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Station Participants:

Joshua Selsby (ISU; Host) – jselsby@iastate.edu
Duane Keisler (Missouri; secretary) - keislerd@missouri.edu
Sarah Reed (UConn) - sarah.reed@uconn.edu
Anna Dilger (Illinios) - adilger2@illinois.edu
Michael Zeece (Nebraska) - michael.zeece@unl.edu
Samer El-Kadi (Virginia Tech) - elkadi@vt.edu
Rob Rhoads (Virginia Tech) - rhoadsr@vt.edu
David Gerrard (Virginia Tech) - dgerrard@vt.edu
Wei Gou (Wyoming) - wguo3@uwyo.edu
Gale Strasburg (Michigan State) - Stragale@anr.msu.edu
William Dayton (Minnesota) - wdayton@umn.edu
Michael E. White (Minnesota) - mwhite@umn.edu
Tracy G. Anthony (Rutgers) - tracy.anthony@rutgers.edu
Shengfa Liao (Mississippi) - sliao@ads.msstate.edu
Tracy L. Scheffler (Florida) - tscheffler@ufl.edu
Daniel Clark for Sandra G. Velleman (Ohio) - velleman.1@osu.edu
Yong soo Kim (Hawaii Station) - ykim@hawaii.edu
Dr. Tizhong Shan (Indiana Station) - tshan@purdue.edu
Deb Hamernik (Administrative Advisor) – dhamernik2@unl.edu

Summary of Minutes of Annual Meeting:

The annual meeting of the NC1184 technical committee meeting was held in the Marion Eugene Ensminger meeting room at Iowa State University on October 24-25, 2014, and was hosted by Dr. Joshua Selsby from the Department of Animal Science, Iowa State University. On October 24th the group was welcomed by Dr. Maynard Hogberg, Professor and Chair of the Department of Animal Science, Iowa State University. Dr. Hogberg shared his welcome with the group and explained the significance of the Marion Eugene Ensminger meeting room and associated gift to the Iowa State University Department of Animal Science. The group then began with oral station reports. A lunch break was then provided by Dr. Selsby and his associates. After lunch Dr. Deb Hamernik (Administrative Advisor) discussed topics for inclusion in the new five-year project. Subsequently a telephone conference call was held with Dr. Mark Mirando, USDA, NIFA, who outlined the current funding situation and gave some statistics on the number of proposals submitted annually and funding rates. Thereafter, a question and answer session was held with Dr. Mirando. The remainder of the day was filled by oral station reports summarizing each station's contributions to the objectives of the NC1184 project. The meeting adjourned and the group met for a dinner and social at the Best Western Hotel in Ames Iowa. The meeting reconvened on October 25th and the remaining stations reported their activities. Following these presentations the meeting adjourned for the year. The 2015 meeting of the NC1184 committee will be hosted by Dr. Duane H. Keisler at the University of Missouri in Columbia Missouri. The group decided that the

2016 meeting of the NC1184 committee will be hosted by Dr. John Gonzalez at Kansas State University.

Accomplishments:

Objective 1: Characterize the signal transduction pathways that regulate skeletal muscle growth and metabolism including the influence of endogenous growth factors and various production practices.

The **Connecticut Station,** the **Illinois station**, the **Missouri station**, the **Mississippi station,** and the **Virginia station** are all working on various aspects of prenatal determinants of muscle growth. More specifically the **Connecticut Station** reported that poor maternal nutrition during gestation can affect pre- and post-natal muscle growth, which contributes to reduced meat production, poor product quality, and increased cost of production in livestock. To characterize the effects of poor maternal nutrition on post-natal muscle growth, pregnant ewes were fed 60%, 100% or 140% of NRC requirements starting at 31 ± 1.4 d of gestation. Lambs were euthanized within 1 d of birth or at 3 mo of age. Muscle was collected from the midpoint of the semitendinosus. At d 1, cross sectional area (CSA) was 47% and 57% greater in OVER and RES, respectively, compared with CON ($P < 0.05$). At 3 mo of age, CSA decreased by 17% and 15% in OVER and RES, respectively, compared with CON ($P < 0.05$). Satellite cells were isolated from the semitendinosus muscle of RES and CON lambs necropsied at 1 d or 3 mo of age and cultured for 24, 48, or 72 h in growth media. At each time point, the percent of total cells immunopositive for Pax7, MyoD, and myogenin was determined. There was no difference in the percent of cells expressing Pax7 at 24, 48 or 72 h. After 24 h of culture, the percent of cells expressing MyoD was 5-fold greater in RES than CON ($P = 0.03$). There was a 3.5-fold increase in the percent of myogenin(+) cells in RES compared with CON after 48 h in culture ($P = 0.04$). After 72 h of culture the percent of myogenin(+) cells in RES was 2-fold less than CON ($P = 0.05$). There were no differences in the percent of Pax7, MyoD or myogenin expressing cells at any time in culture in MPC from 3 mo old lambs ($P > 0.05$). Alterations in the temporal expression of the myogenic regulatory factors are suggestive of precocial differentiation, which may lead to formation of larger and/or fewer muscle fibers.

The **Illinois Station** reported that Insulin-like growth factors (IGF) 1 and 2 regulate prenatal and postnatal skeletal muscle development and growth. While IGF2 is thought to primarily regulate prenatal and not postnatal muscle growth, in swine, a single base pair substitution in intron 3 of IGF2 (g3072A) results in increased lean meat yield in pigs. This SNP disrupts the binding of a transcriptional repressor ZBED6, and lack of that repressor binding is thought to result in increased postnatal IGF2 expression, underlying the phenotype of pigs with a paternal A allele, as IGF2 is maternally imprinted. This suggests that IGF2 may be more involved in postnatal muscle growth than presumed. The Illinois station's ongoing work in this area has established the following new information about IGF2 and other regulators of muscle growth in animals:

1. Increased lean meat yield from pigs with IGF2 paternal A alleles is a result of both increased muscle growth and reduced fat subcutaneous deposition. Lean meat yield at market weight (176 d of age, 127 kg live weight) was increased in pigs with paternal A alleles compared with pigs with paternal G alleles resulting from a reduction in 10^{th} rib back fat depth and an increase in loin eye area at similar hot carcass weights. Body weight was not affected by genotype at any age and muscle weights (longissimus dorsi, semitendinosus and psoas major) were

increased by the paternal A allele only in market weight pigs. Increased muscle weights were not observed at younger ages.

