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NCCC210 Mtg., Boston 2013

General Highlights

- NCCC210 member and ad hoc Attendees: Kichoon Lee, Kola Ajuwon, Min Du, Werner Bergen, Stone Ding, Brynn Voy, James Kinder, Sean Adams, Kim Barnes, Theo Van Kempen, Jack Odle (Could not make it due to inclement weather in the Midwest: R. Scott Rector, Michael Spurlock and Donald Bietz)

- NCCC210 meeting was supported by Nutreco (courtesy of Dr. Van Kempen) and the American Society for Nutrition (ASN)

- The NCCC210 group spearheaded the development of a symposium "Adipose and Lipid Biology: Crossing Taxonomic Boundaries" held at the Experimental Biology 2013 meeting, 4/19/2013 in Boston, MA. The symposium was sponsored by the American Society for Nutrition and supported in part by Proctor & Gamble Pet Care, Nutreco, the North Central Regional Association (NCRA) of Agricultural Experiment Station Directors (NCCC-210 Multi-State Project), the USDA-ARS WHNRC and Human Nutrition National Program NP-107.

- Dr. Bergen is helping lead a Methods Compendium effort by the NCCC210 group, to create a compilation of adipose methodologies for use by the scientific community. The NCCC210 renewal/proposal is also being led by Dr. Bergen, with co-leadership by Dr. Ajuwon and Dr. Ding in an advisory capacity. This document is due in Sept. 2013.

- Dr. Voy was welcomed as a new member to the NCCC210 group. Dr. Kinder was by consensus chosen to continue in the role of NCCC210 Administrative Lead.

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Report 1

USDA-ARS Western Human Nutrition Research Center (WHNRC; Sean H. Adams)

Project Report: A major focus of our laboratory is the biology of adipose tissue, with the ultimate goal of applying this knowledge toward strategies to improve public health via nutritional and physical activity interventions. One aspect that is important to adipose tissue function and ultimately, whole-body insulin sensitivity, is inflammation, since low-grade inflammation in white adipose tissue (WAT) is often associated with obesity and this plays a role in insulin resistance. Such inflammation might be impacted by nutrition (at least in monogastrics), i.e., via increasing intakes of anti-inflammatory phytonutrients, limiting dietary saturated fat intake, limiting calories and hence excess body fat. We have also emphasized comparisons of weight-matched unhealthy vs. healthy obese persons and diet-induced obese rodents, to better understand the metabolic "signatures" specific to health and disease and dissociated from weight. A great deal of this research involves metabolomics analyses that catalog and interpret hundreds of metabolites in the blood or other sites, complementary to observational phenotypes, energy balance measurements, and molecular biological determinations in tissues. This work is outlined in our published work over the last Project Year.

Publications:

