

research in many biomedical and agricultural laboratories (Hanson and Gluckman 2005; Wu et al., 2006; Caton and Hess, 2010; Reynolds and Caton, 2012). Developmental programming is the concept that perturbations during critical developmental periods may have long-term “programmed” impacts on offspring outcomes. While not new, this concept has been elevated to high interest among biological scientists by both early human epidemiological studies (Barker et al., 1989; Barker 1994; Barker 2004) and subsequent follow up work with animal models (Caton and Hess, 2010; Reynolds and Caton, 2012).

Maternal nutritional status is one of the factors implicated in programming nutrient partitioning and ultimately growth, development and function of the major fetal organ systems (Wallace, 1948; Wallace et al., 1999; Godfrey and Barker, 2000; Wu et al., 2006; Caton et al., 2007). Prenatal growth trajectory is sensitive to effects of maternal dietary intake from the earliest stages of embryonic life when the nutrient requirements for conceptus growth are negligible (Robinson et al., 1999). This is relevant because pre-term delivery and fetal growth restriction are associated with greater risk of neonatal mortality and morbidity. Growth restricted neonates are not only at risk of immediate postnatal complications, but may also exhibit poor growth and development, with significant consequences later in life (Barker et al., 1993; Godfrey and Barker, 2000; Barker, 2004; Wu, 2006; Caton and Hess, 2010).

Practical Relevance in Livestock

Within livestock production systems, there is real potential for ruminants to undergo periods of undernutrition (extensive grazing, drought, winter dominance, or high milk output). Decreased growth rate and suboptimal carcasses cost feedlot producers millions of dollars annually (Smith et al., 1995; Gardner et al., 1998). Fetal growth restriction and maternal

undernutrition are implicated in negative impacts upon growth efficiency and body composition (Greenwood et al., 1998, 2000; Wu et al., 2006; Caton et al., 2007; Larson et al., 2009). Recent evidence indicates that maternal nutrient restriction can significantly alter composition of offspring growth in the absence of birth weight differences (Gardner et al., 2005; Ford et al., 2007). Permanent changes in postnatal metabolism induced by maternal nutritional perturbations may present significant challenge to livestock producers because nutritional management decisions are often based on, for example, average body weight of a given group of animals, which may result in undernutrition of animals of below average body weight or overnutrition of animals heavier than average. Therefore, information regarding factors contributing to animal variation, such as developmental programming, has potential to improve efficiency and profitability of rearing programs.

Investigating maternal nutritional effects on offspring growth is very economically relevant to agricultural producers. Weaning weight is an important factor affecting profitability for producers who sell their calves at weaning (Reed et al., 2006). Fetal undernutrition frequently occurs in animal agriculture, leading to reduced fetal growth (Wu et al., 2006). Nutritional requirements of spring-calving beef cows grazing dormant range during late gestation typically exceed the nutrient value of the grazed forage (NRC, 1996; Johnson et al., 1998; Cline et al., 2009; Lardy and Caton, 2010). In addition, gestating ewes on rangeland pastures often experience prolonged bouts of undernutrition (Thomas and Kott, 1995) and the nutrient uptake of grazing ewes in the western United States is often less than 50% of NRC recommendations (NRC, 1985). Maternal nutrition pre- and postpartum has been shown to affect calf growth through weaning (Perry et al., 1991; Beaty et al., 1994; Spitzer et al., 1995; Stalker, 2006), with greater weaning weights produced from cows on a greater plane of nutrition.

In most of the previously mentioned studies offspring were allowed to nurse dams until weaning. The true effect of maternal nutrition on offspring is difficult to decipher when offspring are left nursing their dams until weaning, since pre-partum nutrition and post-partum nutrient supply (lactation) both contribute to postnatal growth and performance. However, some studies, Greenwood et al., 1998, 2001; Caton et al., 2007; Swanson et al., 2008; Neville et al., 2010, and Meyer et al., 2010a) have raised offspring independent of postnatal maternal influences which should eliminate confounding of prenatal and postnatal nutritional supply on offspring performance.

