569 Growth hormone stimulates liver growth by increasing the size of hepatocytes. D. Jia* and H. Jiang, *Virginia Polytechnic Institute and State University, Blacksburg.*

High levels of growth hormone (GH) are known to cause a disproportional increase in liver weight relative to body weight. The mechanism by which GH stimulates liver growth is not clear. In this study, we determined whether GH stimulates liver growth by increasing the number or the size of hepatocytes. We conducted the study in the lit/lit mouse model. The lit/lit mice lack normal GH production because of a mutation in the growth hormone releasing hormone receptor gene. Lit/lit male mice (n = 6), 12–13 weeks of age, were injected (s.c.) daily with 1 mg/g body weight of recombinant bovine GH or an equal volume of vehicle (0.01 *M* $NaHCO_3$) for 2 weeks. Heterozygous (lit/+) male littermates (n = 6) injected with an equal volume of vehicle (0.01 *M* $NaHCO_3$) were used as normal GH controls. Two hours before euthanasia, mice were injected (i.p.) with 5-bromo-2'-deoxyuridine (BrdU) to label the proliferating cells. Both lit/+ mice injected with $NaHCO_3$ and lit/lit mice injected with GH had greater body weight gains ($P < 0.05$) and greater liver weight/body weight percentages ($P < 0.05$) than lit/lit mice injected with $NaHCO_3$. Based on immunohistochemistry, percentages of BrdU-stained hepatocytes were not different between the 3 groups of mice ($P > 0.10$). However, lit/+ mice injected with $NaHCO_3$ and lit/lit mice injected with GH had 30% and 16% less cells per unit liver area than lit/lit mice injected with $NaHCO_3$ ($P < 0.05$), respectively. Hepatocytes in lit/+ mice injected with $NaHCO_3$ and lit/lit mice injected with GH were 43% and 18% larger than those in lit/lit mice injected with $NaHCO_3$ ($P < 0.05$), respectively. Taken together, these data suggest that GH stimulates liver growth not by increasing the number but by increasing the size of hepatocytes.

Key Words: growth hormone, liver, hepatocyte

570 Insulin and insulin-like growth factor-I (IGF-I) receptor phosphorylation in µ-calpain knockout mice. W. Oliver*[1], A. Chishti[2], and C. Kemp[1], [1]*USDA, ARS, U.S. Meat Animal Research Center, Clay Center, NE,* [2]*Tufts University, Boston, MA.*

Numerous cellular processes are controlled by insulin and IGF-I signaling pathways. Due to previous work in our laboratories, we hypothesized that insulin (IR) and type 1 IGF-I (IGF-IR) receptor signaling is decreased due to increased protein tyrosine phosphatase 1B (PTP1B) activity. C57BL/6J mice heterozygous for the µ-calpain deletion were bred and aged-matched control (C) and µ-calpain knockout (KO) mice were killed at 3, 5, and 10 wk of age (n = 48; 24 females and males per age group). Mice studied at 5 and 10 wk were weaned at 21 d of age, housed individually, and given free access to food and water. Mice were killed via cervical dislocation while fasted or allowed to re-feed for one h before sample collection. Trunk blood was collected for insulin and glucose analysis and hind limb muscles were dissected, pooled, and snap frozen in liquid nitrogen. Skeletal muscle abundance of the IR, IGF-IR, and PTP1B and phosphorylation of the IR and IGF-IR were determined. Activity of PTP1B was also analyzed. Feeding increased insulin levels ($P < 0.01$) and the increase was greater at 10 wk of age compared with 3 and 5 wk of age ($P < 0.03$), regardless of sex and genotype. Serum glucose was unchanged by age ($P > 0.43$), but was higher in males compared with females ($P < 0.05$). Glucose was greater in fed compared with fasted mice at 5 and 10 wk of age ($P < 0.001$), but not at 3 wk of age ($P > 0.62$). At 10 wk of age, the feeding-induced increase in glucose was greater in KO compared with C mice ($P < 0.01$). Total IR and IGF-IR was unaffected by genotype, age, sex, or fed status ($P > 0.49$). The phosphorylation of the IR and IGF-IR was unaffected ($P > 0.33$) by age or sex, but feeding increased ($P < 0.01$) phosphorylation of both receptors. IGF-IR phosphorylation was unaffected by genotype ($P > 0.18$). However, IR phosphorylation was decreased in KO mice ($P < 0.01$). In addition, the protein abundance ($P < 0.04$) and activity ($P < 0.01$) of PTP1B was increased in KO mice. These data indicate that µ-calpain regulates phosphorylation of the IR through changes in the activity of PTP1B, which may have implications in glucose metabolism.