2. Increased expression of IGF2 in pigs with paternal A alleles begins as early as late gestation and continues throughout the production phase of pigs. Though expression of IGF2 was similar between genotypes at d60 of gestation, starting at d 90, IGF2 expression was increased in pigs with the paternal A allele compared with pigs with the paternal G allele. At market weight, IGF2 was increased 4-fold in paternal A compared with paternal G pigs.
3. Muscle fiber number may be increased in pigs with paternal IGF2 A alleles. Despite the increase in postnatal IGF2, muscle fiber cross-sectional area of the semitendinosus tended to be smaller in paternal A compared with paternal G pigs. However, there also tended to be more muscle fibers in paternal A pigs. Therefore, it is possible that increased prenatal IGF2 expression noted at d 90 of gestation leads to greater hyperplasia during muscle fiber formation resulting in increased muscle weights observed at market weights. Increased expression of Myf5 and myogenin at d 90 of gestation supports this hypothesis.
4. The heavily muscled phenotype of myostatin null mice may be partially attributable to increased IGF2 expression but not to changes in IGF1. At d21, 42 and 70 of age, IGF2 expression was increased in myostatin null mice compared with wild type while IGF1 was unchanged. The significance of increased IGF2 expression was confirmed by measuring IGF2 protein by ELISA at d 70; IGF2 protein levels were increased more than 2-fold in myostatin null mice compared with wild type. This increase parallels the 2.9 fold increase in expression of IGF2 at similar ages. Though IGF2 expression was unchanged at birth and 7 d of age, IGF1 expression was actually reduced in myostatin null mice compared with wild type at these ages. Therefore, it is unlikely that IGF1 expression contributes to the hypermuscular phenotype of myostatin null mice.
5. Increased IGF2 expression in myostatin null mice is not variant specific. All three IGF2 variants were increased in concert at both d21 and 70 of age in myostatin null mice compared with wild type.
6. ZBED6, a repressor of IGF2 expression, is likely not responsible for the reduction in postnatal IGF2 expression compared with fetal expression. In both pig and mouse models, the expression of ZBED6 was not increased with age, which would be expected if ZBED6 was responsible for reductions in IGF2 expression. In fact, ZBED6 expression in pigs was greatest in muscle at d60 of gestation.
7. Heterozygous mutations of myostatin are viable in pigs with paternal G alleles for IGF2. Using zinc-finger nuclease technology, the Illinois station has engineered mutant myostatin pigs lacking one nucleotide in exon 3 of the myostatin gene. This mutation results in a premature stop codon and is predicted to result in pigs lacking functional myostatin. This mutation was engineered in a wild-type (paternal G) cell line to prevent interference by the IGF2 paternal A allele. Somatic cell nuclear transfer and embryo transfer resulted in the live birth of 7 cloned gilts, heterozygous for the myostatin mutation. At current, 5 of these gilts are pregnant with offspring sired by IGF2 G/A boars. These pregnancies will result in the F1 generation possessing both heterozygous mutations to allow for further breeding to produce myostatin null pigs of both IGF2 paternal G and IGF2 paternal A genotypes.
8. Changes in myostatin expression in adipose tissue may underlie metabolic changes observed in cats based on photoperiod. Myostatin expression in adipose was increased in short-day housed cats compared with long-day house cats. This accompanied changes in other genes involved in cell division and development and protein turnover. In contrast, short-day housed cats had decreased expression of genes related to immune function. The interaction of adipose

tissue and whole body metabolism in determining the body composition of cats and other animals requires more investigation.

9. Differences in the reductions of growth-regulating genes observed with age do not explain muscle growth differences between sexes or genotypes. A set of 7 genes (*Ezh2, Gpc3, Mdk, Mest, Mycn, Peg3,* and *Plagl1*) had been previously implicated in regulating the growth rates of organs as their expression declines with increasing age and growth. However, the Illinois station has determined that while the expression of these genes does decline with age in the muscles of mice, the rate of that decline did not differ between wild type and more heavily muscle myostatin null mice, nor did it differ between male and female mice.

The **Missouri Station** sought to address the impact of environmental heat stress on: 1) reduced gain and impaired feed efficiency; 2) reduced pork quality; and 3) reduced breeding herd efficiency. Furthermore, the Missouri station examined whether environmental heat stress to pregnant sow leads to permanent imprinting of the gestating offspring in terms of their capacity for growth and reproduction, and also the quality of the pork from their carcasses after slaughter. The permanent effects in utero heat stress are important to understand because alleviating heat stress in the gestating sow is amenable to mitigation using existing technology (fans, misters, evaporative cooling. etc.). The Missouri station's efforts have been to characterize the endocrine profile in the offspring (barrows and gilts) of heat stressed or non-heat stressed sows. The endocrine profiles were examined during the finishing phase of the offspring that were fed either a control diet or a diet supplemented with ractopamine. First parity sows suffer from seasonal infertility that is associated with heat stress (HS). The objective was to test the effects of HS on the metabolic and hormonal profiles during gestation and assess carryover effects after farrowing in first parity sows. Nulliparous gilts (n=23) were brought into environmental chambers, inseminated and assigned to one of two ambient temperature treatments, heat stress (HS; n=12; 27 to 37ºC; relative humidity, RH 85 to 55%) or thermoneutral (TN; n=11; 15 to 20ºC; RH 60 to 50%), that were applied after week (wk) 1 of gestation and continued until wk 16. Sows were moved to a thermoneutral farrowing room (23ºC) during the last wk of gestation (wk 17). Rectal and respiration rate [RR; breaths per minute (BPM)] were collected (0900 and 1500h). Blood was collected monthly during gestation [wk 1, wk 5, wk 9, wk 13, and wk 17; samples (S) S1 to S5], weekly after farrowing (lactation wk 1, wk 2, and wk 3; S6 to S8), and 9 d after weaning (S9). Serum growth hormone (GH), IGF1, insulin, NEFA, and glucose were measured. There was an effect of treatment (P<.001) on RT (38.4±.1 vs. 38.1±.1ºC) and RR (58±2 vs. 30±2 BPM) in gestation (HS vs. TN). Gestation was approximately 2 d shorter for HS compared with TN (P<.05) and HS sows had lighter piglets and lighter placenta at birth and lighter piglets at wk 1 and wk 2 of lactation. Serum metabolite and hormone concentrations changed as sows progressed through gestation, lactation, and weaning. Glucose was low in early gestation, increased in late gestation and early lactation, and then declined (63, 61, 62, 68, 73, 71, 68, 66, and 61 mg/dL; S1 to S9; SEM=2; P<.001). The pattern for serum insulin was similar to glucose (.04, .06, .06, .07, .14, .37, .45, .30, and .15; S1 to S9; SEM=.03; P<.001). NEFA were low until lactation (171, 157, 157, 157, 340, 275, 211, 183, and 140 uEq/L; S1 to S9; SEM=23; P<.001). The pattern for IGF1 differed from insulin because IGF1 progressively decreased for the entire gestation and then increased markedly after farrowing (106, 92, 63, 66, 39, 101, 122, 117, 107 ng/mL; S1 to S9; SEM=7; P<.001). Large changes in IGF1 were not explained by changes in circulating GH perhaps suggesting an uncoupling (during gestation) and then recoupling (during lactation) of the somatotropic axis. HS primarily affected glucose and insulin, causing elevated glucose and insulin after farrowing when no treatment was applied (glucose: 70 vs. 64 mg/dL; SEM=1; P<.01; insulin:

.38 vs. .25; SEM=.03; P<.02; HS vs. TN). Regardless of treatment, there were large changes in hormonal and metabolic profiles. HS affected the profile by causing elevated glucose and insulin concentrations. The carry-over effects of gestational HS on glucose and insulin persisted for at least one month (during lactation and after weaning) when sows were no longer under HS conditions.

The **Hawaii station** is investigating myostatin. Myostatin (MSTN) is a potent negative regulator of skeletal muscle mass. The activity of MSTN is suppressed by MSTN propeptide (MSTNPro), the N-terminal part of unprocessed MSTN that is cleaved off during post-translational MSTN processing. Easy availability of MSTNPro is essential to investigate the potential of the protein as an agent to enhance muscle growth in agricultural animal species. The Hawaii station's objectives were 1) to investigate the minimum size of pig MSTNPro (pMSTNPro) for its full MSTN-inhibitory activity, and 2) to produce pMSTNPro fused to the Fc region of pig immunoglobulin G (pMSTNPro-Fc) in *E. coli* in order to examine its potential as an agent to enhance muscle mass in pigs. Constructs of various truncated *pMSTNPro* and few selected truncated *pMSTNPro-Fc* were separately cloned into pMAL-c5X vector downstream of the *maltose-binding protein* (*MBP*) gene, and were expressed in *E. coli*. MBP-pMSTNPro and MBP-pMSTNPro-Fc proteins were purified by affinity chromatography, then their MSTN-inhibitory activities were examined in an *in vitro* reporter gene (pGL-$(CAGA)_{12}$-luciferase) assay system. The Hawaii station found that all of the four truncated forms of MBP-pMSTNPro proteins (42-218, 42-175, 42-115, and 42-98) were expressed in soluble forms in *E. coli*. Amylose-resin affinity chromatography yielded about 10 mg recombinant proteins per liter culture. When measured by the pGL-$(CAGA)_{12}$-luciferase assay, MSTN-inhibitory capacities of MBP-pMSTNPro-(42-218) and -(42-175) were not different from that of un-truncated form of MBP-pMSTNPro, while MSTN-inhibitory capacities of MBP-pMSTNPro-(42-115) and -(42-98) were significantly lower than that of un-truncated form of MBP-pMSTNPro. This result indicates that the fragment containing amino acid residues 42-175 of pMSTNPro is sufficient for full MSTN inhibitory activity. The MBP-pMSTNPro(42-175)-Fc and MBP-pMSTNPro(42-115)-Fc were also expressed in soluble forms in *E. coli*. Measurement of their MSTN-inhibitory activities demonstrated that the Fc fusion did not alter MSTN inhibitory capacity. Current results show that the production of bioactive porcine MSTNpro/Fc fusion protein is possible in *E. coli*, and it remains to be investigated whether the administration of the pMSTNPro-Fc can improve skeletal muscle growth in pigs via suppression of MSTN activity *in vivo*.

