

collected from CSSO cows were longer ( $P = 0.04$ ) compared with embryos collected from CSSO cohorts (2.57 vs. 1.15 cm, respectively; SEM = 0.59). In summary, supplementing beef cows with 100 g of CSSO beginning after AI increased CL and embryo development by d 15 of gestation.

**Key Words:** beef cows, Ca salts of soybean oil, embryo, ovary, pregnancy

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## PHYSIOLOGY AND ENDOCRINOLOGY SYMPOSIUM: PRE- AND POST-NATAL IMPACTS ON OFFSPRING PERFORMANCE

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### 1159 Consequences of early nutritional insults on fetal hepatic glucose metabolism and insulin action.

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Pregnancies complicated by placental insufficiency or reduced maternal nutrient supply produce fetuses with intrauterine growth restriction (IUGR). These in utero insults expose the fetus to a reduced supply of glucose, amino acids, and, in some cases, oxygen. The fetus adapts to these reductions in nutrient supply by reducing insulin secretion, increasing counter-regulatory hormone levels, and developing coordinated tissue-specific adaptations in glucose metabolism. Data from our fetal sheep model of IUGR demonstrate an early activation of hepatic glucose production and increased hepatic gluconeogenic gene expression (*PCK1*, *G6PC*) that are sustained during a hyperinsulinemic-euglycemic clamp, thus demonstrating the development of hepatic insulin resistance. This is liver specific insulin resistance because the IUGR fetus has a robust increase in non-hepatic insulin-stimulated glucose utilization in peripheral tissues. While this early activation of glucose production in utero may be an important adaptive response to produce glucose for other glucose-consuming fetal tissues, uncontrolled and dysregulated hepatic glucose production has adverse consequences postnatally and is a major component to diabetes in humans. The early mechanisms driving dysregulated hepatic glucose production and insulin resistance in the fetal liver are not fully understood. We have found that the AKT protein is robustly phosphorylated in the IUGR liver in response to insulin, yet downstream FOXO1 phosphorylation and nuclear localization is increased. We also find that despite decreased nutrient supply, stress signals like AMPK are not increased in the IUGR fetal liver. In addition to increased glucose production, our recent data also demonstrate decreased mitochondrial oxidation in the IUGR fetal liver and a compensatory increase in hepatic glycolysis, intrahepatic lactate production and utilization, and altered substrate preference for reduced hepatic oxidative metabolism. This combination of metabolic adaptations by the fetal liver may be necessary to activate and sustain GPR. However, decreased hepatic mitochondrial function that persists postnatally may underlie

the development of hepatic steatosis in offspring who were IUGR. Overall, understanding the endocrine and molecular pathways responsible for these early metabolic adaptations in the fetus, will allow for development of targeted strategies to improve liver function in the fetus and improve postnatal growth and performance and decrease risk for diabetes and metabolic disease later in life.

**Key Words:** fetus, metabolic adaptations, nutrition

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### 1160 Alterations in uteroplacental hemodynamics during melatonin supplementation in sheep and cattle. C. O. Lemley\*<sup>1</sup> and K. A. Vonnahme<sup>2</sup>, <sup>1</sup>Mississippi State University, Mississippi State, <sup>2</sup>North Dakota State University, Fargo.

Compromised placental function can result in fetal growth restriction which is associated with greater risk of neonatal morbidity and mortality. Large increases in transplacental nutrient and waste exchange, which support the exponential increase in fetal growth during the last half of gestation, are dependent primarily on the rapid growth and vascularization of the uteroplacenta. We are examining maternal nutritional plane along with therapeutic supplements, such as dietary melatonin, which impact placental vascularization, blood flow, and fetal development. Using a mid- to late-gestation ovine model of intrauterine growth restriction ( $n = 31$ ), we examined uteroplacental blood flow and fetal growth during supplementation with 5 mg of dietary melatonin per day. Maternal nutrient restriction decreased uterine artery blood flow, while melatonin supplementation increased umbilical artery blood flow compared to non-supplemented controls. Although melatonin treatment did not rescue fetal weight in nutrient restricted ewes; we did observe disproportionate fetal size and fetal organ development. Moreover, fetal uptake of branched-chain amino acids was partially rescued by dietary melatonin supplementation. Elevated fetal concentrations of melatonin may result in altered blood flow distribution during important time points of development. These specific melatonin responses on umbilical artery hemodynamics and fetal development may be partially mediated through vascular melatonin receptors. Recently, we examined the effects of supplementing Holstein heifers ( $n = 20$ ) with 20 mg of dietary melatonin per day during the last third of gestation. Uterine artery blood flow was increased by 25% and total serum antioxidant capacity was increased by 43% in melatonin supplemented heifers versus non-supplemented controls. In addition, peripheral concentrations of progesterone were decreased in melatonin supplemented heifers vs. non-supplemented controls. Using an in vitro model, melatonin treatment increased the activity of cytochrome P450 2C, a progesterone inactivating enzyme, which was blocked by treatment with the melatonin receptor antagonist, luzindole. Elucidating the consequences of specific therapeutic supplements on the continual plasticity of placental function will allow us to determine the proper timing