1. A.P. Thomas,T.N. Dunn, J.B. Drayton, P.J. Oort, and S.H. Adams. A high calcium diet containing nonfat dry milk reduces weight gain and associated adipose tissue inflammation in diet-induced obese mice when compared to high calcium alone. *Nutrition & Metabolism* 9:3, 2012
2. J.A. Viscarra, J.P. Vázquez-Medina, R. Rodriguez, C.D. Champagne, S.H. Adams, D.E. Crocker, R.M. Ortiz. Decreased expression of adipose CD36 and FATP1 are associated with increased plasma nonesterified fatty acids during prolonged fasting in northern elephant seal pups (*Mirounga angustirostris*). *J. Exp. Biol.* 215(Pt 14):2455-64, 2012
3. S. Huang, J.M. Rutkowsky, R.G. Snodgrass, K.D. Ono-Moore, D.A. Schneider, J.W. Newman, S.H. Adams, D.H. Hwang. Saturated fatty acids activate TLR-mediated pro-inflammatory signaling pathways. *J. Lipid Res.* 53(9):2002-13, 2012
4. A.P. Thomas,T.N. Dunn, J.B. Drayton, P.J. Oort, S.H. Adams. A dairy-based high calcium diet improves glucose homeostasis and reduces further weight gain in high fat fed mice in the context of pre-existing obesity. 2012 Sep 18. doi: 10.1002/oby.20039 [Epub ahead of print], *Obesity*
5. D. Grapov, S.H. Adams, T.L. Pedersen, W.T. Garvey, K.H. Lok, J.W. Newman. Type 2 diabetes associated changes in the plasma non-esterified fatty acids, oxylipins and endocannabinoids. *PLoS ONE* 7(11): e48852, 2012
6. S. Millership, N. Ninkina, I. Guschina, J. Norton, R. Brambilla, P.J. Oort, S.H. Adams, R.J. Dennis, P.J. Voshol, J.J. Rochford, V.L. Buchman. Increased lipolysis and altered lipid homeostasis protect γ-synuclein null mutant mice from diet-induced obesity. *PNAS* 109(51):20943-8, 2012
7. M.G. Witbracht, M. Van Loan, S.H. Adams, N.L. Keim, K.D. Laugero. Dairy food consumption and meal-induced cortisol response interacted to influence weight loss in overweight women undergoing a 12-week meal-controlled weight loss intervention. *J. Nutrition* 143(1):46-52, 2013
8. P. She, K.C. Olson, Y. Kadota, A. Inukai, Y. Shimomura, C.L. Hoppel, S.H. Adams, Y. Kawamata, H. Matsumoto, R. Sakai, C.H. Lang, C.J. Lynch. Leucine and protein metabolism in obese Zucker rats. *PLoS ONE* 8(3): e59443, 2013
9. T.P. Garcia, S. Müller, R.J. Carroll, T.N. Dunn, A.P. Thomas, S.H. Adams, S.D. Pillai, R.L. Walzem. Structured variable selection with q-values. 2013 Apr 10 [Epub ahead of print], *Biostatistics*

10. D.E. Lackey, C.J. Lynch, K.C. Olson, R. Mostaedi, M. Ali, W.H. Smith, F. Karpe, S. Humphreys, D.H. Bedinger, T.N. Dunn, A.P. Thomas, P.J. Oort, D.A. Kieffer, R. Amin, A. Bettaieb, F.G. Haj, P. Permana, T.G. Anthony, S.H. Adams. Regulation of adipose branched chain amino acid catabolism enzyme expression and cross-adipose amino acid flux in human obesity. 2013 Mar 19 [Epub ahead of print], *Am. J. Physiol: Endocrinol. Metab.*
11. S.H. Adams, K.M. Barnes, and J. Odle. Comparative Metabolic Physiology in the 'omics' Era: A Call to Arms, Paws, Flippers, and Claws (intro to symposium "Adipose and Lipid Biology: Crossing Taxonomic Boundaries" held at the Experimental Biology 2013 meeting, 4/19/2013 in Boston, MA). submitted, *Advances in Nutrition*
12. C. Aguer, O. Fiehn, E.L. Seifert, V. Bezaire, J. Meissen, A. Daniels, K. Scott, J-M. Renaud, M. Padilla, D. Bickel, M. Dysart, S.H. Adams*, M-E. Harper*. Muscle UCP3 overexpression mimics endurance training and reduces circulating biomarkers of incomplete beta-oxidation. submitted with revision, *FASEB J.*
13. Y. Kadota, Y. Kitaura, S.H. Adams, S. Yoshiharu. Regulation of hepatic branched-chain α-keto acid dehydrogenase complex in rats fed a high-fat diet. submitted with revision, *Obesity Res. & Clinical Practice*
14. C. Aguer, C.S. McCoin, T.A. Knotts, R. McPherson, R. Dent, D. Hwang, S.H. Adams*,M-E. Harper*. Acylcarnitines: potential implications for skeletal muscle insulin resistance. submitted, *Diabetes*

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Report 2

The Ohio State University (Kichoon Lee, Jinsoo Ahn)

Establishment of avian transgenic technology: Recently, our laboratory successfully developed, for the first time, transgenic quail exclusively overexpressing a target gene in adipose tissue by using a chicken fatty acid binding protein 4 promoter. Using the avian transgenic technology, novel genes will be overexpressed in adipose tissue of transgenic quail to understand their roles in adipose tissue growth and development. This success leads to a new avenue to open avian transgenesis as an excellent animal model to study gene function for agricultural applications and also to provide significant foundational information for advancing adipocyte biology in general. Importantly, our basic understanding through these studies has implications for other domestic animal species including cattle, sheep and pigs, as well as, for human health, where new knowledge may be applied to obesity and associated diseases.