Maternal Nutrition

Maternal nutrient supply in terms of total nutrient intake or specific nutrient availability can vary widely in practical conditions and be driven by dietary quantity and/or quality issues, perturbations of intake, and environmental conditions. Maternal nutritional status is one of the factors implicated in programming postnatal nutrient partitioning and ultimately growth and development including that of muscle and adipose tissue (Wallace, 1948; Godfrey and Barker, 2000; Armitage et al., 2005; Wu et al., 2006; Taylor and Poston, 2007). Growth restricted infants are not only at risk of immediate postnatal complications, but also may be ‘programmed’ to exhibit poor growth and productivity and also to develop significant diseases later in life (Barker et al., 1993; Godfrey and Barker, 2000; Wu et al., 2006; Caton and Hess, 2010, Reynolds and Caton, 2012). The historical concept is that during pregnancy nutrient partitioning favors the conceptus at the expense of the dam (Barcroft, 1946). However, exposure of pregnant sheep to severe maternal undernutrition at all stages of pregnancy, and particularly during late

gestation, reduces fetal growth (Mellor, 1983; Robinson, 1983; Vincent et al., 1985; Parr et al., 1986).

Nutrient Restriction. Nutrient restriction is broadly defined as any series of events that reduce fetal and/or perinatal nutrient supply during critical windows of development. Nutrient restriction can result from altered maternal nutrient supply, placental insufficiency, deranged metabolism and regulation, physiological extremes, and environmental conditions. From a practical standpoint, maternal nutrient supply and environmental conditions leading to stress responses are the most likely observed causes of nutrient restriction in ruminant livestock. However, in the incidence of multiple births in some breeds can also contribute to physiological extremes and result in reduced nutrient supply to developing fetuses. In this review we will focus primarily on maternal dietary induced nutrient restriction.

Due to the pattern of placental growth in relation to fetal growth during gestation (Redmer et al., 2004), it is important to realize that the effects of nutrient restriction during pregnancy may depend on the timing, level, and (or) length of nutrient restriction. In an excellent review, Luther et al., (2005) indicated that maternal nutrient restriction in sheep during the through mid pregnancy could reduce placenta size and function, while having minimal impacts on fetal body weight near term. In addition, late pregnancy nutrient restriction often will likely reduce fetal weight. Recent data (Reed et al., 2007; Swanson et al., 2008) demonstrates that maternal nutrient restriction during the last two thirds of pregnancy in sheep reduces birth weights. Sletmoen-Olsen et al., (2000) indicated that both low and high levels of metabolizable protein supplementation to mature beef cows reduce birth weights relative to controls fed at projected requirement. In contrast, protein supplementation of cows during the last trimester of pregnancy has been reported to have little effect on birth weights (Martin et al., 2007; Larson et

al., 2009). In addition, a moderate level of total nutrient restriction during the last two thirds of gestation (H. Freetly, personal communication) in mature beef cows has not impacted birth weights. Other data (Spitzer et al., 1995 and Stalker et al., 2007) indicates that increasing body condition before parturition can increase calf birth weight. From the available data it appears that birth weights in sheep are more susceptible to maternal nutrient restriction than beef cattle.

Nutrient Excess. Maternal overnutrition during gestation can also have adverse effects. It has been shown that overnourishing the singleton-bearing adolescent ewe throughout gestation results in rapid maternal growth, and most particularly of maternal adipose tissue, at the expense of the nutrient requirements of the gravid uterus (Wallace et al., 1996, 1999, 2001). In this paradigm, rapid maternal growth results in placental growth restriction, premature delivery of low-birth weight lambs compared with moderately nourished ewes of equivalent age. Data recently generated demonstrates that birth weights are decreased by 9.2% when adolescent ewes are fed 140% of energy requirements from d 40 of pregnancy until parturition (Swanson et al., 2008), which indicates that moderate overnutrition during the last two-thirds of pregnancy can cause moderate fetal growth restriction. Although placental and fetal growth restriction is generally seen only in adolescent, overnourished ewes, gestation length and colostrum yield are negatively affected in adult ewes overnourished throughout pregnancy (Wallace et al., 2005), indicating that the health and growth of offspring from adult animals may also be altered by maternal overnutrition.