and duration for intervention and improvements in offspring growth and development.

**Key Words:** melatonin, umbilical blood flow, uterine blood flow

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**1161 Development of the fetus and fetal reproductive tract in gilts subjected to heat stress from week 4 to 8 of gestation.** C. J. Bernhard\*, T. J. Safranski, M. C. Lucy, W. R. Lamberson, S. G. Moore, L. M. Mayo, and R. Molina-Coto, *University of Missouri, Columbia*.

Gestational heat stress may lead to transgenerational changes in the reproductive capacity of boars and gilts. The objective was to assess fetal and placental development and the development of gonads in conceptuses whose mother was subjected to gestational heat stress (GHS; 28 to 38°C; 65 to 88% relative humidity;  $n = 12$ ) or gestational thermoneutral (GTN; 17 to 22°C; 56 to 65% relative humidity;  $n = 11$ ) conditions during pregnancy. Gilts were housed in the Brody Environmental Chambers from week (wk) 4 to 8 of pregnancy before sacrifice during the eighth wk of gestation for the collection of the reproductive tracts and fetal tissues. During pregnancy, GHS gilts had greater rectal temperature ( $38.5 \pm .04$  vs.  $38.0 \pm .04$ °C;  $P < .001$ ), skin temperature ( $35.5 \pm .2$  vs.  $28.7 \pm .2$ °C;  $P < .001$ ), and respiration rate ( $44.3 \pm 2.6$  vs.  $19.5 \pm 2.7$  breaths per min;  $P < .001$ ) compared with GTN. Sow was the experimental unit for analyses of fetal development. The weight of the pregnant tract ( $12.0 \pm 1.2$  vs.  $12.5 \pm 1.3$  kg), number of viable conceptuses ( $13.8 \pm .8$  vs.  $15.3 \pm .9$ ), the number of non-viable conceptuses ( $.3 \pm .2$  vs.  $.1 \pm .2$ ), the number of mummies ( $.2 \pm .1$  vs.  $.3 \pm .1$ ), and the % survival (number of viable conceptuses/number corpora lutea;  $89 \pm 4$  vs.  $90 \pm 5\%$ ) did not differ ( $P > .10$ ) for GHS vs. GTN (respectively). Upon dissection, the weight of the fetus ( $82.3 \pm 3.6$  vs.  $84.9 \pm 3.8$  g), placenta ( $155.5 \pm 14.7$  vs.  $170.1 \pm 15.6$  g), fetal fluid ( $80.4 \pm 10.0$  vs.  $90.4 \pm 10.6$  g), and placental efficiency (fetal weight/placental weight;  $0.60 \pm .04$  vs.  $0.55 \pm .05$ ) did not differ ( $P > .10$ ) for GHS vs. GTN (respectively). The ratio of male to female fetuses was similar ( $P > .10$ ) for GHS ( $1.3 \pm .3$ ) and GTN ( $1.6 \pm .3$ ). The weight of male fetuses ( $86.2 \pm 3.8$  vs.  $86.4 \pm 4.0$  g), combined testis weight ( $34.2 \pm 1.4$  vs.  $32.8 \pm 1.5$  mg), and combined testis weight as a % of fetal weight ( $.040 \pm .001$  vs.  $.038 \pm .001$ ) did not differ ( $P > .10$ ) for GHS vs. GTN (respectively). The weight of female fetuses ( $81.2 \pm 3.6$  vs.  $83.5 \pm 3.8$  g), combined ovarian weight ( $25.2 \pm 1.0$  vs.  $26.1 \pm 1.1$  mg), and combined ovarian weight as a % of fetal weight ( $.031 \pm .001$  vs.  $.031 \pm .001$ ) did not differ ( $P > .10$ ) for GHS vs. GTN (respectively). The conclusion was that heat stress from wk 4 to 8 of gestation in gilts did not change the growth of the fetus, placenta, ovary, or testis at mid-gestation. Research was supported by the National Pork Board.

