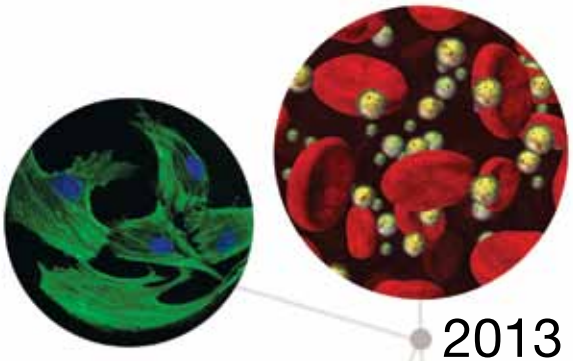
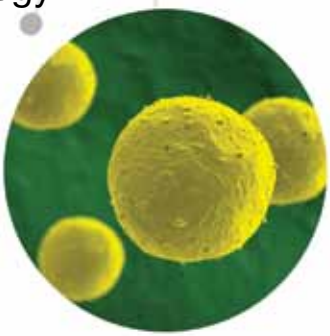


2013

Innovate

Innovations in Animal Growth and Health:

The Next Generation of Cell Biology



Sept. 22 to 24, 2013

Chateau Elan Winery

Braselton, GA

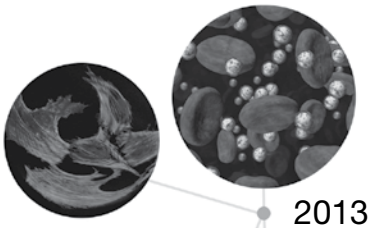
Organized by the American Society for Nutrition

and the American Society of Animal Science

asas.org/innovate2013

Innovate 2014:
Global Food Security
Innovations in Protein Production to Meet the Global Demands of 2050

October 5-7, 2014
Madden's On Gull Lake
Brainerd, MN



2013

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Innovations in Animal Growth and Health:
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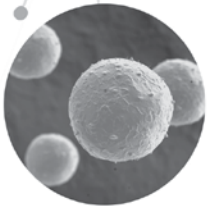


Innovate 2013: Innovations in Animal Growth and Health: The Next Generation of Cell Biology was designed by the American Society of Animal Science (ASAS) and our partner the American Society of Nutrition (ASN) to allow scientists to attend a small intimate program focused on networking and presentation of the latest and greatest new research. The intimate setting is designed to broaden interaction between speaker and attendees.

The meeting will concentrate on the most recent scientific advances in Growth and Development with an emphasis on potential applications. The scientific presentations coupled with the industry perspective of technology application and acceptance will ensure that this conference is "innovative". In fact the Innovate 2013 Program Committee is lovingly referring to this meetings as "not your grandmother's Growth Biology".

Welcome to Innovate 2013.

Jim Sartin
ASAS Past President

SCHEDULE

2013

Innovate

Innovations in Animal Growth and Health:
The Next Generation of Cell Biology

SUNDAY, SEPTEMBER 22, 2013

2:00 pm **Registration Opens - Outside Debussy Ballroom**

5:30 pm **Reception - Atrium Inn**

6:30 pm **Opening Session: Global Acceptance of New Applications and New Technologies - Debussy Ballroom**

Welcome
James Sartin, Auburn University; Teresa Davis, Baylor College of Medicine

Elanco: An Industry Perspective
Dr. Jose Simas, Elanco Animal Health

MONDAY, SEPTEMBER 23, 3013

7:30 am **Breakfast - Outside Debussy Ballroom**

Session 1: Technologies in stem cell and regenerative medicine: theories to "application" - Debussy Ballroom

8:35 am	"SCID" Pig *Chris Tuggle, Iowa State University*
9:20 am	Muscle Fat Crosstalk *Shihuan Kuang, Purdue University*
10:05 am	Break
10:30 am	Translating Stem Cell Biology in GI Research *Mary Estes, Baylor College of Medicine*
11:15 am	Panel Discussion

12:00 pm **Lunch - Atrium Inn**

Session 2: Amino acids: Nutritional regulation of growth - Debussy Ballroom

1:30 pm	mTOR: Nutrient Sensor & Master Regulator of Cell Growth *John Blenis, Harvard College*
2:15 pm	Functional Amino Acids *Guoyao Wu, Texas A & M University*
3:00 pm	Break
3:30 pm	Leucine: A Tonic For Muscle Growth *Teresa Davis, Baylor College of Medicine*
4:15 pm	Panel Discussion

6:30 pm **Reception and Poster Competition - Atrium Inn**

TUESDAY, SEPTEMBER 24, 2013

7:30 am **Breakfast - Outside Debussy Ballroom**

Session 3: Lipid regulation of growth, insulin sensitivity and metabolism - Debussy Ballroom

8:30 am Landscapes of Endogenous Cysteine Proteomes: Functional Protein Regulation by Reversible Cysteine Modifications
Harry Ischiropoulos, University of Pennsylvania

9:15 am Novel Signals Linking Gut Nutrient Sensing to Growth and Metabolism
Doug Burrin, USDA-ARS Children's Nutrition Research Center and Baylor College of Medicine

10:00 am Break

10:30 am Modulation of Animal Growth and Health Via n-6 and n-3 Fatty Acids: Investigations in a Porcine Model
Jack Odle, North Carolina State University

11:15 am **Lunch - Atrium Inn**

Session 4: Microbiome function impacts on malnutrition and metabolic disease - Debussy Ballroom

12:15 pm Microbiome Function Impacts on Nutrition and Metabolic Disease
Bernd Schnabl, University of California San Diego

1:00 pm Dietary Oligosaccharides as Modulators of the Gut Microbiome & Resistance to Infection in the Piglet
Sharon Donovan, University of Illinois

1:15 pm Dietary Manipulation of the Canine and Feline Gastrointestinal Microbiome
Kelly Swanson, University of Illinois

2:30 pm Panel Discussion for sessions 3 & 4

Event ends at 3:00 pm on Tuesday, September 24, 2013

DR. JOHN BLENIS

Dr. John Blenis, Harvard College, has focused his research on defining how Ras and PI3-kinase proto-oncoproteins regulate cellular signaling in normal and cancer cells. His studies on Ras-regulated ERK-MAP kinases and RSK activation revealed how cell surface signals originating from tyrosine kinases or protein kinase C are transmitted by ERK and RSK from the cell surface to the nucleus to alter immediate-early gene expression via ERK and RSK phosphorylation events. His laboratory has shown how several of these induced gene products act as molecular sensors converting subtle differences in ERK and RSK signaling into specific biological responses; molecular mechanisms linking ERK/RSK signaling to regulation of cell migration and other processes involved the metastasis of many cancers; 40S ribosomal S6 protein kinase (S6K) acts downstream of phosphatidylinositols modified by PI3-kinase, which linked tyrosine kinases and downstream serine/threonine phosphorylation events; rapamycin, a natural product now in clinical trials for cancers, blocked S6K activation; and mechanisms by which mTOR and S6K regulate protein synthesis and cell growth and how mTOR/S6K modulate nutrient metabolism to meet the metabolic demands of tumor cells. Many of these pathway components are current and future targets for drug development.