The **Wyoming station's** objective is focusing on molecular signaling mechanisms of muscle protein synthesis and metabolism. Titin, a giant muscle protein, is the "backbone" of the sarcomere. Titin plays a molecular spring-like role in myocyte contractility and integrates diverse myocyte signaling pathways that may regulate protein turnover, hypertrophic activation, and mechanosensing. Titin has three classes of isoforms (N2B, N2BA and N2A). The isoform switching between N2BA and N2B isoforms varies in disease heart, and between N2As in distinct skeletal muscles. However, the regulatory mechanisms of titin isoform switching and the role of titin isoform switching in muscle growth and protein turnover remain elusive. In order to address molecular signaling mechanisms of titin isoform transition, the Wyoming station utilized primary cell cultures and rat models that are lack of RNA binding motif 20 (RBM20), a major regulator of titin isoform transition, and treated these cell and rat models with thyroid hormone (T3). The results demonstrated that T3 can regulate titin isoform switching with RBM20, but cannot without RBM20. PI3K inhibitor LY294002 (LY) can inhibit T3-regulated titin isoform switching in the

presence of RBM20. Western blot indicated that two bands were observed in primary cultures with RBM20. The predicted molecular weight of RBM20 is approximately 135kDa. The size of the upper band was estimated to about 145kDa. The band shifting on gel blot against RBM20 antibody implied that RBM20 is likely posttranslationally modified. The band density analysis also demonstrated that total RBM20 expression was increased with T3 supplementation in the medium. Additionally LY supplementation reduced the total amount of RBM20. These results implied that T3-regulated titin isoform transition could also activate the mTOR pathway and increase gene expression of RBM20. The relative expression of *Rbm20* mRNA was also validated by real-time RT PCR, and the results revealed that the mRNA level was increased with T3 supplementation while it was decreased with LY supplementation. Therefore, the Wyoming station concluded that titin isoform transition triggered by T3 is linked to RBM20 via the PI3K/Akt/mTOR signaling, that is, phosphorylating RBM20 and/or increasing gene expression of RBM20. Next, the Wyoming station will identify the translational modification of RBM20 with mass spectrometry, and also try to understand the regulation of RBM20 gene expression via mTOR pathway. The Wyoming station will also try to find other hormones or growth factors regulating titin isoform transition since titin only expresses larger titin isoform with RBM20 in fetuses, and without RBM20 at all ages.

The **Virginia station** has noted that long term selection for juvenile body size in chickens has created two lines of birds that exhibit divergent postnatal growth patterns. The consequences of the single trait selection on embryogenesis are unresolved. Embryos from the high-weight select (HWS) and low-weight select (LWS) lines were compared at various time points for global growth and developmental differences and possible alterations in myogenesis. Results demonstrate that LWS embryos are larger and form somites more rapidly than HWS embryos. The accelerated rate of embryo growth does not translate to differences in myotome size or muscle gene expression. Muscle progenitor cells were isolated at ED10 from the hindlimbs of HWS and LWS embryos and in vitro parameters of muscle formation were examined. Results find that the HWS muscle cells proliferate to a greater extent and form larger multinucleated myofibers than their LWS counterparts. The cellular basis for disparate myogenic rates is not attributed to different expression of *Pax7*, *Pax3* or the myogenic regulatory factors (*Myf5*, *MyoD*, *MRF4*). The protracted proliferation and differentiation capabilities of the LWS muscle cells may be related to varied expression of the FGF family. FGF2 expression remains elevated during the period of myofiber formation in the LWS and the biphasic expression of FGF4 is distorted in these cells. Summation of these results indicate that selection for reduced postnatal body weight promotes rapid embryogenesis with a reduced rate of fetal myogenesis by comparison to birds selected for a larger postnatal size.

Moreover, the **Virginia station** has noted that recent **s**tudies in mice and zebrafish suggest that the functionally undefined STAC3 gene is specifically expressed in skeletal muscle and plays an important role in skeletal muscle development and contraction. In a recent study, the Virginia station determined the potential role of STAC3 in the proliferation and differentiation of bovine satellite cells. Transfection of bovine satellite cells with STAC3 siRNAs caused a 90% knockdown in STAC3 mRNA expression. Cell proliferation assays revealed that the knockdown did not alter the proliferation rate of bovine satellite cells. Approximately 60% of the cells transfected with STAC3 siRNAs formed myotubes by 72 h of differentiation, whereas that percentage was 40% for those transfected with a negative control siRNA. At 24, 48, and 72 h of differentiation, bovine satellite cells transfected with STAC3 siRNAs had greater mRNA

expression of myogenin, myosin heavy chain 3, and myosin heavy chain 7 than those transfected with negative control siRNA. These results suggest that the STAC3 gene is a negative regulator of the differentiation of bovine satellite cells into myotubes. Similar results were obtained when STAC3 mRNA was knocked down in C2C12 myoblasts. However, STAC3 mRNA expression was increased during myotube differentiation of both bovine satellite cells and C2C12 myoblasts. This increase suggests that STAC3 might have different functions in myotubes than in satellite cells or myoblasts.
Finally, the **Virginia station** noted that metabolic disorders are one of the key factors that trigger the development of cardiovascular diseases and type 2 diabetes, yet the fundamental pathogenic factors and driving regulatory network remain poorly defined. Skeletal muscle accounts for 70-90% of insulin-stimulated glucose disposal, however, studies of the nutrient responsive signaling pathways in skeletal muscle and their contribution to the maintenance of local and systemic metabolic homeostasis are rare. The Virginia station has shown the functional role of O-linked β-D-N-acetylglucosamine (O-GlcNAc) signaling, a nutrient sensor pathway that links cellular protein, carbohydrate, lipid, energy, and nucleotide metabolism, in skeletal muscle and systemic metabolic homeostasis. The Virginia station has generated knockout mice lacking O-GlcNAc transferase (OGT) specifically in skeletal muscle and found that OGT deficient mice exhibit reduced fat deposition, are resistant to high fat diet induced obesity, and have improved systemic insulin sensitivity.