**Key Words:** fetal development, gestation, heat stress

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**1162 The effects of under- and over-feeding ewes during gestation on offspring growth and stem cell function.** K. E. Govoni\*, S. A. Reed, M. L. Hoffman, S. M. Pillai, and S. A. Zinn, *Department of Animal Science, University of Connecticut, Storrs*.

Poor maternal nutrition during gestation has been linked to poor growth and development, metabolic dysfunction, impaired health, and reduced productivity of offspring in many species. Poor maternal nutrition can be defined as an excess or restriction of overall nutrients or specific macro- or micro-nutrients in the mother's diet during gestation. Interestingly, there are several reports that both over- and under-feeding during gestation negatively affect offspring postnatal growth with reduced muscle and bone, increased fat, and metabolic dysregulation through reduced leptin and insulin sensitivity. Our laboratory established a model to evaluate both under- and over-feeding during gestation in one population of ewes to evaluate effects on early postnatal growth of offspring. Specifically, ewes were under-fed (60% of NRC for TDN), over-fed (140% of NRC for TDN), or control-fed (100% of NRC for TDN) beginning at  $d 31 \pm 1.3$  of gestation. Blood and tissue samples were collected at birth and 3 mo of age. As previously reported, during the first 3 mo of postnatal growth, over-feeding during gestation increased body size, circulating growth factors and metabolic hormones in offspring. Both under- and over-feeding altered muscle growth, increased lipid content in the muscle and caused changes in expression of myogenic factors. Although the negative effects of poor maternal nutrition on offspring growth have been well characterized in recent years, the mechanisms are not well established. Our laboratory has focused on elucidating these mechanisms by evaluating changes in gene and protein expression and stem cell function. Through RNA-Seq analysis, we observed changes in expression of genes involved in protein synthesis, metabolism, cell function, and signal transduction in muscle tissue. We recently reported that satellite cells, muscle stem cells, have altered expression of myogenic factors in offspring from under-fed mothers. Mesenchymal stem cells, multipotent cells that contribute to development and maintenance of several tissues including bone, muscle and adipose, have a 50% reduction in cell proliferation ( $P < 0.05$ ) and altered metabolism in offspring from both under- and over-fed mothers. These findings suggest that poor maternal nutrition may alter offspring postnatal growth by programming the stem cell populations. In conclusion, poor maternal nutrition during gestation negatively affects offspring postnatal growth, potentially through impaired stem cell function. Therefore, determining the mechanisms that contribute to fetal programming are critical to identifying novel methods to manage these offspring and improve efficiency of production.

**Key Words:** maternal nutrition, sheep, stem cells

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**1163 Postnatal reproductive development and the lactocrine hypothesis.** F. F. Bartol<sup>1</sup>, C. A. Bagnell<sup>2</sup>, and A. F. George<sup>2</sup>, <sup>1</sup>*Auburn University, Auburn, AL*, <sup>2</sup>*Rutgers University, New Brunswick, NJ*.

