ASAS Western Section Symposium: Perinatal Programming of Offspring Quality 2: Evidence for impacts of maternal nutrition on livestock production

914 Maternal malnutrition induces metabolic reprogramming in offspring. S. P. Ford*[1], L. Zhang[1], L. A. George[1], Y. Ma[1], N. M. Long[1], A. B. Uthlaut[1], and P. W. Nathanielsz[2], [1]*Center for the Study of Fetal Programming, Department of Animal Science, University of Wyoming*, *Laramie,* [2]*Center for Pregnancy and Newborn Research, University of Texas Health Sciences Center*, *San Antonio*.

Evidence suggests that both maternal undernutrition and overnutrition negatively impacts fetal growth and development and postnatal health in mammals. Maternal nutrient restriction from early to midgestation in ewes (NR ewes) results in fetal IUGR (~30% weight reduction) at midgestation compared with ewes fed to NRC requirements (Control, C ewes). If NR ewes are then fed to requirements from mid gestation to term, their offspring are born at weights similar to C ewes, suggesting accelerated fetal growth rate from mid to late gestation. These offspring exhibit increased appetite, insulin resistance and adiposity during post natal development. By 8 mo of age, these NR offspring are heavier, and have increased carcass fat and reduced skeletal muscle mass compared with C offspring. In contrast, if ewes are overfed from 60 d before conception and throughout gestation (obese, OB ewes) their fetuses are macrosomic (enlarged) at midgestation, and then exhibit reduced growth rates from mid-gestation to term. As with lambs from NR ewes, Lambs from OB ewes exhibit birth weights similar to those of ewes fed to requirements. Also similar to NR offspring, OB offspring develop an increased appetite, insulin resistance, and obesity in early adulthood. Interestingly, fetuses gestated by NR and OB ewes experience similar alterations in pancreatic growth and development throughout gestation. At midgestation, both NR and OB fetuses exhibit marked increases in pancreatic β-cell numbers compared with C fetuses, but by late gestation, β-cells numbers have decreased dramatically to reach numbers much lower than that of C fetuses. This talk will compare and contrast the specific prenatal and postnatal impacts of maternal nutrient restriction and overfeeding on conceptus development and offspring health.

Key Words: maternal malnutrition, fetal development, postnatal health

915 Impacts of maternal nutrition in farm animal species on growth characteristics of their offspring. M. Du*, M. J. Zhu, and S. P. Ford, *Department of Animal Science, University of Wyoming*, *Laramie*.

Meat animals spend about one third of their life inside the uterus, and more importantly, all major developmental events are accomplished during the fetal stage. Proper maternal nutrition is crucial for fetal development and the growth characteristics of offspring. Maternal under-nutrition during mid to late gestation reduces birth weight, muscle weight and fatness of offspring at birth, but increases fatness at slaughter, resulting in a permanent impairment of growth performance of offspring. On the other hand, the impact of maternal over-nutrition on fetal development and offspring growth performance is more complicated depending on whether placental function is impaired. If maternal obesity and over-nutrition impairs the function of placenta, the nutrient delivery to fetuses is limited, resulting in poor fetal development and offspring growth characteristics. One the other hand, maternal obesity and over-nutrition without impairment of placental function provides excessive macronutrients to fetuses, which often results in macrosomia and slightly improves the growth performance of offspring. Inflammation associated with maternal obesity may promote adipogenesis (formation of adipocytes) at the expense of myogenesis (formation of muscle cells) and osteogenesis (formation of bone tissue), increasing the fatness but also the marbling of offspring carcasses

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Key Words: maternal nutrition, growth, offspring

916 Maternal nutrition and developmental programming: Impacts on development and function of the gastrointestinal system in offspring. J. S. Caton*, A. M. Meyer, D. A. Redmer, K. A. Vonnahme, and L. P. Reynolds, *Center for Nutrition and Pregnancy, Department of Animal Sciences, North Dakota State University, Fargo*.

Developmental programming is the concept that a perturbation during a sensitive period of development can have lasting consequences in offspring. Maternal nutrition is one of the primary factors impacting growth and development of the gravid uterus, and altered maternal nutrient supply has resulted in compromised offspring. Growth, development, and vascularization of the gastrointestinal tract are often overlooked but are essential processes underlying nutrient uptake and expenditure, immunological competence, neonatal survival, postnatal growth, and metabolic regulation via a cadre of hormones and growth factors. Tissue vascularization is crucial for nutrient transport both to and from the intestine; thus angiogenesis, or the formation of blood vessels, is critical for proper intestinal function. Data indicate that maternal nutrition can alter vascular measurements in late term fetal, early postnatal, and at market weight in ruminant offspring. Additionally, intestinal development during the perinatal period includes growth via cell proliferation, hypertrophy, as well as changes in vascularization. Fetal intestinal growth, inflammatory responses, and/or vascularity, measured near term, have been altered by changes in maternal nutrient supply. In addition, nutrient digestibilities and enzyme activities have been altered in offspring from nutrient compromised dams. Maternal nutritional effects on intestinal vascularity have been accompanied by changes in mRNA expression of vascular endothelial growth factor (VEGF) synthesis, and soluble guanylate cyclase (endothelial nitric oxide, NO receptor), implying possible regulatory roles of VEGF and NO systems. Nutritional modulation or other therapeutics may provide means to stimulate intestinal blood flow and/or angiogenesis, affording potential opportunity to overcome growth or health challenges in compromised ruminants.

Key Words: developmental programming, intestine, maternal nutrition

917 Programming reproductive tract development. F. F. Bartol*[1] and C. A. Bagnell[2], [1]*Auburn University*, *Auburn, AL*, [2]*Rutgers, The State University of New Jersey*, *New Brunswick*.

Female reproductive tract (FRT) development begins prenatally and is completed postnatally. This developmental program is defined by patterns of gene expression in FRT tissues during organizationally critical fetal and perinatal periods. Data for cattle, sheep and pigs show that transient exposure to steroid hormone receptor-modulating agents from birth (postnatal day = PND 0) can disrupt the normal developmental program and alter the developmental trajectory of FRT tissues with lasting consequences. Data for the pig (*Sus scrofa domesticus*) show that postnatal endometrial development and programming are marked by the onset of estrogen receptor (ESR1) expression in nascent glandular

epithelium and stroma by PND 2. Factors affecting ESR1 expression and activation in porcine uterine tissues between birth and PND 14 affect the uterine developmental program and can alter endometrial function and reduce uterine capacity in adults. Maternal effects on neonatal FRT development can be communicated through signals transmitted in milk via a lactocrine mechanism. Studies involving relaxin (RLX), a prototypical milk-borne morphoregulatory factor in the pig, were conducted to test the lactocrine hypothesis for maternal programming of porcine FRT development. Developmental programs were compared for uterine and cervical tissues obtained on PND 2 or PND 14 from gilts allowed to consume colostrum versus those fed a milk replacer from birth, in the presence and absence of exogenous RLX. Results indicated that a lactocrine-driven mechanism, essential to support normal FRT developmental programs, evolves in the uterus and cervix between birth and PND 2. In the absence of lactocrine signaling during this period, normal uterine and cervical ESR1 expression, as well as that of other markers of RLX action and/or estrogen receptor activation including vascular endothelial growth factor-A and matrix metalloproteinase-9, are markedly ($P < 0.01$) reduced by PND 2 and remain low through PND 14, even when replacer-fed gilts are allowed to consume colostrum after PND 2. Lactocrine signaling deserves consideration as an element of the FRT programming equation.

Key Words: female reproductive tract, lactocrine, programming