DR. DOUG BURRIN

Dr. Doug Burrin received his B.S. degree (1981) in animal science from Purdue University and his M.S. (1983) and Ph.D. (1987) degrees from the Department of Animal Science at University of Nebraska. He was a postdoctoral fellowship at the Children's Nutrition Research Center, Houston, where he has been a faculty member since 1987. Dr. Burrin is recognized for his expertise in human and domestic animal nutrition and gastroenterology. He has been a leader in using pigs as a translational, dual-purpose animal model, and his research has benefited human pediatric gastroenterology and swine nutrition. His recent work is focused on determining how nutritional support, enteral versus parenteral, affects gut and liver function and susceptibility to disease in early development. Dr. Burrin has authored more than 160 publications, 25 invited reviews and book chapters; mentored 25 postdoctoral fellows; served on various editorial boards and national grant review panels; and has received grant support from federal and privately-sponsored sources. He received the Mead Johnson Award from the American Society for Nutrition, the Animal Growth and Development Award from the American Society of Animal Science, and the Department of Pediatric Research Mentorship Award at Baylor College of Medicine.

DR. TERESA DAVIS

Dr. Teresa Davis is a Professor of Pediatrics at the USDA/ARS Children's Nutrition Research Center at Baylor College of Medicine in Houston, Texas, and is the current Past President of the American Society for Nutrition (ASN). She is internationally recognized for her studies on the nutritional regulation of protein metabolism and growth. Dr. Davis has received the Stokstad Award from ASN, the Animal Growth and Development Award from the American Society of Animal Science, the Centennial Leader Award from the University of Tennessee, and the Research Mentor Award from Baylor College of Medicine. She is a Fulbright Distinguished American Scholar and a Distinguished Foreign Expert in China. Dr. Davis is an Associate Editor for the *Journal of Nutrition* and has been appointed its Editor-in-Chief for 2014-2019.

DR. SHARON M. DONOVAN

Dr. Sharon M. Donovan is the Melissa M. Noel Endowed Chair in Nutrition and Health at the University of Illinois, Urbana-Champaign. She received her B.S. and Ph.D. degrees in Nutrition from the University of California, Davis, completed a postdoctoral fellowship in Pediatric Endocrinology at Stanford University School of Medicine, and then accepted a faculty position at the University of Illinois, Urbana, in 1991. She is actively involved in professional societies and served as the President of the American Society for Nutrition (ASN) from June 2011 to June 2012. Her research has been focused on neonatal intestinal development and development of clinically-efficacious therapies, including optimized formulas, to enhance gut function of infants, and on impact of human milk oligosaccharides and synthetic prebiotics on intestinal development and gene expression. She uses a variety of porcine models of human disease, including parenteral nutrition, rotavirus diarrhea, and inflammation. Dr. Donovan has published over 120 peer-reviewed articles, invited reviews, and book chapters and has garnered ~ $20,000,000 in research funding from NIH, USDA, private industry, and foundations. Dr. Donovan has received several awards in recognition of her research, including the Mead Johnson Award and the Norman A. Kretchmer Award from ASN.

DR. MARY K. ESTES

Dr. Mary K. Estes, Cullen Endowed Chair of Molecular and Human Virology and Professor in the Department of Molecular Virology and Microbiology (MVM) and Medicine-Gastroenterology, Baylor College of Medicine, is founding Director of the Digestive Diseases Center, which supports multi-institutional research in the Texas Medical Center. She is also founding Co-Director of a graduate program in Translational Biology and Molecular Medicine. Dr. Estes developed virus-like particle vaccines for gastroenteritis viruses and discovered new mechanisms of pathogenesis now being targeted for drug discovery. Dr. Estes has served on various committees devoted to research and vaccine development including the NIH's Virology Study Section, Research Advisory Committee to the Texas State Coordinating Board, U.N. Development Programme for Vaccine Development, WHO Diarrheal Diseases Vaccine Steering Committee, U.S.-Japan Cooperative Medical Sciences Program, and FDA Vaccines and Related Biological Products Advisory Committee. She is an elected Fellow of the American Academy of Microbiology and the AAAS and a member of the Institute of Medicine, the National Academy of Science, and the Academy of Medicine, Engineering and Science of Texas. She and her husband, who is a marine geologist enjoy sailing, fishing, and being grandparents.

DR. HARRY ISCHIROPOULOS

Dr. Harry Ischiropoulos is a professor at the University of Pennsylvania. His laboratory focuses on biological chemistry and signaling pathways of nitric oxide in the cardiovascular and neuronal systems. They have developed and applied novel biochemical and molecular methodologies in partnership with mass spectroscopy-based approaches to uncover nitric oxide-mediated post-translational protein modifications. Original work discovered tyrosine nitration as an oxidative modification mediated by nitric oxide under physiological conditions and in disease states. They also determined that the selective modification of cysteine residues by nitric oxide equivalents to generate S-nitrosocysteine regulates the function of enzymes participating in glycolysis, gluconeogenesis, tricarboxylic acid cycle, oxidative phosphorylation, and β-oxidation of fatty acids. The acquisition of endogenous S-nitrosocysteine proteomes has also enabled studies to explore the structural determinants that guide selective modifications of cysteine residues as well as to appreciate the global influence of nitric oxide on mitochondria function, bioenergetics, and metabolic processes.

DR. SHIHUAN KUANG

Dr. Shihuan Kuang received his Ph.D. degree from University of Alberta in 2002 and was a postdoctoral fellow in the U.S. and Canada. He became a member of the faculty of Animal Sciences, Purdue University, at the rank of assistant professor in 2008, and was promoted to associate professor in 2013. Dr. Kuang's basic research in adult stem cells and muscle-fat interaction translate to applications in both animal agriculture and human health. Specifically, he studies muscle stem cells that are responsible for muscle growth and meat production, as well as muscle repair and proper muscle function. In addition, his laboratory investigates the differentiation and plasticity of intramuscular fat that contributes to meat marbling and helps to rebuild damaged muscles. Furthermore, his team has recently identified a muscle hormone secreted from the double-muscled mouse that modulates the plasticity adipose tissues and insulin sensitivity. Dr. Kuang has published over 50 refereed journal papers and book chapters, served on the editorial board or as a referee for various journals and grant panels. His research has been supported by National Institutes of Health, American Cancer Society, Muscular Dystrophy Association, and United States Department of Agriculture.

DR. JACK ODLE

Dr. Jack Odle received his B.S. degree from Purdue University and M.S. and Ph.D. degrees from University of Wisconsin-Madison. He was an Assistant Professor at the University of Illinois; joined the Department of Animal Science at North Carolina State University in 1995; and was named William Neal Reynolds Distinguished Professor in 2005. At NCSU, he is a member of the Genomics, Biotechnology and Nutrition Faculties, the Center for Gastrointestinal Biology & Disease, and the Center for Comparative Medicine. Dr. Odle's uses pigs as a model for human infants, and his research encompasses nutritional biochemistry of the neonate and challenges that face both production agriculture and medical science. Dr. Odle has assisted the Bill & Melinda Gates Foundation and Scientists without Borders in seeking means to improve global maternal and infant health. He has received numerous awards, including two Young Investigator Awards, AFIA Nonruminant Nutrition Award, and American Society of Animal Science, Animal Growth and Development Award. Dr. Odle has garnered >$7,000,000 in research funding, published research in 335 papers, abstracts, and technical reports, and served on various grant review panels and journal editorial boards. He and his wife, Willa, have two children and one grandson and reside in Raleigh, NC.

DR. BERND SCHNABL

Dr. Bernd Schnabl is an Assistant Professor in Gastroenterology at the University of California, San Diego, School of Medicine. His main interest as a physician scientist is the pathogenesis of chronic liver disease resulting in liver fibrosis and cirrhosis. His laboratory at UCSD is particularly interested in the contribution of the gut microbiome, metagenome, and metabolome to the onset and progression of liver disease.