The **Kansas Station** has continued to characterize cellular and molecular events that regulate postnatal muscle growth and composition. Within the past year, two studies were conducted at the K-State . The first study involved isolation of satellite cells from post mortem porcine skeletal muscle. The current dogma suggests that viable satellite cells cannot be readily isolated from skeletal muscle after extended post-mortem storage. This complicates study design requiring special accommodations to collect tissue samples shortly after slaughter in commercial abattoirs. The ability to isolate progenitor cells from samples that have gone through slaughter and fabrication would aid greatly in alleviating this logistical barrier facilitating novel studies and approaches to understanding postnatal satellite cell activity. Recently, satellite cells have been isolated from humans and mice up to 17 and 14 days post mortem, respectively. The Kansas Station sought to investigate the amount and viability of porcine satellite cells isolated from loin muscles at 1, 24, and 72 hours post mortem. The Kansas Station observed decreases in cell yield of 55% and 66% at 24 and 72 hours, respectively. However, as time post-mortem increased the percentage of cells expressing Pax7 and Myf5 increased. The second study examined nutrient gene interactions in adipose tissue. In this study, iodine value (IV), a measure of fatty acid (FA) unsaturation, is currently the industry standard for assessing pork fat quality. However, variation exists between the anatomical locations of the fat depots with respect to IV. Additionally, variation exists in the response of these depots to the iodine value product (IVP; IV of feed × percentage of fat in feed) of dietary feedstuffs. In addition to their direct impact, the composition of dietary fats can affect endogenous FA synthesis which contributes to the ultimate IV of AT depots. A total of 160 finishing pigs (PIC 327 × 1050; initially 45.4 kg) were used in an 84-d experiment to evaluate the effects of dietary fat source and feeding duration on growth performance, carcass characteristics, lipogenic gene expression, and carcass fat quality in swine. Dietary treatments were a corn-soybean meal control diet with no added fat or a 3 × 3 factorial arrangement of treatments with main effects of fat source (4% tallow, 4% soybean oil, or a blend of 2% tallow and 2% soybean oil (blend) and feeding duration (d 0 to 42, 42 to 84, or 0 to 84). On d 0, 41, and 81, one pig from each pen was selected for backfat, belly, and jowl biopsy collection.

Added fat increased ($P < 0.05$) IV compared to control diets. Across all three depots a duration × fat source interaction ($P < 0.05$) showed increased poly unsaturated fatty acids (**PUFA**) and decreased mono unsaturated fatty acids (**MUFA**) with added dietary fat. For backfat there was a feeding period × fat source interaction ($P = 0.05$) for blend and soy bean oil where feeding soy bean oil in period 2 increased *sterol regulatory element binding protein-1c* mRNA expression. In belly samples, *fatty acid synthase* and *acetyl-CoA carboxylase* (**ACC**) mRNA expression were increased ($P < 0.05$) in control versus added fat diets. In jowl samples, added fat in period 1 increased *ACC* and *peroxisome proliferator activated receptor gamma* mRNA expression compared to period 2 ($P < 0.05$). Control diets resulted in decreased ($P < 0.05$) d 42 *acyl-CoA oxidase* expression compared to diets with added fat. In conclusion feeding soybean oil negatively impacts the fatty acid composition in terms of IV in finishing pigs.

The **New Jersey (Rutgers) Station** reports that phytoecdysteroids such as 20 hydroxyecdysone (20HE) are ingredients in various nutritional supplements marketed to enhance physical performance and lean body mass. I*n vitro* studies suggest 20HE may activate protein synthesis via a phosphoinositide-3-kinase-Akt signaling mechanism. To confirm these findings *in vivo* and evaluate downstream activation of mammalian target of rapamycin complex 1 (mTORC1) signaling, overnight fasted 4-8 week old male rats (n=5-6 per group) were randomized to one of three study designs: 1) gavaged with 0, 10, 50, 200 mg/kg 20HE and euthanized 30 min post-gavage; 2) gavaged with 200 mg/kg 20HE or excipient and euthanized at 30 min, 60 min, 120 min, 240 min post-gavage, and 3) administered excipient or 200 mg/kg 20HE alone or in combination with 1.35 g/kg L-leucine and euthanized 30 min post-gavage. Independent evaluation of 20HE confirmed purity as reported by Sigma before solubilization in 3% DMSO or 70% Labrasol® as excipient. Phosphorylation of Akt at Thr308 and Ser473, mTOR at Ser2448, ribosomal S6 kinase (S6K1) at Thr389, and eukaryotic initiation factor 4E binding protein 1 (4E-BP1) at Thr 37/46 were evaluated by immunoblot. Leucine administered in 3% DMSO or Labrasol robustly increased phosphorylation of mTOR and 4E-BP1 but not Akt in muscle and liver. In contrast, at all doses and times, 20HE did not significantly increase phosphorylation of Akt, mTOR or 4E-BP1 as compared to excipient controls. Furthermore, oral ingestion of 20HE led to measurable levels in the blood but not dose-reactive dampened mTORC1 signaling by leucine in both muscle and liver. These data do not support the idea that anabolic effects of 20HE are acutely mediated by mTORC1 and do not support the supplementation of such components in the feed of animals to promote muscle growth.

The **Iowa station** reported that that activation of the PGC-1α pathway via gene transfer decreased muscle injury in dystrophic skeletal muscle using a mouse model of Duchenne muscular dystrophy. To make these observations translatable to humans the Iowa station used quercetin, an orally available and safe PGC-1α activator. The Iowa station found that a 0.2% quercetin diet decreased muscle damage in the diaphragm and the heart. Their next step was a long term experiment where mdx mice were fed a quercetin enriched diet for 12 months. The full data set has not yet been analyzed statistically. The Iowa station found that quercetin treated mdx mice were more active than untreated mice and even more active than healthy mice while untreated mdx mice were less active than both healthy and quercetin-treated mdx mice. This was a surprising result and may suggest these mice are more active because they have the capacity to be more active. Alternatively, it may suggest that quercetin is driving activity through some unknown mechanism. In vivo respiratory function was also measured during the 12 month period using whole body plethysmography. Descriptively, the mdx mice performed worse than the C57 mice,

as expected. The quercetin treatment provided clear benefits through approximately 8 months of age and then respiratory function appeared become quercetin insensitive. It is unclear if this is a result of a negative feedback system where the pathway driven by quercetin was deactivated and led to reduced muscle function or if quercetin only slowed disease progression and by 8 months of age dystrophic muscle had accumulated sufficient injury so as to impair muscle function. Regardless, muscle function in treated mice, albeit clearly declining, was still better than untreated mdx mice for most measures. More importantly, quercetin prevented cardiac failure in dystrophic hearts. This latter point is critical because heart failure is rapidly becoming the leading cause of death in these patients.

The **Minnesota station** has examined the mechanism of Estradiol-17β (E2) enhanced muscle growth in feedlot steers. In feedlot steers, estradiol-17β (E2) and combined E2 and trenbolone acetate (TBA) (a testosterone analog) implants enhance rate and efficiency of muscle growth; and, consequently, these compounds are widely used as growth promoters. Although the positive effects of E2 on rate and efficiency of bovine muscle growth are well established, the mechanisms involved in these effects are not well understood. Combined E2/TBA implants result in significantly increased muscle satellite cell number in feedlot steers. Additionally, E2 treatment stimulates proliferation of cultured bovine satellite cells (BSC). Studies in nonmuscle cells have shown that binding of E2 to G protein-coupled estrogen receptor (GPER)-1 results in activation of matrix metalloproteinases 2 and 9 (MMP2/9) resulting in proteolytic release of heparin binding epidermal growth factor-like growth factor (hbEGF) from the cell surface. Released hbEGF binds to and activates the epidermal growth factor receptor (EGFR) resulting in increased proliferation. In order to assess if GPER-1, MMP2/9 and/or hbEGF are involved in the mechanism of E2-stimulated BSC proliferation, we have examined the effects of G36 (a specific inhibitor of GPER-1), CRM197 (a specific inhibitor of hbEGF), and MMP-2/MMP-9 Inhibitor II (an inhibitor of MMP2/9 activity) on E2-stimulated BSC proliferation. Inhibition of GPER-1, MMP2/9 or hbEGF suppresses E2-stimulated BSC proliferation ($P < 0.001$) suggesting that all of these are required in order for E2 to stimulate BSC proliferation. These results strongly suggest that E2 may stimulate BSC proliferation by binding to GPER-1 resulting in MMP2/9-catalyzed release of cell membrane-bound hbEGF and subsequent activation of EGFR by binding of released hbEGF. Additionally, treatment of BSC cultures with AG1478 (a specific inhibitor of EGFR tyrosine kinase activity) suppresses E2-stimulated BSC proliferation ($P < 0.05$). Additionally, E2-stimulated proliferation is completely suppressed ($P < 0.05$) in BSC in which EGFR expression is silenced by treatment with EGFR small interfering RNA (siRNA). These results indicate that EGFR is required in order for E2 to stimulate proliferation in BSC cultures. Both AG1478 treatment and EGFR silencing also suppress LR3-IGF-1- (an IGF1 analogue that binds normally to the IGFR-1 but has little or no affinity for IGF binding proteins) stimulated proliferation in cultured BSC ($P < 0.05$). Even though EGFR siRNA treatment has no effect on IGFR-1β mRNA expression in 2 cultured BSC, IGFR-1β protein level is substantially reduced in BSC treated with EGFR siRNA. These data suggest that EGFR silencing results in post transcriptional modifications that result in decreased IGFR-1β protein levels. Although it is clear that functional EGFR is necessary for E2 stimulated proliferation of BSC, the role of EGFR is not clear. Transactivation of EGFR may directly stimulate proliferation or EGFR may function to maintain the level of IGFR-1β which is necessary for E2 stimulated proliferation. It also is possible that the role of EGFR in E2-stimulated BSC proliferation may involve both of these mechanisms.

