Maternal Plane of Nutrition and Impact on the Offspring[1,2]

J. S. Caton[3†‡] and B. W. Hess[*]
[‡]Center for Nutrition and Pregnancy, Animal Sciences Department
North Dakota State University
[*] Department of Animal Science, University of Wyoming

Introduction

Maternal nutritional plane (Barker, 2004; Wu et al., 2006) 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) 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. Fetal intrauterine growth restriction (**IUGR**) is an issue in relevant livestock species in certain circumstances (Wu et al., 2006) and can be precipitated by numerous forms of perturbed maternal nutrition. 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; Caton and Hess, 2010) 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 relevant livestock species. Emphasis will also be placed on growth, development, metabolic, and performance outcomes, timing of nutritional perturbations, potential long-term production consequences, and areas for future research.

---

[1] This manuscript is an updated summary presentation of a previously larger work presented at the Grazing Livestock Conference and published in those proceedings; Caton, J. S. and B. W. Hess. 2010. Maternal plane of nutrition: Impacts on fetal outcomes and postnatal offspring responses. Invited Review. Pages 104-122 in Proc. 4th Grazing Livestock Nutrition Conference. B. W. Hess, T. Delcurto, J. G. P. Bowman, and R. C. Waterman eds. West Sect. Am. Soc. Anim. Sci., Champaign, IL.

[2]The authors would like to thank the numerous collaborators that have contributed to the data and concepts contained within this manuscript. Appreciation is also expressed the organizing committee of the 22nd Florida Ruminant Nutrition Symposium. This project was partially supported by the National Research Initiative Competitive Grants no. 2005-35206-15281 and 2009-35206-05253 from the USDA Cooperative State Research, Education and Extension Service.

[3]Contact at: Department of Animal Sciences, 185 Hultz Hall, North Dakota State University Fargo, ND 58108-6050; Phone: 701-231-7653; Email: joel.caton@ndsu.edu.

Maternal Nutrition Plane 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). 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.

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; Caton and Hess, 2010). The prenatal growth trajectory is sensitive to direct and indirect 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 especially 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 et al., 2006). Because of accumulating data and potential ramifications, the concept that a maternal stimulus, including plane of nutrition may result in developmental programming responses that impact neonatal, growth and developmental, and adult responses is gaining traction in animal agriculture.

Practical Relevance in Livestock

Within livestock production systems, there is real potential for ruminants to undergo periods of undernutrition (extensive grazing or high milk output) or overnutrition (overfeeding) during gestation. 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; Neville et al., 2010). 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 et al., 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 during gestation on offspring is difficult to decipher when offspring are left nursing their dams until weaning, since pre-partum nutrition and postpartum nutrient supply (lactation) both contribute to postnatal growth and performance. However, some studies (Greenwood et al., 1998; Caton et al., 2007; Swanson et al., 2008; Neville et al., 2010, 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

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, 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 hand 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; Stalker et al., 2006) indicate 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 over nourishing 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 demonstrate 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 are 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; Boland and Lonergan, 2005). Changing nutrient supply during the peri-conceptual period can have profound impacts on reproduction. 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% 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.

Fetal Organ Development and Postnatal Function

Nutrient restriction to the developing conceptus, regardless of the reason (maternal nutrient restriction, environmental conditions, caruncleectomy or other experimental models), often results in impaired fetal organogenesis and (or) development. The degree of compromised internal organ growth is usually more sever 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., 2009; Caton et al., 2009).

In our laboratories we 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. (2009), Caton et al. (2009), Neville et al. (2010), and Meyer et al. (2010a,b).

Muscle Development and Product Quality

Other more traditionally thought of as “production oriented” tissues like muscle and adipose also appear responsive to programming affects in utero. Maternal nutritional status is one of the factors impacting nutrient partitioning and ultimately growth and development of fetal skeletal muscle (Rehfeldt at al., 2004; Wallace, 1948; Wallace et al., 1999; 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 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 mammalian target of rapamycin (mTOR) signaling, which is crucial for determining nutrient availability.

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). Visceral adipose tissue accumulation represents a significant inefficiency in meat animal production, considering that much of the visceral fat is discarded at slaughter. While regulation of lipogenesis has depot-specific characteristics as well, relatively, few studies have addressed the biological mechanisms by which maternal nutrition alters adipose tissue growth and development in offspring. In addition, little data are available that addresses the long-term consequences of altered adipose tissue development on body composition and carcass value.

