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PROGRAM BOOKLET

2025
PERINATAL BIOLOGY SYMPOSIUM

Perinatal Programming: Challenges and Solutions for Optimizing Long-Term Health

VIEWLINE RESORT SNOWMASS
SNOWMASS, CO
AUGUST 16 – 19

SAFETY STATEMENT

The Perinatal Biology Symposium is committed to providing a safe, productive, and welcoming environment for all conference participants and staff. Participants are asked to maintain professionalism at the symposium.

Per policy, unallowable behaviors include but are not limited to:

- Harassment, intimidation, or discrimination in any form, including threats or stalking behaviors.
- Physical or verbal abuse of any participant, volunteer, staff/service member, or guest.
- Disparagement of gender, sexual orientation, disability, body, race, religion, or nationality.
- Inappropriate/unprofessional imagery in public spaces or presentations.
- Disruption of presentations or other events.
- Inappropriate or vulgar dress.

The Perinatal Biology Symposium and its partners will maintain a zero-tolerance policy for any form of discrimination or harassment.

If you experience or witness harassment or other unallowable behaviors, we ask that you inform the conference organizers at the bottom of this page so that appropriate action can be taken.

The hosts reserve the right to take necessary action, including unrefunded removal from the meeting and prohibited attendance at future meetings.

For questions, concerns, or complaints related to this policy, please contact the conference organizers.

Information on how to file a complaint with HHS Office for Civil Rights can be found on their webpage (HHS.gov). HHS Office for Civil Rights complaints may be filed independently of any action by or communication with the hosts. Likewise, seeking assistance from the organizers does not prohibit filing complaints with HHS Office for Civil Rights.

Snowmass Village offers the following summer safety tips:

1. **Sun Protection**: Apply SPF 30+ sunscreen generously and reapply every two hours on exposed skin. Wear long sleeves, pants, and a hat/helmet, even on warm days. Use sunglasses/goggles with 100% UV protection.

2. **Hydration**: Drink plenty of water. Altitude can increase the risk of dehydration. Carry a water bottle or hydration pack and drink frequently. Stay hydrated, don't wait until you're thirsty to drink. Drink water throughout the day.

3. **Wildlife Awareness**: Be cautious. Keep a safe distance from wildlife, especially moose, mountain lions, and bears. Never feed wildlife. Feeding wildlife can be dangerous for both you and the animals. Be aware of your surroundings. Stay on marked trails and avoid approaching animals, especially if they appear startled or agitated. If you encounter a bear, make noise, appear large, and slowly back away.

4. **Terrain Awareness**: Choose appropriate trails. Select trails that match your skill level. For bike safety, wear a helmet, eye protection, and gloves, and consider padded bike shorts. Stay on marked trails. Avoid closed trails and areas. Be visible. If you stop, choose a visible spot and avoid obstructing others.

Thank you,

2025 Perinatal Biology Symposium Organizing Committee

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2025
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THANK YOU TO OUR SPONSORS

WELCOME!

Dear Scientists,

Welcome to Snowmass! And thank you for being a part of the preeminent meeting for perinatal biologists around the globe. By our calculations, the 2025 meeting will mark the 8th iteration of the symposium, although this number is debatable and could be as high as 10th or 11th... it's a long story. (See the "*About the Perinatal Biology Symposium*" section at the end of this booklet if you enjoy long stories). What's not debatable is the success of this triennial meeting-of-the-minds and the benefits it has bestowed to our respective and collective fields of study since its fledgling start in 2000 (or was it 2007? 2004?). The goal of the symposium is to provide a mechanism that effectively facilitates ***transdisciplinary scientific interactions***, which are essential for building and developing productive translational research endeavors. If you have attended PNB before, you already know that the meeting brings together ***global experts*** in reproductive success, maternofetal medicine, developmental programming of health and disease, and neonatal care in humans, agricultural animals, wildlife, and animal models, which is the key to its effectiveness. If this is your first go-around, here is a quick overview of the symposium's objectives that help make it the one-of-a-kind meeting that it is:

OBJECTIVE 1. COMMUNICATE NEW SCIENTIFIC DISCOVERIES THAT ADVANCE PERINATAL OUTCOMES IN HUMANS & AGRICULTURAL ANIMALS. Speakers and attendees represent broad expertise in the cellular mechanisms for normal and pathological maternal status, placental function, and fetal growth and development during pregnancy. These scientists are integral to understanding how intrauterine environments impact fetal and postnatal health and vigor, as well as long-term maternal well-being. Other speakers and attendees bring knowledge in broad areas of neonatal health and interventive care. The talks and poster presentations you will see include postnatal nutritional, supplemental, and pharmacological strategies for common conditions such as premature birth, low birthweight, and insufficient lactation in newborn infants, livestock, animal research models, and even wildlife animals.

OBJECTIVE 2. DISCUSS & DISSEMINATE NOVEL RESEARCH APPROACHES FOR ADDRESSING EXISTING & EMERGING CHALLENGES TO PERINATAL SUCCESS. This is a broad one, for sure. Here's what it means. As new issues related to maternal, fetal, and newborn health arise, new methods for studying and resolving them must be created in kind. Some challenges stem from conditions that have emerged anew or worsened, like climate change, poor nutrition and lifestyle, and global poverty patterns. Other challenges have arisen simply from new or better information about common elements. Examples of this include chemical fire retardants, high-fat foods, and microbiome-influencing medications. Cutting-edge research tools and technologies like omics, genetic animal models, and experimental induction of disease states are paramount to meeting these challenges. This meeting facilitates collaborations that extend the impact of research efforts beyond the skills and expertise of any individual scientist. PNB's unique schedule provides formal and informal opportunities for discussions necessary for creating these collaborations.

OBJECTIVE 3. STRENGTHEN THE NEXT GENERATION OF PERINATAL SCIENTISTS BY FOSTERING THE GROWTH OF TRAINEES & EARLY-CAREER INVESTIGATORS. Since its inception, organizers of the Perinatal Biology Symposium have prioritized providing unique career development opportunities for young scientists beyond what is offered at most other national/international scientific meetings. Each day begins with trainee workshops on career-building topics like effective scientific writing, the publication process, managing diverse teams, or starting a new research lab. Scientific sessions include talks by trainees and early-career investigators, who will present their work right alongside established scientists. Trainees pay less than one-half the standard rate for registration but are welcome at all Symposium activities, including banquets, team-building and social activities, and the business meeting. Moreover, trainees can have their novel research recognized by awards and invitations to present in the scientific sessions.

We are very pleased you have chosen to attend this unique meeting. We hope that your experience is as exciting as we have found it to be over the years! Please let us know if there is anything we can do to make your experience an even better one!

-The Organizing Committee

***P.S. KEEP AN EYE OUT FOR THE PERINATAL BIOLOGY SPECIAL COLLECTION THAT WILL BE PUBLISHED IN THE *JOURNAL OF ANIMAL SCIENCE* IN EARLY 2026, FEATURING ORIGINAL CONTRIBUTIONS FROM MANY OF THE 2025 PBN SPEAKERS!

2025 Perinatal Biology Symposium

Perinatal Programming: Challenges and Solutions for Optimizing Long-Term Health

August 16 – 19, 2025

Day 1 – August 16th

5:00–7:45 pm Registration
5:30–6:30 pm Opening reception
6:30–6:45 pm Welcome and opening remarks: Organizing Committee
6:45–7:45 pm **2025 Keynote Address:** Teresa Davis, PhD (Texas A&M University)
Impact of Prematurity on the Nutritional Regulation of Growth

Day 2 – August 17th

6:45–8:15 am Continental Breakfast
7:00–8:00 am **Trainee Workshop 1:** Effective Scientific Writing
Chair: Sarah Reed, PhD (University of Connecticut)
Panelists: Sean Limesand, PhD (University of Arizona)
Kurt Albertine, PhD (University of Utah School of Medicine)

SESSION I – Early Influences on Growth & Reproductive Competence
Chairs: Rebecca Swanson, PhD (South Dakota State University)
Ryan Ashley, PhD (New Mexico State University)

8:15–8:45 am Jay Ramadoss, PhD (Wayne State University)
Sexual Dimorphism in E-cigarette-Induced Developmental Lung Immune Adaptations
8:45–9:15 am **Early Career Speaker**: Caitlin Cadaret, PhD (Colorado State University)
Investigating the Impacts of Maternal Environment on Lamb Performance & Reproductive Dynamics
9:15–9:45 am **Early Career Speaker**: Upasna Sharma, PhD (University of California-Santa Cruz)
Intergenerational Transmission of Paternal Environmental Effects via Sperm Small RNAs in Mice
9:45–10:15 am **Early Career Speaker:** Amy Desaulniers, PhD (University of Nebraska-Lincoln)
Developmental Origins of Boar Fertility: Consequences of In Utero Heat Stress

10:15–10:45 am Break

SESSION II – Health Consequences of Placental Insufficiency
Chairs: Rosa Luna-Ramirez, PhD (University of Arizona)
Sarah Reed, PhD (University of Connecticut)

10:45–11:15 am Emilyn Alejandro, PhD (University of Minnesota)
Unlocking Metabolic Fate: How Placental Insulin/IGF-1 & mTOR Signaling Shape Offspring Health
11:15–11:45 am Larry Reynolds, PhD (North Dakota State University)
Maternal Nutrition & Developmental Programming – Lessons from Livestock Models
11:45–12:15 pm Helen Jones, PhD (University of Florida)
Sexual Dimorphism in Placental Insufficiency
12:15–12:30 pm **Trainee Talk**–TBD
TBD
12:30–12:45 pm **Trainee Talk**–TBD
TBD

12:45–6:00 pm **Informal Networking** / Go Enjoy the Mountains!

SESSION III – MATERNAL HEALTH/DIET & PLACENTAL FUNCTION

Chairs: Caitlin Cadaret, PhD (Colorado State University)
Kurt Albertine, PhD (University of Utah School of Medicine)

6:00–6:30 pm Theresa Powell, PhD (University of Colorado Anschutz Medical Campus)
Maternal-Placental-Fetal Cross Communication: Impact on Pregnancy Outcomes & Life-long Health

6:30–7:00 pm Sathish Kumar Natarajan, PhD (University of Nebraska-Lincoln)
Palmitoleate Protects against Zika Virus-Induced Endoplasmic Reticulum Stress & Apoptosis in Neurons

7:00–7:30 pm **Early Career Speaker:** Jennifer Thompson, PhD (University of Calgary)
Revisiting the Adipocyte Number Hypothesis & Its Implications for Developmental Programming

7:30–8:00 pm **Trainee Flash Talks:** TBD (x 5)
Overview: 5-minute research overviews, 1-2 questions each

8:00–9:30 pm **POSTER SESSION I**

Day 3 – August 18th

6:45–8:15 am Continental Breakfast

7:00–8:00 am **Trainee Workshop 2:** WHAT TO KNOW ABOUT THE PUBLISHING PROCESS

Chair: Teresa Davis, PhD (Texas A&M University)
Panelists: Sarah Reed, PhD (University of Connecticut)
Sean Limesand, PhD (University of Arizona)

SESSION IV – ART, REPRODUCTIVE ENDOCRINOLOGY, & OFFSPRING HEALTH

Chairs: Nicole Tillquist, PhD (University of Connecticut)
Dusty Yates, PhD (University of Nebraska-Lincoln)

8:15–8:45 am Monica Mainigi, MD (University of Pennsylvania)
Assisted Reproductive Technologies & Adverse Perinatal Outcomes: The What, Why, & How?

8:45–9:15 am **Early Career Speaker:** Lisa Vrooman, PhD (Oregon Health Science Center)
Investigating Placental Protein Changes Associated with ART in a Mouse Model

9:15–9:45 am **Early Career Speaker:** Colin Conine, PhD (University of Pennsylvania)
The Immune System Regulates Sperm RNAs to Drive Transgenerational Epigenetic Inheritance in Mice

9:45–10:15 am **Early Career Speaker:** Maria Gracia Gervasi, PhD (University of Connecticut)
Metabolic Manipulation of Spermatozoa Improves Sperm Function with Implications for ART

10:15–10:45 am **Early Career Speaker:** Jessye Wojtusik, PhD (Omaha's Henry Doorly Zoo & Aquarium)
From Rhinos to Sea Stars: Innovative Applications of ART to Support Wildlife Conservation

10:45–11:00 am Break

SESSION V – REGULATION OF IMPLANTATION & PLACENTATION

Chair: Rachel Gibbs, PhD (North Dakota State University)
Ryan Ashley, PhD (New Mexico State University)

11:00–11:30 am Soumen Paul, PhD (University of Kansas Medical Center)
Arginine Methyl Transferase & Epigenetic Equilibrium in Trophoblast Development to Prevent Early Pregnancy Loss

11:30–12:00 pm Jane Cleal, PhD (University of Southampton)
Maternal Exposures & Molecular Regulation of the Human Placenta & Endometrium

12:00–12:30 pm Mike Soares, PhD (University of Kansas Medical Center)
Trophoblast Cells at the Uterine-Placental Interface

12:30–12:45 pm **Trainee Talk**–TBD
Overview: TBD

12:45–1:15 pm **Trainee Flash Talks:** TBD (x 5)
Overview: 5-minute research overviews, 1-2 questions each

1:15–6:00 pm **Free Time and Networking**

6:00–7:30 pm **Conference Dinner and Presentation of Awards**

7:30–8:30 pm **DJP Barker Memorial Lecture:** Laura Brown, MD (University of Colorado Anschutz Medical Campus)
Reduced Myogenesis in Fetal Growth Restriction: Basic Mechanisms & Future Therapies

8:30–9:45 pm **POSTER SESSION II**

Day 4 – August 19th

6:45–8:15 am Continental Breakfast

7:00–8:00 am **Trainee Workshop 3:** Tips for Starting Your Independent Research Lab
Chair: Stephanie Wesolowski, PhD (University of Colorado Anschutz Medical Campus)
Panelists: Allison Meyer, PhD (University of Missouri)
Rachel Gibbs, PhD (North Dakota State University)

SESSION VI – The Role of Nutrition in the Development of the Newborn
Chairs: Amelia Tanner, PhD (University of Colorado Anschutz Medical Campus)
Virginia Winn, MD, PhD (Stanford University)

8:15–8:45 am Susan Carlson, PhD (University of Kansas School of Medicine)
DHA & Preterm Birth: What We've Learned Since the 2018 Cochrane Review

8:45–9:15 am Paula Meier, PhD (Rush University School of Medicine)
Mothers' Own Milk Provides Personalized Nutrition & Protection to Optimize Short/Longterm Health in Term/Preterm Infants

9:15–9:45 am **Early Career Speaker:** Hannah Hollinger, PhD (University of Wyoming)
The Pre-Ruminant Microbiome: Why Do We Care & Where Does It Come From?