The **Mississippi station** has conducted two experiments collaboratively with Dr. Kehe Huang in China to study the effects of yeast-culture-based feed additives on animal growth performance. Furthermore, animal trials using finishing pigs were conducted at Mississippi State University to study the effects of dietary lysine on the plasma concentrations of amino acids, some metabolites and hormones, and the skeletal muscle gene expression profile. The Mississippi station collaborative project with China found that dietary supplementation of a Se-enriched probiotic can enhance the antioxidative capacity, enhance the thyroid function, produce a healthy gastrointestinal ecosystem, and improve the growth performance of post-weanling piglets raised under high ambient temperature. The Mississippi station project conducted in Mississippi showed 5 patterns of plasma amino acid concentration change: (1) The plasma lysine concentration was positively correlated to the dietary lysine concentration, (2) the plasma asparagine concentration in the lysine deficient or adequate group was lower than that in the lysine excess group, (3) the plasma concentration of alanine, glutamic acid, glycine, or leucine in the lysine deficient group was respectively lower than that in the lysine adequate or excess group, (4) the plasma concentration of arginine, histidine, citruline, threonine, valnine, isoleucine, or phenylalanine in the lysine deficient group was respectively higher than that in the lysine adequate or excess group, and (5) there were no differences in the plasma concentration of aspartic acid, β-alanine, cystine, glutamine, methionine, ornithine, proline, serine, taurine, tryptophan, or tyrosine among three treatment groups. These results indicated a complex metabolic relationship among amino acids in finishing pigs, and why a single dietary lysine concentration change affects the plasma concentrations of the other 12 amino acids is being analyzed. The Mississippi station is currently studying the effect of dietary lysine on the plasma concentrations of selected metabolites and the skeletal muscle gene expression profile.

The **North Carolina station** has reported that loss of muscle mass, or sarcopenia, is nearly universal in cirrhosis and adversely affects patient outcome. The underlying cross-talk between the liver and skeletal muscle mediating sarcopenia is not well understood. Hyperammonemia is a consistent abnormality in cirrhosis due to impaired hepatic detoxification to urea. The North Carolina station observed elevated levels of ammonia in both plasma samples and skeletal muscle biopsies from cirrhotic patients compared with healthy controls. Furthermore, skeletal muscle from cirrhotics had increased expression of myostatin, a known inhibitor of skeletal muscle accretion and growth. In vivo studies in mice showed that hyperammonemia reduced muscle mass and strength and increased myostatin expression in wild-type compared with postdevelopmental myostatin knockout mice. The North Carolina station postulated that hyperammonemia is an underlying link between hepatic dysfunction in cirrhosis and skeletal muscle loss. Therefore, murine C2C12 myotubes were treated with ammonium acetate resulting in intracellular concentrations similar to those in cirrhotic muscle. In this system, the North Carolina station demonstrated that hyperammonemia stimulated myostatin expression in a NF-κB-dependent manner. This finding was also observed in primary murine muscle cell cultures. Hyperammonemia triggered activation of IκB kinase, NF-κB nuclear translocation, binding of the NF-κB p65 subunit to specific sites within the myostatin promoter, and stimulation of myostatin gene transcription. Pharmacologic inhibition or gene silencing of NF-κB abolished myostatin up-regulation under conditions of hyperammonemia. The North Carolina station work provides unique insights into hyperammonemia-induced myostatin expression and suggests a mechanism by which sarcopenia develops in cirrhotic patients.

The **Michigan station** has continued studies on molecular mechanisms responsible for development of pale, soft, exudative turkey meat. Deep transcriptome RNA sequence analysis was used analyzed differential gene expression in the two categories of meat. Turkey breasts (n = 43) were previously classified as normal or PSE using marinade uptake as an indicator of quality (high = normal; low = PSE). Total RNA from breast muscle samples with the highest (n = 4) and lowest (n = 4) marinade uptake were isolated and sequenced using the Illumina GA(IIX) platform. The results indicated differential expression of 494 loci (false discovery rate < 0.05). Changes in gene expression were confirmed using quantitative real-time PCR. Pathway analysis of differentially expressed genes suggested abnormalities of calcium homeostasis and signaling pathways regulating actin cytoskeleton structure as well as carbohydrate metabolism and energy production in PSE samples. Dysregulation of postmortem glucose oxidation in PSE turkey was suggested by both dramatic downregulation of pyruvate dehydrogenase kinase, isozyme 4 (PDK4) mRNA, the most downregulated gene, and a decrease in the protein product (P = 0.0007) as determined by immunoblot analysis. These results support the hypothesis that differential expression of several genes and their protein products contribute to development of PSE turkey meat.

Objective 2: Characterize the cellular and molecular basis of myogenesis.

The **Virginia Station** has continued to focus on heat stress and immune challenge as costly issues to the swine industry causing significant loss in production and health including reduced efficiency in muscle accretion and energy utilization. Alterations to metabolism and immune response may participate in these shortcomings. The study objectives were to examine the metabolic profiles and immune status of swine subjected to a dual challenge of thermal stress and porcine reproductive and respiratory syndrome virus (PRRSV). To determine this, pigs were subjected to four treatments: thermoneutral (22° C; TN), thermo-neutral PRRSV infected (TP), heat stress (HS), and heat stress PRRSV infected (HP), during two experimental phases. The first phase consisted of infecting half the experimental group with PRRSV while the rest remained infection free in thermo-neutral conditions. A second phase further divided infected and non-infected into heated conditions for three days of constant heat (35° C) or TN conditions. Blood was collected prior to each phase and before sacrifice to analyze for metabolites. At sacrifice liver and longissimus dorsi skeletal muscle samples were collected for molecular and metabolic analyses. Pigs in challenged conditions had increased body temperatures, reduced feed intake and body weights compared to controls, with greatest detriment to dual challenged pigs. In addition, challenged pigs had increased markers of muscle degradation. In challenged pigs, differences ($p<0.05$) were observed in the metabolic and cytokine gene expression profiles suggesting heat stress blunts the immune response of viral infection in muscle and liver. In conclusion, heat stress and immune challenge directly and indirectly affect metabolism and cytokine expression and both variables may contribute to decreased growth parameters.