DR. JOSE SIMAS

Dr. Jose Simas joined Elanco in 2000 in Brazil, where he held a technical role for cattle products. He moved to the U.S. in 2002 in a global technical support role for Global marketing. In 2004, Dr. Simas took on the responsibility for the Argentina and Chile operations. He moved to Vienna, Austria, in November 2005 with sales, marketing, and technical responsibility for Elanco's operations in Central-Eastern Europe/North Africa/Middle Eastern markets.

Dr. Simas is currently responsible for the Companion Animal and Food Animal Production Product Development teams in the R and D organization of Elanco Animal Health. In the past 12 years, he has held several positions internationally and at the corporate office with the technical, sales and marketing, and R and D organizations. Dr. Simas received a B.S. from the Federal University of Lavras in Brazil. He has M.S. and Ph.D. degrees in Animal Nutrition and Physiology from the University of Arizona and was a postdoctoral fellow at the University of Sao Paulo in Brazil.

DR. KELLY SWANSON

Dr. Kelly Swanson received his Ph.D. degree in Nutritional Sciences at the University of Illinois (UI) in 2002. After receiving postdoctoral training in functional genomics, he became Assistant Professor in the Department of Animal Sciences at UI in 2004. He was promoted to Associate Professor with indefinite tenure in 2009. He is also a member of the Division of Nutritional Sciences and Department of Veterinary Clinical Medicine. Dr. Swanson teaches companion animal nutrition to veterinary, undergraduate, and graduate students, and has been named to the "List of Teachers Ranked as Excellent" 10 times over the past 7 years. On campus, he is the Pre-Vet Club faculty advisor, has served as mentor for many undergraduate research projects, and serves on many campus committees. Dr. Swanson has established himself as a leader in companion animal nutrition and nutrigenomics, primarily in the areas of gastrointestinal health and obesity. His research program has gained international recognition, highlighted by over 70 invited lectures at scientific meetings in 9 countries, many international research collaborations, invited review articles, awards, and service on 3 editorial boards. To date, he has published 93 peer-reviewed manuscripts and invited reviews, 6 book chapters, 48 conference proceedings, and over 140 scientific abstracts.

DR. CHRISTOPHER TUGGLE

Dr. Christopher Tuggle: Ph.D. Biochemistry, University of Minnesota, 1986. Professor, Molecular Genetics, Department of Animal Science and Chair, Interdepartmental Genetics Program, Iowa State University, Ames.

The areas of focus of Dr. Tuggle's research group include functional genomics and bioinformatics of the pig genome, especially as it relates to understanding control of appetite, reproduction, and the immune response. They use bioinformatic analysis of transcriptomic data to find important regulatory pathways controlling responses in these fields through comparison to similar data in human and other animals. They are interested in using these data to find gene expression patterns correlated with animal-to-animal phenotypic differences that can be used to select for superior animals. Finally, the Tuggle research group wants to apply porcine immune response molecular phenotypes as a model for improving human disease diagnosis. One of the most exciting projects in this area is the serendipitous discovery at Iowa State University of a mutation that causes Severe Combined Immune Deficiency in pigs. This novel finding will permit the development of a large-animal model for studying stem cells in many organ systems, including the immune system. Such a "humanized" pig model would be useful in studying how to combat human-specific diseases and in improving vaccines.

DR. GUOYAO WU

Dr. Guoyao Wu is a University Distinguished Professor, University Faculty Fellow, and AgriLife Research Senior Faculty Fellow in the Department of Animal Science, Texas A&M University. He received his B.S. degree in Animal Science from South China Agricultural University (1978-1982); M.S. degree in Animal Nutrition from China Agricultural University (1982-1984); and M.S. and Ph.D. degrees in Animal Biochemistry from University of Alberta in Canada (1984-1986 and 1986-1989, respectively). Dr. Wu undertook postdoctoral training in Biochemistry and Nutrition at McGill University (1989-1991) and Memorial University of Newfoundland (1991) in Canada. His research interests include biochemistry, nutrition and physiology of amino acids and proteins in animals at molecular, cellular, and whole body levels. He has published 396 papers in peer-reviewed journals, 48 book chapters, and 1 book entitled *Amino Acids: Biochemistry and Nutrition* (CRC Press, USA). His research has been supported by American Heart Association, Chinese Academy of Sciences, International Council of Amino Acid Sciences, International Glutamate Technical Committee, Juvenile Diabetes Research Foundation, National Natural Science Foundation of China, NIH, USDA, and industry. Dr. Wu is a member and elected Fellow of American Association for the Advancement of Science. He has received numerous prestigious awards from Canada, China, and the U.S.

POSTER PRESENTATIONS

P01 **A Porcine Model Studying Prophylaxis and Treatment of Neonatal Enteric Disease with Long-chain PUFA.** *S. Jacobi*, A. J. Moeser, L. Xi, J. Odle, and A. Blikslager, North Carolina State University, Raleigh, NC*

POSTER COMPETITION

P02 **Characterization Of A Novel Bacterium Capable Of Increasing Circulating Triglycerides In Swine.** *J. R. Donaldson*[*1], J. A. Carroll[2], T. B. Schmidt[3], T. R. Callaway[4] and J. G. Wilson[1], [1]Mississippi State University, Mississippi State, MS, [2]USDA-ARS, Lubbock, TX, [3]University of Nebraska, Lincoln, NE, [4]USDA-ARS, College Station, TX*

P03 **Increasing growth in broilers by serotonin modulation: The effects of selective serotonin reuptake inhibitors.** *W. J. Croom, Jr.*[*1], C. W. Nash[1], M. Koci[1], B. W. McBride[2], B. Clemmons[1] and J. T. Brake[1], [1]North Carolina State University, Raleigh, NC, [2]University of Guelph, Guelph, ON, Canada*

P04 **Poor Maternal Nutrition During Gestation Alters Postnatal Muscle Development in Lambs.** *S. A. Reed*, M. L. Hoffman, J. S. Raja, S. A. Zinn, and K. E. Govoni, University of Connecticut, Storrs, CT*

P05 **Effects of supplemental lysine and methionine in combination with zilpaterol hydrochloride on muscle fiber type, size and beta-adrenergic receptors in finishing feedlot cattle.** *J. E. Hergenreder*[*1], A. D. Hosford[1], P. W. Rounds[2] and B. J. Johnson[1], [1]Texas Tech University, Lubbock, TX, [2]Kemin Agrifoods North America Inc., Des Moines, IA*

P06 **Whole transcriptome profiling reveals differential expression of genes associated with monamine synthesis and transport that are accentuated by exogenous insulin in the hypothalamus of chickens selected for low or high body weight.** *W. Zhang*, S. Kim, R. Settlage, W. McMahon, M. A. Cline, P. B. Siegel, L. H. Sumners, and E. R. Gilbert, Virginia Polytechnic Institute and State University, Blacksburg, VA*

P07 **Effect of Maternal Docosahexaenoic Acid Supplementation on Foal Growth and Mare Reproductive Performance.** *A. M. Adkin*, L. K. Warren, and C. J. Mortensen, University of Florida, Gainesville, FL*

P08 **Chickens selected for low (LWS) or high (HWS) body weight differ in expression of metabolic flexibility-related genes in skeletal muscle and white adipose tissue.** *S. Zhang*, R. P. McMillan, W. Zhang, M. W. Hulver, P. B. Siegel, M. A. Cline, and E. R. Gilbert, Virginia Polytechnic Institute and State University, Blacksburg, VA*