Comparative analysis of microarray databases to identify tissue specific genes: Microarrays are established technologies that can provide large-scale gene expression data through measurements of transcript abundance of various tissues that are available in the NCBI web site. However, there have been no attempts to integrate these valuable microarray databases to identify novel sets of adipose tissue-specific genes that might have important functions in adipose tissue growth and development. Adipose-specific genes have important functions in adipose development and fat accretion. Although about 100-200 signature genes are expressed in specific tissues, so far, less than about 30 adipose-specific genes have been identified; and these genes have important functions in the processes of adipocyte development and metabolism. However, there is a critical need to develop strategies for efficiently identifying additional new adipose-specific genes to understand their functions, thus, filling a significant void in our comprehension of fat accretion in food animals. In addition to extensively analyzing these data sets to identify adipose-specific genes in the mouse and human, we recently performed a microarray analysis, using the Affymetrix chicken GeneChips containing 28,000 transcripts (80% of the total transcripts) and identified new sets of adipose-specific genes in chickens. The selected adipose-specific genes for chicken were compared to the data for the mouse and human. The selected candidate genes were also exclusively expressed in adipose tissues of human and mice, demonstrating conservation of tissue-specific expression of genes across species of animals. We expect that these novel genes will open new studies that will lead to high impact discoveries in the adipocyte biology area.

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Publications

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3. Park YJ, Mohamed ES, Oh SA, Yoon SJ, Kwon WS, Kim HR, Lee MS, Lee K, Pang MG. 2012. Sperm Penetration Assay as an Indicator of Bull Fertility. J Reprod Dev. 2012. 58(4):461-6.
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Report 3

Washington State University (Michael Dodson, Min Du)

Dodson Laboratory. The main focus of the Dodson laboratory is to develop isolation methods for mature adipocytes so that individual cells may be cloned in a manner to insure that no (potential) "rider" stem cell may exist in ceiling cultures. Purity issues are evident in most of the research in this area, suggesting that contaminating SV cells may provide proliferative-competent cells that are now being termed DFAT cells. The two figures (below) show elementary methods being employed and the flat-bottomed dishes being used for ceiling culture. In 2013, we are conducting a bio-substratum study to optimize mature adipocyte isolation (in progress).