The **Virginia Station** has also pursued the effects of intrauterine growth restriction (IUGR) on changes in body composition and meat quality, reduced growth performance, increased morbidity and mortality, and abbreviated muscle development. The aim of this study was investigate how IUGR alters muscle protein synthesis and degradation. Birth weight was used as the criterion for selection, with normal birth weight (NBWT) and IUGR pigs considered within ±0.5 and – 2 standard deviations below litter average. All animals used in this protocol were litter-mates and sex matched. Pigs in NBWT and IUGR groups were fed for 21 days (5 meals/d) equivalent

amounts (ml•kg^{-1} body weight•d^{-1}) either a protein adequate formula resembling sow milk (NBWT, IUGR; 5.1% crude protein, 4,790 kJ/kg), and pigs in the third group were fed the same protein adequate diet supplemented (IUGRAA) with 0.65% branched-chain amino acids (1:0.65:0.55 leucine: valine: isoleucine). At 14 days of age, piglets were surgically fitted with indwelling catheters into the jugular vein, carotid artery, and inferior vena cava using sterile techniques. During the last 8h of the 21 d feeding period, pigs were infused with [$^{2}H_5$]phenylalanine and [$^{2}H_2$]tyrosine and amino acid net balances were measured across the hindquarters for the last 4h. At the end of tracer infusion pigs were fed and sacrificed 60 minutes later. Weight gain was greater for NBWT than for IUGR and IUGRAA pigs and resulted in heavier body weights throughout the study (P<0.05). Insulin and blood IGF-1 concentrations were not different among the three groups. Branched chain amino acid supplementation increased plasma concentrations (P<0.05), however there was no effect of supplementation on protein turnover. Protein synthesis was stimulated following a meal in all three groups and was significantly higher in NBWT compared to IUGR and IUGRAA pigs (P<0.05), while there was no temporal effect on protein degradation. This resulted in a higher protein deposition rate in NBWT compared to IUGR and IUGRAA pigs (P<0.05). Work is ongoing to examine the differences in protein synthesis signaling in skeletal muscles of IUGR and NBWT pigs.

The **Connecticut Station** reported that an important issue in maintaining muscle mass is the prevention of atrophy during periods of disease or disuse. IL-6 has multiple effects on satellite cell activity, depending on the context and environment in which expression is increased. C26 colon carcinoma induces IL-6 dependent muscle atrophy. To determine if this environment inhibits satellite cell function, the tibialis anterior (TA) of tumor bearing animals was injured via cardiotoxin injection two weeks after inoculation with C26 cells. A subset of animals was treated with an IL-6 receptor antagonist to inhibit IL-6 signaling. The cross sectional area (CSA) of uninjured TA muscles in tumor bearing was smaller than uninjured control muscles by 21 days after C26 inoculation ($P < 0.05$). Further, both seven and ten days following injury the CSA of injured muscles from TB animals was decreased compared to that of injured muscle from healthy animals, indicating a deficit in muscle regeneration ($P < 0.05$). While there was no difference in the number of Pax7+ cells in non-regenerating muscle from control and tumor bearing animals, there were fewer Pax7+ and MyoD+ cells in tumor bearing animals at both 3 and 7 days after injury ($P < 0.05$). Inhibition of IL-6 signaling did not alter Pax7 or MyoD mRNA expression in healthy or regenerating muscle in control or TB animals. These data suggest that cachexia inhibits muscle regeneration and satellite cell function during muscle regeneration. Ongoing work is investigating the effect of the IL-6 receptor antagonist on the activation and proliferation of satellite cells during muscle regeneration in tumor bearing animals.

The **Texas Station** has utilized a unique cattle population to investigate differential gene expression and signal transduction in skeletal muscle resulting from inheritance of *Bos taurus* or *Bos indicus* alleles of genes associated with skeletal muscle growth, development and proteolysis. One objective of this project is to identify networks of genes in cattle that are critical for production of consistently tender and highly palatable beef, and for the effectiveness of electrical stimulation of carcasses. The Texas station's second objective is to integrate gene expression phenotype (microarray data) with SNP array data to refine QTL, and identify distinct genetic contributions that distinguish *Bos indicus* from *Bos taurus* cattle to influence tenderness and other meat quality traits. The Texas station is also interested in identifying genetic factors that are important for growth efficiency. Emphasis has been placed on identifying regions of the genome

that harbor genes for traits that directly impact the consumer, such as marbling and tenderness. For mapping genes associated with production efficiency and nutrient utilization, a resource population of multiple F_2 families of Nellore-Angus, Brahman-Angus, and Brahman-Hereford was generated at the Research Station in McGregor, TX. DNA has been collected for all animals, and phenotype has been scored for multiple characteristics including disposition, feed intake, age at puberty, and carcass and meat traits of steers. Current work examines non-coding RNA influence on these phenotypes. The Outputs: Network gene expression analyses combined with genotype and other expression analyses resulted in the identification of additional genes that affect shear force in beef.

The **Ohio station's** current research efforts have focused on understanding the mechanism of how the heparan sulfate family of proteoglycans (specifically the syndecan and glypican families) may be involved in the regulation of muscle growth properties. Fibroblast growth factor 2 (FGF2) is a potent stimulator of muscle cell proliferation and a strong inhibitor of muscle cell differentiation. Heparan sulfate proteoglycans function as a low affinity receptor for FGF2 thus permitting a high affinity interaction of FGF2 with its receptor. The work for this year summarizes progress in determining the mechanisms of syndecan-4 and glypican-1 in regulating muscle growth properties mediated by the adult myoblast or satellite cell population. This research was done in collaboration with the South Dakota Station (Dr. Doug McFarland). More specifically, syndecan-4 core protein is composed of extracellular, transmembrane, and cytoplasmic domains. Syndecan-4 is a cell membrane-associated heparan sulfate proteoglycan that forms oligomers in muscle satellite cells. The syndecan-4 oligomers activate protein kinase Cα (PKCα) through the syndecan-4 cytoplasmic domain and may regulate the activation of ras homolog gene family member A (RhoA), a signal transduction molecule down-stream of PKCα which is thought to influence cell migration. However, little is known about the function of the syndecan-4 cytoplasmic domain in satellite cell migration and RhoA activation. To study the function of the syndecan-4 cytoplasmic domain, clones of syndecan-4 and syndecan-4 without the cytoplasmic domain (S4C) were used in over-expression studies, and small interference RNAs targeting syndecan-4 or RhoA were used in knockdown studies. Satellite cell migration was increased by syndecan-4 over-expression but decreased by the knockdown or deletion of the syndecan-4 cytoplasmic domain. The RhoA protein was activated by the over-expression of syndecan-4, but not with the deletion of the syndecan-4 cytoplasmic domain. Co-transfection (syndecan-4 overexpression and RhoA knockdown) and rescue (the knockdown of syndecan-4 and the treatment with Rho activator II) studies demonstrated that S4 mediated satellite cell migration was regulated through the activation of RhoA. The cytoplasmic domain of syndecan-4 is required for cell migration and RhoA activation which will affect muscle fiber formation. Alternatively, glypican-1 is a cell membrane heparan sulfate proteoglycan composed of a core protein with covalently attached glycosaminoglycan, and N-linked glycosylated (N-glycosylated) chains, and is attached to the cell membrane by a glycosylphosphatidylinositol (GPI) linkage. Glypican-1 is a regulator of FGF2 and its expression increases with the differentiation of turkey satellite cells. The GPI anchor of glypican-1 can be cleaved resulting in glypican-1 being secreted or shed into the extracellular matrix environment. To investigate the role of glypican-1 shedding and the glycosaminoglycan and N-glycosylated chains in regulating the differentiation of turkey myogenic satellite cells the following constructs were made. A glypican-1 construct without the GPI anchor was cloned into the mammalian expression vector pCMS-EGFP, and glypican-1 without the GPI anchor and glycosaminoglycan and N-glycosylated chains were also cloned and transfected into turkey myogenic satellite cells to measure the effect on differentiation, myotube formation in the presence of FGF2, and the

expression of the myogenic transcriptional regulatory factors, MyoD and myogenin. The removal of the glypican-1 GPI region increased differentiation and myotube formation in the presence of exogenous FGF2. The increase in differentiation was supported by the elevated expression of myogenin. In general, the shedding of glypican-1 during differentiation increased MyoD and myogenin expression showing that the GPI anchor is important in regulating the state of satellite cell replication and differentiation. These data demonstrated that the shedding of glypican-1 from the satellite cell surface acts as a positive regulator of satellite cell differentiation and sequesters FGF2, permitting further differentiation.