9:45–10:00 am **Trainee Talk**–TBD
Overview: TBD

10:00–10:15 am Break

SESSION VII – Environmental Exposure & Toxins during Pregnancy
Chairs: Allison Meyer, PhD (University of Missouri)
Stephanie Wesolowski, PhD (University of Colorado Anschutz Medical Campus)

10:15–10:45 am Clyde Wright, MD (University of Colorado Anschutz Medical Campus)
Is the Developing Lung Susceptible to Acetaminophen Toxicity?

10:45–11:15 am Carrie McCurdy, PhD (University of Oregon)
Effect of Metformin Use during Pregnancy on Fetal Skeletal Muscle Growth & Metabolism in Rhesus Macaques

11:15–11:45 am Michael Golding, PhD (Texas A&M University)
Paternal Drinking & the Epigenetic Influences on Mitochondrial Function, Placental Dysfunction, & Structural Birth Defects

11:45–12:15 pm Almudena Veiga-Lopez, DVM, PhD (University of Illinois-Chicago)
Placental Vulnerability at the Molecular Level: The EGFR Axis under Chemical Stress

12:30–1:00 pm Conference Closure–Final comments and election of the next Organizing Committee

ABSTRACTS

**Alphabetical by presenting author*

1. Unlocking metabolic fate: How placental insulin/IGF-1 and mTOR signaling shape offspring health.

Emilyn U. Alejandro*
University of Minnesota Medical School, Minneapolis, MN, USA

Both genetic and environmental factors contribute to the development of Type 2 diabetes (T2D). Hyperinsulinemia is frequently observed in pregnant women with prediabetes, obesity, or gestational diabetes, and their offspring are at increased risk of developing T2D. However, there is a lack of longitudinal studies examining the long-term metabolic outcomes in offspring of hyperinsulinemic mothers. Moreover, the mechanistic link between maternal hyperinsulinemia and the programming of metabolic disease in offspring remains poorly understood. The prevailing view is that insulin does not cross the placenta to regulate fetal growth directly. Nonetheless, maternal insulin can function as a growth factor and anabolic hormone by binding to insulin receptors (IR) and insulin-like growth factor 1 receptors (IGF1R) on the placenta. This interaction can drive key placental functions, including nutrient transport to the fetus. As a result, maternal insulin may indirectly alter fetal development by modifying placental nutrient delivery to fetal metabolic tissues, potentially causing permanent changes that predispose offspring to T2D in adulthood. This seminar will examine the metabolic phenotypes of offspring born to hyperinsulinemic murine dams. It will also highlight findings on metabolic outcomes in offspring from otherwise normal pregnancies where insulin or IGF1 receptors were selectively deleted in the placenta during gestation. Finally, evidence from genetic mouse models demonstrating that both loss- and gain-of-function alterations in placental mTOR signaling can significantly influence susceptibility to type 2 diabetes will be presented. These effects appear to be mediated through changes in placental nutrient transport and subsequent altered nutrient sensing by fetal pancreatic beta cells in the offspring. Collectively, these observations support the concept that insulin/mTOR signaling in the placenta integrates maternal metabolic signals with fetal nutrient exposure, ultimately programming the metabolic health of the offspring.

2. Evaluation of the neonatal hepatic innate immune response to *E. coli*-induced systemic infection.

L.T. Atahualpa*, M. Grayack, M. Solar, N. Balasubramaniyan, L. Zheng, C.J. Wright
Neonatology, University of Colorado Anschutz Medical Campus Department of Neonatology, Aurora, CO, USA

Purpose of Study: Neonates are more susceptible to systemic infections than adults. There is a growing recognition that in adults, the hepatic innate immune response is a key first line of defense against systemic gram-negative bacterial (GNB) infection, such as *Escherichia coli* (*E. coli*). This robust innate immune response will initiate a pro-inflammatory response, activating local and systemic responses, thus mitigating the threat of *E. coli* and preventing systemic infection. Whether an attenuated hepatic innate immune response contributes to the increased risk of infection in the neonatal period is unknown. **Methods Used:** Postnatal day 3 (P3) and adult (6-8 weeks) C57BL/6J (B6) mice were exposed to *E. coli* (105 CFU/g, IP x 1 and 5 hrs). Hepatic tissue was assessed for neutrophil accumulation [myeloperoxidase (MPO) DAB staining] and *E. coli* [Colony Forming Units (CFU)]. Hepatic mRNA was assessed for induction of pro-inflammatory primary response genes and Acute Phase proteins (APP) using RT-qPCR. **Summary of Results:** An evaluation of the MPO DAB staining helped determine hepatic neutrophil accumulation and revealed a decrease in baseline recruitment in P3 compared to adult mice. In adults, we expected to see a gradual increase in hepatic neutrophil recruitment throughout the passing time points. In contrast, hepatic neutrophilic count stayed stagnant in *E. coli*-exposed neonates. In adults, *E. coli* hepatic collections had no significant increases throughout the time points. In contrast, consistent with impaired neutrophil recruitment, *E. coli* burden in P3 mice significantly increases by the 5-hour timepoint. *E. coli* CFU quantification assessed that P3 mice have a significant growth of *E. coli* while the CFU for adult mice showed no significant growth. A contributing factor to the recruitment of hepatic neutrophils and *E. coli* growth in the liver is pro-inflammatory genes. We evaluated common pro-inflammatory genes *Il6*, *Il1β*, and *Cxcl1,* known to express at significant levels in adult mice. In bacteremic adult mice, we observed an increase in the pro-inflammatory genes after 1 hour of *E. coli* exposure. In contrast, P3 mice do not express the same level of significant expression, and rather demonstrate a delayed initial response. We observed a decrease in hepatic neutrophilic recruitment and significant *E. coli* growth in the P3 mice coupled with an initial delay in the pro-inflammatory transcriptional response of P3 mice, showing a significant increase at the later 5-hour time point. The APR, activated by the pro-inflammatory transcriptional response, showed a parallel behavior, also exhibiting a delayed and stunted expression of the APR genes *Crp*, *Lbp*, and *Saa1*. **Conclusions:** In response to systemic GNB infection, the neonatal hepatic innate immune response is attenuated when compared to adults. This includes decreased hepatic neutrophil accumulation, a delay in pro-inflammatory and APR transcription, and increased *E. coli* growth. We speculate that impaired neonatal hepatic innate immune response to *E. coli* exposure increases susceptibility to infection.

3. Metabolic response to premature lipid exposure in fetal cardiomyocytes.

Neeka Barooni*, Leena Kadam, Sonnet S. Jonker
Oregon Health & Sciences University, Center for Developmental Health, Portland, OR, USA

Introduction: Fetal plasma lipid concentrations are low, and cardiomyocytes rely on carbohydrates as their primary fuel source. Circulating lipid levels may be prematurely elevated by maternal dyslipidemia or parenteral nutrition in preterm neonates. In late-term fetal sheep, maximal respiration was lower in cardiomyocytes exposed to Intralipid infusion compared to Controls. It is unclear whether fetal cardiomyocyte metabolism is altered by Intralipid exposure during mid-gestation. **Objective:** Test the hypothesis that maximal oxygen consumption rate (OCR) is lower in cardiomyocytes from mid-gestation fetal sheep exposed to 8-day Intralipid treatment

compared to fetuses receiving vehicle infusion *in utero*. **Methods:** Mid-gestation fetal sheep received Intralipid 20® or vehicle infusion from 89 to 97 days of gestation. Left ventricular cardiomyocytes were isolated and cultured. Maximal OCR was measured in cardiomyocytes with or without additional exposure to palmitic acid *in vitro*. **Results:** There was a significant interaction among fetal sex and *in vivo* Intralipid treatment for maximal OCR in cardiomyocytes cultured in standard media (P=0.018); OCR was 30% lower in males receiving Intralipid treatment compared to male Controls (P=0.037). Maximal OCR was similar between Control and Intralipid cardiomyocytes exposed to palmitic acid *in vitro*. **Conclusion:** These findings indicate that mid-gestation Intralipid treatment reduces cardiomyocyte OCR in a sex-dependent manner. Similarities between Control and Intralipid cardiomyocytes treated with palmitic acid *in vitro* suggest that the metabolic response to lipid substrate is not compounded by prior lipid exposure. (Supported by NIH R01HL146997)

4. Fetal c-reactive protein rs1205 genotype is associated with maternal pre-eclampsia.

L.G. Best[1,2*], C. Azure[2], H. Davis[2], L. Jeanotte[2], S. LaRocque[2], S. Poitra[2], J. Poitra[2], S. Standish[2], T.J. Parisien[2], K. Morin[2]
[1]University of North Dakota, Grand Forks, ND, USA; [2]Natural Sciences, Turtle Mountain Community College, Belcourt, ND, USA

Introduction: Maternal variants including C-Reactive Protein, *CRP* rs1205 have previously been associated with risk of pre-eclampsia (PE). These findings were replicated in two non-American Indian populations. The rs1205 T allele is associated with reduced serum levels of CRP. Our objective was to determine if the fetal rs1205 genotype contributed to maternal risk of PE independent of maternal rs1205 genotype. **Methods:** Only offspring of both case and control mothers heterozygous for rs1205 were enrolled, thus controlling for maternal genetic influence at this locus. Offspring were then genotyped for rs1205 by TaqMan assay. Association was assessed by chi-square and multivariate logistic regression. **Results:** Offspring of 10 of 45 normal pregnancies and 11 of 24 PE pregnancies exhibited the rs1205 C allele recessive genotype (Pearson chi square p=0.042). Multivariate logistic regression analysis adjusted for maternal age, nulliparity and BMI demonstrates an odds ratio of 3.603, p=0.043, 95% CI 1.042-12.457 for the fetal, C recessive genotype. **Discussion:** Among 69 women, heterozygous for the rs1205 allele, both chi-square and multivariate adjusted logistic analysis shows significant association of PE among pregnancies with fetal rs1205 C recessive genotypes. This is consistent with previous findings of reduced risk associated with this maternal genotype, and with a pathophysiologic model wherein increased placental CRP expression increases risk of PE. (Supported by NIGMS P20GM103442)

5. Reduced myogenesis in fetal growth restriction: basic mechanisms and future therapies.

Laura D. Brown[1*], Tristan B. Dear[1], Neeka Barooni [2] Carrie E. McCurdy[2], Michael L. Armstrong[1], Nichole A. Reisdorph[1], Kendall Mesch[1], Jane Stremming[1], Saif I. Al-Juboori[1], Evgenia Dobrinskikh[1], Eileen I. Chang[1]
[1]University of Colorado Anschutz Medical Campus, Aurora, CO, USA; [2]University of Oregon, Eugene, OR, USA

Introduction: Redistribution of blood flow to vital organs in fetal growth restriction (FGR) occurs at the expense of skeletal muscle growth. Infants born after FGR have lower muscle mass that persists into adulthood. **Objective:** Understand the mechanisms for how myogenesis rates are slowed in the FGR fetus so that therapies may be developed to support muscle growth before deficits become permanent. **Methods:** Pregnant ewes were exposed to elevated temperature to induce placental insufficiency, and FGR fetuses were compared to control (CON) fetuses (n=19/group). At 0.9 gestation, fetal lambs were infused with EdU and hindlimb muscles were harvested to measure myofiber size, type, and vascularity by immunohistochemistry; proliferation and differentiation rates by single cell dissociation and flow cytometry; and leucine utilization in isolated myofibers by ^{13}C tracing. **Results:** Fetal weight and summed muscle weights were ~40% less in FGR vs. CON (P<0.005). Slow twitch oxidative myofiber composition, myofiber cross-sectional area, and capillary relative to myofiber number were reduced in FGR (P<0.05). Fewer myoblasts expressed EdU and were committed to early stages of differentiation in FGR (P<0.05); however, more myoblasts were in late differentiation (P<0.005) indicating impairments in myoblast fusion. Fractional contribution of leucine to intact protein in isolated myofibers was 19% lower in FGR (P<0.05). **Conclusion:** Fetal myogenesis is adversely impacted by placental insufficiency, including slower myoblast proliferation and differentiation rates, slower hypertrophic growth, fewer oxidative myofibers, and reduced angiogenesis. Targeting multiple regulators of myogenesis will likely be required to augment muscle growth in the FGR fetus. (Supported by NIH HD079404)

6. Investigating the impacts of maternal environment on lamb performance and reproductive dynamics.

Caitlin N. Cadaret[1*], Rachael M. Stucke[1], Julianna S. Messina[1], Ryne D. Haggard[1], Ellen A. Roberts[1], Alexandria P. Snider[2], Terry E. Engle[1]
[1]Department of Animal Sciences, Colorado State University, Fort Collins, CO, USA; [2]USDA-Agricultural Research Service, US Meat Animal Research Center, Clay Center, NE, USA

(Supported by USDA 2024-67015-42353)

7. Maternal hypoxia at mid-gestation lowers uterine oxygen delivery and uptake and placental but not fetal weight.

Alejandro A. Candia[1]*, Ramón A. Lorca[2], Julie A. Houck[2], German A. Arenas, Lorna G. Moore[2], Colleen G. Julian[3], Stephanie R. Wesolowski[1]