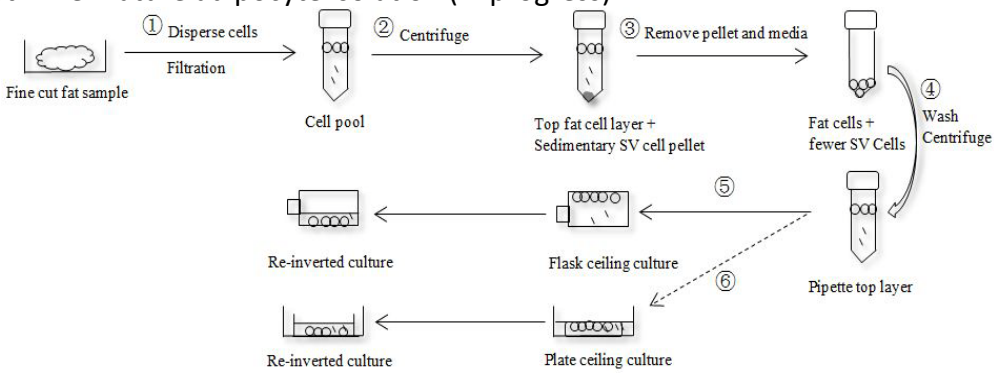

Figure 1. Mature adipocyte isolation and plate/flask ceiling culture. ① In the hood, collect 5 g of fine cut (about 3 mm pieces) fat fragments into a 50 ml tube containing 20 ml of 0.25% collagenase I (Gibco) solution. Disperse cells gently on a rocker at 37 °C for 1 h, followed by filtering through a 1000 µm mesh into a new 50 ml tube. ② Centrifuge the cell pool at 186 x g for 10 min. ③ Remove the underlying pellet (SV cells) and media. ④ Wash the fat layer by 20 ml DMEM/F12 + 10% HS, followed by centrifugation at186 x g for 10 min. ⑤ (flask ceiling culture) Pipette the top layer into a BD Falcon™ T-12.5 cm² cell culture flask. Fill the flask with DMEM/F12 + 10% HS. The medium-filled flask was tightly closed and inverted for 4-6 d. Then the flask was re-inverted and exposed to a small amount of traditional basal media.⑥ (plate ceiling culture) Pipette the top fat layer into a 150 mm × 25 mm cell culture dish containing small amount of media (DMEM/F12 + 10% HS). Then put an inverted 100 mm × 15 mm cell culture dish (ceiling) into the big dish by forceps. Add proper amount of media and chase bubbles out, allowing the solution surface contact with the inner surface of the small dish. Cover the lid of big dish (container) and incubate for 4-6 d for plate ceiling culture. Re-invert the small dish and expose it to small amount of traditional basal media.

Figure 2. Initial attempt at plate ceiling culture. [A] Pipette top layer of the 1st tube into a Nunclon® 100 mm × 20 mm cell culture dish (container). Then put three inverted BD Falcon™ 35 mm × 10 mm dishes into the big dish by forceps. Add proper amount of media (DMEM/F12 + 10% HS) and chase bubbles out, allowing its surface contact with the inner surface of the small dishes (the bottom of the small dishes then became the 'ceiling'). [B] Pipette top layer of the 2nd tube into a Nunclon® 150 mm × 20 mm cell culture dish. Then put an inverted Nunclon® 100 mm × 10 mm lid into the big dish by forceps. Add proper amount of media (DMEM/F12 + 10% HS) and chase bubbles out, allowing its surface contact with the inner surface of the lid. [C] Pipette top layer of the 3rd tube into a BD Falcon™ T-12.5 cm² cell culture flask. Fill the flask with DMEM/F12 + 10% HS. The medium-filled flask was tightly closed and inverted (the bottom of the flask then became the 'ceiling').

Du Laboratory. Our lab is focusing on studying epigenetic regulation of early adipogenic commitment, focusing on the impact of maternal nutrition on Zfp423 expression and adipogenic commitment. The following is the summary of our studies during the last year. Maternal obesity (MO) has long-term impacts on offspring health, including predisposition to obesity and type 2 diabetes (T2D). Zfp423 is the key transcription factor committing multipotent cells to the adipogenic

lineage; exceptionally dense CpG sites were observed in the Zfp423 promoter. We hypothesized that MO enhances adipogenic differentiation during fetal development through inducing epigenetic changes in the Zfp423 promoter and elevating its expression. Female mice (one month old) were fed either a control (Con, 10% of energy from fat) or obesogenic (OB, 45% of energy from fat) diet for two months to induce obesity and, then, mated and maintained in their respective diets during pregnancy. Fetal primordial somatic tissue was harvested at E14.5, at stage when the early adipogenic commitment is initiated but not the late stage adipogenic differentiation. The mRNA expression of Zfp423 was 3.6-fold higher in OB compared to Con fetal tissue, which was associated with lower DNA methylation in the Zfp423 promoter; consistently, the protein content of Zfp423 was more than 2.7-fold higher in OB compared to Con tissue. We further observed that repressive histone methylation (H3K27me3) was lower, while the active histone methylation (H3K4me3) was higher in the Zfp423 promoter of OB tissue. In addition, the binding of Enhancer of Zeste 2 (EZH2), which is the catalytic component of polycomb repressive complex 2, was lower in the Zfp423 promoter despite the EZH2 content was higher in OB fetal tissue. In summary, MO enhanced adipogenic differentiation during fetal development, at least partially through reducing DNA methylation of the Zfp423 promoter. Because DNA methylation is stable, the reduction in Zfp423 methylation is expected to permanently enhance adipogenic differentiation of progenitor cells, programming adiposity later in life.