PERTURBATIONS OF FETAL DEVELOPMENT AND OFFSPRING RESPONSES IN RUMINANT LIVESTOCK

Periconceptional Nutrition and Oocyte Quality

Nutritional status is a major factor influencing an animal's ability to reproduce (Robinson, 1990; Webb et al., 1999; O'Callaghan et al., 2000). Nutrition has a significant impact on numerous reproductive functions including hormone production, fertilization, and early embryonic development (Boland et al., 2001; Armstrong et al., 2003; Boland and Lonergan, 2005). Changing nutrient supply during the peri-conceptual period can have profound impacts on reproduction. Nutritional status has been correlated with embryo survival and is a key factor influencing efficiency in assisted reproductive technologies (Armstrong et al., 2003; Webb et al., 2004). Recent studies (Borowczyk et al., 2006; Grazul-Bilska et al., 2006) have investigated the effects of maternal plane of nutrition prior to superovulation and in vitro fertilization on oocyte quality and embryonic development. In the first study (Borowczyk et al., 2006) control ewes were fed at maintenance and an underfed group was fed at 60 % of controls. After eight weeks ewes were superovulated, oocytes collected and evaluated, in vitro fertilization rates of oocytes determined, and viability to morula and blastocysts states of embryos determined. There were no differences in the number of healthy oocytes collected from control and restricted ewes. However, at fertilization, restricted ewes produced oocytes that fertilized more poorly. In addition, morulas and blastocysts, were less in the underfed group.

In a follow-up study, these researchers added an additional over fed treatment (Grazul-Bilska et al., 2006). In the second study, which was also with ewes, these researchers had a control group, which maintained body weight and condition score (adiposity) through an 8 week pre-superovulation period, an overfed group that increased adiposity, and a restricted group that lost weight. The overfed group was fed ad libitum and the restricted group was fed 60 percent of controls. After 8 weeks ewes were superovulated and oocytes collected. There was no

difference in the number of characterized healthy oocytes in control, overfed, and restricted ewes. However, both the overfed and the restricted ewes had reduced successful fertilizations, morulas, and blastocysts, indicating that maternal nutrition (both inadequate and excess) before mating can have profound impacts on oocyte quality and fertilization rates.

Placental Development

A primary role of the placenta is to provide for physiological interface, including nutrient and waste exchange between the fetal and maternal systems (Meschia, 1983; Reynolds and Redmer, 1995; Reynolds et al., 2010). Therefore, adequate placental circulation is imperative to successful pregnancy and is exemplified by observed close relationships among fetal weight, placental size, and uterine and umbilical blood flows during normal pregnancies (Reynolds et al., 2005a, 2005b, 2006; 2010). In the ewe, cotyledonary growth is exponential during the first 10 to 11 wk of pregnancy, with significant slowing until term (Stegeman, 1974; Ferrell et al., 1976). In the cow, the cotyledonary growth progressively increases throughout gestation (Reynolds et al., 1990; Vonnahme et al., 2007).

Uterine and umbilical blood flows, which represent the circulation to maternal and fetal portions of the placenta, respectively (Ramsey, 1982; Mossman, 1987), increase exponentially throughout gestation, essentially keeping pace with fetal growth (Reynolds and Redmer, 1995; Magness, 1998). For example, in sheep the absolute rate of uterine blood flow increases by approximately 3-fold throughout the last half of pregnancy (Meschia, 1983). Over a similar interval of gestation, uterine blood flow in cows increase by 4.5-fold (Reynolds, 1986).

In sheep studied during late gestation, uterine or umbilical blood flows, or both, are reduced in every model of compromised pregnancy in which they have been evaluated

(Reynolds et al., 2006). These models of compromised pregnancy include overfed adolescents, underfed adolescent and adult dams, as well as environmental heat-stress, hypoxic stress, and multiple fetuses.

In addition, although vascular development of the placenta also is decreased in several of the models of compromised pregnancy, it is increased in others (Reynolds et al., 2006).

Interestingly, the models in which placental vascularity is increased show no effect on fetal size (high dietary Se, or hypoxic stress) or were associated with long-term genetic selection (Romanov vs. Columbia genotype), suggesting an adaptive placental response that preserves the fetal nutrient supply.

Fetal Organ Development and Postnatal Function

Nutrient restriction to the developing conceptus, regardless of the reason (maternal nutrient restriction, environmental conditions, carunclectomy or other experimental models), often results in impaired fetal organogenesis and (or) development. The degree of compromised internal organ growth is usually more severe with increasing extremes of nutrient restriction. Timing of nutrient deprivation can also result in differential effects of fetal organ systems, with restriction during early to mid pregnancy, a critical time for fetal organ growth and development, being more likely to compromise function when compared with moderate levels of restriction during late pregnancy. Various organ systems may respond differently to specific timing and severity of nutrient restriction because of differing growth trajectories and maturation time points. In fact, recent data indicate that both low and high planes of maternal nutrition can impact growth of numerous fetal organs (Reed et al., 2007; Carlson et al., 2008; Caton et al., 2009)

Our laboratories have a particular interest in intestinal development, growth, and function. Intestinal tissues are important to livestock production because of their role in nutrient uptake, immunocompetence, and their disproportional use of energy (and other nutrient resources) in relation to their contribution to overall body mass. A detailed discussion of fetal and offspring response to perturbations during gestation are beyond the scope of this effort. However, readers are referred to Reed et al., 2007; Carlson et al., 2008; Caton et al., 2009; Neville et al., 2010, and Meyer et al., 2010a,b).