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). 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).

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), indicate that visceral adipose tissue may contribute to abnormal nutrient metabolism.

Postnatal Performance Measurements

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 lamb growth through d 19 of age (Meyer et al., 2010b). 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 average daily gain (**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 recent report (Neville et al., 2010) growth, digestibility, and nitrogen (**N**) retention were studied in offspring from dams receiving different levels of nutrition and selenium (**Se**) during gestation. As in the work of Meyer et al. (2010b) mentioned above, lambs in these studies were removed from ewes at birth and raised independently. Neville et al. (2010) reported 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 dry matter intake (**DMI**) resulted in the similar responses in gain to feed (**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 dams supplemented with high levels of Se 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 G:F in steer calves during the finishing period when dams received protein supplements during late gestation.

Developmental Programming of Offspring Born into Conventional Grazing Livestock Production Systems

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).

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 d 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).

Summary and Conclusions

Accumulating data is providing traction for relevance of developmental programming concepts in livestock. Maternal nutrition during gestation is a major determinant to 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 gestation nutrition restriction is not severe. Improper nutrition during the perinatal period will increase offspring mortality. Feeding supplemental lipid during late gestation seems to be an effective strategy to help the neonatal ruminant combat cold stress. 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 maternal nutritional perturbations during gestation on both short and long-term metabolism, health, production, product quality, and economics in ruminant livestock species.

References

Armitage, J. A., I. Y. Khan, P. D. Taylor, P. W. Nathanielsz, and L. Poston. 2004. Developmental programming of the metabolic syndrome by maternal nutritional imbalance: how strong is the evidence from experimental models in mammals? J. Physiol. 561:355-377.

Banta, J. P., D. L. Lalman, F. N. Owens, C. R. Krehbiel, and R. P. Wettemann. 2006. Effects of interval-feeding whole sunflower seeds during mid to late gestation on performance of beef cows and their progeny. J. Anim. Sci. 84:2410-2417.

Barcroft, J. 1946. Researches on Pre-Natal Life. Blackwell Scientific Pub., Oxford.

Barker, D. J. P. 2004. Developmental origins of well being. Philos. Trans. R. Soc. Lond. B. Biol. Sci. 359:1359-1366.

Barker, D. J. P. 1994. Mothers, babies and disease in later life. BMJ Publishing Group, London.

Barker, D. J. P., P. D. Gluckman, K. M. Godfrey, J. E. Harding, J. A. Owens, and J. S. Robinson. 1993. Fetal nutrition and cardiovascular disease in adult life. Lancet 2341:938-941.

Barker, D. J. P., K. M. Godfrey, C. Osmond, and A. Bull, 1992. The relation of fetal length, ponderal index and headcircumference to blood pressure and the risk of hypertension in adult life. Paediat. Perinat. Epidemiol. 6:35-44.

Barker, D. J., P. D. Winter, C. Osmond, B. Margetts, and S. J. Simmonds. 1989. Weight in infancy and death from ischaemic heard disease. Lancet 2:577-580.

Bauman, D. E., J. H. Eisemann, and W. B. Currie. 1982. Hormonal effects on partitioning of nutrients for tissue growth: role of growth hormone and prolactin. Fed. Proc. 41:2538-2544.

Beaty, J. L., R. C. Cochran, B. A. Lintzenich, E. S. Vanzant, J. L. Morrill, R. T. Brandt, Jr., and D. E. Johnson. 1994. Effect of frequency of supplementation and protein concentration in supplements on performance and digestion characteristics of beef cattle consuming low-quality forages. J. Anim. Sci. 72:2475-2486.

Bedi, K. S., A. R. Birzgalis, M. Mahon, J. L. Smart, and A. C. Wareham. 1982. Early life undernutrition in rats. 1. Quantitative histology of skeletal muscles from underfed young and refed adult animals. Br. J. Nutr. 47:417-431.

Bellows, R. A. and R. E. Short. 1978. Effects of precalving feed level on birth weight, calving difficulty and subsequent fertility. J. Anim. Sci. 46:1522-1528.

Bellows, R. A., R. E. Short, D. C. Anderson, B. W. Knapp, and O. F. Pahnish. 1971. Cause and effect relationships associated with calving difficulty and calf birth weight. J. Anim. Sci. 33:407-415.