WASHINGTON STATION
PUBLICATIONS

Research Papers [including finalized citations from last year]:

1. Das, A. K., Q. Y. Yang, X. Fu, J. F. Liang, M. S. Duarte, M. J. Zhu, G. D. Trobridge, and **M. Du**. (2012). AMP-activated protein kinase stimulates myostatin expression in C2C12 cells. *Biochemical and Biophysical Research Communications*, 427: 36-40.
2. Dodson, M.V., S. Boudina, E. Albrecht, L. Bucci, M. Fernyhough-Culver, S. Wei, W.G. Bergen, A.J. Amaral, N. Moustaid-Moussa, S.Poulos and G.J. Hausman. 2013. A long journey to effective obesity treatments: Is there light at the end of the tunnel? *Experimental Biology and Medicine* doi: 10.1177/1535370213477603
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12. Jellyman, J. K., M. S. Martin-Gronert, Cripps, R. L., Giussani, D. A., Ozanne S. E., Q. W. Shen, M. Du, A. L. Fowden, and A. J. Forhead. (2012). Effects of cortisol and dexamethasone on insulin signaling pathways in skeletal muscle of the ovine fetus during late gestation. *PLOS one*, 7: e52363.
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Teaching/Advising/Learning Papers:

Drake, S.D., S. Wei, l. Hansen, B. Rohde, C. Harris, B. Moranville, W. Lewis, M. Blyzka, E. Miller, W.G. Bergen, K.H. McKeever, E.A. Greene, A.L. Reitmeier and M.V. Dodson. in preparation. Effects of clenbuterol/cimaterol on quarter horses and racehorses. *Equine and Comparative Exercise Physiology*

Bowie, J.M., H.K. Floren, J.K.B. Gentry, L.E. Hansen, C.L. Harris, M.A. Jackson, W.C. Lewis, J.L. Mutch and M.V. Dodson. 2013. Sarcomere in the classroom: Learning with undergraduate groups. *NACTA Journal* 57(2): [in press]

Dodson, M.V. and S. Wei. 2012. What are we doing right? *NACTA Journal* 57(1):96-97

Book Editting:

Yin, J. (Editor in Chief), M. Du (Associate Editor in Chief), and Y. Jin (Associate Editor in Chief). (2012). Animal Muscle Biology and Meat Science (动物肌肉生物学与肉品科学). *China Agricultural University Press*, Beijing, China.

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Report 4

University of Missouri (Randy Scott Rector)

My primary research interest is to examine the mechanisms responsible for nonalcoholic fatty liver disease (NAFLD) development and progression. NAFLD is a chronic, progressive liver disease that affects 75-100% obese adults and 50-75% of obese children. Despite these staggering numbers, causes of NAFLD, the factors that trigger disease progression and effective treatment strategies remain poorly defined.

Taking a translational approach, my lab utilizes multiple animal models and studies in humans to address these important questions. Completed and ongoing studies range from utilizing murine models with primary gene defects in mitochondria to the use of rodent models of obesity to better understand single gene vs. polygenic contributions to NAFLD. This work has shown that hepatic mitochondrial dysfunction is an initiating event in the development of both NAFLD and hepatic insulin resistance. We also have shown that daily exercise given at an early age can completely prevent NAFLD development in these rodent models, in part through enhancement of hepatic lipid metabolism. Other studies are underway to examine the effects of exercise and pharmacological therapies on treating NAFLD and hepatic insulin resistance.

My laboratory also is examining the potential negative consequences of childhood obesity on NAFLD development and progression. To address this question, we have developed a large animal model of childhood obesity, which has a longer development period compared with rodents and allows for invasive testing not allowed in pre-obese children. We have found that juvenile Ossabaw swine when fed a high fat/high fructose corn syrup diet style diet (to an age equivalent to adolescence) rapidly develop obesity, insulin resistance and advanced NAFLD. This represents an exciting model and mechanistic studies are underway to examine adipose tissue and liver of these animals. To compliment this work, another ongoing project involves the characterization of the inflammatory status of different fat depots (subcutaneous and intra-abdominal) in obese children (tissues taken during bariatric surgery) and then correlating these findings with systemic inflammation and NAFLD presence and severity.