In other studies, the **Ohio station** examined the effect of thermal stress and growth selection on satellite cell proliferation and differentiation in turkeys. Initial studies have begun on determining the effect of thermal stress both hot and cold on turkey satellite cell proliferation and differentiation. Studies are using satellite cells isolated from a randombred control line 2 (RBC2) turkey that has been maintained without selection for any trait and a line F which was selected from the RBC2 line for 16-wk body weight. Results have shown that decreased temperature reduced proliferation and differentiation in both the RBC2 and F lines whereas increased temperature increased both proliferation and differentiation in both lines. Proliferation rate but not differentiation was affected by growth selection. Myotube width during differentiation increased with temperature in both the F and RBC2 lines but the increase in diameter was not affected by growth selection.

The **North Carolina station** reported that myogenesis is facilitated by 4 myogenic regulatory factors (MRF) and is significantly inhibited by myostatin (MSTN). The objective of the current study was to examine embryonic gene regulation of MSTN/MRF, and subsequent manipulations of protein synthesis, in broiler embryos under induced hyperammonemia. Broiler eggs were injected with ammonium acetate solution 4 times over 48 h beginning on either embryonic day (ED) 15 or 17. Serum ammonia concentration was significantly increased ($P < 0.05$) in ammonium acetate injected embryos for both ED17 and ED19 collected samples when compared with sham-injected controls. Expression of mRNA, extracted from pectoralis major of experimental and control embryos, was measured using real-time quantitative PCR for MSTN, and MRFs myogenic factor 5 (MyF5), myogenic determination factor 1 (MyoD), myogenin (MYOG), and myogenic regulatory factor 4 (MRF4). A significant reduction ($P < 0.01$) in MSTN expression accompanied increased serum ammonia concentration in both ED17 and ED19 collected samples. There was an increase in MyF5 expression ($P < 0.05$) in ED17 collected samples, supporting increased myoblast proliferation. In both ED17 and ED19 collected samples, MRF4 was decreased ($P = 0.05$) in ammonium acetate injected embryos suggesting increased myoblast proliferative activity. No significant difference was seen in expression of MyoD or MYOG for either age group. MSTN protein levels were evaluated by Western blot analysis, and also showed decreased MSTN expression ($P < 0.05$). Overall, it appears possible to inhibit MSTN expression through hyperammonemia, which is expected to increase embryonic myogenesis and postnatal muscle growth.

The **Indiana Station** reported progress in two projects. First, Lkb1 is indispensable for skeletal muscle development, regeneration and satellite cell homeostasis. Stk11, commonly known as Lkb1, is a tumor suppressor that regulates cellular energy metabolism and stem cell function. Satellite cells are skeletal muscle resident stem cells that maintain postnatal muscle growth and repair. Here, we used MyoDCre and Lkb1flox/flox mice to delete Lkb1 in all embryonic myogenic

progenitors and their descendant satellite cells and myofibers. Subsequently, the Lkb1 conditional knockout mice develop severe muscular dystrophy characterized by central nucleated myofibers, reduced mobility, growth retardation and premature death. Although tamoxifen-induced postnatal deletion of Lkb1 in satellite cells using Pax7CreER mice bypasses the muscular dystrophy phenotype, Lkb1-deficient satellite cells lose their regenerative capacity cell-autonomously. Strikingly, Lkb1 null satellite cells fail to maintain quiescence in non-injured resting muscles and exhibit accelerated proliferation but reduced differentiation kinetics. At the molecular level, Lkb1 limits satellite cell proliferation through the canonical AMPK/mTOR pathway, but facilitates differentiation through phosphorylation of GSK-3β, a key component of the WNT signaling pathway. Together, these results establish a central role of Lkb1 in muscle stem cell homeostasis, muscle development and regeneration. In the second project, it was reported that Notch signaling deficiency underlies age-dependent depletion of satellite cells in muscular dystrophy. Duchenne muscular dystrophy (DMD) is a devastating disease characterized by muscle wasting, loss of mobility and death in early adulthood. Satellite cells are muscle-resident stem cells responsible for the repair and regeneration of damaged muscles. One pathological feature of DMD is the progressive depletion of satellite cells, leading to the failure of muscle repair. Here, we attempted to explore the molecular mechanisms underlying satellite cell ablation in the dystrophin mutant mdx mouse, a well-established model for DMD. Initial muscle degeneration activates satellite cells, resulting in increased satellite cell number in young mdx mice. This is followed by rapid loss of satellite cells with age due to the reduced self-renewal ability of mdx satellite cells. In addition, satellite cell composition is altered even in young mdx mice, with significant reductions in the abundance of non-committed (Pax7+ and Myf5-) satellite cells. Using a Notch-reporter mouse, we found that the mdx satellite cells have reduced activation of Notch signaling, which has been shown to be necessary to maintain satellite cell quiescence and self-renewal. Concomitantly, the expression of Notch1, Notch3, Jag1, Hey1 and HeyL are reduced in the mdx primary myoblast. Finally, we established a mouse model to constitutively activate Notch signaling in satellite cells, and show that Notch activation is sufficient to rescue the self-renewal deficiencies of mdx satellite cells. These results demonstrate that Notch signaling is essential for maintaining the satellite cell pool and that its deficiency leads to depletion of satellite cells in DMD.

Objective 3. Mechanisms of protein assembly and degradation in skeletal muscle.

The **Nebraska Station** used the bovine stearnomandibularis muscle to investigate the fraction of myofibrils termed easily releasable myofilaments (ERM). The method for isolating ERM was essentially that described by Neti and co-workers with a few modifications. Isolated ERM were characterized by electron microscopy, proteomics and Western blotting. Considerable difference was observed in the 2-dimensional electrophoretic separation patterns of myofibrillar and ERM fractions. The most striking differences were observed in alpha actin between myofibril and ERM samples. ERM fractions contained multiple alpha actin spots that migrated to a more alkaline region of the separation than its typical pI of 5.3. This change in alpha actin mobility was not observed in ERM from muscle tissue held 7 days post-mortem. MS analysis also identified the presence of ubiquitin tagging in alpha actin. These findings are consistent with arginine modified residues in alpha actin. Additionally, Western blotting of myofibril separations using antibodies to ubiquitin, MuRF1, and TRIM32 (E3 ubiquitin ligases) demonstrated the presence of these members of the ubiquitin proteasome system.

Impact Statements

- Poor maternal nutrition during gestation has significant and long lasting impacts on muscle development in the offspring, which negatively affect meat production and quality, and increase the cost of production. Understanding the mechanisms which lead to decreased muscle mass may allow development of strategies to improve production efficiency in livestock.

- Alterations in the temporal expression of the myogenic regulatory factors are suggestive of precocial differentiation, which may lead to formation of larger and/or fewer muscle fibers.

- Insulin-like growth factors (IGF) 1 and 2 regulate prenatal and postnatal skeletal muscle development and growth.

- Increased lean meat yield from pigs with IGF2 paternal A alleles is a result of both increased muscle growth and reduced fat subcutaneous deposition.

- Increased expression of IGF2 in pigs with paternal A alleles begins as early as late gestation and continues throughout the production phase of pigs.

- Muscle fiber number may be increased in pigs with paternal IGF2 A alleles.

- The heavily muscled phenotype of myostatin null mice may be partially attributable to increased IGF2 expression but not to changes in IGF1.

- Increased IGF2 expression in myostatin null mice is not variant specific

- ZBED6, a transcriptional repressor of IGF2 expression, is likely not responsible for the reduction in postnatal IGF2 expression compared with fetal expression.

- Heterozygous mutations of myostatin are viable in pigs with paternal G alleles for IGF2.