Muscle Development and Product Quality

Maternal nutritional status is one of the factors impacting nutrient partitioning and ultimately growth and development of fetal skeletal muscle (Strickland et al., 2004; Rehfeldt et al., 2004; Wallace, 1948; Wallace et al., 1999a; Godfrey and Barker, 2000). Skeletal muscle has a lower priority in nutrient partitioning compared to the brain and heart during development, rendering it potentially more vulnerable to nutrient perturbed nutrient supply (Bauman et al., 1982; Close and Pettigrew, 1990). The fetal period is crucial for skeletal muscle development, because no net increase in the number of muscle fibers occurs after birth (Glore and Layman, 1983; Greenwood et al., 2000; Nissen et al., 2003). Numerous studies carried out in a range of mammalian species have shown that maternal undernutrition during gestation can significantly reduce the number of both muscle fibers and nuclei in the offspring (Bedi et al, 1982; Ward and Strickland, 1991). Zhu et al. (2004) observed that nutrient restriction from early to mid-gestation resulted in a reduction in the number of fetal skeletal muscle fibers, which might be related to a down-regulation of signaling a system crucial for determining nutrient availability, mammalian target of rapamycin (mTOR). Bee (2004) also observed differences in fiber type in

semitendinosus muscle where progeny from the low-energy diets had fewer fast glycolytic fibers than the offspring of sows fed a high-energy diet during the first 50d of gestation. These results correspond with results of Schantz and Hutchinson (1983), where a reduction of fast glycolytic fibers but retained slow oxidative fiber numbers in energy deficient animals.

Muscle fiber type and size not only affect prenatal and postnatal growth, they also can have an impact on end-product quality. Percentage of slow muscle fibers present can affect meat color (Monin and Ouali, 1992) and speed of tenderization that occurs postmortem (Ouali, 1990). Fiber diameter has an impact on meat tenderness (Maltin et al., 1997). Altering fiber type will also impact glycogen storage capabilities, which has an impact on water-holding capacity (Fernandez and Thornberg, 1991). These product quality traits have a large impact on the acceptability and consumer demand of animal food products.

Adipose Tissue Development and Postnatal Accumulation

In sheep and cattle, adipose tissue development begins during gestation, but the vast majority of adipose tissue growth occurs postnatally (Mersmann and Smith, 2005). Perirenal adipose tissue represents the primary adipose tissue depot at birth in sheep (Muhlhausler et al., 2002). The perirenal adipose depot undergoes rapid and considerable expansion in the first month of life, during which time the adipose tissue transitions from having a thermoregulatory function to one of energy storage (Clarke et al., 1997). Adipose tissue development during the neonatal period is accompanied by a well-characterized pattern of gene expression as adipose tissue expresses the proteins required for the synthesis of triacylglycerol from glucose and fatty acids (Mersmann and Smith, 2005).

The role of maternal nutrition in programming postnatal body composition and growth trajectory may involve permanent alterations in adipocyte morphology, cellularity, and (or) metabolism (Taylor and Poston, 2007). Recent data examining the effects of maternal protein restriction has revealed marked changes in the characteristics of visceral adipose tissue in the offspring, such as upregulation of genes associated with preadipocyte proliferation, adipocyte differentiation, lipogenesis, and angiogenesis, and a concomitant downregulation of anti-angiogenic factors (Guan et al., 2005). These data in rodents suggest that, in meat animals, maternal nutrition may alter the trajectory of visceral adipose tissue growth of offspring and prove detrimental to the value of the carcass at slaughter.

In sheep, the effects of maternal undernutrition on birth weight and fetal adipose tissue mass have been inconsistent and depend on the timing, level, and (or) length of dietary restriction (Bispham et al., 2003; Symonds et al., 2004). A more consistent observation in lambs from undernourished ewes is that the characteristics of fetal adipose tissue are altered. Other studies have documented that total visceral (Gardner et al., 2005) and perirenal (Ford et al., 2007) adiposity of the offspring is increased by maternal undernutrition, which was accompanied by insulin resistance compared with offspring from adequately-nourished ewes (Gardner et al., 2005; Gnanalingham et al., 2005; Ford et al., 2007; Caton et al., 2007; Vonnahme et al., 2010).

