Meat Science and Muscle Biology Symposium: In Utero Factors that Influence Postnatal Muscle Growth, Carcass Composition, and Meat Quality

376 Fetal programming of skeletal muscle mitochondrial function and insulin sensitivity: Perspectives from non-human primates and mouse models. J. A. Houck[1], K. L. Grove[2], and C. E. McCurdy*[1], *[1]Department of Pediatrics, University of Colorado, [2]National Primate Research Center, Oregon Health and Sciences University.*

Maternal obesity is associated with an increased and earlier risk for the offspring to develop early onset-obesity. In skeletal muscle from either obese adults or lean adult offspring of mothers with type 2 diabetes, reduced mitochondrial density, increased muscle triglyceride accumulation and impaired oxidative function have all been observed and the traits have been linked to skeletal muscle insulin resistance. Accordingly, it has been suggested that the fetal environment may have a profound effect on programming of skeletal muscle metabolism and subsequently have a substantial effect on life-long muscle growth, muscle self-renewal/repair and substrate partitioning. This talk will focus on the influence of maternal diet and obesity on offspring mitochondrial function, fiber typing and insulin sensitivity from both mouse models and a non-human primate model of maternal obesity. Female Japanese Macaques were fed either a control (10% fat) or a high fat/calorie diet (35% fat; HFD) for 2–4 years. Skeletal muscles were collected from fetuses (F-CON or F-HFD) terminated early-3rd trimester. Mitochondrial biogenesis, electron transport activity, and gene expression were analyzed in whole muscle homogenates and isolated mitochondria. Insulin-stimulated 2-deoxyglucose uptake (2DG) was measured in isolated muscle strips from F-CON and F-HFD at sub-maximal [0.3 nM] and maximal [12 nM] doses. Similar studies were performed in mouse models of maternal obesity to investigate post-weaning skeletal muscle mitochondrial development, persistence of insulin resistance and muscle progenitor cell populations. Mitochondrial copy number and citrate synthase activity were significantly reduced in male, but not female, fetal skeletal muscle from F-HFD vs. F-CON. Additionally, key transcriptional regulators of mitochondrial biogenesis, PGC1α, PGC1β, tFAM and Mitofusin-2, were also significantly downregulated in F-HFD. Analysis of maximal mitochondrial complex activity showed significant increase in mitochondrial complex activities and increased ROS damage with maternal obesity. Ex vivo insulin-stimulated 2DG uptake at submaximal and maximal insulin doses was significantly reduced in both male and female rectus femoris and soleus of F-HFD, demonstrating impaired insulin sensitivity and responsiveness that persists at 1 year of age. Additionally in mouse models, we find that maternal obesity leads to a significant shift in the offspring skeletal muscle progenitor cell population favoring adipogenic/fibrogenic precursor cells. Our results demonstrate that the intrauterine environment has profound effects on mitochondrial function and insulin sensitivity in fetal skeletal muscle. Impairments in these 2 key skeletal muscle metabolic functions would undoubtedly predispose the offspring to metabolic complications and poor or impaired muscle growth. It will be essential to determine the extent to which these effects persist in postnatal life. This work was supported by NIH grant K12 HD057022 (CEM) and DK090964 (CEM, KLG).

377 Manipulating mesenchymal progenitor cell differentiation to optimize performance and carcass value of beef cattle. M. Du,* *Department of Animal Sciences, Washington State University, Pullman.*

Beef cattle are raised for their lean, and excessive fat accumulation accounts for large amounts of waste. On the other hand, intramuscular fat or marbling is essential for the palatability of beef. In addition, tender beef is demanded by consumers, and connective tissue contributes to its background toughness. Recent studies show that myocytes, adipocytes and fibroblasts are all derived from a common pool of progenitor cells during the embryonic stage. It appears that during the early developmental stage, multipotent mesenchymal stem cells (MSCs) first diverge into either myogenic or adipogenic/fibrogenic lineage; myogenic progenitor cells further develop into muscle fibers and satellite cells, while adipogenic/fibrogenic lineage cells develop into the stromal-vascular fraction of skeletal muscle, which contains adipocytes, fibroblasts and resident fibro/adipogenic progenitor cells. Strengthening myogenesis (formation of muscle cells) enhances lean growth, promoting intramuscular adipogenesis (formation of fat cells) elevates marbling, and reducing intramuscular fibrogenesis (formation of fibroblasts and synthesis of connective tissue) improves overall tenderness of beef. Nutritional, environmental and genetic factors affect MSC differentiation; however, up to now, our knowledge regarding mechanisms governing MSC differentiation remains rudimentary. Altering MSC differentiation through nutritional management of cows, or fetal programming, is a promising method to improve cattle performance and carcass value.