[1]Department of Pediatrics, University of Colorado School of Medicine, Aurora, CO, USA; [2]Department of Obstetrics and Gynecology, University of Colorado School of Medicine, Aurora, CO, USA; [3]Department of Biomedical Informatics, University of Colorado School of Medicine, Aurora, CO, USA

(Supported by NIH-R01-HD134949)

8. DHA and preterm birth: what we've learned since the 2018 Cochrane Review.

Susan E. Carlson[1]*, J.Thomas Brenna[2]

[1]University of Kansas Medical Center, Kansas City, KS, USA; [2]The University of Texas, Austin, Austin, TX, USA

Introduction: A 2018 Cochrane Review concluded strong evidence that omega-3 fatty acids, including docosahexaenoic acid (DHA, 22:6n-3), prevented preterm birth (PB<37wks gestation) by 11% and early PB (EPB<34wks gestation) by 42%, however, it did not determine dose or who was most likely to benefit from DHA supplementation. **Objective:** The Assessment of DHA on Reducing Preterm Birth (ADORE) multicenter trial enrolled 1100 women between 12 and 20wks gestation and assigned them to 200 or 1000 mg/d DHA. Red blood cell phospholipid (RBC-PL) DHA and DHA intake (DHA-Food Frequency Questionnaire) were assessed at baseline. **Results:** The higher dose reduced PB (PP=0.95) and EPB (PP=081). A secondary analysis found low DHA at baseline (<6%total fatty acids) or low DHA intake (<150mg/d) predicted who had reduced EPB (PP=0.93) with 1000mg/d. Adherence to high dose (RBC-PL ≥8%) decreased PB by 58% (PP=0.95) and EPB by 65% (PP=0.94) in the low baseline DHA group. Black participants with high baseline DHA provided 1000mg had 6.4% PB and 2% EPB, below US rates. Our latest finding is that a FADS1 Insertion allele that increase arachidonic acid synthesis predicts women who benefit from high dose DHA supplementation; an allele found in 85.5% of Black and 48.6% of Other Races participating in ADORE. **Conclusion:** Identification of the FADS I allele is a precision medicine strategy for primary prevention of PB and EPB. (Supported by NIH HD083292)

9. Impact of placental insufficiency-induced growth restriction on the fetal sheep heart transcriptome.

Eileen I. Chang[1]*, María B. Rabaglino[2], Jane Stremming[1], Stephanie R. Wesolowski[1], Paul J. Rozance[1], Laura D. Brown[1]

[1]Department of Pediatrics, Section of Neonatology, Perinatal Research Center, University of Colorado Anschutz Medical Campus, Aurora, Colorado, USA; [2]Department of Population Health Sciences, Veterinary Medicine, Utrecht University, Utrecht, Netherlands

(Supported by NIH UL1TR002535)

10. Maternal exposures and molecular regulation of the human placenta and endometrium.

Jane K. Cleal*

The University of Southampton, UK

Introduction: The placenta mediates the effects of changes to the maternal environmental during gestation on fetal development and both fetal and maternal lifelong health. These studies investigate the effects of exposures during pregnancy on human placental function, gene expression and epigenetics and the subsequent associations with fetal development. **Methods:** We use *ex vivo* systems including placental perfusion, villous and trophoblast culture, multi-scale imaging, epigenetic, proteomic and transcriptome analysis as well as placental samples from our Southampton cohort studies. **Results:** Findings include, maternal vitamin D levels, smoking and poor diet during pregnancy associate with placental gene expression of key nutrient transporters. These placental transporters relate to fetal and neonatal growth and body composition as well as maternal body composition. We show localized placental activation of vitamin D that induces rapid effects on the placental transcriptome, epigenome and proteome that effect placental function and thereby fetal development, independent of vitamin D transfer. **Conclusions:** Local vitamin D metabolism may also impact upon the interplay at the maternal-fetal interface. The structure and molecular profile of the endometrium during the window of implantation is being investigated in relation to the maternal environment. Cell population profiles and interactions along with endometrial gland cilia function

and extracellular vesicles production are targeted as potential biomarkers to predict pregnancy outcome. This work involves endometrial organoid culture, 3D imaging and single cell transcriptomic analysis. These data demonstrate a complex interplay between vitamin D and the placenta and endometrium and may inform future interventions using vitamin D to support fetal development and maternal adaptations to pregnancy. (Supported by The Gerald Kerkut Trust, Wellbeing of Women, Wessex Medical Research, The Rosetree's Trust)

11. Hepatocyte cell-intrinsic mechanisms for increased gluconeogenic capacity in the growth-restricted fetus.

Kiarra J. Coger[1]*, Molly M. McGuckin[1], Dong Wang[1], Ken Jones[2], Paul J Rozance[1], Laura D. Brown[1], Stephanie R. Wesolowski[1]
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(Supported by NIH-R01-DK108910 and HD079404)

12. The immune system regulates sperm RNAs to drive transgenerational epigenetic inheritance in mice.

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It was long assumed that sperm's sole function was to transport the paternal genome. However, it is now increasingly understood that the male gamete also transmits molecules, including RNAs, to the egg during fertilization, leading to non-genetically inherited phenotypes in offspring. In particular, small non-coding RNAs have been identified as key transmitters of paternal epigenetic information in some of the best-characterized examples of transgenerational epigenetic inheritance, such as RNA interference (RNAi) in *C. elegans*, and paramutation in plants and mice. Furthermore, in rodents, sperm small RNAs have been shown to causally transmit paternal epigenetically inherited phenotypes. In mice, multiple studies have demonstrated that sperm small RNAs are crucial for embryonic development. Despite these findings, the molecular processes that enable sperm RNAs to act as heritable information—altering developmental trajectories and inducing adult phenotypes—remain largely unknown in any organism. Using a variety of model systems, including mice, *C. elegans*, and embryonic stem cells, we are investigating the molecular mechanisms by which sperm small RNAs program embryonic development and transmit non-genetically inherited phenotypes to offspring. Additionally, we recently discovered that, in mice, the immune system and microbiome can regulate the accumulation of small RNAs in sperm. This occurs through the regulation of RNA packaging into extracellular vesicles in the epididymis, which then fuse with sperm, delivering their RNA cargo. Fascinatingly, this process leads to the transmission of epigenetically inherited phenotypes both intergenerationally (to F1 progeny) and transgenerationally (to F2 progeny and beyond).

13. Postnatal ω-3 polyunsaturated fatty acid supplementation partially resolves adiponectin receptor 2 deficits in skeletal muscle of IUGR-born lambs.

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Introduction: Intrauterine growth restriction (IUGR) causes heightened inflammatory sensitivity in muscle that impairs insulin-stimulated metabolism. This may be facilitated by disruption of adiponectin signaling, which is an insulin sensitizer and is mutually antagonistic with inflammatory signaling. **Objective:** This study evaluated the impact of supplementing anti-inflammatory ω-3 polyunsaturated fatty acids (PUFA) on circulating adiponectin and muscle receptors in IUGR lambs. **Methods:** Maternal heats stress was used to induce unsupplemented (n=11) and ω-3 PUFA-supplemented (n=12) IUGR lambs. Controls (n=12) were born to pair-fed thermoneutral ewes. Blood was collected at 1 and 4 weeks of age to determine adiponectin (ELISA). *Semitendinosus* muscle was collected at necropsy (day 28) to quantify the adiponectin receptors, CDH13 and AdipoR2 (immunoblot). **Results:** *Semitendinosus* CDH13 content did not differ among groups. AdipoR2 tended to be 63% less (P=0.09) for unsupplemented IUGR lambs than controls and was intermediate for ω-3 PUFA-supplemented IUGR lambs. Blood plasma adiponectin was 28% greater (P=0.04) for unsupplemented IUGR lambs (3.4±0.2 ng/mL) than for controls (2.7±0.1 ng/mL) and was intermediate for ω-3 PUFA-supplemented IUGR lambs (3.0±0.1 ng/mL), regardless of age. **Conclusions:** These findings demonstrate a potential deficit in adiponectin signaling in IUGR neonatal muscle. Partial resolution of the deficit following ω-3 PUFA supplementation indicate that it was partially due to heightened inflammatory tone. The greater circulating adiponectin, which was also improved by ω-3 PUFA, was paradoxical but may have been a compensatory response to reduced adiponectin sensitivity. Nevertheless, these findings illustrate how anti-inflammatory intervention may improve muscle-centric IUGR metabolic outcomes. (Supported by USDA 2019-67015-29448, 2020-67015-30825)

14. Effects of vitamin and mineral supplementation fed to F0 beef heifers throughout pregnancy on hepatic gene expression of the F2 generation.

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Supplementing pregnant F0 beef heifers with vitamins and minerals throughout pregnancy has been shown to alter development of F1 offspring, but questions of whether outcomes persist across generations remain. In this study, F0 dams were managed on one of two dietary treatments throughout pregnancy: 1) basal diet (CON, n=7), or 2) basal diet with vitamin and mineral supplement (VTM, n=8). At parturition, F1 heifers were managed as a single group throughout the experiment. All F1 heifers were bred via artificial insemination using female-sexed semen from a single sire and confirmed pregnant with female calf. At d 250 of gestation, F1 dams were harvested for collection of F2 fetal liver samples. Liver tissues from F2 fetuses underwent total RNA isolation and sequencing. After data quality control, reads were mapped using the STAR aligner and final statistical analyses were conducted with R (v4.4.2). Through differential expression analysis with DESeq2, we identified 269 differentially expressed genes (*P- value* ≤ 0.05 and |log2FC| ≥ 0.5). The most prevalent affected genes included *SHH*, *EDN1*, *LEF1*, *SFRP1*, *ESR1*, *AR*, and *BMP7*, which are involved in key biological pathways related to hepatic function – including energy metabolism, lipid biosynthesis, and liver health; as well as muscle function – particularly satellite cell proliferation critical for postnatal muscle growth and skeletal tissue regeneration. These findings suggest that providing vitamin and mineral supplementation to F0 dams during pregnancy induces effects on the F2 generation, including greater expression of genes that may enhance hepatocyte development and myocyte proliferation. (Supported by the USDA 2022-67016-36479).

15. Impact of prematurity on the nutritional regulation of growth.

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Extrauterine growth restriction is common in premature infants and is largely attributed to reduced lean mass accretion. This reduced lean growth likely contributes to their increased lifelong risk for obesity and type 2 diabetes. There is critical need to identify mechanisms for the reduced growth and to develop targeted nutritional therapies to improve lean accretion. We showed in neonatal pigs born at term that post-meal surges in insulin and amino acids independently activate insulin and amino acid signaling pathways that stimulate mechanistic target of rapamycin complex 1 (mTORC1)-dependent translation initiation and protein synthesis in skeletal muscle. These anabolic actions promote more efficient use of amino acids for muscle protein synthesis and sustain rapid early postnatal growth rates. In contrast, preterm birth reduces relative weight gain and blunts the protein synthetic response in muscle to feeding. The reduced protein synthesis response in the preterm is due to reduced insulin- and amino acid-induced activation of mTORC1. This anabolic resistance to feeding likely contributes to the high prevalence of extrauterine growth restriction and reduced lean mass in prematurity. Supplementation with leucine, an essential amino acid and potent agonist of mTORC1, enhances translation initiation and protein synthesis in skeletal muscle and overall lean growth of neonatal pigs born at term. In the preterm, leucine supplementation also increases mTORC1 activity and protein synthesis, but higher doses are required. This suggests that the anabolic resistance of preterm muscle can be overcome by nutritional therapies targeted to stimulate mTORC1 signaling and protein synthesis and promote lean gain.

16. Skeletal muscle microvascular architecture is unaltered in hypoxic fetal sheep.

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Introduction: Late gestation fetal sheep with placental insufficiency-induced fetal growth restriction (PI-FGR) have reduced vascularity and proportions of slow-twitch oxidative myofibers in tibialis anterior (TA) muscle. Sustained experimental hypoxemia in late gestation, a feature of PI-FGR, limits glucose utilization and oxidation and lowers anabolic hormones, yet does not reduce fetal weight or muscle mass. **Objective**: We hypothesized that sustained hypoxemia disrupts oxidative myofiber development and microvascular growth in TA muscle. **Methods**: Pregnant ewes were assigned to control (CON; n=5) or hypoxemia (HOX; n=10) at ~125 days gestation (term=149 days) for ~9-days. Myofiber area, type, and vascularity were quantified in TA muscle using immunofluorescence and Visiopharm® software. Data are expressed as mean±SD. Student's t-test was used; $P<0.05$ was significant. **Results**: Fetal PaO_2 was 15% lower in HOX than CON (14.9±1.7 vs. 17.5±2.8; $P=0.0427$), modeling the hypoxemia in PI-FGR fetuses. Myofiber proportion, size, vascular area, arteriole and venule area, capillary area, and capillary to myofiber ratios were similar between groups. **Conclusions**: In contrast to the PI-FGR fetus, sustained late gestation hypoxemia does not alter myofiber size, vascular architecture, or oxidative myofiber proportion in TA muscle. Thus, reduced myofiber and vascular development in the PI-FGR fetus may be caused by more severe or prolonged hypoxemia, or secondary to other factors following limited nutrient utilization and growth restriction. Alternatively, the absence of myofiber or vascular effects in response to hypoxemia may reflect fetal mechanisms to defend its oxidative metabolism and growth. (Supported by NIH R01-DK108910 and HD709404)

17. Developmental origins of boar fertility: consequences of *in utero* heat stress.

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18. Metabolic manipulation of spermatozoa improves sperm function with implications for Assisted Reproductive Technologies.