Publications

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Station Reports
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Report 5

Univ. of Tennessee (Brynn Voy)

In collaboration with Drs. Jean Simon and Joelle Dupont of the Institut National de la Recherche Agronomique, we used Affyemtrix-based expression profiling to characterize the effects of either short term (five hour) feed withdrawal or insulin deprivation (via injection of an anti-insulin antibody) on adipose tissue metabolism in young (21 day-old) broiler chicks. Loss of insulin signaling had relatively modest effects on adipose expression profiles, consistent with a minimal role for insulin in regulation of adipose metabolism in avians. In contrast, fasting exerted broad effects on metabolic pathways, including suppression of genes involved in glucose and lipid metabolism, and upregulation of genes mediating protein catabolism and fatty acid oxidation. Fasted broilers showed a significant increased in adipose content of β-hydroxybutyrate, supporting the concept of increased fatty acid oxidation. Fasting also suppressed genes in each step of adipogenesis, from mesenchymal stem cell commitment through lipid deposition in mature adipocytes, suggesting that both adipocyte hypertrophy and hyperplasia are dynamically tied to feed intake in young, rapidly growing broilers. Adipose gene expression profiles were analyzed in adult broilers and compared to those of both meat-type Fayoumi chickens and egg-type Leghorns as two representative lean lines of chickens to determine if pathways upregulated by fasting were also enhanced in genetically-driven leanness. These lines, obtained through collaboration with Dr. Sue Lamont (Iowa State University) were specifically selected as models of leanness because they are the parents of an advanced intercross population that may be used in the future for systems genetics approaches to fat deposition. Leaner chickens, like fasted broilers, exhibited both increased levels of plasma non-esterified fatty acids and upregulation of genes involved in fatty acid oxidation. In combination, these studies suggest that leanness may be due in part to enhanced fat catabolism in adipose tissue. Ongoing efforts include ex vivo adipose culture to identify specific pathways that promote fatty acid oxidation in broilers.

Publications

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Report 6

Auburn University (Werner Bergen, Terry Brandebourg)

The Bergen Laboratory

Our research aims in recent years have been revolved around the timing and regulation of differentiation of perimysial-intramuscular preadipocytes to mature adipocytes. The canonical pathway of differentiation regulation starting with C/REB β, δ induction followed PPARγ expression followed by initiation of adipogenesis in IMF fat cells has been well established. There are still poorly understood regulatory transcription factors which are active during commitment of progenitor cells to preadipocytes that may be important very early in differentiation of IMF fat cells. There are a number of zinc-finger protein transcription factors that have been shown to either prolong onset of differentiation or directly promote differentiation. We have obtained loin muscle samples from at various ages during post-weaning/growing to finishing stages in beef cattle (170-310 days of age). We note as have others that by harvest (slaughter) expression of regulatory factors in the early commitment and differentiation in loin muscle tissue such as DKL-1, PPARγ and SREBP-1c differ little between various dietary and management treatments at termination of the finishing trial. We are now exploring the role of zinc –finger protein (ZFP 423) and other ZFP expression in regulation of differentiation of IMF fat cells. We are aware that at 170 days of age, the effects of age and nutritional treatments on ZFP expression may have run its course. Securing tissue samples at ages well below our cut-off may enhance our understanding of the role of ZFPs and DKL-1 in IMF fat cell biology.

The Brandebourg Laboratory

My research program aims to facilitate better control of body composition by focusing upon the regulation of feed efficiency and adipose tissue development. A significant part of this effort involves the development of the pig as a translational model for obesity-induced metabolic disease.

My beef cattle research program seeks to maximize the return of receipts to Alabama cattlemen by enhancing the sustainability of forage-finishing production systems in the Southeast. To accomplish this, our research aims to enhance carcass quality and decrease the breakeven point necessary for grass-based beef production systems. We focus on three main issues: 1) better understanding the regulation of marbling development in beef cattle with the goal of increasing marbling score in grass-fed animals while limiting undesirable fat cover in other areas of the carcass, 2) using a genomic approach to better understand the molecular control of feed efficiency, and 3) developing increased tolerance to heat stress. These aims have been pursued during the past year by conducting growth trials and cell culture experiments coupled with the application of molecular biology techniques to measure gene expression, cell signaling and the endocrine function of adipose tissue. Importantly, achieving these goals would have obvious benefits to cow-calf producers in the state by increasing heifer performance and calf value and ultimately would help position Alabama cattleman to increase return of receipts by increasing the sustainability of pasture-based calf to finish production systems in the region.