Key Words: µ-calpain, insulin receptor, protein tyrosine phosphatase 1B

571 Ractopamine hydrochloride and estradiol/trenbolone acetate implants alter live performance and carcass components of heifers during the finishing phase. M. A. Jennings*[1], T. R. Young[1], J. T. Cribbs[1], B. C. Bernhard[1], A. D. Hosford[1], T. L. Harris[1], M. J. Anderson[1], G. J. Vogel[2], J. A. Scanga[2], M. F. Miller[1], and B. J. Johnson[1], [1]*Texas Tech University, Lubbock,* [2]*Elanco Animal Health, Greenfield, IN.*

Objectives were to evaluate the interaction of ractopamine hydrochloride (Optaflexx, RH) and timing of terminal implant administration on growth performance, carcass characteristics, and meat quality of finishing heifers. A 2 × 3 factorial complete block design was used with 2 levels of RH and 3 terminal implant windows. British x Continental heifers (n = 216; initial BW = 341.6 kg) were blocked by BW and randomly allotted to 54 pens (9 pens/treatment; 6 pens/block; 4 heifers/pen). Main effects were time of implant [TE-200 with Tylan® (200 mg TBA + 24 mg E_2) administered 140 d from slaughter (TI140); 100 d (TI100); or 60 d (TI60)] and RH (0 or 200 $mghd^{-1}d^{-1}$). Individual BW and DMI were collected at 0, 40, 80, 112, and 140 d. No interactions ($P > 0.10$) between main effects were detected. Average daily gain (0.14 kg/d), predicted carcass ADG (0.24 kg/d), HCW (5.6 kg) were increased ($P < 0.05$) by RH, but DMI was unchanged ($P > 0.10$). Heifers fed RH tended ($P \leq 0.09$) to have a larger LM area (2.45 cm^2 difference) and reduced marbling score. Prime and Choice carcasses were decreased ($P < 0.05$) by 16.5% with RH supplementation. No affect of RH was found on 12th-rib fat and KPH ($P > 0.10$). No differences ($P > 0.10$) in Warner-Bratzler shear force (WBS) were detected at 3, 7, and 21 d aging postmortem however WBS values of RH steaks at 14 d were higher (0.45 kg; $P < 0.05$). From 0 to 40 d, ADG of TI140 (0.34 kg/d) and TI100 (0.18 kg/d) groups was increased ($P < 0.05$) compared with TI60. From 40 to 80 d, TI100 had a greater ADG ($P < 0.05$) than all implant groups. The TI60 had a higher ADG ($P < 0.05$) than TI100 and TI140 from 80 to 112 d. Predicted carcass ADG mirrored live ADG advantages ($P < 0.05$). No differences ($P > 0.10$) in DMI, final BW, carcass characteristics, or WBS values across all aging periods were observed among implant strategies. Results from this study demonstrated that heifers fed RH had increased ADG, carcass ADG, and HCW. Also, this study indicated the terminal implant window, before RH feeding, did not affect performance or carcass quality.