P09 **Effect of L-Arginine Supplementation on Broodmare Uterine Involution and Foal Growth.** *A. M. Mesa* and C. J. Mortensen, University of Florida, Gainesville, FL*

P10 **Role of bovine Sirt1 in hepatic energy metabolism regulation.** *Y. Ghinis-Hozumi*[1], L. González-Dávalos[2], A. Antaramian[3], E. Piña[4], F. Villarroya[5], A. Shimada[2], A. Varela-Echavarría[3] and O. Mora[2], [1]Programa de Posgrado en Ciencias de la Producción y de la Salud Animal, FES-Cuautitlan, UNAM, México, Mexico, [2]RuMeN, FES-C, UNAM, Querétaro, Mexico, [3]Instituto de Neurobiología, UNAM, Querétaro, Mexico, [4]Facultad de Medicina, UNAM, México, Mexico, [5]IBUB, Universidad de Barcelona, Barcelona, Spain*

P11 **Maternal High Fat Diet Alters The Offspring Gastrointestinal Functions.** *C. de La Serre*, University of Georgia, Athens, GA*

P12 **Palmitoleic (C16:1) acid alters glucose and insulin metabolism in obese lambs.** *T. A. Burns*, N. M. Long, M. Alende, G. Volpi Lagreca, A. K. G. Kadegowda, M. C. Miller, and S. K. Duckett, Clemson University, Clemson, SC*

P13 **Metabolism of c9,t11- and t10,c12- CLA in 3T3-L1 adipose cell culture: novel metabolites.** *J. S. Church*[1], T. D. Turner[1], W. J. Meadus[2], D. C. Rolland[2], C. Mapiye[2], M. E. Dugan[2] and P. D. Duff[2], [1]Thompson Rivers University, Kamloops, BC, Canada, [2]Agriculture and Agri-Food Canada, Lacombe, AB, Canada*

P14 **Development of Novel Antibody Based Diagnostic Tests for Detection of Mycoplasma synoviae in Chickens.** *V. Durairaj* and N. M. Ferguson-Noel, The University of Georgia, Athens, GA*

POSTER PRESENTATIONS

P01 **A Porcine Model Studying Prophylaxis and Treatment of Neonatal Enteric Disease with Long-chain PUFA.** *S. Jacobi*, A. J. Moeser, L. Xi, J. Odle, and A. Blikslager, North Carolina State University, Raleigh, NC*

Dietary intervention is a potential therapy of enteric disease. Two objectives were investigated using *in vivo* and *in vitro* models of enteric disease. First, if supplementation of arachidonic acid (ARA) affects intestinal barrier repair in ischemic-injured ileum. Piglets consumed diets with 0% ARA, 0.5% ARA, 5% ARA, or 5% eicosapentaenoic acid (EPA). Following dietary enrichment, ileum segments were subjected to *in vivo* ischemia, and then control and ischemic loops were mounted on Ussing chambers. Transepithelial electrical resistance (TER) was measured over a recovery period. Ischemia-injured tissues from piglets fed the 5% ARA and 5% EPA exhibited enhanced recovery (% increase in TER = 13±13, 21.6±12, 59.1±12, 50.8±13, for 0% ARA, 0.5% ARA, 5% ARA, and 5% EPA, respectively, $P < 0.05$). Additionally, histology revealed reduced histological lesions of ischemic tissue from 5% ARA piglets verses other treatments ($P < 0.05$). Second, if PUFA enrichment of flagellin-challenged neonatal enterocytes would alter proinflammatory response. Porcine jejunal epithelial cells were supplemented with 30 μM PUFA for 96h. Cells were then stimulated with flagellin and cell RNA and media were harvested. Following supplementation ARA and EPA incorporation increased from 0.96 to 5.47 and 0.03 to 1.23 percent of FA, respectively ($P < 0.05$). TNFα mRNA expression was numerically increased ~2-fold from 0-24h, and ARA and EPA treatment overall increased TNFα mRNA. However, there was a decrease in protein secretion from 0-24h ($P < 0.05$), and ARA numerically decreased TNFα protein secretion compared to control ($P = 0.08$). These data demonstrate increased supplementation of long-chain-PUFAs has a protective and therapeutic effect on ischemic-injured ileum, and potentially modulate proinflammatory immune response in epithelial cells.

POSTER COMPETITION

P02 **Characterization Of A Novel Bacterium Capable Of Increasing Circulating Triglycerides In Swine.** *J. R. Donaldson*[*1], J. A. Carroll[2], T. B. Schmidt[3], T. R. Callaway[4] and J. G. Wilson[1], [1]Mississippi State University, Mississippi State, MS, [2]USDA-ARS, Lubbock, TX, [3]University of Nebraska, Lincoln, NE, [4]USDA-ARS, College Station, TX*

Weanling pigs are at a high risk of succumbing to illness primarily due to an immature immune system and insufficient supply of available energy at the time of weaning. Solutions have been investigated to supplement feed with alternate energy sources, yet limitations still arise with the utilization of these sources by young pigs due to their relatively immature gastrointestinal (GI) systems. Therefore, the objective of this study was to evaluate whether orally providing bacteria that produce large amounts of lipids to swine could increase the concentrations of circulating triglycerides (TAGs) and thus available energy. A novel strain of *Enterobacter sp.* previously identified by our research group was analyzed as a potential source of lipids in pigs. Thirty-six weaned pigs 30 days of age were randomly assigned to treatment groups, which consisted of daily oral supplementation for 5 days with: 1) *Enterobacter sp.* (JD6301; $1x10^{10}$ CFU); 2) an alternate form of this bacterium (JD8715; $1x10^{10}$ CFU) that secretes TAGs into the surrounding environment; or a control of PBS. Serum samples were collected every 6 hours and analyzed for non-esterified fatty acids, TAGs, free glycerol, and glucose concentrations. Fecal samples were collected daily to assess bacterial shedding by viable plate counts. At the conclusion of the trial, GI contents were collected and analyzed for colonization patterns of JD6301 or JD8715. Circulating TAGs increased by 84 hour in comparison to PBS controls for pigs supplemented with either JD6301 (P = 0.04) or JD8715 (P = 0.01). Both forms of *Enterobacter sp.* were present in the GI tract with minimal shedding observed, suggesting that these bacteria can colonize within the GI tract. These data indicate that oleaginous bacteria can be used as a source of utilizable lipids by weanling pigs. Further research is needed to determine whether this correlates with improved immune function in the presence of pathogens.