- Differences in the reductions of growth-regulating genes observed with age do not explain muscle growth differences between sexes or genotypes.

- In gestating sows, the endocrine pattern for IGF1 progressively decreased as gestation progressed and then increased markedly after farrowing. Large changes in IGF1 were not explained by changes in circulating GH perhaps suggesting an uncoupling (during gestation) and then recoupling (during lactation) of the somatotropic axis.

- Environmental heat stress on gestating sows greatly affected glucose and insulin endocrine profiles which resulted in elevated glucose and insulin after farrowing.

- Myostatin is a potent negative regulator of skeletal muscle mass, yet myostatin propeptide fused to the Fc region of pig immunoglobulin G may be a pharmacological approach for improving skeletal muscle growth in pigs via suppression of myostatin activity.

- Titin, a giant muscle protein, is the “backbone” of the sarcomere, and titin isoform switching occurs during muscle growth and protein turnover. Recent observations provides evidence that titin isoform transition is triggered by thyroid hormones linked to RNA binding motif 20 (RBM20; a major regulator of titin isoform transition) via the PI3K/Akt/mTOR signaling, which is phosphorylating RBM20 and/or increasing gene expression of RBM20.

- In chickens, selection for reduced postnatal body weight promotes rapid embryogenesis with a reduced rate of fetal myogenesis by comparison to birds selected for a larger postnatal size.

- The STAC3 gene is specifically expressed in skeletal muscle and plays an important role in skeletal muscle development and contraction. Recent results suggest that the STAC3 gene is a negative regulator of the differentiation of bovine satellite cells into myotubes.

- In knockout mice lacking O-GlcNAc transferase (OGT) specifically in skeletal muscle, OGT deficient mice exhibit reduced fat deposition, are resistant to high fat diet induced obesity, and have improved systemic insulin sensitivity.

- Dogma suggests that viable satellite cells cannot be readily isolated from skeletal muscle after extended post-mortem storage. Recent work in the pig indicates that post mortem satellite cell yield decreases 55% and 66% at 24 and 72 hours, respectively. However, as time post-mortem increased the percentage of cells expressing Pax7 and Myf5 increased.

- Iodine value (IV), is a measure of fatty acid (FA) unsaturation and is currently the industry standard for assessing pork fat quality. Variation exists between the anatomical locations of the fat depots with respect to IV. Fat depot IV were affected also by duration of feeding, percentage of fat in the diet, and degree of saturation of the fats in the diet.

- Phytoecdysteroids such as 20 hydroxyecdysone (20HE) are ingredients in various nutritional supplements marketed to enhance physical performance and lean body mass. Current findings do not support the idea that anabolic effects of 20HE are acutely mediated by mTORC1 and do not support the supplementation of such components in the feed of animals to promote muscle growth.

- Activation of the PGC-1α pathway via gene transfer decreased muscle injury in dystrophic skeletal muscle using a mouse model of Duchenne muscular dystrophy. Quercetin, an orally available and safe PGC-1α activator was tested as an alternative method of activating the PGC-1 α pathway. It was found that a 0.2% quercetin diet decreased muscle damage in the diaphragm and the heart in a mouse model of Duchenne muscular dystrophy.

- Environmental heat stress and immune challenge to growing pigs directly and indirectly affect metabolism and cytokine expression and both variables may contribute to decreased growth parameters.

- An important issue in maintaining muscle mass is the prevention of atrophy during periods of disease or disuse. IL-6 has multiple effects on satellite cell activity, depending on the context

and environment in which expression is increased. Current observations suggest that the muscle wasting syndrome associated with disease or disuse is through inhibition of muscle regeneration and satellite cell function during muscle regeneration.

- An expression network and gene variants have been identified that are associated with shear force variability that remains in beef after electrical stimulation of carcasses. These findings can be applied to improve selection strategies for the beef cattle industry.

- Mass spectroscopy analysis of myofibrillar proteins revealed the presence of ubiquitin tagging in alpha actin.

- Estradiol (E2) and combined E2 and trenbolone acetate (TBA) (a testosterone analog) implants are routinely used to enhance muscle growth in feedlot steers [1,2]. According to the National Animal Health Monitoring System (NAHMS) (2000), 97% of feedlot cattle receive at least one steroid implant during their lifespan. Results of the studies at the Minnesota Station will provide information on the mechanism by which widely used E2 implants stimulate bovine muscle growth. In the future this kind of fundamental information will help in developing new, safe and effective strategies to increase rate and efficiency of muscle growth in beef cattle.

- A Se-enriched probiotics product developed by the Mississippi station has the potential to be used as a new feed additive to promote swine muscle growth.

- Deep transcriptome RNA sequencing has revealed several differentially expressed genes, most notably pyruvate dehydrogenase kinase isozyme 4, that are associated with development of pale, soft, exudative turkey meat.

Collaborative Grants

Collaborative NIFA-AFRI Grant between Ohio State University with the Michigan State Station (Gale Strasburg) Strasburg, G., Velleman, S.G. Reed, K., NIFA AFRI, “Influence of Thermal Challenge on Turkey Muscle Development and Meat Quality,” $975,000, 3/2014-2/2017.

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Theses:

1. Diana Clark, MS. Effects of maternal and postnatal infection with porcine reproductive and respiratory syndrome virus on muscle growth and development in piglets. University of Illinois, September, 2014.
2. Daniel Clark, PhD. Regulation of skeletal muscle growth and gene expression by insulin-like growth factors and myostatin. University of Illinois, August 2014.
3. Kayla Leanne Brooks, “The Safety and Adequacy of Galactooligosaccharides and Fructooligosaccharides in Infant Pig Formula” (M.S. degree conferred August 2014).
4. Kirsten Seelenbinder, “Effects of heat stress and porcine reproductive and respiratory syndrome virus on metabolism” (M.S. degree conferred August 2014).
5. Allysa Stern. The Effect of Hyperammonemia on Myogenesis in Broiler Embryos. (M.S. degree conferred 2014)

Other publications and presentations:

1. Ross JW, Selsby JT, Nonneman DJ. Genetic Modification of Pigs: Expanding their Utility as Biomedical Models. National Breeders Roundtable Annual Conference, Conference Proceedings, pp 32-38, 2013.
2. Johnson, J.S., M. Abuajamieh, M.V. Sanz-Fernandez, J.T. Seibert, S.K. Stoakes, J.W. Ross, J.T. Selsby, N.K. Gabler, H. Xin, M.C. Lucy, T.J. Safranski, R.P. Rhoads, and L.H. Baumgard. 2013. Heat stress alters energy metabolism during pre- and postnatal development.

XXIII International Reunion on Production of Meat and Milk in Hot Climates. Mexicali, Mexico. Pp. 38-50.

3. Kaiser A, Johnson A, Selsby JT, and Stalder KJ. Independent Study 490A: Positive Reinforcement Training Piglets to Stand in a Container and Follow a Human. AS-Leaflet-R2914.pdf, 2014.
4. Selsby JT. 17th annual meeting of the Iowa Physiological Society. The Physiologist, 57: 74-75, 2014.
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6. TriBeta Seminar - PGC-1α-mediated protection of dystrophic skeletal muscle, Feb. 13, 2014
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8. Quercetin-mediated protection of dystrophic skeletal muscle. Duchenne Alliance Annual Meeting, March 28, 2014.
9. Quercetin as a novel therapeutic approach for Duchenne muscular dystrophy. Undergraduate Science Seminar Series, Drake University, September 12th, 2014.
10. Strasburg, GM. Turkey skeletal muscle growth, development, and meat quality. Seminar presentation at Iowa State University Department of Animal Science, March 25, 2014.
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12. Liao, S. F., M. A. Crenshaw, N. Regmi, and T. Wang. 2014. To improve the efficiency of swine production through basic and applied nutrition research. Oral presentation at the annual meeting of Mississippi Pork Producers Association on Mar. 15 (Louisville, MS).