Key Words: stem cell, myogenesis, adipogenesis

378 In utero nutrition related to fetal development, postnatal growth, and pork quality. N. Oksbjerg,* *Aarhus University-Foulum, Dept. of Food Science, Tjele, Denmark.*

Sub-optimal in utero nutrition gives rise to small birth weights (IUGR) and may cause long lasting effects on postnatal performance and economical loss in meat production. The objectives are to summarize the most important results of our research in this area. Our results show that increased (ad lib) global maternal feed intake in various windows had no main effects on performance and meat quality of the offspring. However, muscle growth rate was reduced in small pigs when the sow was feed ad lib from d 25 to 50 of gestation. On the other hand, when sows were fed a diet containing 30% less protein, the daily gain of the offspring tended to decrease. The meat quality traits, pH_{45} and shear force as well as calpastatin level, were increased and decreased, respectively, in entire male pigs, but not in female pigs following a maternal low protein diet. Comparing small and heavy pigs selected at slaughter at the same age or at birth (IUGR) showed that the small pigs had lower daily gain, fewer muscle fibers, and mean fiber area and increased kg feed/kg gain. Estimated content of DNA/fiber was lowest in the small pigs. Also shear force and calpastatin were increased in pigs born small. When blood was drawn at d 110 of gestation from the umbilicus cord and subjected to NMR metabolomics analyses, it was shown that the smaller fetuses had a lower concentration of glucose and a higher concentration of inositols compared with the heaviest littermates. Proteomic analyses of muscle samples identified 5 proteins which were differentially expressed depending on sex. In conclusion, our results suggest that pigs which were born small had lower performance due to a lower muscle fiber number and satellite cell proliferation which were at least partly due to reduced nutrition. Dietary manipulation of the pregnant sows also programmed the meat quality of the offspring dependent on the sex.

Key Words: maternal nutrition, offspring performance, muscle fiber traits

379 Maternal nutrition on pasture mediates long-term consequences for offspring primarily through effects on growth early in life of beef cattle. P. L. Greenwood,* L. M. Cafe, and D. L. Robinson, *Australian Cooperative Research Centre for Beef Genetic Technologies, and NSW Department of Primary Industries, UNE, Armidale NSW, Australia.*

Long-term consequences for offspring due to dam nutrition, beyond those due to variation in early-life growth were studied. Hereford cows were mated in consecutive years to Piedmontese or Wagyu sires. When confirmed pregnant, cows (n = 513) entered low or high pasture quality and availability treatments until parturition and (or) weaning. At weaning (7 mo), offspring (n = 240) within steer and heifer cohorts were selected for further study into 4 early-life growth groups (Low-Low, Low-High, High-Low and High-High), resulting in multi-model distributions based on maternal nutrition and offspring growth to birth and to weaning. A stepwise regression procedure specified covariates including birth (B) and weaning (W) weight, age at measurement (A), and effects of nutrition during pregnancy (P) and lactation (L), calf sex (S), year (Y), sire breed (G), and first order interactions of factors and with covariates. B was affected by P, and W by L and by P over and above differences due to B. For feedlot entry (26 mo) and exit (30 mo) weights and for carcass weight (C), there were significant linear effects of B and W, and effects of G, S and Y, but no effects of P or L beyond those due to differences in B and W. Average differences of 6 kg in B of calves due to P and 55 kg in W due to L both translated into differences of ~20 kg in C, 11–12 kg in retail yield and 6 kg of fat trim, but did not affect beef tenderness. When adjusted to a constant C, heavier W cattle had more fat trim and less retail yield. There were few interactions however at the same C there was a significant G x W interaction for rib eye area: Piedmontese-sired, Low 95.7 vs. High 88.4 cm^2; Wagyu-sired, both groups 85.7 cm^2. Feedlot feed intake (n = 142) was affected by B and W but not when corrected for weight at start of intake test. FCR was affected by W and L, and whether dams were lactating in early pregnancy. Maternal nutrition did not affect RFI. Overall, there were few long-term influences of maternal nutrition on offspring beyond those related to early-life growth within our pasture-based system. Optimal maternal nutrition to maximize capacity to re-breed has greater economic impact than longer-term consequences for offspring.

Key Words: birth weight, fetal programming, nutrition