P03 **Increasing growth in broilers by serotonin modulation: The effects of selective serotonin reuptake inhibitors.** *W. J. Croom, Jr.*[*1], C. W. Nash[1], M. Koci[1], B. W. McBride[2], B. Clemmons[1] and J. T. Brake[1], [1]North Carolina State University, Raleigh, NC, [2]University of Guelph, Guelph, ON, Canada*

Preliminary studies in our laboratories suggest that administration of a selective serotonin reuptake inhibitor (SSRI) to growing broiler chicks (n=60; hatch to 21d) significantly increases growth, feed intake and feed efficiency. We administered a first generation SSRI, fluoxetine (Ft; Prozac®), subcutaneously over alternating breast muscles daily. Two levels of Ft equivalent to human dosages of 10mg and 20mg on a $BW^{.75}$ basis were administered to 20 broiler chicks, each, for 21 days. Amounts of Ft injected were adjusted every 3 days to account for growth. Controls (n=20) were administered excipient (0.9% saline). Bird weights (BW) and pen feed intake were recorded daily. At 17 - 21 days of age, three birds from both treatments and control were randomly selected and whole bird oxygen consumption as well as organ weights were measured. Ft increased BW 9% ($p<0.05$), feed intake 5% ($p<0.05$), and feed/gain 6% ($p<0.01$). Additionally, there was a trend for decreased oxygen consumption ($p<0.08$) with Ft administration. No changes in organ weights/kg BW were detected with the number of birds sampled. The mechanisms of growth enhancement seen with Ft administration have not yet been elucidated. Increases in feed intake and decreased resting metabolic rate may explain the observed growth. Additionally, the data were not clear as to which organs or physiological systems the increased energy associated with increased feed intake and decreased metabolic rate was partitioned. Stepwise linear regression analysis of the data suggested that increases in small intestinal and cecal weights/kg BW accounted for a significant amount of variation ($p<0.03$) associated with increased body mass. Similarly both cecal weight/kg BW and *Pectoralis* major muscle weight/kg BW accounted for significant variation associated with decreased whole bird oxygen consumption ($p<0.05$). Additionally, it is possible that mild sedation and the associated decreased stress on the birds may be responsible for growth enhancement. Further studies are planned by our group to more fully describe the effects and mechanisms of SSRI's on broiler growth.

P04 **Poor Maternal Nutrition During Gestation Alters Postnatal Muscle Development in Lambs.** *S. A. Reed*, M. L. Hoffman, J. S. Raja, S. A. Zinn, and K. E. Govoni, University of Connecticut, Storrs, CT*

Poor maternal nutrition, both over and under feeding, has negative consequences on muscle development. Potential mechanisms for these defects include the formation of fewer muscle fibers during development and/or alterations in regulators of muscle growth. We hypothesized that poor maternal nutrition would reduce muscle growth, reduce satellite cell number and alter gene expression in lambs. To test these hypotheses, 36 pregnant ewes were assigned to 100% (CON), 60% (RES) or 140% (OVER) of NRC requirements for ewes pregnant with twin lambs from d 31 ± 1.3 of gestation until parturition, at which time lambs were allowed to nurse for colostrum. Lambs were necropsied within 24 h of birth (n = 18) or maintained on a control diet until 3 mo of age (n = 16). Semitendinosus muscle was collected at necropsy for satellite cell isolation, immunohistochemistry, protein analysis and gene expression analysis. Data were analyzed using ANOVA. Muscle fiber cross sectional area (CSA) was increased 58% in RES and 47% in OVER lambs at birth ($P < 0.01$); however at 3 mo, CSA was decreased compared with CON lambs (15% and 17%, respectively; $P < 0.01$). At birth, lambs from poorly nourished ewes exhibited greater numbers of Type IIb fibers and fewer Type I fibers than CON lambs ($P < 0.001$), but exhibited no differences at 3 mo ($P = 0.9$). Poor maternal nutrition did not affect whole muscle mRNA expression of Pax7, MyoD or myogenin at birth or 3 mo ($P \geq 0.48$). At birth, myostatin mRNA expression was 2.9 ± 0.5-fold greater in OVER ($P = 0.07$) but was not different at 3 mo ($P = 0.36$). Follistatin mRNA expression was increased 1.7 ± 0.3 fold in RES ($P = 0.06$), and 1.9 ± 0.4 fold in OVER lambs at birth ($P = 0.04$) but not at 3 mo ($P = 0.37$). There was no difference in myostatin or follistatin protein expression ($P > 0.3$). Relative to CON, the number of Pax7(+) satellite cells was greater at birth in RES (55%; $P = 0.10$) and OVER (78%; $P = 0.05$) lambs, but was not different at 3 mo ($P = 0.8$). Poor maternal nutrition did not affect Pax7, MyoD or Myf5 gene expression in satellite cells at birth ($P \geq 0.2$). In conclusion, poor maternal nutrition alters prenatal and postnatal muscle development of offspring which may be the result of altered gene expression and satellite cell number.

P05 **Effects of supplemental lysine and methionine in combination with zilpaterol hydrochloride on muscle fiber type, size and beta-adrenergic receptors in finishing feedlot cattle.** *J. E. Hergenreder*[*1], A. D. Hosford[1], P. W. Rounds[2] and B. J. Johnson[1], [1]Texas Tech University, Lubbock, TX, [2]Kemin Agrifoods North America Inc., Des Moines, IA*

Beta-adrenergic receptors (β-AR) are a member of the G-coupled protein receptor family and are responsible for binding endogenous catecholamines and synthetically produced β-adrenergic agonists (β-AA). These β-AAs are established growth promoters that increase muscle accretion via hypertrophy of the muscle fiber, decrease lipogenesis, and improve feed efficiency when fed to finishing cattle. However, studies report desensitization of cells after extended exposure to β-AAs in part due to internalization of β-ARs. Little is known about the limiting amino acid requirements during this phase of rapid muscle growth and what their effects may be on β-ARs. Therefore, our objective was to evaluate the effects of feeding encapsulated amino acids in combination with zilpaterol hydrochloride (ZH) on muscle fiber type, size, and β-AR density in continental crossbred steers. Steers (n=180; initial BW=366 kg) were blocked by weight and randomly assigned to pens and 1 of 5 treatments including: 1) no amino acids and no ZH (Cont-); 2) no amino acids and ZH (Cont+); 3) encapsulated lysine supplement (Lys; LysiPEARL™) and ZH; 4) encapsulated methionine (Met; MetiPEARL™) and ZH; 5) encapsulated lysine and methionine (Lys+Met) and ZH. Zilpaterol HCl was fed for the last 20 d of the finishing period with a 3 d withdrawal. Lysine and Met were top dressed daily for 134 d. Steers were harvested at a commercial abattoir, and one steer per pen was selected for longissimus muscle histology analysis. Longissimus muscle samples were cryosectioned (10 μm) and immunoflourescence stained. Myosin heavy chain (MHC) type-I, IIA, IIX were identified and area was measured. Nuclei density per mm^2, β-1 adrenergic receptor (β-1AR), β-2 adrenergic receptor (β-2AR) densities per mm^2 were determined via Nikon imaging software analysis. Lys+Met MHC-I fiber area was increased compared to Lys and Met ($P \leq 0.05$). Lys+Met MHC-IIX fiber size was increased compared to Cont- ($P \leq 0.05$). The MHC-IIA fiber area tended to be greater for Lys+Met compared to Met and Cont- ($P < 0.15$). Proportion of MHC-IIA fibers were increased in Cont+ compared to Cont- and Met ($P \leq 0.05$). Negative control steers tended to have greater nuclei density compared to Lys+Met steers ($P < 0.15$). There was no difference in β-1AR or β-2AR densities between all treatments ($P > 0.15$). Muscle hypertrophy occurs with ZH administration, and its effect is enhanced with the addition of encapsulated amino acids during the finishing period. However, nuclei, β-1AR, and β-2AR densities were unchanged.