Ford et al. (2007) recently reported that expanded perirenal fat mass in offspring from nutrient-restricted ewes is associated with a reduction in semitendinosus and longissimus muscle mass as a proportion of hot carcass weight. These data, coupled with relative insulin resistance in the offspring from nutrient-restricted dams (Gardner et al., 2005; Ford et al., 2007; Vonnahme et al., 2010), indicate that visceral adipose tissue may contribute to abnormal nutrient metabolism.

Postnatal Performance Measurements

Growth (an increase in the number and size of cells or in mass of tissue) and development (changes in the structure and function of cells or tissues) of the calf are complex biological events. There are numerous growth and development pathways that may influence productive characteristics of ruminants. Growth and development of the bovine fetus, which can be influenced drastically by restricted nutrition during the latter half of gestation (Dunn, 1980) has well-studied consequences for survival. Likewise, pre-weaning plane of nutrition or level of maternal milk production effects on growth to market weights of cattle are well characterized (Berge, 1991). However, investigations on effects of various treatments or management practices might also need to be made in early- to mid-gestation (Anthony et al., 1986). Long-term consequences of more specific nutritional influences during specific stages of fetal and neonatal calf development remain to be determined.

When lambs were separated from their dams and raised on an identical management system regardless of maternal treatment, maternal nutritional plane during gestation continued to affect on lamb growth through d 19 of age (Meyer et al., 2010a). Specifically, lambs born to ewes restricted to 60% of control nutrient intake weighed less than controls at multiple time points up to necropsy at 19 d. Also, lamb ADG was decreased in lambs from restricted ewes compared with controls and those from over fed ewes from birth to d 19. In agreement with the current study, Neville et al., (2010) recently reported that lambs born to restricted ewes weighed less birth than lambs born to control-fed or over fed dams with some differences persisting to both weaning and 180 d of age. Others have reported effects of maternal nutrition during

gestation on postnatal growth of cattle (Stalker et al., 2006; Martin et al., 2007; Larson et al., 2009), which tends to agree with our data with lambs.

In a recently report (Neville et al., 2010) growth, digestibility, and N retention were studied in offspring from dams receiving different levels of nutrition and Se during gestation. As in the work of Meyer et al., (2010a) mentioned above, lambs in these studies were removed from ewes at birth and raised independently. They report (Neville et. al., 2010) that male offspring from ewes not supplemented with Se had greater ADG then those receiving high Se during gestation. Conversely, male offspring from over fed ewes not supplemented with Se had lower ADG compared with those receiving high Se during gestation. No differences in DMI resulted in the similar responses in G:F as were found in ADG. These authors also reported reductions in diet digestibility in growing lambs from ewes fed high Se during gestation when compared with those born to ewes fed adequate Se. No differences were observed in nitrogen retention. These authors speculated that lambs from HSe dams either had increased passage rates or decreased digestive efficiencies. Others (Martin et al., 2007) found greater residual feed intake in female calves from dams that received a protein supplement during late gestation and an early lactation hay diet. In addition, Stalker et al. (2006) and Larson et al. (2009) found no differences in ADG, DMI, or Gain:Feed in steer calves during the finishing period when dams received protein supplements during late gestation.

Many extensive beef cattle production systems are designed for cattle to receive the majority of their nutrients from grazed forages. Forage quality is often poor, particularly in dry and winter seasons, and may be inadequate to support optimal nutrition for growth, pregnancy, and lactation without provision of supplemental nutrients. Supplementation of nutrients may not occur for cows during early through mid-gestation because nutrients required to support the

growing and developing fetus appear minimal, especially when compared with later stage of gestation and early to mid-lactation. Bovine fetal undernutrition is quite likely to occur during late pregnancy, particularly if cows do not receive supplemental energy and protein. Low precalving plane of nutrition of the cow has been associated with low birth weight of the calf (Bellows et al., 1971), and calf birth weight was increased in heifers and cows in response to dietary supplementation with protein and energy concentrates during late gestation (Clanton and Zimmerman, 1970; Bellows and Short, 1978).