Key Words: anabolic steroid, beef cattle, β-agonist

572 The use of terminal implants and β-agonists to alter blood components and myogenic mRNA and protein levels. T. L. Harris*[1], A. D. Hosford[1], M. A. Jennings[1], M. J. Anderson[1], G. J. Vogel[2], and

B. J. Johnson[1], [1]*Department of Animal and Food Sciences, Texas Tech University, Lubbock,* [2]*Elanco Animal Health, Greenfield, IN.*

Two commonly used growth promotants in the beef industry are β-agonists and anabolic steroid hormones. Each has been shown to increase lean muscle deposition in cattle, but much is unknown on how steroid implants and β-agonists work in combination. We provided a terminal implant [TE-200 with Tylan (200 mg TBA + 24 mg E_2)] to heifers at 140 d (TI140), 100 d (TI100), or 60 d (TI60) from slaughter in a 140 d trial (TI100 and TI60 animals were also implanted on 0 d with Component TE-IH). Cattle were then treated with ractopamine hydrochloride (Optaflexx RH) the final 28 d of the trial. Heifers either received 0 mg/head/d RH or 200 mg/head/d of RH. Five animals/treatment were subjected to longissimus muscle (LM) biopsies on 0, 40, 80, 112 d and at slaughter on 140 d, and were used to isolate mRNA of myogenic related genes and protein quantification of the β_1-adrenergic receptor (β1 AR) and β_2-adrenergic receptor (β2 AR). On the same days, blood samples were taken from 18 animals/treatment to assess changes in plasma blood urea nitrogen (BUN), nonesterified fatty acids (NEFA) and progesterone due to treatments. Relative mRNA levels of myosin heavy chain (MHC) IIX, AMPKα, and IGF-I were increased ($P < 0.05$) in animals receiving TI100 over the other 2 implant dates after RH was fed to animals. After RH administration MHC IIA mRNA levels tended to decrease ($P = 0.09$) due to RH. An interaction between TI d and RH administration caused an increase ($P < 0.05$) in MHC IIA mRNA level in the TI60/RH treatment group over all other treatments except the TI100/no RH treatment group. Protein intensity of the β2 AR was decreased ($P < 0.05$) by the latest TI d (TI60) during RH feeding, while β1 AR protein intensity tended to be lower ($P < 0.10$) in animals fed RH. Plasma urea nitrogen levels were reduced ($P < 0.05$) after terminal implants and RH feeding, while progesterone was decreased ($P < 0.05$) by RH alone, and NEFA levels were unaffected. Treatments were shown to cause a biological response to muscle growth by showing a possible shift toward the more efficient MHC IIX, decreasing plasma urea nitrogen, and increasing β2 AR levels.

Key Words: β-agonist, implant, muscle growth

573 Transcriptional regulation of M. longissimus dorsi during nutritional restriction and compensatory growth in Aberdeen Angus × Holstein Friesian steers. S. M. Keady, A. G. Doran, C. J. Creevey, D. A. Kenny, and S. M. Waters*, *Teagasc, Animal and Bioscience Department, Grange, Dunsany, Co. Meath, Ireland.*

The objective of this study was to examine changes in muscle gene expression of growing steers during a period of dietary energy restriction followed by a period of realimentation. Crossbred Aberdeen Angus × Holstein Friesian (n = 24) steers were assigned to one of 2 feeding treatments. Over a 99-d period, 1 group (n = 12) was offered a high energy control diet consisting of concentrates ad libitum and 7 kg of grass silage per head daily. The second group (n = 12) was offered an energy restricted diet consisting of grass silage ad libitum plus 0.5 kg of concentrate per head daily. From the end of the differential feeding period (99 d), both groups of animals were offered a total mixed ration (grass silage:concentrate ratio of 80:20) for 200 d (viz, the realimentation period). Muscle biopsies were collected at 2 time points (end of the differential feeding period (d 99) and during the realimentation period (d131)). RNA was extracted and the muscle transcriptome was examined using RNaseq. Sequence reads were aligned to the Bovine genome. Differentially expressed genes and over-represented pathways were identified using the DESeq and GOseq respectively. During the differential feeding period, 17 over-represented pathways were identified including the peroxisome proliferator activated receptor signaling, glycolysis/gluconeogenesis and lipid/lipoprotein metabolism pathways indicating reduced energy intake and fat tissue accumulation occurring in muscle tissue during the restriction phase. During the realimentation period, 164 differentially expressed genes were annotated to 9 over-represented pathways including starch and sucrose metabolism, carbohydrate digestion and absorption and TGF-β signaling pathway. It is hypothesized that the signaling effects of the TGF-β pathway were reduced thereby promoting accelerated cell growth and proliferation in muscle tissue of animals experiencing compensatory growth. This information can be exploited in genomic breeding programmes to assist selection of cattle with a greater ability to compensate following a period dietary restriction.