P06 **Whole transcriptome profiling reveals differential expression of genes associated with monamine synthesis and transport that are accentuated by exogenous insulin in the hypothalamus of chickens selected for low or high body weight.** *W. Zhang*, S. Kim, R. Settlage, W. McMahon, M. A. Cline, P. B. Siegel, L. H. Sumners, and E. R. Gilbert, Virginia Polytechnic Institute and State University, Blacksburg, VA*

Chickens from lines selected for low (LWS) or high (HWS) body weight for over 50 generations display differences in food intake, body composition and central insulin sensitivity. The LWS are hypophagic and lean, whereas the HWS are compulsive feeders with excess adipose tissue accumulation, and are resistant to the effects of insulin on food intake. We hypothesized that there were differences in gene expression in the hypothalamus, a region of the brain that ultimately regulates appetite, between LWS and HWS chickens, and that these differences are accentuated by exogenous insulin. The objective of this study was to measure whole transcriptomic changes in LWS and HWS hypothalamus in response to insulin treatment. Ninety-day old female LWS and HWS were intraperitoneally injected with insulin (80 μg/kg BW) or vehicle (n=5). Total RNA was isolated and quality assessed with an Agilent 2100 Bioanalyzer. RNA-Seq libraries were prepared with Illumina TruSeq DNA Library Preparation kits, followed by sequencing on a HiSeq 1000 using 101 SR. RNA-Seq reads were run through the Ensembl RNA-Seq pipeline using the chicken genome assembly (Ver. 4.0) and available cDNA for assembling and annotation. Differentially expressed genes (DEGs) were determined using the DESeq program with FDR<0.05 and fold changes of DEGs validated using real-time PCR. Total 361 DEGs were identified, of which 132 were revealed for HWS insulin-injected as compared to HWS vehicle-injected chickens, 196 for LWS insulin versus HWS insulin, 152 for LWS vehicle versus HWS vehicle, and 26 for LWS insulin versus LWS vehicle-treated chickens. Real time PCR results for 9 randomly selected genes were highly correlated with DESeq (R^2=0.88, P<0.0001). Among the DEGs, groups of genes related to appetite regulation, monoamine synthesis, transport and nutrient sensing were differentially expressed in response to exogenous insulin between HWS and LWS. The HWS chickens showed an acute response to insulin treatment with greater expression of genes involved in serotonin and dopamine synthesis and transport. This extensive overview of gene expression in response to exogenous insulin in diverse populations provides insight into appetite regulation and the role of insulin in food intake.

P07 **Effect of Maternal Docosahexaenoic Acid Supplementation on Foal Growth and Mare Reproductive Performance.** *A. M. Adkin*, L. K. Warren, and C. J. Mortensen, University of Florida, Gainesville, FL*

Maternal nutrition during gestation and lactation can influence the development and growth of both dam and offspring. Supplementation of the diet with omega-3 (*n*-3) fatty acids (FA) has been shown to increase gestation length, follicular growth, birth weight, and neonate viability in livestock, but has not been evaluated in horses. The objective of this study was to investigate foal growth, and postpartum reproductive performance in mares supplemented with docosahexaenoic acid (DHA). Twenty gravid, stock breed mares were randomly assigned to one of two dietary treatments: an *n*-3-rich fat supplement containing an algae source of DHA (n=10; Releira®, Arenus, St. Charles, MO) or a placebo fat supplement formulated to mimic the *n*-6:*n*-3 FA ratio (10:1) of the basal grain concentrate (n=10). Mares were supplemented from 90 d prior to expected foaling through 74 d lactation. Researchers were blinded to treatment. On average the DHA supplement provided 18.6, 10.5, and 2 g/d of total fat, total *n*-3 FA, and DHA, respectively. The basal diet included grain concentrate, bahiagrass pasture and Coastal bermudagrass hay. Milk and plasma collected from mares and foals was analyzed for FA composition. Parturition-related events were documented and foal body weight (BW) was recorded from birth to 2 mo of age. Postpartum mares underwent daily transrectal Doppler ultrasonography to measure folliculogenesis. Data were analyzed using a one-way ANOVA or a mixed model ANOVA with repeated measures using time, treatment and time*treatment as fixed effects and horse within treatment as a random variable. DHA-supplemented mares had a higher concentration of DHA in plasma ($P < 0.0001$) and milk ($P < 0.0001$) compared to placebo mares. Foals exposed to maternal DHA supplementation had a higher plasma DHA concentration ($P < 0.0001$) and a shorter latency to stand ($P = 0.09$) and nurse ($P = 0.02$) at birth compared to foals born to placebo-supplemented mares. Dietary treatment had no effect on gestation length (339.3 ± 2.7 d), latency of labor, latency of placental expulsion, placental weight, foal BW, or the growth of follicles ($P > 0.10$). Results indicate that low-level DHA supplementation does not alter gestation length, foal birth weight, or postpartum folliculogenesis, but does enhance neonate viability. Future research should investigate the clinical impacts of DHA supplementation on uterine health, fertility, and the long term effects on offspring growth and development. The authors would like to acknowledge Dr. Kenneth Kopp and Novus Nutrition Brands for support of this research.

P08 **Chickens selected for low (LWS) or high (HWS) body weight differ in expression of metabolic flexibility-related genes in skeletal muscle and white adipose tissue.** *S. Zhang*, R. P. McMillan, W. Zhang, M. W. Hulver, P. B. Siegel, M. A. Cline, and E. R. Gilbert, Virginia Polytechnic Institute and State University, Blacksburg, VA*

The Virginia lines of chickens are a unique model of anorexia and obesity that have resulted from more than 55 generations of selection for low (LWS) or high (HWS) body weight. We hypothesized that hyperphagia and obesity in juvenile HWS chickens are associated with metabolic inflexibility reflected by differences in expression of metabolic flexibility-related genes. We collected hypothalamus, liver, pectoralis major, gastrocnemius, abdominal fat, clavicular fat and subcutaneous fat from 28- and 56-day old LWS and HWS chickens (n=10, 5 males and females). The mRNA abundance of pyruvate dehydrogenase kinase 4 (*PDK4*), forkhead box O1A (*FoxO1*), peroxisome proliferator-activated receptor γ (*PPARγ*) and *PPARγ* co-activator α (*PGC1α*) was measured by real-time PCR. Expression data calculated using the DDC_T method were analyzed within each tissue by ANOVA using JMP 10.0. The statistical model included the main effects of sex, genetic line, age and the interactions between them. Means were separated using Tukey's Test. In 56-day old chickens, there was greater ($P < 0.05$) *PDK4* mRNA in HWS than LWS in pectoralis major, gastrocnemius, abdominal fat and subcutaneous fat, while lower ($P = 0.02$) *PDK4* abundance in clavicular fat, irrespective of age. There was an interaction of sex by line ($P = 0.002$) on *PDK4* mRNA in hypothalamus, where there was greatest expression in HWS females. Abundance of *FoxO1* showed a similar pattern as *PDK4*, where HWS chickens had greater *FoxO1* mRNA than LWS in the two skeletal muscles ($P < 0.01$) and at 56 days in subcutaneous fat ($P = 0.02$). In both clavicular fat and hypothalamus, there was an age x sex x line interaction, where *FoxO1* mRNA was greatest in LWS males at 28 and 56 days of age, respectively. Expression of *PPARγ* mRNA was greater ($P < 0.05$) in LWS than HWS in clavicular fat and subcutaneous fat at both ages, and at 28 days in the skeletal muscles. For *PGC1α*, in gastrocnemius there was greatest expression in HWS males ($P = 0.03$). The results suggest that the LWS chickens have greater metabolic flexibility and fatty acid oxidation efficiency due to down-regulation of pyruvate dehydrogenase to accommodate the influx of acetyl CoA from fatty acid oxidation in skeletal muscle and white adipose tissue. There appears to be coordinate regulation of *PDK4* and *FoxO1* mRNA. These data also demonstrate that different white adipose tissue depots and skeletal muscle types have distinct metabolic profiles.