Nutritional inadequacy is likely to occur in beef cattle production systems in which cows are managed to produce a calf annually. In these systems, with an average gestation length of 285 days, a cow must conceive within 80 days postpartum to produce a calf on a yearly basis. In lactating heifers and cows beginning at the second or greater parity, milk output generally peaks at about 2 months postpartum, but feed intake usually takes at least 2 months or more to reach its maximum, which results in negative energy balance during early to mid-lactation (Bauman and Currie, 1980). The low plane of nutrition will be more evident if cows are managed to calf during the dry or winter seasons when poor forage quality is more probable.

Offspring born with low birth weights are less viable and adjust less rapidly to the extrauterine environment (Cundiff et al., 1986). Furthermore, low birth weight is associated with high neonatal morbidity and mortality rates. In reviewing 13 datasets in which researchers placed cows on a low plane of nutrition during late gestation, Dunn (1980) noted that the incidence of neonatal calf mortality increased 5 percentage units if cows were underfed. More recent data summarized by Wu et al. (2006) showed that preweaning deaths of heifer calves born alive in the United States was 10.5%, with 70% of the deaths occurring within the first 7 days after birth.

Incidence of neonatal mortalities may be linked to less thriftiness and reduced immune transfer for calves born to cows that were undernourished during gestation (Odde, 1988).

If the low birth weight calf survives the early neonatal period, it is possible that it will grow slower than calves of normal birth weight at all stages of postnatal growth. Calves born 35% lighter due to severely restricted maternal nutrition from day 80 to 90 of pregnancy to parturition remained smaller at any given postnatal age compared with well-grown or better nourished counterparts (Greenwood and Cafe, 2007). However, it is difficult to ascertain whether or not this response represents permanent stunting or simply delayed of attainment of mature size. When differences in birth weight were less severe, however, post-weaning growth was not significantly affected by birth weight (Cafe et al., 2006a). Nonetheless, influences of nutrition during mid and late pregnancy on calf weaning weight have been demonstrated, irrespective of effects on fetal growth (Cafe et al., 2006b; Stalker et al., 2006). Although effects of variable nutrition during mid and (or) late pregnancy on weight at birth seem to be overcome by adequate nutrition within 56 days postpartum (Freetly et al., 2000), a cursory review of several datasets would suggest that, while not statistically significant in each experiment, absolute weight of cattle at the time of harvest is consistently less if calves are born lighter or are lighter at weaning (Freetly et al., 2000; Banta et al., 2006; Stalker et al., 2006).

The major nutritional factors affecting pre-weaning calf growth and composition at weaning are the lactational performance of the dam and the quality and availability of nutrients from pasture and (or) supplementation before and after parturition. In summarizing data of Greenwood et al. (2005; 2006), Greenwood and Cafe (2007) illustrated that a difference in weaning weight of 73 kg resulted in a 40 kg difference in live weight and 24 kg in carcass weight at 30 months of age. The low weaning weight cattle grew more rapidly during backgrounding

and at a similar rate in the feedlot. Similarly, Cafe et al (2006a) reported some compensatory growth occurred in steers restricted in growth from birth to weaning, then backgrounded to the same feedlot entry weight. Thus, nutritional restriction to weaning limits compensatory growth, which results in smaller cattle and carcasses at an equivalent age end points. Effects of early-postpartum nutrition on growth, intake and efficiency of steers (Stalker et al., 2006) and heifers (Martin et al., 2007) in the feedlot soon after weaning were not evident, which was consistent with literature reviewed by Berge (1991) in which feed conversion efficiency was not necessarily affected by plane of nutrition before weaning.

SUMMARY AND CONCLUSIONS

Data provides strong evidence for developmental programming in livestock. Maternal nutrition during gestation is a major determinant of fetal growth. Consequently, improper nutrition during gestation creates situations during critical windows of development where offspring may be predisposed to long-term complications. Specifically, inadequate nutrition during early to mid-gestation alters development of a variety of fetal tissues, which may affect subsequent health but effects on postnatal growth performance and carcass characteristics seem to be equivocal. A low plane of nutrition during mid to late gestation may reduce the ruminant offspring's absolute body weight at birth through slaughter, but again, effects on postnatal growth performance and carcass characteristics are less affected if nutrient restriction during gestation is not severe. Improper nutrition during the perinatal period will increase offspring mortality. A low plane of nutrition during lactation may reduce absolute body weight throughout the growing period because calves experiencing lower planes of nutrition during the first 6 months of life are less apt to compensate later in life. Additional research efforts in these

areas are needed to quantify degree of impact of in appropriate maternal nutrition during gestation on both short and long-term metabolism, health, production, product quality, and economics in ruminant livestock species.

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