Bispham, J., G. S. Gopalakrishnan, J. Dandrea, V. Wilson, H. Budge, D. H. Keisler, F. Broughton Pipkin, T. Stephenson, and M. E. Symonds. 2003. Maternal endocrine adaptation throughout pregnancy to nutritional manipulation: consequences for maternal plasma leptin and cortisol and the programming of fetal adipose tissue development. Endocrinology 144:3575-3585.

Boland, M. P. and P. Lonergan. 2005. Effects of nutrition on fertility in dairy cows. Adv. Dairy Techn. 15:19-33.

Boland, M. P., P. Lonergan, and D. O'Callaghan. 2001. Effect of nutrition on endocrine parameters, ovarian physiology, and oocyte and embryo development. Theriogenology 55:1323-1340.

Borowczyk, E., J. S. Caton, D. A. Redmer, J. J. Bilski, R. M. Weigl, K. A. Vonnahme, P. P. Borowicz, J. D. Kirsch, K.C. Kraft, L. P. Reynolds, and A. T. Grazul-Bilska. 2006. Effects of plane of nutrition on in vitro fertilization and early embronic development in sheep. J. Anim. Sci. 84:1593-1599.

Bull, S. and W. E. Carroll. 1937. Principles of Feeding Farm Animals. The MacMillan Company, New York.

Cafe, L. M., D. W. Hennessy, H. Hearnshaw, S. G. Morris, and P. L. Greenwood. 2006a. Influences of nutrition during pregnancy and lactation on birth weights and growth to weaning of calves sired by Piedmontese or Wagyu bulls. Aust. J. Exp. Agric. 46:245-255.

Cafe, L. M., H. Hearnshaw, D. W. Hennessy, and P. L. Greenwood. 2006b. Growth and carcass characteristics at heavy market weights of Wagyu-sired steers following slow or rapid growth to weaning. Aust. J. Exp. Agric. 46:951-955.

Carlson, D. B., J. J. Reed, P. P. Borowicz, J. B. Taylor, L. P. Reynolds, T. L. Neville, D. A. Redmer, K. A. Vonnahme, and J. S. Caton. 2009. Effects of dietary selenium supply and timing of nutrient restriction during gestation on maternal growth and body composition of pregnant adolescent ewes. J. Anim. Sci. 87:669-680.

Caton, J. S. and B. W. Hess. 2010. Maternal plane of nutrition: Impacts on fetal outcomes and postnatal offspring responses. in: Proc. 4th Grazing Livestock Nutrition Conference. West Sect. Am. Soc. Anim. Sci. 104-122.

Caton, J. S., J. J. Reed, R. Aitken, J. S. Milne, P. P. Borowicz, L. P. Reynolds, D. A. Redmer, and J. M. Wallace. 2009. Effects of maternal nutrition and stage of gestation on body weight, visceral organ mass, and indices of jejunal cellularity, proliferation, and vascularity in pregnant ewe lambs. J. Anim. Sci. 87:222-235.

Caton, J., K. Vonnahme, J. Reed, T. Neville, C Effertz, C. Hammer, J. Luther, D. Redmer, and L. Reynolds. 2007. Effects of maternal nutrition on birth weight and postnatal nutrient metabolism. in: Proc. International Symposium on Energy and Protein Metabolism. EAAP Publication 124:101-102.

Clanton, D. C. and D. R. Zimmerman. 1970. Symposium on pasture methods for maximum production in beef cattle: Protein and energy requirements for female beef cattle. J. Anim. Sci. 30:122-132.

Cline, H. B., B. W. Neville, G. P. Lardy, and J. S. Caton. 2009. Influence of advancing season on dietary composition, intake, site of digestion, and microbial efficiency in beef steers grazing a native range in western North Dakota. J. Anim. Sci. 87: 375-383.

Close, W. H. and J. F. Pettigrew. 1990. Mathematical models of sow reproduction. J. Reprod. Fertil. Suppl. 40:83-88.

Cramption, E. W. and L. E. Lloyd. 1959. Fundamentals of Nutrition. W. H. Freeman and Company. San Francisco.

Cundiff, L. V., M. V. Macneil, and K. E. Gregory. 1986. Between- and within-breed analysis of calving and survival to weaning in beef cattle. J. Anim. Sci. 63:27-33.

Dunn, T. G. 1980. Relationship of nutrition to successful embryo transplantation. Theriogenology. 13:27-39.