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Introduction: Despite advances in Assisted Reproductive Technologies (ART), obtaining developmentally competent embryos remains a major challenge. We previously demonstrated that modulating sperm metabolism through Sperm Energy Restriction and Recovery (SER) enhances sperm function and improves fertilization and embryo development rates following in vitro fertilization in mice. **Objective:** To optimize SER in bovine and equine species. **Methods:** Cryopreserved sperm were incubated in either complete medium (control) or nutrient-deficient medium (ST), followed by recovery with metabolic substrates (SER). Sperm function was assessed by evaluating motility and capacitation-associated markers, including phosphorylation of PKA substrates (pPKAs), tyrosine phosphorylation (pY), and intracellular Ca^{2+} levels. Control and SER bovine sperm were then used for intracytoplasmic sperm injection (ICSI). **Results:** SER improved sperm motility in both species, while no differences were observed in pPKAs or pY levels in either species. ST equine sperm showed higher intracellular Ca^{2+} levels and a greater percentage of live, acrosome-reacted cells compared to controls (14.4% vs. 6.6%, $p < 0.05$). In bovines, ICSI using SER sperm yielded higher 2-cell embryo rates than controls (50% vs. 16%, $p < 0.05$), and 17% of these embryos developed to the blastocyst stage, whereas none did in the control. SER also enhanced egg activation post-ICSI, with more oocytes displaying greater than 3 Ca^{2+} peaks (40% vs. 10%, $p < 0.05$). **Conclusion:** SER improves sperm function in both bovine and equine species and enhances egg activation after bovine ICSI. These findings suggest that targeting sperm metabolism may increase the efficiency of in vitro embryo production across species. (Supported by USDA 2022-67016-41939)

19. Paternal drinking and the epigenetic influences on mitochondrial function, placental dysfunction, and structural birth defects.

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Emerging research reveals that epigenetic mechanisms of paternal inheritance are a significant driver of adverse developmental outcomes, including those associated with fetal alcohol spectrum disorders (FASDs). In recognition of the urgent need to understand the combined effects of maternal and paternal alcohol consumption, our laboratory established a preclinical multiplex mouse model to facilitate the comparison of alcohol-induced developmental defects in offspring resulting from maternal, paternal, and dual parental exposures. Our findings reveal that both maternal and paternal alcohol consumption independently impair placental development and alter craniofacial patterning in a dose-dependent manner. Strikingly, in the male offspring, we observed an interaction between maternal and paternal alcohol use, with adverse developmental outcomes in the dual-parental offspring exceeding those induced by either maternal or paternal alcohol use alone. Our ongoing experiments reveal that parental alcohol exposure heritably disrupts offspring mitochondrial complex I activity in the placenta and fetal brain. These deficits persist into adulthood, resulting in elevated oxidative stress, chronic inflammation, and premature cellular aging in the brain and liver. Our results underscore that chronic toxicant-induced mitochondrial stress, particularly in males, programs enduring bioenergetic dysfunction that elevates the risk of birth defects and long-term disease. These findings emphasize the critical importance of considering both maternal and paternal health in preconception planning.

20. Using anogenital distance, a prenatal marker of androgen exposure, to predict pregnancy by artificial insemination in yearling beef heifers.

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Anogenital distance (AGD), distance from the anus to the clitoris is programmed during fetal life by testosterone concentrations. In rodents, fetal testosterone increases AGD and reduces reproductive performance in the adult life. We hypothesized that yearling beef heifers with a shorter AGD during the gynecological examination have a greater probability of pregnancy by artificial insemination (AI)

than heifers with longer AGD. Yearling heifers (n = 554) were submitted to estrous synchronization, gynecological examination (pre pubertal vs. pubertal) and AGD measures to the top of the clitoris (AGD-C) or to the dorsal commissure of the vulva (AGD-T). At CIDR withdrawal, heifers received an estrus patch. AI was conducted using 5 bulls. 30 days after AI pregnancy diagnosis was performed. 85% of the heifers were pubertal and 62% displayed estrus. AGD-C and AGD-T were positively associated (P< 0.001, R^2= 0.1982). For each unit of increase in body weight AGD-C increased 0.0064 cm (P= 0.0003). AGD-C was positively associated with the probability of puberty (P= 0.0019) and pregnancy in heifers without a corpus luteum upon gynecological examination (P= 0.10). ROC curve analysis found a poor area under the curve (0.59) for any AGD to predict pregnancy/AI. In conclusion, it looks like in adult life the AGD effects on fertility are mostly a consequence of the increase in body weight the prenatal effects on the AGD are diluted over time and are not associated with reproductive outcome in adults.

21. Long-term effects of reduced docosahexaenoic acid placental transfer on offspring neurobehavioral outcomes in mice.

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Introduction: Docosahexaenoic acid (DHA) is critical for fetal brain development. Inadequate supply during the perinatal period has been associated with an impaired neurological function in children. DHA is transported from the mother to the fetus via the placenta by the Major Facilitator Superfamily Domain Containing 2a (MFSD2a) transporter. **Objective:** We hypothesize that placenta-specific knockdown (KD) of MFSD2a in mice causes neurobehavioral changes in adult offspring. **Methods**: We generated placenta-specific KD of MFSD2a using a mouse model of trophectoderm lentivirus transfection and embryo transfer. Neurobehavioral functions such as cognition, memory, motor skills and anxiety-like behaviors as well as social interaction were tested at 3 months of age in male and female mice from placental MFSD2a KD and non-coding transfection (SCR) pregnancies using Open field, Double H maze, Three-Chamber Sociability and Rotarod test. **Results**: Adult mice from pregnancies with placental MFSD2a KD demonstrated a significant increase of anxiety-like behavior (n=34 SCR and n=39 MFSD2a KD, p-value<0.0001) and an impaired mobility (n=41 SCR, n=46 MFSD2a KD, p-value<0.001) predominantly in females. No significant differences were observed in memory or social behavior. **Conclusion**: Our results suggest that placental DHA transfer by MFSD2a during pregnancy is critical for long-term neurodevelopment. (Supported by NIH HD104644)

22. The pre-ruminant microbiome: why do we care and where does it come from?

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Introduction: Despite ruminant livestock being born with a non-functional rumen, the early rumen microbiome can have lasting impacts on animal performance. Identifying key sources of colonization of this early rumen microbiome are imperative to harnessing the programming potential of this microbial niche to improve long term health and performance. **Objectives:** The objective of this collective work is to identify maternal influences on colonization of the neonatal gut microbiome. We hypothesize that environmental, genetic, and pathogenic disruptions during gestation and early lactation will have lasting impacts on the offspring, partially via impacts on the gut microbiome. **Methods:** Research projects involving beef cattle and range sheep have been employed to characterize maternal influence on the neonatal gut microbiome. In all studies, microbial characterizations have been completed utilizing amplicon sequencing of the 16S rRNA and analysis via QIIME2. Alpha- and beta-diversity metrics in additional to differences in relative abundance of taxa have been evaluated. **Results:** It is apparent across all studies that the neonate does in fact have a unique microbiome immediately after birth. Data also indicates that maternal factors impact offspring rumen microbiome including nutritional status of the dam during gestation and early lactation and mammary health. Furthermore, these studies have revealed several potential sources of colonization of the neonatal gut. **Conclusion:** These data from several projects highlight opportunities for programming of the rumen microbiome to enhance long term health and performance.

23. Vitamin and mineral supplementation in beef heifers and the impacts on intestinal vascularity and proliferation in neonatal calves at 30 hours of age.

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24. Sexual dimorphism in placental insufficiency.
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25. Physiological response to fetal intravenous lipid emulsion in mid-gestation.
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26. Embryo transfer outcomes and placental vascular development in sheep: breed interactions.
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Infertility is a major barrier to reproductive efficiency in livestock. Early pregnancy is a critical window for placental vascular development, which is essential for embryonic survival and fetal growth. This study evaluated the individual and combined effects of embryo and ewe breed on pregnancy establishment and placental vascularization in sheep. A total of 85 embryo transfers (ET) were performed using straight-bred and reciprocal combinations of Rambouillet (Rambo) and Romanov (Romo) ewes. Pregnancy was assessed on day 24 of gestation. Straight-bred groups showed the greatest pregnancy rates: Rambo × Rambo (31.3%) and Romo × Romo (26.7%). The reciprocal Rambo × Romo group had the lowest pregnancy rate (11.8%), while Romo × Rambo was intermediate (22.7%), indicating a tendency for breed-dependent effects (P=0.10). To assess vascular development, uterine cross-sections were collected at slaughter on day 25 and stained with CD31 and CD34 to quantify capillary area density (CAD) and capillary number density (CND) in the caruncle (CAR), fetal membrane (FM), and inter-caruncular (ICAR) regions. compared to the other groups, Rambo × Romo had increased CAD in CAR and ICAR regions (P<0.01), while Romo × Romo had reduced CND in CAR (P<0.05), indicating differences in vascular development due to embryo and ewe breed. These vascular differences may underlie reduced pregnancy success after ET. This study highlights a link between fertility and early placental vascularization, offering insight into mechanisms impacting pregnancy maintenance. Understanding these effects could lead to breeding strategies to improve reproductive outcomes in sheep.

27. Gestation stage and lactation impacts on nutrient retention in multiparous sows.
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Introduction: Understanding nutrient retention during gestation and lactation is important for understanding sow nutrient demands. Little information is available regarding nutrient retention of multiparous sows during these phases. **Objective:** Assess nutrient apparent total tract digestibility (ATTD) days 40 to 44 and 96 to 100 of gestation and mid-lactation. **Methods:** During the gestation collection periods, sows were housed in individual metabolism crates. Total feces and urine was collected. Titanium dioxide (TiO2) was used as an indigestible marker in lactation. The study used 48 parity 2 through 6 sows. Gestation and lactation diets contained 1,000 FTU/kg of phytase and 50 mcg/kg of 25-OH D3. **Results:** The ATTD of DM, N, GE, fat, total and insoluble fiber were not different (P>0.10) between stages of gestation but were lower in lactation than gestation (P<0.001). Ash, Ca and P ATTD were greater in later gestation than early gestation (P<0.001). Ash and Ca ATTD were lower in lactation than either stage of gestation (P<0.05) with P ATTD being similar in early gestation and lactation. The ATTD of K (P<0.05), Na and Mg (P<0.001) were lower in lactation than gestation. Gestation Zn, Cu, Mn and Fe ATTD were positive with no difference by stage of gestation (P>0.10). Lactation ATTD of Zn, Cu, Mn and Fe was negative and significantly lower than during either stage of gestation (P<0.01). **Conclusion**: Late gestation appears to drive Ca and P retention while lactation appears to result in reduced Ca retention and net loss of Zn, Cu, Mn and Fe.

28. Serum 25-OH D3 concentration changes in sows as a result of 25-OH D3 addition and mineral source.

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Introduction: Vitamin D is essential for growth and development including processes beyond those associated with bone. Vitamin D status can be low in reproducing sows. The vitamin D receptor is a Zn dependent receptor. Thus available mineral may be necessary to optimize vitamin D status. **Objective:** Assess impact of mineral source fed during gestation and lactation on sow serum 25-OH D3 concentration. **Methods:** Thirty-two multiparous sows (16 reps/treatment) were fed 100 ppm Zn, 20 ppm Cu and 40 ppm Mn either solely in the hydroxy form (ITM), or, as a 50:50 blend of metal methionine hydroxy analogue bis-chelate minerals and ITM (MHAC:ITM). All diets were supplemented with 50 mcg/kg 25-OH D3 and 1,000 FTU/kg phytase. Sows had not previously received 25-OH D3. Serum samples were obtained at insemination, gestation d-103, and 21-d post-farrow and analyzed for 25-OH D3 concentration. **Results:** At breeding serum 25-OH D3 averaged 26.4 ng/ml (P=0.20). Breeding 25-OH D3 was used as a covariate. Day 103 25-OH D3 was 47.0 and 40.4 ng/ml for ITM and MHAC:ITM respectively (P=0.20). Weaning 25-OH D3 was 78.2 ng/ml for MHAC:ITM which tended (P<0.10) to be greater than the 66.3 ng/ml in ITM serum. The d-103 to weaning rise in 25-OH D3 tended (P<0.10) to be greater in MHAC:ITM (+16.5 ng/ml) than ITM (+4.5 ng/ml). **Conclusion:** Supplementing 50 mcg/kg 25-OH D3 can increase vitamin D status within 1 parity. A MHAC:ITM blend fed throughout gestation and lactation tended to result in a greater weaning serum 25-OH D3.

29. Maternal mineral source impact on piglet serum mineral and carcass composition at harvest.

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Introduction: Offspring from sows fed Zn, Cu and Mn in the metal methionine hydroxy analogue chelate (MHAC) form vs. sulfate form have been shown to have greater loin eye area at harvest. Data is lacking compared with other mineral sources. **Objective:** Assess the impact of maternal feeding of MHAC or glycinate (Gly) minerals on offspring serum mineral concentration, loin depth and fat depth. **Methods:** From 6 weeks pre-breeding through first gestation, diets were supplemented with 80 mg/kg Zn, 10 mg/kg Cu and 20 mg/kg Mn. From first through third lactation, Zn, Cu and Mn were provided at 50 mg/kg Zn, 10 mg/kg Cu and 20 mg/kg Mn. During parity 3, serum from 1 piglet/litter at 4-d, weaning and 14-d post-weaning was obtained from 20 litters/treatment. At each parity, muscle and backfat depth was determined for approximately 1,000 pigs/treatment. **Results:** There was no treatment (P>0.15) nor treatment × time interaction (P>0.22) for serum mineral concentration. Serum Fe, Zn, Cu and Mn were highest at weaning (P<0.001). Cu and Zn concentrations were 34% and 60% lower respectively at 14-d post-weaning than at 4-d of age. Parity 2 and 3 MHAC offspring had 0.5 mm (P<0.01) and 1.0 mm (P<0.001) more muscle depth at 122 kg. Backfat was lower in parity 1 (-0.2 mm; P<0.01) and parity 3 (-0.4 mm; P<0.001) MHAC offspring. **Conclusions:** Offspring serum mineral concentration was highest at 4-d of age. Maternal trace mineral source appears to provide an opportunity to impact offspring loin and fat depth at harvest.