Station Reports
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Report 7

West Virginia University (Kimberly Barnes)

Our lab has the primary focus of the effect of dietary lipids on lipid metabolism and body composition. We currently have several on-going projects:
The first is on the interaction of dietary CLA with other dietary lipids on body fat, and specifically on lipolysis. We have established that mice fed coconut oil and CLA lose more body fat than those fed soy oil and CLA, and that the greater loss of body fat involves increased lipolysis. We have also established that 3T3-L1 adipocytes exposed to the fatty acids from coconut oil respond to CLA supplementation with a greater increase in lipolysis than those exposed to the fatty acids from soy oil. Work is on-going to identify the signaling pathway(s) that may be responsible for the increased lipolysis.
The second on-going project is on comparing the effect of vegetarian sources of omega-3 fatty acids to fish oil, on body fat and serum lipids. We have established that the vegetarian sources of algae oil (high in DHA) and yeast oil (high in EPA) are not as effective as fish oil at reducing serum lipids or body fat or increasing tissue omega-3 content, even when fed in combination with equal dietary DHA and EPA to the fish oil diet. More recent work has focused on changes in hepatic gene expression of enzymes and transcription factors involved in lipid metabolism and a further characterization of the oils to try to explain the observed differences.
In addition, we have done some work recently with a horse model. First we determined the effect of fish oil vs krill oil on metabolic markers and serum and muscle fatty acids. And we have a collaborative project on-going with Middle Tennessee State University, focused on the metabolic effects of weight loss in overweight horses. As part of the preliminary data for this project characterization of horse mitochondrial function has been made.

Major findings from this year's work include:

1. Coconut oil + CLA-induced lipolysis in 3T3-L1 adipocytes does not appear to involve protein kinase A, and instead may be the result of inhibition of the phospholipase C signaling pathway.
2. Fish oil, but not algae or yeast oil, feeding causes reduced mRNA expression of HMG-CoA Reductase and numerically reduced SREBP2 mRNA expression, which could account for at least part of the reduction in serum total cholesterol.
3. Tissue concentrations of DHA and EPA are less in algae or yeast oil-fed mice, respectively, compared to fish oil-fed mice. The lesser DHA incorporation could be the result of increased fecal excretion of DHA in algae oil-fed mice. The apparent poorer absorption of DHA from algae oil may be the result of differences in esterification position, as fish oil DHA is largely esterified at the sn-2 position, while algae oil DHA is more evenly distributed between positions.
4. Krill oil and fish oil increased tissue omega-3 fatty acids to a similar degree even though krill oil supplemented horses had increased serum EPA.
5. There are fewer differences in mitochondrial oxidation rates between subpopulations in the horse than the mouse.

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Report 8

Purdue University (Kolapu Ajuwon)

The main focus of this station continues to be to understand the role of inflammation and extracellular matrix genes in the regulation of adipocyte differentiation. Our additional interest include study of the role of fiber and fermentation-produced volatile fatty acids on whole body metabolism and well as in utero programming of postnatal metabolism and growth.

Publications

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Report 9

National Taiwan University (Shih-Torng [Stone] Ding)

In the last year, my laboratory worked on three aspects related to adipocyte biology.

First, amelioration of obesity-associated inflammation by n-3 PUFA was demonstrated by cell culture studies and an in vivo pig model (Chen et al., 2012; Chang et al., 2012). We also worked on utilizing mesenchymal stem cells to treat metabolic diseases (Lin et al., 2013).

Secondly, we used a zebrafish model to demonstrate the function of a putative adipocytokine, cysteine sulfinic acid decarboxylase. We described its involvement in synthesize taurine and its physiological effect (Chang et al., 2012).

Finally, we also worked on the utilization of adipose derived stem cells. Protocol and system to differentiate these stem cells into glucose responsive beta cell-like insulin secreting cells were established.

Publications

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