Fernandez, X. and E. Thornbert. 1991. A review of the causes of variation in muscle glycogen content and ultimate pH in pigs. J. Muscle Foods 2:209-235.

Ford, S. P., B. W. Hess, M. M. Schwope, M. J. Nijland, J. S. Gilbert, K. A. Vonnahme, W. J. Means, H. Han, and P. W. Nathanielsz. 2007. Maternal undernutrition during early to mid- gestation in the ewe results in altered growth, adiposity, and glucose tolerance in male offspring. J. Anim. Sci. 85:1285-1294.

Freetly, H. C., C. L. Ferrell, and T. G. Jenkins. 2000. Timing of realimentation of mature cows that were feed-restricted during pregnancy influences calf birth weights and growth rates. J. Anim. Sci. 78:2790-2796.

Gardner, D. S., K. Tingey, B. W. M. Van Bon, S. E. Ozanne, V. Wilson, J. Dandrea, D. H. Keisler, T. Stephenson, and M. E. Symonds. 2005. Programming of glucose-insulin metabolism in adult sheep after maternal nutrition. Am. J. Physiol. Regul. Integr. Comp. Physiol. 289:R947-R954.

Gardner, B. A., H. G. Dolezal, F. N. Owens, L. L. K. Bryant, J. L. Nelson, B. R. Schutte, and R. A. Smith. 1998. Impact of health on profitability of feedlot steers. in: Oklahoma State University Animal Science Research Report 102-108.

Glore, S.R. and D. K. Layman. 1983. Cellular growth of skeletal muscle in weanling rats during dietary restrictions. Growth 47:403-410.

Gnanalingham, M. G., A. Mostyn, M. E. Symonds, and T. Stephenson. 2005. Ontogeny and nutritional programming of adiposity in sheep: potential role of glucocorticoid action and uncoupling protein-2. Am. J. Physiol. Regul. Integr. Comp. Physiol. 289:R1407-1415.

Godfrey, K. M. and D. J. Barker. 2000. Fetal nutrition and adult disease. Am. J. Clin. Nutr. 71(Suppl):1344S-1352S.

Grazul-Bilska, A. T., E. Borowczyk, W. Arndt, J. Evoniuk, M. O'neil, J. J. Bilski, R. M. Weigl, J. D. Kirsch, K. C. Kraft, K. A. Vonnahme, D. A. Redmer, L. P. Reynolds, and J. S. Caton. 2006. Effects of overnutrition and undernutritionn on invitro fertilization (IVF) and early embryonic development in sheep. Biol. Reprod. 267:131. (Abstr.)

Greenwood, P. L. and L. M. Cafe. 2007. Prenatal and pre-weaning growth and nutrition of cattle: long-term consequences for beef production. Anim. 1:1283-1296.

Greenwood, P. L., A. S. Hunt, J. W. Hermanson, and A. W. Bell. 2000. Effects of birth weight and postnatal nutrition on neonatal sheep: II. Skeletal muscle growth and development. J. Anim. Sci. 78:50-61.

Greenwood, P. L., A. S. Hunt, J. W. Hermanson, and A. W. Bell. 1998. Effects of birth weight and postnatal nutrition on neonatal sheep: I. Body growth and composition, and some aspects of energetic efficiency. J. Anim. Sci. 76:2354-2367.

Hanson, M. A. and P. D. Gluckman. 2005. Developmental processes and the induction and cardiovascular function: conceptual aspects. J. Physiol. 565:27-34.

Johnson, J. A., J. S. Caton, W. W. Poland, D. R. Kirby, and D. V. Dhuyvetter. 1998. Influence of season dietary composition, intake, and digestion by beef steers grazing mixed-grass prairie in western North Dakota. J. Anim. Sci. 76:1683-1690.

Lardy, G. P. and J. S. Caton. 2010. Beef Cattle Nutrition in Commercial Ranching Systems. in: Ruminant Nutrition Production Systems. UNESCO Books. (in press)

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.

Luther, J. S., D. A. Redmer, L. P. Reynolds, and J. M. Wallace. 2005. Nutritional paradigms of ovine fetal growth restriction: implications for human pregnancy. Human Fertil. 8:179-187.

Martin, J. L., K. A. Vonnahme, D. C. Adams, G. P. Lardy, and R. N. Funston. 2007. Effects of dam nutrition on growth and reproductive performance of heifers calves. J. Anim. Sci. 85:841-847.