30. Offspring microflora is affected by parity and organic acid use.

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Introduction: Primiparous sows can differ in microbial diversity compared with mature sows. Organic acids influence gut microflora when fed directly to the pig. Impact of organic acid blends (OAB) when fed to sows of differing parities on offspring microflora is lacking. **Objective:** Evaluate the impact of 0% or 0.3% OAB fed to young (parity 1 and 2) and adult (≥ parity 3) sows on offspring microflora. **Methods:** An OAB containing 2-hydroxy-4-(methylthio) butanoic acid was fed from 50-d of gestation through lactation. Feces was collected at lactation d-20 from 40 pigs/treatment with 2 pigs/litter sampled. Microbial DNA was extracted with 16s RNA amplification of the V3/V4 region. **Results:** Primary effects were between 0% OAB young sows compared with adult sows and 0.3% OAB young sows. Alpha diversity (P<0.05) and richness (P<0.01) were greater in pigs from adult sows compared to young sows. Feeding 0.3% OAB to young sows increased diversity and richness to levels similar to that of adult sows while simultaneously repressing *Anaerotignum lactatifermentans* (P<0.05) and *Bacteroides uniformis* (P<0.05), two species naturally suppressed in piglets from adult sows (P<0.05). Family *Sphaerochaetaceae* was higher in adult sows' offspring and young 0.3% OAB offspring (P<0.05) than young 0% OAB offspring and was supported by a corresponding increase in class *Spirochaetia* (P<0.05) in adult sows's offspring and 0.3% OAB young offspring. **Conclusions:** These results indicate 0.3% OAB fed to young sows increased offspring bacterial diversity and richness and altered microbial profiles to be more similar to offspring from adult sows.

31. Prenatal oxygen and glucose therapy rescues the immature β cell population and their transcriptomic signature in growth-restricted fetal sheep.

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Introduction and objective: Placental insufficiency (PI) reduces fetal oxygen and glucose concentrations, leading to fetal growth restriction (FGR), decreased β-cell mass, and reduced insulin production. Single-cell RNA sequencing (scRNA-seq) of fetal sheep islets identified different stages of β-cell maturation. FGR fetuses exhibited a higher mature-to-immature β-cell ratio compared to controls. We

tested the hypothesis that supplemental oxygen and glucose to FGR fetus normalizes the immature β-cell population. **Methods:** PI-FGR was induced by maternal hyperthermia. The oxygen and glucose therapy was delivered via maternal oxygen insufflation and fetal glucose infusion (FGR-OG, n=5) for 7-10 days. FGR-AS received air and saline infusions (n=5) and control fetuses were developed under thermoneutral conditions (n=3). Pancreatic islet cells were isolated for scRNA-seq (10X Genomics). Differential gene expression (DESeq2, P<0.05) and pathway enrichment (KOBAS) were performed on the pseudo-bulked immature β-cell transcriptomes. **Results:** FGR groups were growth restricted compared to controls (P<0.01). The immature-to-mature β-cell ratio was reduced in FGR-AS islets (1.7:1) compared to control (5.7:1) and FGR-OG (8.7:1) islets. Transcriptomic analysis of immature β-cells from FGR-AS fetus revealed upregulation of ribosome, oxidative phosphorylation, proteosome, metabolic pathways, spliceosome and RNA polymerase compared to control β-cells. In contrast, oxidative phosphorylation, spliceosome, ribosome, and proteosome were downregulated in FGR-OG compared to FGR-AS β-cells. No enriched pathways were detected between control and FGR-OG β-cells. **Conclusion:** FGR accelerates β-cell maturation based on the augmentation of gene expression in metabolic pathways. Moreover, immature β-cells in FGR-OG fetuses preserve the immature β-cell pool because their transcriptomic profile was similar to immature β-cells from control fetuses, which together indicates the oxygen glucose therapy partially rescue dysregulated metabolic programming in pancreatic β-cells. (Supported by NIH R01-DK084842)

32. Assisted Reproductive Technologies and adverse perinatal outcomes: the what, why and how?

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Greater than 200,000 children in the United States are born yearly with the aid of assisted reproductive technologies (ART). Recent epidemiological studies have suggested that these treatments are associated with an increased risk of adverse perinatal outcomes, including fetal growth restriction, low birth weight, preterm labor, preeclampsia and some rare genetic and epigenetic diseases. Given that many ART treatments including in vitro fertilization (IVF) utilize multiple clinical and laboratory interventions to generate a cohort of embryos capable of implantation and development, it is critical to examine each intervention individually in order to assess its relationship, if any, to the described adverse perinatal outcomes. Both human and animal studies can provide us with significant insights into the clinical procedures as well as the molecular mechanisms that may be playing in role in the adverse outcomes associated with IVF. By analyzing these data, we can not only can we modify current protocols to minimize the maternal and neonatal risk, but we can gain understanding of the critical cells and processes that play a role in pregnancy establishment and maintenance.

33. Effect of metformin use during pregnancy on fetal skeletal muscle growth and metabolism in rhesus macaques.

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Introduction: Metformin use in pregnancy is increasingly common with indications beyond treatment of diabetes. Metformin crosses the placenta and bioaccumulates at near equimolar concentrations as maternal circulation. Concerningly, in adults, metformin can suppress mitochondrial complex (C) I activity, limiting oxidative metabolism and ATP production. **Objective:** Examine the effect of metformin on fetal growth and muscle metabolism in Rhesus macaques. **Methods:** Twice-daily metformin (MET, 10 mg/kg) or placebo was given at 30 days of pregnancy with a chow (mCD) or a Western-style diet (mWD) with fetal muscles (n=25F/17M) collected at g145 (of g164). Respiration was measured with carbohydrate or lipid substrates in permeabilized fiber bundles (PFB) from gastrocnemius and soleus or isolated mitochondria (mitos) from rectus femoris. **Results:** Female mCD+MET offspring had reduced weight compared to mWD+MET (p=.02), with no differences in males. Muscle cross-sectional area was reduced with MET, independent of diet, in gastrocnemius, but not soleus, suggesting fiber-type specific effects. In mCD, MET decreased CI reliance (p=.03) in gastrocnemius and soleus PFB. Increased fat oxidation and respiratory capacity in mWD was blunted by MET in PFB. Lipid oxidation (p=.03) was lower in mWD+MET mitos concomitant with reduced ATP production. CI and CI+II carbohydrate-respiration was higher in mCD+MET vs. mCD mitos (p=.04) but without greater ATP production. **Conclusion:** Metformin resulted in subtle changes in fetal weight and was principally associated with reductions in glycolytic muscle fiber size, mitochondrial respiration and ATP generation. As muscle mass correlates with functional capacity and insulin sensitivity throughout life, caution is warranted in using metformin during pregnancy. (Supported by NIH DK128187)

34. Mothers' own milk provides personalized nutrition and protection to optimize short- and long-term health outcomes in term and preterm infants.

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The human infant is the most immature of all mammals at birth, yet boasts the kingdom's largest and most complex brain, which grows exponentially between 35 weeks of gestation and 2 years of age. Mothers' own milk (MOM; excludes donor human milk; DHM) has

evolved to provide personalized nutrition and bioactive components that prioritize brain growth, development and neuroprotection, as well as maturation of other body organs and metabolic and immunomodulatory pathways. Because of high MOM lactose and lipid to support the brain, MOM protein is the lowest among mammals, programming slow body growth, which is linked to lower obesity risk into adulthood. Clinical studies, albeit mostly observational due to inability to ethically randomize diet, have demonstrated improved long-term outcomes consistent with these components, including lower risks of non-communicable chronic diseases and higher measures of neurocognitive outcome. MOM components vary markedly as a function of lactation phase, with lactoferrin, secretory IgA, leptin, growth factors, gut colonizing components and other bioactives highest in the first postpartum month, suggesting programming and protection roles. Although MOM has not evolved to support optimal growth of preterm infants, necessitating exogenous fortification, the immature mammary gland secretes many bioactive components in higher concentrations and for a longer duration postpartum, with several studies suggesting a critical window for receipt of high-dose MOM during the first month post-birth in preterm infants. DHM is significantly less effective due to maternal donation during later lactation phases, storage, freeze-thaw cycles and pasteurization. Research priorities for feeding preterm infants will be summarized.

35. Regulators of branched-chain amino acid catabolism and activators of muscle protein synthesis are reduced in skeletal muscle of fetal growth-restricted sheep.

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Introduction: In a sheep model of placental insufficiency and fetal growth restriction (FGR), rates of branched-chain amino acid (BCAA) uptake into fetal hindlimb skeletal muscle and BCAA incorporation into muscle protein synthesis (MPS) were lower compared to normally growing controls. However, intracellular concentrations of BCAAs were higher in FGR muscle. **Objective:** Test the hypothesis that molecular pathways regulating BCAA catabolism and MPS are down-regulated in FGR skeletal muscle. **Methods:** Pregnant ewes were housed in elevated temperatures to induce placental insufficiency and FGR and compared to controls (CON; n=15/group). At 0.9 gestation, protein was isolated from fetal biceps femoris muscle and analyzed by Western Blot for regulators of BCAA catabolism (BCAT2, BCKDH, BCKDK, KLF15, PPM1K) and MPS (AKT, mTOR, RPS6, 4E-BP1). Student's t-test was used; $P<0.05$ was considered significant. **Results:** In the BCAA catabolism pathway, protein expression of BCAT2 was 37% lower ($P<0.01$); phosphorylated and total BCKDH were 31% and 27% lower, respectively ($P<0.05$); and KLF15, an activator of BCKDH, was 15% lower ($P<0.05$) in FGR vs. CON. In the MPS pathway, total AKT was 20% lower ($P<0.05$) and the ratio of phosphorylated to total RPS6 was 41% lower ($P<0.05$) in FGR vs. CON. **Conclusion:** Molecular regulators of BCAA catabolism and MPS were reduced in FGR muscle, indicating less BCAA flux into catabolic pathways and decreased incorporation of BCAA into protein. We speculate that elevated intramuscular BCAAs are the result of decreased utilization by muscle and are instead used to promote fetal survival during FGR pregnancies. (Supported by NIH R01HD079404)

36. Impact of feeding different selenium (Se) sources to pregnant and lactating ewes on lamb Se enrichment and serum biochemistry profiles.

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Introduction: Global pre-weaning lamb mortalities have remained above 10%, a detriment to farm profits. Selenium (Se) is a trace mineral vital to several functions in sheep. Se supplementation during late pregnancy and lactation, particularly organic Se supplementation, may produce more robust lambs. **Objective:** To determine how differences in maternal Se supplementation impact lamb health within 10 days postpartum (ppd 10). **Methods:** Ewes (n=110) were enrolled in a feeding trial from gestation day (gd) 110 to ppd 10, and supplemented with either no Se, 0.3 mg/day inorganic Se, or 0.3 or 0.6 mg/day organic Se. Lambs only received Se via maternal nursing. Lamb serum was collected on ppd 0, 2, and 10 to assess maternal transfer of Se, and muscle samples were collected on ppd 10 to assess lamb Se stores. Serum glutathione peroxidase (GPx) levels, thyroid hormone triiodothyronine (T3) levels, and a complete ovine 23-parameter biochemistry panel were assessed on ppds 0 and 10. **Results:** Maternal organic Se supplementation significantly increased Se levels in lamb serum ($P<0.0001$) and muscle ($P<0.0001$) as compared to other treatments. Organic Se supplementation significantly increased GPx activity at birth ($P>0.001$), but T3 levels were not affected. From the biochemical panel, serum levels of non-esterified fatty acid, potassium, chloride, albumin, total bilirubin, creatine kinase, aspartate aminotransferase, and glutamate dehydrogenase showed significant treatment differences. **Conclusions:** Organic Se supplementation to ewes during late gestation and lactation improved lamb Se status and antioxidant capacity at birth. Other serum biochemistry parameters were only marginally affected by Se treatment. (Supported by NSERC 401814)

37. Palmitoleate protects against Zika virus infection-induced endoplasmic reticulum stress and apoptosis in neurons.

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Zika virus (ZIKV) infection during pregnancy is associated with the development of fetal complications such as microcephaly. We have recently demonstrated that palmitoleate protects against ZIKV-induced apoptosis in placental trophoblasts. In the present study, we hypothesize that palmitoleate prevents ZIKV infection-induced endoplasmic reticulum (ER) stress and apoptosis in neurons. Neurons were infected with 0.1-1 multiplicity of infection of recombinant MR766 or PRVABC59 strains of ZIKV for an hour followed by treatment of palmitoleate (100 μM-200 μM) for different post-infection time points. Apoptosis was measured by nuclear morphological changes, caspase 3/7 activity, and immunoblot analysis of pro-apoptotic mediators. Activation of ER stress markers and viral envelope levels were detected using qRT-PCR and immunoblot analysis. Infectious virus particles were measured by using plaque assay. ZIKV infection to neuronal cells showed increased levels of pro-apoptotic markers like cleaved-PARP, cleaved caspase-3, Bim, and Puma, whereas decreased levels of anti-apoptotic markers such as Mcl-1, Bcl-1, and Bcl-xL. Further, we observed activation of three arms of ER stress namely: inositol requiring enzyme 1 alpha (IRE1), protein kinase-like ER kinase (PERK), and activating transcription factor (ATF6) pathways with ZIKV infection. Treatment of palmitoleate dramatically decreased ZIKV infection-induced increase in percent apoptotic nuclei and caspase 3/7 activity. Further, treatment of palmitoleate decreased cleaved PARP and PUMA protein expressions. Treatment of palmitoleate reduced ZIKV-induced ER stress activation as evidenced by decreased levels of phosphorylated forms of IRE1 and eukaryotic initiation factor 2 alpha; decreased expressions of cleaved ATF6, spliced X-box associated protein 1 and C/EBP homologous protein compared to ZIKV infection alone. Further, treatment of palmitoleate attenuated ZIKV envelope levels and infectious titer in SH-SY5Y and primary fetal cortical neurons isolated from humanized STAT2 knockin mice. These data suggest that palmitoleate supplementation protects against ZIKV-induced neuronal ER stress, apoptosis and decreases Zika viral load thereby mitigates neuronal damage.