Maternal contributions to development begin at conception. Prenatal conditions that evolve in utero through the course of gestation define the environment in which embryogenesis and fetoplacental development occur. Genotype notwithstanding, maternal effects on development from the time of conception can program cell fate and dictate offspring phenotype as defined by various aspects of performance and health, including fertility and fecundity. Maternal effects on development do not end at birth, but extend into postnatal life through signals communicated from mother to offspring in first milk (colostrum). Transmission of bioactive factors from mother to offspring as a specific consequence of nursing defines a lactocrine mechanism. The female reproductive tract (FRT) is not fully formed at birth. Data for both ungulate species and mice indicate that disruption of the developmental program during critical organizational periods of neonatal life can have lasting effects on the form and function of FRT tissues, including the uterus. Radial patterning of the uterine wall, reflected by differentiation and proliferation of nascent endometrial glands, is a postnatal event in most mammals. Both uterine growth and histogenesis proceed in an ovary-independent manner shortly after birth, suggesting that extra-ovarian inputs are important in this process. Data for the pig indicate that lactocrine signals constitute one source of such uterotrophic support. Disruption of lactocrine signaling by feeding gilts porcine milk replacer instead of colostrum for 2 d from birth (postnatal day = PND 0) retarded uterine gland genesis by PND 14. Differences in endometrial and whole uterine gene expression patterns between colostrum- and replacer-fed gilts were evident by PND 2, when RNA sequencing revealed over 800 differentially expressed, lactocrine-sensitive genes. Organizationally relevant, lactocrine-sensitive processes, pathways, and networks identified through transcriptomic studies included cell adhesion, cell-cell signaling, cytokine-receptor interactions, integrin cell surface interactions, ESR1 and Hedgehog signaling, and the plasminogen activating network. Lactocrine-sensitive expression of nine microRNAs with 115 potential mRNA targets was also identified. Results provide evidence of lactocrine-mediated, epigenetic effects on multiple elements of the uterine developmental program. A single oral dose of colostrum given at birth affects endometrial cell behaviors associated with uterine wall development by 12 h postnatal. Evidence that minimal colostrum consumption at birth is associated with reduced lifetime fecundity in adult sows indicates that lactocrine programming can affect reproductive efficiency. Data support a role for lactocrine signaling in regulation of postnatal reproductive tract development and function.

**Key Words:** development, lactocrine programming, reproductive tract, uterus

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**1164 Supplementation of corn-dried distiller's grains plus solubles to gestating beef cows fed low quality forage: neonatal calf performance.**

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We have previously reported that corn-dried distiller's grains plus solubles (DDGS) supplementation to low quality forage during late gestation results in a tendency for heavier calves at birth and larger weaning weights compared to no DDGS (CON). To investigate if birth and weaning weight differences were due to metabolite or hormonal status, calf blood samples were collected during early life. Multiparous beef cows ( $n = 27$ ;  $674 \pm 17$  kg) were divided randomly into 2 pens equipped with Insentec feeders to monitor individual intake of corn stover and silage. For 10 wk, both treatment groups were fed the basal diet for ad libitum intake while one group was supplemented (SUP;  $n = 12$ ) with DDGS at 0.3% of BW during the last third of gestation. Following parturition, all cows received the same diet for an additional 8 wk. At calving (0 and 24 h) and weekly for 56 d, blood samples were obtained from calves for analysis of NEFA, urea, glucose, cortisol, thyroxine ( $T_4$ ) and triiodothyronine ( $T_3$ ). From 0 to 24 h, NEFA concentrations did not differ between treatment groups, but were greater at 0 h ( $571$  vs.  $366 \pm 40$   $\mu$ M,  $P < 0.01$ ). Neither urea nor cortisol differed by treatment or hour at 0 and 24 h, however, treatment interacted with time for glucose, where SUP calves had the greatest concentration at 24 h ( $6.83 \pm 0.32$  mM). Glucose decreased from birth onward ( $P < 0.01$ ), with treatments beginning to separate near d 56. Both  $T_3$  and  $T_4$  decreased ( $P < 0.01$ ) from birth to d 56. Neither NEFA nor cortisol differed ( $P \geq 0.95$ ,  $P \geq 0.35$ , respectively) by day or treatment. Urea was influenced by day ( $P = 0.06$ ), but with little overall deviation from concentrations at birth. While there was no impact of diet on dystocia ( $P = 0.39$ ), calves from SUP cows were heavier ( $P = 0.01$ ) at birth than CON calves, which may have impacted their greater glucose concentration at 24 h. It appears that heavier weaning weights observed in calves from SUP cows were not influential on the measured metabolites and hormone profiles at birth and through early life.