P09 **Effect of L-Arginine Supplementation on Broodmare Uterine Involution and Foal Growth.** *A. M. Mesa* and C. J. Mortensen, University of Florida, Gainesville, FL*

L-Arginine is an essential amino acid in horses and has been shown to influence equine, porcine and human reproduction amongst other species after being orally supplemented. The objectives of this experiment were to evaluate reproductive parameters on mares supplemented with L-Arginine thought their last 90 days of gestation and first two weeks postpartum, and to verify if supplementation had an effect on the size and growth rate of resulting offspring. For this purpose, 16 light-horse mares were randomly divided in two groups: 8 mares supplemented with 0.05% of their estimated daily intake with L-Arginine and 8 mares fed an isonitrogenous equivalent supplement of urea. Gestation length, days to uterine clearance and days to first postpartum ovulation were compared between groups. Uterine body depth, diameter of the gravid and non-gravid horns, and length of the largest pocket of uterine fluid was recorded daily via transrectal ultrasound to estimate uterine involution. Weekly measurements of foal weight, height, and cannon bone circumference were recorded. Arginine treatment had no effect on gestation length ($P=0.583$), with a mean ±SEM of 338.8 ± 3.59 days for the arginine treatment and 336.1 ± 2.98 days for the isonitrogenous control. However, L-arginine supplemented mares cleared postpartum fluid quicker (6.8 ± 0.53 days; $P < 0.05$) compared to isonitrogenous control mares (9.0 ± 0.38 days). Additionally, mares supplemented with L-arginine had a tendency for overall smaller diameter of fluid present in the postpartum uterus ($P \leq 0.05$). Days to first postpartum ovulation were not affected by dietary treatment. Treatment had no effect on uterine body depth, and formerly gravid and non-gravid uterine horn diameters. Arginine treatment had no effect on any of the foal's measured parameters (weight, height, cannon ball diameter) during the 9 week evaluation period. L-Arginine supplementation fed at 0.05% of daily intake during the last 90 days of gestation and early postpartum in mares decreased uterine fluid accumulation postpartum, yet did not appear to have any effect on uterine involution or on any of the foal growth parameters measured.

P10 **Role of bovine Sirt1 in hepatic energy metabolism regulation.** *Y. Ghinis-Hozumi*[*1], *L. González-Dávalos*[2], *A. Antaramian*[3], *E. Piña*[4], *F. Villarroya*[5], *A. Shimada*[2], *A. Varela-Echavarría*[3] *and O. Mora*[2], [1]*Programa de Posgrado en Ciencias de la Producción y de la Salud Animal, FES-Cuautitlan, UNAM, México, Mexico,* [2]*RuMeN, FES-C, UNAM, Querétaro, Mexico,* [3]*Instituto de Neurobiología, UNAM, Querétaro, Mexico,* [4]*Facultad de Medicina, UNAM, México, Mexico,* [5]*IBUB, Universidad de Barcelona, Barcelona, Spain*

Sirt1, a Class III NAD+-dependent histone and non-histone protein deacetylase, has been widely proven to regulate energy homeostasis, especially under low nutrient availability. This enzyme deacetylates proteins involved in the regulation of glucose and lipid metabolism. Among its target proteins, we count the transcription factors (TFs) from the Forkhead box O family, as well as the transcriptional coactivator PGC-1α.

FoxO TFs regulate energy metabolism, development, aging, apoptosis, and cellular response to oxidative stress, among other biological functions. These proteins exert their effects through post-translational modifications such as phosphorylation, acetylation, and ubiquitination. They are responsive to different external stimuli such as growth factors, insulin, nutrient availability, oxidative stress, and others.

PGC1α plays a key role in energy metabolism regulation, mitochondrial biogenesis, and adaptive thermogenesis, mainly. This coactivator promotes gluconeogenesis and fatty acid oxidation in liver under nutrient deprivation conditions.

Since these genes (Sirt1, FoxO1, FoxO3a, and PGC1α) are all correlated with energy metabolism, especifically with the promotion of expression of genes involved in gluconeogenesis and fatty acid oxidation, the aim of this work was to determine how they behave in bovines, used as model organism for ruminant species. Therefore, we incubated liver slices for 60 min. in Krebs-Ringer Buffer (KRB) with no nutrients and with propionate and glucagon (induction group, KRB-I). Besides, we incubated liver slices with KRB or KRB-I with and without adding 40 or 80 μM resveratrol, a natural polyphenol known to activate Sirt1. We then quantified the expression of these genes by real-time PCR.

We found that Sirt1 expression was decreased by the incubation time, while expression of the other genes of interest was increased. Incubation with KRB with 40 μM resveratrol promoted the highest expression of Sirt1 and the FoxOs, while KRB-I resulted in the highest expression of PGC1α, all with respect to the control.

No previous reports on the effects of resveratrol on bovine Sirt1 expression are available. Therefore, since we could notice that addition of propionate and glucagon tended to have a negative effect on the expression of the studied genes, we can elucidate that, in ruminants, their normal metabolic state does not require a high expression of gluconeogenic or lipolytic genes. However, after 60 min. of incubation with no other nutrient than propionate being available, we saw a higher expression of these genes with respect to the control and this was probably because they were regulating the cell metabolism gene expression to ensure survival.

P11 **Maternal High Fat Diet Alters The Offspring Gastrointestinal Functions.** *C. de La Serre*, University of Georgia, Athens, GA*

Maternal obesity is associated with an increased risk for obesity in the offspring. Recent work in a rodent model has shown that consumption of high fat food leads to delocalization of tight junction proteins and an increase in gastrointestinal (GI) permeability. Alteration in GI functions is correlated with weight gain. In the present study we investigated the effect of maternal obesity on the offspring gastrointestinal functions. Mice were fed a high fat diet six weeks prior to mating and during gestation and lactation. GI permeability was measured via oral dextran gavage in the offspring at post-natal day 21, maternal obesity leaded to a significant increase in GI permeability. Maternal high fat feeding also affected tight junction protein localization. Alteration of GI functions is associated with weigh gain and systemic inflammation and can contribute to the development of obesity

P12 **Palmitoleic (C16:1) acid alters glucose and insulin metabolism in obese lambs.** *T. A. Burns*, N. M. Long, M. Alende, G. Volpi Lagreca, A. K. G. Kadegowda, M. C. Miller, and S. K. Duckett, Clemson University, Clemson, SC*