38. Glucagon inhibits trophoblast secretion of placental lactogen *in vitro.*

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39. Colostrum intake promotes mammary cell proliferation in neonatal piglets.

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Introduction: Greater colostrum intake increases milk production during lactation in mature sows and cows. Although, we know that the number of mammary epithelial cells drives milk production during lactation, the mechanism however, is not clearly understood. Thus, we aimed to evaluate how neonate colostrum and formula intake administered at varying levels, affect epithelial and stromal cell proliferation in the neonatal mammary gland. **Methods:** We randomly assigned piglets to one of six treatment groups: bottle-fed pooled colostrum at 20% (COL 20, n=10) or 10% (COL 10, n=10) of body weight, milk replacer at 20% (MR 20, n=10) or 10% (MR 10, n=10) of body weight, suckled *ad libitum* on the sow (SOS, n=9), or not fed (zero hour, ZH, n=8). All groups were euthanized 24 hours postnatally except ZH which were euthanized immediately after birth. Mammary tissues were obtained, fixed, and stained with Ki67 to identify proliferating cells using ImageJ and analyzed statistically with SAS software. **Results:** Colostrum significantly increased epithelial cell proliferation ($P = 0.05$) compared to formula. Higher feeding dose also significantly increased epithelial cells ($P = 0.0003$) and stroma cells ($P=0.0005$) than lower dose. Notably, stroma proliferation was significantly higher ($P < 0.05$) in COL 20 compared to other groups including COL 10, MR 20, MR 10, SOS and ZH. **Conclusion:** These findings indicate that sufficient colostrum intake promotes proliferation of epithelial and stromal cells in the neonatal mammary gland, suggesting a potential role in enhancing the gland's future lactation capacity. (Funded by USDA 2110002200)

40. Fetal hepatic consequences of metformin exposure *in utero.*

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Introduction: Metformin is used in adults with diabetes and by pregnant women. However, metformin crosses the placenta and may restrict fetal growth. In primary fetal hepatocytes, we have demonstrated that metformin decreases oxygen consumption and glucose production, consistent with its effects in adults, yet induces fetal-specific metabolic stress. **Objective:** Determine whether metformin, in combination with Western diet (WD), impairs oxidative metabolism, injuring the fetal liver. **Methods:** Pregnant Rhesus macaques received vehicle (VEH) or metformin (MET) and a healthy control diet (CD) or WD (n=10/group) from 0.2 to 0.85 gestation. Cesarean deliveries were performed, cord blood samples obtained, fetal weight measured, and fetal hepatocytes isolated and studied. **Results**: Hepatocytes from MET-exposed versus VEH fetuses had higher oxygen consumption rates with greater maximal (P=0.015) and spare respiratory capacity (P=0.05). There was a trend in liver tissue for increased MFN2 protein (P=0.09) and *PGC1A* gene expression (P=0.06), supporting increased mitochondrial fusion and biogenesis. MET-exposure did not affect WD-induced liver triglyceride accumulation, nor did it affect cord blood glucose or oxygen levels. However, histological analysis showed increased collagen with MET and WD exposure. Furthermore, WD+MET fetuses had increased blood lactate concentrations. Fetal weight was similar across groups; however, CD+MET versus CD+VEH female fetuses weighed less (interaction, P=0.02). **Conclusions**: Metformin is associated with fetal metabolic stress, resulting in mitochondrial remodeling and increased oxygen consumption capacity. Further, increased lactate may underlie collagen deposition and hepatocellular injury. Ongoing studies are underway to understand if liver metabolic effects are linked with sex-dependent growth effects. (Supported by NIH-R01-DK128187)

41. Arginine methyl transferase and epigenetic equilibrium in trophoblast development to prevent early pregnancy loss.

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Introduction: Recurrent pregnancy loss (RPL) is the failure of two or more clinically relevant pregnancies, being experienced by ~2.5% of women. The molecular pathogenesis of the early trophoblast progenitor cells and differentiation in RPL is poorly understood. The Protein Arginine Methylation (PRMT1) is the one of the major epigenetic modifiers in mammalian placentation. Defective PRMT1 is associated with pathological complications and dictates the equilibrium of trophoblast stem cells and other lineages of trophoblast, extravillous trophoblast stem cells (EVT) and Syncytiotrophoblast (ST) cells. Our main aim is to understand the role of PRMT1 in maintaining a balance between cell fate choice of TSCs and other trophoblast cells in early and pathological pregnancies. We investigate how PRMT1 is contributing to the defective development of trophoblast progenitors and aim to dissect out the conserved transcriptional programming in mammalian placentation. **Methods:** Discarded, deidentified first-trimester placental tissues from elective termination and placental tissues from patients with idiopathic RPLs were obtained at the University of Kansas Medical Center with consent from patients. We procured ***Prmt1***$^{tm1a(EUCOMM)Wtsi}$ mouse**,** that harbors the Knock-out first (**tm1a**) allele developed by the Knockout Mouse Project, and we also have generated *Prmt1* conditional ready mice by crossing the *Prmt1*^{tm1a} mice with mice ubiquitously expressing Flp recombinase. **Results:** Subset of idiopathic RPLs placenta is associated with strong reduction of PRMT1 expression in trophoblast progenitor populations. Using genetic mouse model and human trophoblast stem cells (hTSC), we provide evidence that a conserved PRMT1 is essential to maintain trophoblast progenitors and progression of pregnancy including mammals. Our mechanistic analysis provides evidence that PRMT1 regulates transcription of key genes by directly modifying histone arginine 4 methylation and their chromatin loci. **Conclusions:** Depletion of PRMT1 in human TSCs impairs their self-renewability and activates ST transcriptional program. Impaired expression of PRMT1 in RPL and hTSC is associated with the loss of TEAD4, a hippo pathway component. PRMT1 abrogation leads to the loss of trophoblast progenitors at E7.5 in post-implantation mouse embryo. Together, PRMT1 acts as the gatekeeper of trophoblast stem cells and propensity to differentiate in other lineages.

42. A polygenic risk score associated with gestational diabetes mellitus.

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Introduction: Gestational diabetes mellitus (GDM) is a state of hyperglycemia during pregnancy. GDM can increase risk of birth complications, and subsequent type 2 diabetes mellitus in the mother and offspring. Risk factors such as diet, BMI, and family history have demonstrated strong association with GDM but no clear pathophysiology has been ascertained. **Methods:** A diagnosis of GDM was abstracted from participant medical records. Analysis was conducted on 41 women with and 320 without GDM. Genotypes of 7 genetic variants were available. Additive and dominant genetic models were evaluated by chi-square and multivariate logistic regression methods. A genetic risk score comprised of total risk alleles among the 7 variants was also evaluated. **Results:** Multivariate logistic regression showed significant, independent, positive associations between body-mass index (BMI), age, the posited genetic risk score and GDM. Genetic variant rs1421085 was associated with GDM in multivariate analysis (OR 2.12, 1.08-4.15, p=0.029. The polygenic risk score showed association with GDM, odds ratio 1.27, 95% CI 1.09 - 1.48, p=0.003. **Discussion:** This relatively small cohort replicated a previously proposed polygenic risk score, as well as established risk factors for GDM (age and BMI). A previous meta-analysis in the literature reported a degree of heterogeneity between the 3 large cohorts analyzed, suggesting that the effect of these variants may differ according to genetic background. **Conclusion:** We validate a previously published polygenic risk score for GDM in an ethically unrelated population. (Supported by NIGMS P20GM103442)

43. Maternal-placental-fetal cross communication: impact on pregnancy outcomes and life-long health.

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The role of the placenta in delivering nutrients and oxygen to the fetus in order to support growth and development is well established. How these processes are regulated and how resulting placental functional changes contribute pregnancy pathology remains unclear. Pregnancy complications and pre-term birth are recognized as major contributors to life-long health risk. The placenta communicates to maternal organs and we have shown that placental exosomes are one factor modulating maternal insulin sensitivity. Maternal factors, such as adipokines, hormones and nutrients are critical signals to the placenta and modulate its' growth and function. In obese mouse pregnancies, improving adiponectin levels was sufficient to restore appropriate placental function, fetal growth was normalized and long-term health of offspring dramatically improved. Using a mouse model of placental specific gene manipulation, we have reduced expression of placental nutrient transporters to demonstrate that specific nutrients such as essential amino acids and long chain poly unsaturated fatty acids play critical roles in overall fetal growth and brain development. Additionally, we have recently shown that the placenta releases factors that are likely involved in fetal angiogenesis, neurogenesis, lung, liver and pancreatic function. We have developed a working model in prematurely born guinea pig pups to demonstrate that placental factors are critical for successful fetal organ development. This opens up a potential therapeutic target for infants born early and have lost the contribution of the placenta. The presentation will focus on the importance of cross talk of mother- placenta- fetus for healthy outcomes at birth and life-long health.

44. Sexual dimorphism in E-cigarette-induced developmental lung immune adaptations.

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Electronic cigarette (e-cig) use during pregnancy has become a major health concern in recent years and is perpetuated by the perception that e-cigs are less harmful than traditional combustible cigarettes. An extensive knowledge gap persists regarding their health impact when aerosolized, especially during pregnancy. Recent reports indicate e-cig vaping leads to heightened risk to respiratory infection in nonpregnant adults, but there is limited understanding in the neonatal population. We hypothesized that fetal exposure to e-cig aerosols alters the developing lung immune environment in a sex-specific manner, resulting in deficient antiviral innate immune responses and increased risk to respiratory viral infections. Using our well-established pregnant rat model, we utilized a custom-engineered e-cig system with a commercial e-cig unit and atomizer that offered a translational inhalation delivery method and generated vapor profiles directly comparable to human vaping. Our data indicate a major separation of immune-related genes between male and female fetal lungs, with sex-specific deficits in innate immune pathways, including the neutrophil system. Flow cytometry analysis of fetal lungs confirmed sex specific changes in innate immune cell populations following e-cig exposure, primarily in neutrophils. RNA-seq data showed that both viral-sensing pathways, and type I IFN signaling pathways, including IRFs, interferon receptors, interferon stimulated genes (ISGs), were downregulated in a sex-specific manner after e-cig exposure, suggesting impairment in antiviral responses. Importantly, sex-specific enhanced susceptibility to neonatal viral infection in the e-cig exposed developing lung was noted. Sex-specific innate immune alterations were compounded by altered morphometrics, and e-cig altered both neonatal lung development and function in a sex- specific manner. These data provide novel information in a growing area focused on e-cig effects on the offspring lung and its influence on appropriate fetal/neonatal immune responses and highlights the importance of examining sexual dimorphism in developmental adaptations. These findings emphasize the need for translational studies to better understand and manage respiratory infections that show clinical differences between males and females. (Supported by NIH HL151497, AA23520, AA23035)

45. Vitamin and mineral supplementation in pregnant F0 beef dams may induce persistent changes in the hepatic transcriptome of F1 heifers.

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Supplementation with vitamins and minerals (VTM) in the diet of pregnant F0 dams influences offspring development; however, long-term effects on metabolic organs, including the liver, remain unclear. This study evaluated hepatic gene expression in F1 heifers born to F0 dams fed a basal diet with VTM (n = 8) or without VTM (CON; n = 7) throughout gestation. After birth, F1 heifers were managed as a single group and inseminated with sexed female semen from a single sire. At 250 days of gestation and ~22 months of age, F1 heifers were harvested and liver samples collected. Total RNA was extracted, sequenced, and reads aligned using STAR after quality control. Differential expression analyses were performed in R (v4.4.2) using the DESeq2 package, and over-representation of KEGG pathways and biological processes was carried out using ShinyGO v.0.76. A total of 13,990 genes were tested, of which 486 were differentially expressed ($P \leq 0.05$; $|\log2FC| \geq 0.5$), including 241 upregulated and 245 downregulated in the VTM group. Upregulated genes by maternal VTM supplementation included *RPL23*, *SOX4*, *PTGS2*, *NDUFC1*, *UQCRH*, and *RPS28*, associated with pathways including ribosome and processes like cell proliferation and oxidative phosphorylation. Downregulated genes, including *EIF2AK2*, *ESR1*, *SLC7A1*, and *PIK3CB*, were associated with processes related to regulation of cell population proliferation and organonitrogen compound biosynthetic process. These results indicate that VTM supplementation in F0 dams may induce persistent changes in

hepatic gene expression in the offspring, with potential metabolic implications during critical physiological stages, such as during gestation in F1 heifers. (Supported by USDA 2022-67016-36479)

46. Maternal nutrition and developmental programming – lessons from livestock models.

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Our laboratories study “problems of pregnancy,” which include infertility, poor pregnancy outcomes (often reflected by fetal growth restriction and the accompanying low birthweight), and premature birth. These problems of pregnancy underpin the high incidence of low birthweight in humans (>20 million low-birthweight infants [15 to 18% of all births] per year worldwide); they also contribute to neonatal morbidity and mortality as well as developmental programming of the surviving offspring. Similarly, among livestock in the U.S. low birth weight contributes to high rates of neonatal mortality (≈10% of all births) and developmental programming of the offspring. Although there are consequences for development of many fetal organs, this talk will focus on the placenta because of its critical role in supporting pregnancy establishment, fetal growth and development, and timing of parturition. It also will focus on livestock models, as studies using livestock (e.g., cattle, horses, pigs, and sheep) have led to an improved understanding of mechanisms contributing to poor fertility, poor fetal growth/development, and premature birth. In addition, although the talk will focus primarily on the mechanisms by which maternal nutritional stress contributes to problems of pregnancy, it also will briefly discuss other stressors including multiple fetuses, maternal age, environmental stress, breed/ethnicity, and, especially, assisted reproductive technologies. Lastly, the talk will discuss recent studies focused on strategies designed to overcome the negative consequences of maternal nutritional stress and will conclude with suggestions for future research directions.