Key Words: ‘ruminant nutrient, muscle, mRNA expression

574 Ruminal and adipose gene expression in beef steers selected for diverse feed intake and gain phenotypes. A. K. Lindholm-Perry*, L. A. Rempel, K. E. Hales, W. T. Oliver, H. C. Freetly, and L. A. Kuehn, *USDA, ARS, U.S. Meat Animal Research Center, Clay Center, NE.*

Limited information exists regarding genes responsible for phenotypic variation in feed efficiency. To determine whether cattle feed intake or growth phenotypes are related to transcript abundance of genes expressed in rumen and adipose, variation in 5 candidate genes from 2 seasons (fall and spring 2012) of steers (n = 32) with differential feed intake and gain phenotypes was examined. Gain and intake were plotted against each other within season and the 4 most extreme animals were selected from each of the 4 Cartesian quadrants relative to the mean of the 2 traits (ADFI: 6.8 to 17.3 kg/d; ADG: 1.0 to 2.4 kg/d). Transcript abundance of candidate genes fatty acid snythase (FASN), fat mass and obesity (FTO) and DNA protein kinase (DNA-PK) in subcutaneous adipose tissue collected near the tailhead and Rho-gamma (RHOG), and protein kinase, AMP-activated, gamma 2 non-catalytic subunit (PRKAG2) in the papillae collected from the cranial sac of the rumen was analyzed. Total RNA was extracted and transcribed into cDNA for use with quantitative real-time PCR. Raw data was normalized with a standard curve generated from a pooled sample from adipose tissue (FASN, FTO, DNA-PK) or was normalized against a housekeeping gene using a pooled sample from rumen tissue (RHOG, PRKAG2). Resulting data were analyzed by season for relationships between transcript level and gain or intake. Relative expression of FASN, FTO and DNA-PK was correlated with each other in adipose tissue (r = 0.63–0.83, $P \leq 0.002$), while rumen expression of RHOG and PRKAG2 was not correlated (r = 0.228, $P = 0.2$). Data were blocked by season to account for diet contribution of either dry rolled corn or high moisture corn. In fall animals on a dry rolled corn diet relative expression of PRKAG2 in the rumen and FTO in adipose was correlated ($P \leq 0.04$) with intake. In spring animals on a high moisture corn diet, RHOG in the rumen was correlated with intake ($P = 0.02$). Variation in response by season is likely due to a change in diet between season affecting rumen function and adipogenesis. USDA is an equal opportunity provider and employer.

Key Words: beef cattle, feed intake, gain

575 Identification of the SH3 and cysteine-rich domain 3 (STAC3) gene as a novel regulator of myogenesis in cattle. Y. Zhang*, X. Ge, D. E. Gerrard, and H. Jiang, *Virginia Polytechnic Institute and State University, Blacksburg.*