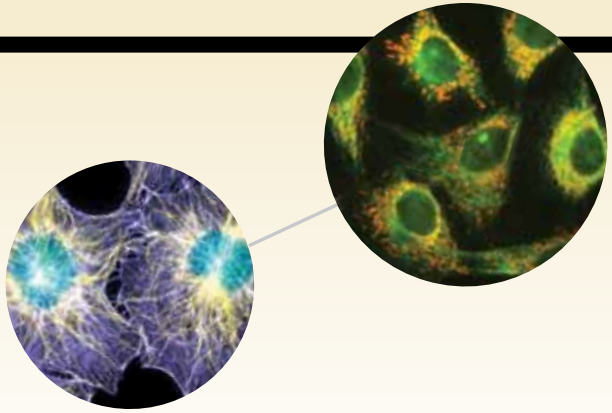
Palmitoleic (C16:1) acid has been proposed to function as a lipokine and alter insulin sensitivity. The objective of this study was to assess C16:1 effects on glucose and insulin tolerance in obese sheep. Southdown wethers (86.7 ± 1.5 kg BW; n = 4) with indwelling jugular catheters were used in a crossover design. Treatments were intravenous infusion of two doses of C16:1, 0 (CON) or 5 (LIPO) mg/kg BW in 40% (v/v) ethanol, immediately followed by a glucose (0.25 g/kg) tolerance test. The design was repeated with CON and LIPO treatments immediately followed by an insulin (0.02 μU/kg) challenge. Catheters were inserted 1 d prior to first infusion. Lambs were fasted 18 h prior to treatment with 44 h rest between tests. Blood samples were collected at -15 min, immediately prior to infusion, and serially for 4 h. Plasma was analyzed for fatty acids, glucose, and insulin using GLC, colorimetric assay, and commercial RIA, respectively. Repeated measures data were analyzed using Proc Mixed procedure. Plasma C16:1 was increased ($P < 0.01$) in LIPO compared with CON lambs. At 2 min post-infusion, percent C16:1, mg of C16:1/mL of serum, and ratio of C16:1/C16:0 was maximal with 9.5-, 10.9-, and 10.6-fold increase over baseline, respectively. By 30 min post-infusion, plasma C16:1 levels had returned to baseline. In addition, C17:0 and C18:0 (mg/mL) increased ($P < 0.05$) in LIPO compared with CON lambs. During the glucose tolerance test, C16:1-administration increased ($P < 0.05$) peak and overall glucose concentrations in LIPO lambs compared with CON. Glucose peaked at 2 min post-infusion and returned to baseline in both treatment groups by 180 min. During the insulin test, LIPO lambs had increased ($P < 0.05$) peak and overall plasma insulin compared with CON; in addition, glucose was greater ($P < 0.05$) in LIPO compared with CON lambs. Insulin peaked at 2 min post-infusion and returned to baseline in both treatment groups by 20 min. In conclusion, fatty acid profiles indicated rapid removal of C16:1 from plasma after pulse dose infusion. In addition, C16:1 infusion appears to affect insulin signaling to alter plasma glucose in obese lambs.

P13 **Metabolism of c9,t11- and t10,c12- CLA in 3T3-L1 adipose cell culture: novel metabolites.** *J. S. Church*[*1], *T. D. Turner*[1], *W. J. Meadus*[2], *D. C. Rolland*[2], *C. Mapiye*[2], *M. E. Dugan*[2] *and P. D. Duff*[2], [1]*Thompson Rivers University, Kamloops, BC, Canada,* [2]*Agriculture and Agri-Food Canada, Lacombe, AB, Canada*

Adipose tissue is an active regulatory tissue producing cytokines with immunomodulating effects that can be influenced by fatty acids (FA). Conjugated linoleic acid (CLA), with *c*9,*t*11-CLA being the predominant isomer in ruminant products, and *t*10*c*12-CLA largely of industrial origin, purportedly have substantial anti-arithrogenic, anticarcinogenic and anti-obesity effects. The fate of CLA's has not been investigated in adipose tissue, prompting investigations into effects on the FA profile. Mature 3T3-L1 cells were incubated with 70µM *c*9,*t*11-CLA, *t*10,*c*12-CLA or 18:2n-6. Lipid profiles of cells harvested at 144h were analysed by GC-FID, and FA with conjugated bonds were separated and collected by Ag^{+}-HPLC then analysed by GC-MS-EI to identify novel metabolites. Cultures incubated with *c*9,*t*11-CLA and 18:2n-6 had similar lipid content, whereas *t*10,*c*12-CLA induced a two-fold reduction. Conjugated diene metabolites with 16-, and 20-carbons were identified for *c*9,*t*11-CLA, similar *t*10,*c*12-CLA metabolites were also present, along with a 14-carbon diene. Previously unreported conjugated trienes assumed to be *c*6,*c*9,*t*11/*c*6,*t*10,*c*12-18:3 and *c*8,*c*11,*t*13/*c*8*t*12*c*14-20:3 were identified. Indices for desaturation, elongation and β-oxidation activity were affected differently by the CLA isomers. Stearoyl-coA desaturase (SCD) is a key factor in lipogenesis, and *t*10*c*12-CLA reduced the desaturation index, supportive of its known delipidation effects. Higher indices for β-oxidation corresponding to *t*10*c*12-CLA further support these delipidation effects. *c*9*t*11-CLA seemed to be metabolised more readily, first undergoing desaturation to a triene, then elongation to a 20-carbon conjugate, possibly competing for eicosanoid production pathways. Alternatively, *t*10*c*12-CLA was also readily desaturated to a triene, where it tended to accumulate without further elongation. Triene metabolites of both CLA isomers underwent β-oxidation, however, the *c*9*t*11-CLA metabolite seemed to undergo greater β-oxidation, possibly due to less overall functional metabolism of the *t*10*c*12-CLA isomer. Elongase indices suggest both CLA isomers were elongated to 20-carbon conjugates, however, *c*9*t*11-CLA seemed more readily desaturated to 22-carbon conjugate. Present results indicate that CLA isomers can undergo further desaturation and elongation in adipose tissues, possibly producing precursors which can act as substitutes for 20:3n-6 or 20:3n-3 for eicosanoid production. Further effects of these metabolites need to be investigated.

P14 **Development of Novel Antibody Based Diagnostic Tests for Detection of Mycoplasma synoviae in Chickens.**
V. Durairaj and N. M. Ferguson-Noel, The University of Georgia, Athens, GA*

Mycoplasma synoviae (*M. synoviae*) is an infectious pathogen in chickens affecting synovial membrane of the joints and bursa. *M. synoviae* also affects the respiratory system and causes subclinical/clinical signs. *M. synoviae* causes devastating economic losses in the poultry industry. *M. synoviae* is transmitted by vertical and horizontal routes. Aerosol exposure is one of the common routes for transmission which causes mucosal insult in the trachea and stimulates the production of IgA antibodies. This further leads to a systemic infection resulting in production of IgG antibodies. Serological assays are used as first line diagnostic assays in monitoring and surveillance programs. The objectives of this research were to develop diagnostic assays for *M. synoviae* by the detection of (a) IgA antibodies by a fluorescence antibody culture test (FACT) and (b) IgG antibodies by the determination of serum neutralization titer. For the FACT test *M. synoviae* colonies were grown on Frey's modified agar in a 48 well plate. Tracheal supernatant was used as a primary antibody followed by a secondary anti-chicken IgA antibody conjugated with FITC. Based on presence and absence of immunofluorescence signals, the positive and negative samples were identified respectively. FACT was conducted on the tracheal swabs collected from three-week-old experimentally infected SPF chickens. The samples (blood, choanal cleft swabs, tracheal swabs) were collected on alternate days from 3-days post infection to 11-days post infection. The samples were analyzed by serum plate agglutination test, hemagglutination inhibition test, ELISA, conventional culture test, real-time PCR and FACT. Positive reactions were observed as early as 3 days by FACT, conventional-culture test and real-time PCR. For the second objective of this research, serum neutralization antibody titers against *M. synoviae* were determined. Serum neutralization assay was performed by adding a standard concentration of *M. synoviae* culture to diluted levels of serum. The results of this assay were read ten days later based on color changing units. No color change indicated presence of neutralizing antibodies and color change to yellow indicated absence of neutralizing antibodies. This assay provides both quantitative and qualitative results.



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