47. Transcriptomic effects of increased maternal estradiol on fetal organs in late-gestation ewes.

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The World Health Organization estimates that 15 million babies are born preterm each year, with one newborn dying every 40 seconds. Understanding the mechanisms that initiate normal parturition is critical to identifying disruptions associated with preterm birth. In sheep, activation of the fetal hypothalamic-pituitary-adrenal axis, resulting in cortisol secretion, is believed to be the initial trigger for labor. We previously established a sheep model in which estradiol (E2) administration reliably induces parturition, enabling controlled investigation of labor mechanisms. In this study, we used this model to investigate the transcriptomic effects of E2 on fetal organ maturation. Pregnant ewes at late gestation (days 139–142) were randomly assigned to an E2-treated (n = 6; 4 Silastic® implants × 50 mg each) or a control (n = 6; 4 empty implants) group. Implants were inserted subcutaneously in the axillary region, and ewes were euthanized 26 h post-treatment for tissue and blood collection. Samples of the fetal hypothalamus, pituitary, adrenal gland, and lungs were collected for RNA sequencing. Estradiol concentration was assessed in maternal systemic blood, umbilical vein, and fetal vein samples. Maternal E2 levels increased five-fold in treated ewes ($p \leq 0.01$), while umbilical and fetal E2 levels were not affected relative to controls. Differentially expressed genes (DEGs) ($p \leq 0.05$; $|\log2FC| \geq 1$) were identified in all fetal tissues: 4 (hypothalamus), 240 (pituitary), 5 (adrenal), and 2 (lungs). These findings suggest that maternal E2 indirectly influences fetal organ gene expression, particularly in the pituitary, potentially contributing to fetal organ maturation and preparedness for birth. (Supported by USDA 2021-67015-34277)

48. The impact of leucine supplementation in milk replacer on muscle proteome and metabolism in neonatal dairy calves.

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Introduction: Early postnatal nutrition plays a critical role in regulating muscle growth and metabolism. Leucine, a branched-chain amino acid, activates the mTOR signaling pathway and promotes protein synthesis, yet its specific effects on the muscle proteome in neonatal dairy calves remain unclear. **Objective:** To investigate the influence of leucine supplementation in milk replacer (MR) in muscle proteomic profiles at day 28 of treatment. **Methods:** Thirty-five newborn Holstein heifer calves were randomly assigned to one of three treatment groups: Control (no added amino acids), Leucine-supplemented (5% in MR), and Alanine-supplemented (isonitrogenous to Leucine). Calves received equal MR volumes for 56 days. Muscle biopsies collected on day 28 underwent proteomic analysis using Data-Independent Acquisition (DIA) via Orbitrap Exploris 480, with protein quantification performed using Spectronaut. Protein–protein interaction (PPI) networks and Gene Ontology (GO) enrichment were analyzed with STRING v12.5. **Results:** In the Leucine vs. Control group, 25 proteins were upregulated ($P \leq 0.05$), and 15 were upregulated in Leucine vs. Alanine, with enrichment in amino acid and nucleotide metabolism, suggesting enhanced protein synthesis and cell proliferation (FDR ≤ 0.05). Steroid hormone response proteins (dehydroepiandrosterone, estradiol, progesterone) and caseins were enriched in both comparisons (FDR ≤ 0.05), implying a role for hormonal regulation in muscle development. In the leucine-supplemented groups, proteins associated with catabolic processes were downregulated (FDR ≤ 0.05), suggesting a shift towards anabolic metabolism. **Conclusion:** Leucine supplementation promoted an anabolic muscle environment, enhancing proteome remodeling and metabolic efficiency. (Supported by SBARE)

49. Does fetal sex influence placental abnormalities in polycystic ovary syndrome (PCOS) patients conceiving with fertility treatments (FT)?

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Introduction: Sex-specific differences in fetal-placental signaling are well established. Female (F) fetuses favor glucocorticoid-regulated pathways, enhancing placental reserve but limiting growth. Male (M) fetuses prioritize androgen-driven signaling, promoting growth at the cost of adaptability. In PCOS, an androgen-mediated condition, these adaptations may be exaggerated, potentially altering placental histopathology. **Methods:** We retrospectively reviewed placental pathology data from singleton livebirths (1/2004–4/2022) conceived with FT (n=1381). PCOS patients (Rotterdam criteria; n=181) were grouped by fetal sex (M=90; F=91). Placental findings were categorized as anatomic, inflammatory, infectious, or vascular by a blinded perinatal pathologist using Amsterdam Workshop Consensus definitions. Comparisons were made using parametric/nonparametric tests. Logistic regression calculated crude and adjusted odds ratios (aOR), controlling for maternal age, BMI, race, gestational age, FT type, and gestational diabetes (GDM). **Results:** Baseline characteristics of PCOS patients who delivered M vs F fetuses did not differ significantly [age, mean (SD)—M: 32.8 (3.1) vs F: 33.0 (3.8), p=0.75; BMI— M: 26.2 (5.6) vs F: 26.2 (5.7), p=0.89; nulliparity, n (%)—M: 67 (74.4%) vs F: 75 (83.3%), p=0.14; FT—Ovulation Induction /Intrauterine Insemination—M: 40(44%) vs F: 48 (53.3%); In Vitro Fertilization—M: 51 (56%) vs F: 42 (46.7%), p=0.21; GDM—M: 14 (15.4%) vs F: 8 (8.9%), p=0.18]. There were no differences in anatomic, infectious, or vascular abnormalities by fetal sex in crude or adjusted models. Inflammatory abnormalities—villitis of unknown etiology, deciduitis, and intervillositis—were more frequent in F placentas on crude analysis [F: 20 (23%) vs M: 9 (9.9%), OR 1.14 (95% CI 1.02–1.27); p=0.01], but not after adjustment [aOR 1.07 (95% CI 0.91–1.26); p=0.4]. **Conclusion:** This study suggests that fetal sex does not significantly impact placental pathology in PCOS pregnancies. These findings provide reassurance that fetal sex differences may not be a major factor in placenta-mediated pregnancy complications in PCOS patients undergoing FT. Further research should explore the potential influence of androgen exposure on placental function and long-term fetal outcomes in this population.

50. Associations between maternal extreme heat exposure and inflammatory biomarkers in early and late pregnancy.

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Introduction: Maternal heat exposure has been linked to adverse pregnancy outcomes. The impact of high ambient temperature exposure on inflammation during human pregnancy remains largely unknown. **Objective:** Determine the association between preconception and trimester-wise exposure to excessive heat stress (HS) with α1-acid glycoprotein (AGP) and C-Reactive Protein (CRP) at 12 and 34 weeks of gestation. **Methods:** This secondary analysis included women with serum concentrations of AGP (n = 160), and CRP (n = 143) collected at both 12 and 34 weeks of pregnancy from Women First Preconception Maternal Nutrition Trial conducted in Thatta, Pakistan. Excessive HS was categorically defined as having > 20 days with average maximal daily temperature > 39°C in each period, 90 days preconception (PreC), trimester 1 (T1), or trimester 2 (T2). Multiple linear regression was used to assess relationships between HS and each inflammatory marker in separate models for 12- and 34-weeks assessments, including adjustment for maternal characteristics, intervention arm, cluster, maternal anemia status, $PM_{2.5}$ levels, and 12-week AGP or CRP (for 34-week outcomes). **Results:** Exposure to HS during PreC increased 34-week AGP by 0.10 µg/mL compared to no HS exposure (p= 0.045). Exposure to HS compared to no exposure was positively associated with 34-week CRP (mg/L) during PreC (ß= 1.90, p= 0.015), T1 (ß= 2.06, p= 0.009), and T2 (ß= 1.93, p= 0.020). No significant associations were observed between exposure to HS and inflammatory markers at 12 weeks. **Conclusion:** Findings suggest that preconception and early pregnancy HS may contribute to late-gestation inflammation.

51. Intergenerational transmission of paternal environmental effects via sperm small RNAs in mice.

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Introduction: Transmission of parental traits to offspring is the most fundamental process for the perpetuation of life on earth and is vital for the process of evolution. Although there is mounting evidence from worms to humans suggesting that parental environment can influence phenotypes in offspring, the mechanism of such intergenerational inheritance remains deeply mysterious. Our previous studies implicated sperm small RNAs in intergenerational epigenetic inheritance of paternal environmental effects. We found that cleavage products of tRNAs, known as tRNA-derived small RNAs or tRNA fragments (tRF), are highly abundant in mature sperm and

environmental conditions alter their levels. A 5' fragment of tRNA-Valine-CAC-2 (tRFValCAC) as one of the most abundant tRNA fragments in mature mouse sperm. tRFValCAC is enriched in sperm during epididymal maturation, and extracellular vesicles (EVs) secreted by epididymal epithelial cells can deliver tRFValCAC to sperm. **Objective:** Here, we investigated how tRFValCAC is delivered to sperm and what functions it carries out upon deposition in the embryo at fertilization to elucidate the mechanism of sperm tRF-mediated intergenerational inheritance. **Methods:** We used a combination of RNA-sequencing, assisted reproduction, and embryo microinjections to study the dynamics and functions of tRFValCAC. **Results:** Our studies demonstrate that heterogeneous nuclear ribonucleoprotein A/B (hnRNPAB) binds tRFValCAC in the epididymis and regulates its abundance in EVs, thereby modulating its levels in sperm. Inhibition of tRFValCAC in preimplantation embryos alters transcript abundance of genes involved in RNA splicing and mRNA processing, dysregulates alternative splicing, and delays preimplantation development. **Conclusions:** Our work revealed that a sperm-enriched tRF regulates early embryonic gene expression and the pace of preimplantation development, providing a potential mechanism of sperm small RNAs-mediated intergenerational inheritance. (Supported by NIH grant 1DP2AG066622-01 and Searle Scholars Program award number 20-SSP-109)

52. Placenta-specific RNA interference of iodothyronine deiodinase 2 in sheep.

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53. Trophoblast cells at the uterine-placental interface.

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The uterine-placental interface is a dynamic site where uterine and trophoblast cells cooperate to establish a protective environment conducive to the redirection of resources facilitating development of the embryo. The rat and human possess a uterine-placental interface characterized by deep trophoblast cell infiltration into the uterine parenchyma. Invasive trophoblast cells direct changes in uterine immune, endothelial, smooth muscle, glandular epithelial, and stromal cell constituents, and effectively anchor the placenta to the uterus and restructure uterine spiral arteries. In the human, these invasive trophoblast cells are referred to as extravillous trophoblast cells. Trophoblast cell invasion and trophoblast-directed uterine spiral artery remodeling are critical events in the establishment of pregnancy. Failures in trophoblast-guided uterine transformation lead to obstetrical complications, including early pregnancy loss, preeclampsia, intrauterine growth restriction, and pre-term birth. Therefore, studying molecular mechanisms regulating development and function of the invasive trophoblast/extravillous trophoblast cell lineage is clinically relevant and is of considerable importance. Our research approach involves identification of candidate conserved regulatory pathways controlling invasive trophoblast/extravillous trophoblast cell lineage development using comparative transcriptomic approaches, evaluating the importance of the regulators using trophoblast stem cell models, and testing critical hubs within the pathways using relevant in vivo rat models. (Supported by NIH HD020676, HD105734, HD112559, and the Sosland Foundation)

54. Gilt uterine gland density is affected pre- and post-puberty by maternal immune stimulation in late gestation.

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Uterine glands provide critical nutrients during pregnancy, but uterine gland number is fixed shortly after birth. Thus, we sought to determine if maternal immune stimulation would decrease uterine gland density in offspring. Pregnant sows were treated on day 77 of gestation with lipopolysaccharide (LPS), vaccine (VAC), or saline (CON). Postnatal gilts were sampled from each treatment group. Reproductive tracts were collected from gilts pre-puberty (LPS, n = 4; VAC, n = 6; CON, n = 6) and post-puberty (LPS, n = 3; VAC, n = 5; CON, n = 7). Uteri were weighed and cross sections were collected from four non-sequential mid-horn segments, fixed, embedded, and sectioned. Sections were stained using hematoxylin and eosin, imaged, and assessed for the number of deep and superficial uterine glands per area (density). Data were analyzed by ANOVA for the fixed effect of treatment. Statistical significance was considered at $P \leq 0.05$. In the pre-pubertal phase, there were no differences in deep or superficial uterine gland density among

treatment groups. However, uterine weight tended to be reduced in gilts from the VAC group compared with the CON and LPS groups. In the post-pubertal phase, there were no differences in deep or superficial uterine gland density among treatment groups. However, uterine weight was increased in the VAC and LPS groups compared with the CON group. Gestational immune stimulation does not affect uterine gland density in offspring; however, it does alter uterine weight, and thus affects the total number of uterine glands, and perhaps secretory capacity.

55. Maternal 3-hour placental lactogen infusions in sheep increase fetal glucose uptake in hyperglucagonemic fetuses by regulating placental utilization.

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56. Revisiting the adipocyte number hypothesis and its implications for developmental programming.

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The strongest predictor of childhood obesity is being born to a mother who was obese during pregnancy, which is now the case for ~30% of pregnancies in Canada and the USA. Therefore, women's metabolic health during pregnancy is an important target for preventative measures aimed at addressing the obesity crisis. Our lab is exploring the role of adipose progenitors in programming early life adiposity and mediating the heightened risk for metabolic syndrome in offspring born to pregnancies complicated by obesity. The setpoint of adiposity is determined in early life by the number of adipocytes arising from a developmental pool of adipose progenitors that are specified to the adipocyte fate prior to birth. After puberty, adipocyte numbers remain stable throughout the rest of life. A pool of adipose progenitors residing in adult adipose depots serves as a reservoir to support adipocyte turnover, which is increased in obesity, and thereby plays a critical role in preserving metabolic health. Using a mouse model of diet-induced maternal obesity, our lab has shown that a perturbation in developmental adipogenesis, raises the setpoint of early life adiposity and predisposes to later-life adipose dysfunction and metabolic syndrome. Predisposition of offspring to metabolic dysfunction is sex-dependent, which our data demonstrate to be an estrogen-mediated sex difference arising at puberty rather than a programming effect. Together, our work provides new insight into the relationship between maternal obesity and childhood metabolic health and the modifying influence of sex. (Funded by the Canadian Institutes of Health Research, Heart and Stroke Foundation of Canada, Natural Sciences and Engineering Research Council of Canada, and Diabetes Canada)

57. Nutrient restriction during gestation influences fetal small intestine weight, histomorphology, and microbial diversity.