**Key Words:** beef cows, metabolites, neonatal life

**1165 The effects of nutritional restriction on endogenous retroviruses and placentation during the first 50 d of gestation in beef heifers.**

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The objectives of this study were to evaluate the effects of maternal nutrient restriction and day of gestation on mRNA expression of *syncytin-Rum1*, bovine endogenous retrovirus K1 (*BERV-K1*), interferon-tau (*INF-τ*), and pregnancy specific protein B (*PSP-B*). At breeding (0 d), crossbred heifers ( $n = 49$ ; ~15 mo of age; initial BW = 324.9 kg) were assigned to dietary treatments, control (fed to gain 0.45 kg/d BW gain) or restricted (60% of control). Heifers were ovariohysterectomized at d 16, 34, or 50 resulting in a 2 × 3 factorial. Non-bred, non-pregnant heifers ( $n = 6$ ; NP), on the control diet, were ovariohysterectomized as baseline controls on d 16 of the estrous cycle. The tissues collected consisted of pregnant horn caruncle (P-CAR), pregnant horn inter-caruncle (P-ICAR), non-pregnant horn caruncle (NP-CAR), non-pregnant horn inter-caruncle (NP-ICAR), and fetal membrane (chorioallantoic; FM). Relative gene expression was calculated using the delta delta Ct method with  $\beta$ -actin as the reference gene and NP as the control tissue. Data were analyzed using PROC GLM of SAS with the model including d of gestation, nutritional treatment, and their interaction. There was significant d of gestation × nutrition interaction for expression of *BERV-K1* in NP-CAR and *INF-τ* in FM while all other interactions were not significant ( $P > 0.08$ ). Expression of *INF-τ* was influenced by d of gestation and nutritional treatment in FM, with d 16 restricted being greatest (5781 fold;  $P < 0.01$ ) followed by d 16 control FM (3324 fold); the remaining d and treatments were not different. In FM, *BERV-K1* was greatest ( $P < 0.01$ ) on d 34 (2961 fold) compared with d 16 and 50 (5 and 1861 fold, respectively). *Syncytin-Rum1* increased ( $P = 0.04$ ) in FM throughout the first 50 d (375 fold) of gestation. *Syncytin-Rum1* expression in P-ICAR was greatest ( $P = 0.01$ ) at d 16; however, *syncytin-Rum1* expression in P-CAR tended ( $P = 0.09$ ) to be greater at d 50. Expression of *PSP-B* increased ( $P < 0.01$ ) throughout early gestation until d 50 in both NP-CAR (316 fold) and P-CAR (18,215 fold). Although nutritional restriction did not influence endogenous retrovirus expression in maternal or fetal tissues, it did influence *INF-τ* expression. These data suggest that both *BERV-K1* and *syncytin-Rum1* may interact with *PSP-B* during the establishment of the fetomaternal interface and syncytial plaques.

**Key Words:** beef heifers, early gestation, endogenous retroviruses, nutrient restriction

**PHYSIOLOGY, ENDOCRINOLOGY,  
AND EXTENSION SYMPOSIUM:  
ENHANCING ADOPTION OF  
REPRODUCTIVE MANAGEMENT TOOLS  
FOR BEEF AND DAIRY PRODUCERS**

**1166 History of the development of the Beef Reproduction Task Force (BRTF) and impacts of the BRTF on beef cattle reproductive management.**

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The Beef Reproduction Task Force (BRTF) was formed during a period of evolving science that resulted in systems to allow producer-acceptable results with a single fixed-timed insemination. The group organized and developed goals to enhance productivity and profitability of U.S. beef herds by integrating research and extension efforts with the intent of more effectively transferring the use of reproductive technologies to the field. A key early step was to coordinate efforts in identifying effective breeding management protocols for beef cattle and to clarify their associated acronyms. A short-list of recommended protocols and their acronyms for synchronization of estrus and ovulation in beef cattle was developed based on results from peer-reviewed, published research and a comprehensive review of data collected from the field. The list of recommended protocols was developed by the BRTF in cooperation with veterinarians and representatives from associated industries. The synergies of this larger industry-centered working group have resulted in ideas for research and broader educational reach. Together the group has planned and hosted 17 in-depth meetings at locations in key cow-calf areas across the country since 2002. These “Applied Reproductive Strategies in Beef Cattle” workshops targeted beef producers, AI industry personnel, veterinarians, allied industry representatives, and academicians. A national media sponsor has provided online coverage of the meetings ([www.appliedreprostrategies.com](http://www.appliedreprostrategies.com)) since 2008. The effectiveness of the team was recognized with the NIFA Partnership Award for