Maltin, C. A., C. C. Warkup, K. R. Matthews, C. M. Grant, A. D. Porter, and M. I. Delday. 1997. Pig muscle fibre characteristics as a source of variation in meat quality. Meat Sci. 47:237-248.

Maynard, L. A. and J. K. Loosli. 1956. Animal Nutrition. McGraw-Hill Book Company, Inc., New York.

Mersmann, H. J., and S. B. Smith. 2005. Development of white adipose tissue lipid metabolism. in: Biology of Metabolism in Growing Animals. Elsevier, Philadelphia.

Meyer, A. M., J. J. Reed, T. L. Neville, J. B. Taylor, C. J. Hammer, L. P. Reynolds, D. A. Redmer, K. A. Vonnahme, and J. S. Caton. 2010a. Effects of nutritional plane and selenium supply during gestation on ewe and neonatal offspring performance, body composition, and serum selenium. J. Anim. Sci. 88:1786-1800.

Meyer, A. M., J. J. Reed, K. A. Vonnahme, S. A. Soto-Navarro, L. P. Reynolds, S. P. Ford, B. W. Hess, and J. S. Caton. 2010b. Effects of stage of gestation and nutrient restriction during early to mid-gestation on maternal and fetal visceral organ mass and indices of jejunal growth and vascularity in beef cows. J. Anim. Sci. 88:2410-2424.

Monin, G. and A. Ouali. 1992. Muscle differentiation and meat quality. in: Developments in Meat Science. Elsevier Applied Science, London.

Morrison, F. 1949. Feeds and Feeding: A Handbook for the Student and stockman. 21st Edition. The Morrison Publishing Company, Ithaca.

Neville, T. L., J. S. Caton, C. J. Hammer, J. J. Reed, J. S. Luther, J. B. Taylor, D. A. Redmer, L. P. Reynolds, and K. A. Vonnahme. 2010. Ovine offspring growth and diet digestibility are influenced by maternal Se supplementation and nutritional intake level during pregnancy despite a common postnatal diet. J. Anim. Sci. 88:3645-3656.

Nissen, P. M., V. O. Danielson, P. F. Jorgensen, and N. Oksbjerg. 2003. Increased maternal nutrition of sows has no beneficial effects on muscle fiber number or postnatal growth and has no impact on the meat quality of the offspring. J. Anim. Sci. 81:3018-3027.

NRC 1996. Nutrient requirements of beef cattle. 7th Ed. Natl. Acad. Sci. Washington, D.C.

NRC 1985. Nutrient requirements of sheep. Natl. Acad. Sci., Washington, D.C.

NRC 1970. Maternal Nutrition and the Course of Pregnancy. Natl. Acad. Sci.. Washington, D.C.

O'Callaghan, D., H. Yaakub, P. Hyttel, L. J. Spicer, and M. P. Boland. 2000. Effect of nutrition and superovulation on oocyte morphology, follicular fluid composition and systemic hormone concentrations in ewes. J. Reprod. Fertil. 18:303-313.

Odde, K. G. 1988. Survival of the neonatal calf. Vet. Clin. N. Am.: Food Anim. Pract. 4:50-58.

Ouali, A. 1990. Meat tenderization: possible causes and mechanisms. A review. J. Muscle Foods. 1:129-165.

Perry, R. C., L. R. Corah, R. C. Cochran, W. E. Beal, J. S. Stevenson, J. E. Minton, D. D. Simms, and J. R. Brethour. 1991. Influence of dietary energy on follicular development, serum gonadotropins, and first postpartum ovulation in suckled beef cows. J. Anim. Sci. 69:3762-3773.

Reed, J. J., M. A. Ward, K. A. Vonnahme, T. L. Neville, S. L. Julius, P. P. Borowicz, J. B. Taylor, D. A. Redmer, A. T. Grazul-Bilska, L. P. Reynolds, and J. S. Caton. 2007. Effects of Selenium supply and dietary restriction on maternal and fetal body weight, visceral organ mass, cellularity estimates, and jejunal vascularity in pregnant ewe lambs. J. Anim. Sci. 85:2721-2733.

Reed, J. J., A. L. Gevlin, G. P. Lardy, M. L. Bauer, and J. S. Caton. 2006. Effect of creep feed supplementation and season on intake, microbial protein synthesis and efficiency, Ruminal fermentation, digestion, rate of passage and performance in nursing calves grazing native range in southeastern North Dakota. J. Anim Sci. 84:411-423.