Myogenesis is the process of formation of myofibers from myoblasts. The objective of this study was to identify novel regulators of this

process in cattle. We searched the gene expression databases for genes preferentially expressed in skeletal muscle but without known functions. This search led to the identification of the SH3 and cysteine rich domain 3 (STAC3) gene. Through RT-PCR, we confirmed that STAC3 was exclusively expressed in skeletal muscle in adult cattle. We next determined the effect of STAC3 gene knockdown on the differentiation of bovine satellite cells into myotubes in culture. Bovine satellite cells were isolated from adult cattle skeletal muscle by Pronase digestion, expanded in medium containing 10% fetal bovine serum, and were then transfected with STAC3 small interfering RNA (siRNA) or scrambled siRNA. Immediately following the transfection, bovine satellite cells were induced to differentiate into myotubes in medium containing 2% horse serum. At d 3 of differentiation, the cells were stained with Giemsa and 4',6-diamidino-2-phenylindole (DAPI) to qualify fusion rates. In addition, total RNA and total protein were isolated to quantify gene expression. Analyzed by a real-time RT-PCR analysis, STAC3 siRNA caused a 92% reduction in STAC3 mRNA expression compared with scrambled siRNA in bovine satellite cells ($P < 0.01$). Analyzed by a Western blotting analysis, STAC3 siRNA caused a 76% reduction in STAC3 protein expression compared with scrambled siRNA ($P < 0.01$). Of those cells transfected with STAC3 siRNA, 57% formed myotubes, whereas 42% of those transfected with scrambled siRNA formed myotubes by d 3 of differentiation ($P < 0.01$). Furthermore, bovine satellite cells transfected with STAC3 siRNA had greater mRNA expression of myotube markers, myogenin, myosin heavy chain 3, and myosin heavy chain 7, compared with those transfected with scrambled siRNA ($P < 0.01$). These data together suggest that STAC3 is an inhibitory regulator of differentiation of bovine satellite cells.

Key Words: satellite cell, cattle, differentiation

576 Metabolomic profile of the small for gestational age piglet following arginine supplementation. C. M. Getty*, A. A. Baratta, and R. N. Dilger, *University of Illinois, Urbana.*

Large profit losses in the swine industry can be attributed to morbidity and mortality of piglets before weaning, especially in the small for gestational age (SGA) piglet (Sus scrofa). Recent evidence suggests sow's milk contains insufficient concentrations of arginine to support optimal growth and health of piglets. Thus, our objective was to assess global metabolomic profiles and the potential for arginine supplementation to promote growth of SGA (≤0.9kg body weight) and average for gestational age (AGA, 1.3–1.5 kg body weight) piglets. Piglets were selected in littermate pairs at processing to receive either L-arginine (Arg, n = 8) or an isonitrogenous control (L-alanine, Ala, n = 8), weighed daily to assess growth rate, and blood was collected at 15–17 d of age. Overall, differences ($P < 0.05$) were noted between treatments for metabolic pathways involving energy (i.e., TCA cycle), amino acids, nucleotides, and fatty acids. Interestingly, serotonin levels in SGA-Arg piglets were lower than in any other group, which may be explained by altered tryptophan metabolism as compared with the SGA-Ala piglets. Although fatty acid oxidation was higher in SGA piglets, Arg supplementation reduced phosphatidylcholine hydrolysis in the SGA-Arg piglet, which may indicate impaired cell membrane formation. Increased nucleotide turnover, indicating an increase in DNA damage and cell death, was also noted in the SGA piglet. However, Arg supplementation reduced these effects to levels comparable to the AGA piglet. Moreover, changes in glucose metabolism suggested the ability to extract energy from dietary sources may have been compromised in the SGA piglet, but partially rescued by Arg supplementation. In terms of growth, piglets dosed with Arg weighed 22.3% and 12.7% ($P = 0.0032$) less at d 16 compared with Ala-dosed piglets in both the SGA and AGA groups, respectively. We conclude that a reduction in the growth potential of SGA piglets is associated with alterations in multiple metabolic pathways, and further reduction due to Arg supplementation may have resulted from perturbations in phospholipid and tryptophan metabolism.

Key Words: arginine, intrauterine growth restriction, metabolite