Rehfeldt, C., I. Fiedler, and N. C. Stickland. 2004. Number and size of muscle fibers in relation to meat production. in: Muscle Development of Livestock Animals: Physiology, Genetics, and Meat Quality. CAB Int., Wallingford.
Robinson, J. J, K. D. Sinclair, and T. G. McEvoy. 1999. Nutritional effects on foetal growth. J. Anim. Sci. 68:315-331.
Robinson, J. J. 1990. Nutrition in the reproduction of farm animals. Nutr. Res. Rev. 3:961-966.
Sletmoen-Olson, K. E., J. S. Caton, L. P. Reynolds, and K. C. Olson. 2000. Undegraded intake protein supplementation: I. Effects on forage utilization and performance of periparturient beef cows fed low-quality hay during gestation and lactation. J. Anim. Sci. 78:449-455.
Smith, G. C., J. W. Savell, H. G. Dolezal, T. G. Field, D. R Gill, D. B. Griffin, D. S. Hale, J. B. Morgan, S. L. Northcutt, and J. D. Tatum. 1995. The final report of the National Beef Quality Audit. Colorado State Univ. Fort Collins; Texas A&M Univ., College Station; and Oklahoma State Univ., Stillwater, OK. NCA, Eaglewood, CO.
Spitzer, J. C., D. G. Morrison, R. P. Wettemann, and L. C. Faulkner. 1995. Reproductive responses and calf birth and weaning weight as affected by body condition at parturition and postpartum weight gain in primiparous beef cows. J. Anim. Sci. 73:1251-1257.
Stalker, A. L., D. C. Adams, T. J. Klopfenstein, D. M. Feuz, and R. N. Funston. 2006. Effects of pre- and postweaning nutrition on reproduction in spring calving cows and calf feedlot performance. J. Anim. Sci. 84:2582-2589.
Swanson, T. J., C. J. Hammer, J. S. Luther, D. B. Carlson, J. B. Taylor, D. A. Redmer, T. L. Neville, J. J. Reed, L. P. Reynolds, J. S. Caton, and K. A. Vonnahme. 2008. Effects of gestational plane of nutrition and selenium supplementation on mammary development and colostrum quality in pregnant ewe lambs. J. Anim. Sci. 86:2415-2423.
Symonds, M. E., S. Pearce, J. Bispham, D. S. Gardner, and T. Stephenson. 2004. Timing of nutrient restriction and programming of fetal adipose tissue development. Proc. Nutr. Soc. 63:397-403.
Taylor, P. D. and L. Poston. 2007. Developmental programming of obesity in mammals. Exp. Physiol. 92:287-298.
Thomas, V. M. and R. W. Kott. 1995. A review of Montana winter range ewe nutrition research. Sheep and Goat Res. J. 11:17-24.
Wallace, J. M., J. S. Milne, and R. P. Aitken. 2005. The effect of overnourishing singleton-bearing adult ewes on nutrient partitioning to the gravid uterus. Br. J. Nutr. 94:533-539.
Wallace, J. M., D. A. Bourke, P. Da Silva, and R. P. Aitken. 2001. Nutrient partitioning during adolescent pregnancy. Reproduction 122:347-357.
Wallace, J. M., D. A. Bourke, and R. P. Aitken, 1999. Nutrition and fetal growth: paradoxical effects in the overnourished adolescent sheep. J. Reprod. Fertil. 54(Suppl. 1):385-399.
Wallace, J. M, R. P. Aitken, and M. A. Cheyne. 1996. Nutrient partitioning and fetal growth in rapidly growing adolescent ewes. J. Reprod. Fertil. 107:183-190.

Wallace, L. R. 1948. The growth of lambs before and after birth in relation to the level of nutrition. J. Agricult. Sci. 38:243-300.
Ward, S. S. and N. C. Stickland. 1991. Why are slow and fast muscles differentially affected during prenatal undernutrition. Muscle Nerve 14:259-267.
Webb, R., R. G. Gosden, E. E. Telfer, and R. M. Moor. 1999. Factors affecting folliculogenesis in ruminants. Anim. Sci. 68:257-284.
Wu, G., F. W. Bazer, J. M. Wallace, and T. E. Spencer. 2006. Intrauterine growth retardation: implications for the animal sciences. J. Anim. Sci. 84:2316-2337.
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.

SESSION NOTES