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Introduction: Proper development of the small intestinal epithelium and establishment of a diverse microbiome is critical for transport and absorption of nutrients, and gut health, but may be negatively impacted by restricted nutrition during gestation. **Objective**: To determine if maternal nutrient restriction impairs morphological characteristics and microbial community of fetal small intestine, multiparous Dorset ewes pregnant with twins were fed 100% (n=8) or 60% (n=7) of requirements from d 30 – d 130 of gestation. Ewes were euthanized at d 130 of gestation, and fetal measurements and samples were collected. Fetuses are referred to as CON (n=8 female; n=8 male) or RES (n=9 female; n=5 male), corresponding to their dam's diet. DNA was extracted from jejunal mucosal scrapings and v4 of 16S was amplified and sequenced to determine microbial diversity. **Results**: Small intestine weight (g/kg BW) in RES-females was 11.5% lighter than CON-females (P=0.04). In the duodenum, villi height (VH; μm) were 20.2% and 21.6% shorter in RES-males relative to CON-males and RES-females (P≤0.04). In the jejunum, VH were 7.9% and 10.6% shorter in RES-males relative to CON-females and RES-females (P≤0.03). In the ileum, VH were 11.5% shorter in CON-males relative to CON-females (P=0.005). A Shannon Index for α-diversity revealed that RES-males have decreased diversity relative to CON-females (P=0.013) and a Bray-Curtis analysis for β-diversity had a main effect of treatment (P=0.04) indicating a difference in species composition. **Conclusion**: Maternal nutrient restriction during gestation influences fetal offspring small intestine development and microbial environment in a sex-specific manner. (Supported by USDA-NIFA 2023-67012-39740)

58. Effects of gestational nutrition in an earlier pregnancy on growth of subsequent offspring in grazing beef cattle.

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Introduction: Human health effects due to birth order have been extensively studied. This topic is less researched in livestock, likely due to pronounced confounding of birth order with age of dam in a k-strategist managed to conceive “early and often”. Emerging evidence indicates a need to better understand longitudinal effects of gestational nutrition on health and well-being of future progeny in livestock. **Objective:** To determine the effects of beef cow stocking rate (SR; a proxy for cow nutrition) at three different production cycle periods on performance measures in offspring. **Methods:** 1,900 records from calves born over a 30-year period to *Bos taurus* x *B. indicus* cross cows at three SR (high, moderate, low) and periods (previous gestation, current gestation, current suckling) were analyzed. Calf weight and performance data were collected during the suckling period, the feedlot period, and from carcasses. **Results:** Nutrition during gestation and suckling affected calves as expected; i.e. measures from high SR calves were generally lower ($P < 0.05$) than those from moderate and low. High SR during the pregnancy immediately previous to that of a given calf had no effect ($P > 0.1$) on calf birth weight but generally depressed measures of feedlot/carcass performance compared to moderate and low ($P < 0.05$). Specifically, hot carcass weight (kg) was 376 ± 2.5, 390 ± 1.3, and 386 ± 2.9, for high, moderate and low SR, respectively. **Conclusion:** Intrauterine conditions of an earlier pregnancy affected outcomes for future offspring at the point of harvest. (Funded by Texas Cattle Feeders Association)

59. Placental vulnerability at the molecular level: The EGFR axis under chemical stress.

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Increasing evidence suggests that exposure to endocrine-disrupting chemicals (EDCs) during pregnancy can adversely affect maternal and fetal health. The placenta, a transient yet essential endocrine organ, is a sensitive target of such exposures. Among EDCs, bisphenol S (BPS)—a common replacement for bisphenol A—is frequently detected in pregnant individuals. Through in vivo models, primary human trophoblasts, and 3D microfluidic platforms, our work has shown that BPS disrupts placental function by antagonizing the epidermal growth factor receptor (EGFR), impairing trophoblast fusion and invasion. Building on these findings, we have recently investigated a mixture of EGFR-targeting environmental chemicals and demonstrated that this mixture alters trophoblast cell function and disrupts mitochondrial bioenergetics. These studies identify EGFR as a molecular target of environmental chemicals and illustrate how receptor-level disruptions may contribute to placental dysfunction. This talk will highlight mechanistic and functional findings on EGFR-targeting chemicals and their implications for pregnancy outcomes.

60. Investigating placental protein changes associated with Assisted Reproductive Technologies in a mouse model.

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Pregnancies utilizing Assisted Reproductive Technologies (ART) are associated with several complications including an increased risk of preeclampsia, placental abruption, and morbidly adherent placentas. Our work along with others has shown that placentas from mice conceived by in vitro fertilization (IVF) display overgrowth, impaired blood vessel development, altered gene expression and DNA methylation. These changes are associated with impaired fetal growth. We sought to identify the placental protein differences with IVF to inform potential interventions for improving ART procedures. Placental protein differences among IVF and spontaneously-conceived mouse concepti were analyzed in a sex-specific manner (n=4-5 placentas/sex/experimental group/timepoint) by data-independent acquisition mass spectrometry at five timepoints covering shortly after placental formation through term (E11.5, 12.5, 14.5, 16.5, and 18.5). Peptides below 1% FDR were included, and statistical analysis was performed using Two-way ANOVA and Tukey HSD post hoc tests, and adjustment for multiple hypothesis testing. We observed normal, dynamic abundance changes for placental proteins over development. IVF induces an overall reduction in the abundance of several placental proteins at the earliest timepoint, E11.5. Several of the identified affected proteins are known to be important for placenta development and epigenetic regulation. Intriguingly, we observed most placental protein changes were observed among the female concepti but not the male concepti. To our knowledge, this is the first proteomic analysis of mouse placentas at multiple timepoints along gestation. We are currently conducting spatial transcriptomics experiments to determine if protein changes are associated with specific placental compartments.

61. Daily supplementation of ω-3 polyunsaturated fatty acids to IUGR-born neonatal lambs improved skeletal muscle growth and body composition.

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Introduction: Muscle growth is impaired in intrauterine growth-restricted (IUGR) offspring by programming that includes elevated inflammatory tone. The resulting body composition changes reduce production efficiency in livestock and compromise long-term health

in humans. **Objective:** Evaluate whether anti-inflammatory ω-3 polyunsaturated fatty acid (PUFA) supplements improve body composition in IUGR-born lambs. **Methods:** Pregnant ewes were housed under thermoneutral or hyperthermic conditions to produce control (n=12) or IUGR lambs. From birth, IUGR lambs were supplemented (oral bolus) 0.42 g/kg ω-3 PUFA Ca^{2+} salts (IUGR+ω-3; n=12) or placebo (IUGR; n=11). Biometrics were assessed weekly. Loin ultrasounds were conducted on day 23. Muscle weights were determined at necropsy on day 28. **Results:** Crown circumference/BW, abdominal circumference/BW, and cannon bone length/BW were greater (P<0.05) for IUGR lambs than for controls or IUGR+ω-3 lambs. Body length/BW was greater (P<0.05) for IUGR lambs than for controls and was intermediate for IUGR+ω-3 lambs. IUGR lambs had 14% less (P<0.05) ultrasound-estimated subcutaneous fat and 22% smaller (P<0.05) loin cross-sectional areas. Both were recovered in IUGR+ω-3 lambs. Ultrasound-estimated loin depth was less (P<0.05) for IUGR lambs (16.5±0.4 mm) than for controls (14.6±0.5 mm) and was intermediate for IUGR+ω-3 lambs (15.6±0.6 mm). At necropsy, IUGR lambs had 20 – 23% lighter (P<0.05) *biceps femoris*, *semitendinosus*, *gastrocnemius*, *flexor digitorum superficialis,* and *longissimus dorsi* muscles than controls. These deficits were partially or completely recovered in IUGR+ω-3 lambs. **Conclusions:** Supplementing ω-3 PUFA improved muscle growth and body composition of IUGR-born lambs, demonstrating the role of inflammatory programming in IUGR growth outcomes. (Supported by USDA 2019-67015-29448, 2020-67015-30825)

62. Investigating maternal physiological responses to pregnancy in a guinea pig model of increased maternal stress.
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Introduction: Chronic maternal stress during pregnancy can have lasting impacts on both maternal health and fetal development, yet the underlying maternal physiological adaptations remain poorly understood. **Objective**: Investigate increased chronic maternal stress, through food insecurity, on maternal physiological parameters at two gestational (GD) timepoints. **Methods**: Female Hartley guinea pigs were fed either an *ad libitum* (Control) or a restricted diet (MNR) from 4 weeks prior to pregnancy until sacrifice. MNR dams were provided food at 1000h daily which was generally consumed within 4-6h leaving a period of ~18-20h without food. Dams were euthanized at GD35-38 (mid-pregnancy: Control n=7, MNR n=6) or GD57-63 (near-term: Control n=6, MNR n=6) and weights recorded. Maternal plasma was analyzed for various metabolic markers. Statistical significance was determined using generalized linear modelling. **Results**: While maternal weight (minus fetal and maternal-fetal interface weight) and maternal-fetal interface weight increased between timepoints (P<0.001, both respectively), they remained similar between MNR and Control. MNR maintained litter size but had reduced fetal weight at both mid-pregnancy and near-term (13-19%; P=0.014). Between mid-pregnancy and near-term, maternal cortisol (P=0.041), glucose (P=0.001), and calcium (P=0.004) increased, while progesterone (P<0.001), lactate (P<0.001), and sodium (P=0.021) decreased. MNR dams showed elevated cortisol (53-85%, P=0.016), progesterone (13-35%, P=0.036), and BUN (8-38%, P=0.012) and reduced cholesterol (24-32%, P=0.019) compared to Controls at both gestational timepoints. **Conclusion**: Chronic maternal stress from before pregnancy leads to a metabolic state characterized by elevated cortisol and altered energy utilization (increased BUN, decreased cholesterol), suggesting metabolic adaptations to prioritize maternal survival but with compromised fetal growth. (Supported by NIH K99HD109458 & R01HD090657)

63. From rhinos to sea stars: innovative applications of Assisted Reproductive Technologies to support wildlife conservation.
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64. Late gestation maternal feed restriction alters specific rumen microbiota of the neonatal beef calf.
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Introduction: Feed restriction during late gestation in beef cattle is a relevant issue in the inter- mountain west due to the overlap of limited and low-quality feed resources with heightened energy requirements associated with late gestation and early lactation. Little is known how this period of potential restriction may impact the rumen microbiota or the neonatal calf. **Objectives:** The objective of this research was to asses the effects of maternal feed restriction on offspring rumen microbiota. We hypothesize that there will be microbial profile differences between calves born to control fed versus feed restricted cows and that those differences persist through one month of age. **Methods:** Mature cows (n=30) were provided 100% of intake requirements (CON) or 70% of the intake volume (FR). Restriction began 60d prior to calving and continued through one-month post-parturition. Eight animals from each treatment were randomly selected for sampling. Rumen fluid was collected from the calf at birth (RFd1), d7 (RFd7), and d28 (RFD28) post- parturition. DNA was isolated using a lysis buffer and mechanical bead-beating procedure and purified using the QIAamp DNA Stool Mini Kit (Qiagen). Amplicon sequencing of the 16S rRNA V4 region was completed on the MiSeq and analyzed with ANCOM-BC plugin within QIIME2. **Results:** At birth, the phylum Fibrobacterota was enriched in FR calves (q=0.003) where Sumerlaeota was depleted (q=0.03) compared

to CON calves. Desulfobacterota was enriched in FR calves (q=0.02) and Deferribacterota was depleted (q=0.01) relative to CON calves at d7. At d 28, the phylum Patescibacteria was depleted (q≤0.001) in FR calves compared to CON calves. Results indicate feed restriction during gestation could influence calf rumen profiles with potential impacts on functions such as degradation of cellulose, sulfur cycling, and utilization of iron. More research is needed to determine if these differences have implications in long-term animal health and performance.

65. Is the developing lung susceptible to acetaminophen toxicity?

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Acetaminophen exposures in the perinatal period are ubiquitous. In addition to being the most commonly used drug during pregnancy, clinicians have increasingly prescribed acetaminophen (APAP) for patients in the neonatal intensive care unit (NICU). Acetaminophen has been shown to reduce post-operative opiate burden and may provide similar efficacy for closure of the patent ductus arteriosus (PDA) as nonsteroidal anti-inflammatory drugs (NSAIDs). However, while APAP exposures have spread to a highly vulnerable population of increasingly less mature infants, robust pharmacokinetic and pharmacodynamic data for APAP are lacking. Concerningly, preclinical studies suggest that perinatal APAP exposures may result in unanticipated adverse effects that are unique to the developing lung. I will discuss the clinical observations linking APAP exposures to adverse respiratory outcomes and the preclinical data demonstrating a developmental susceptibility to APAP-induced lung injury. I will discuss how clinical observations linking perinatal APAP exposures to pulmonary injury have been taken to the bench to produce important insights into the potential mechanisms underlying these findings.

66. Exogenous oxygen and glucose supplementation remodels the mitochondrial proteome in skeletal muscle of FGR fetal sheep.

Weicheng Zhao[1]*, Daniel Chrisenberry[1], Mariangel Varela[1], Rosa Luna-Ramirez[1], Paul Langlais[2], Laura Brown[3], Sean Limesand[1]

[1]School of Animal and Comparative Biomedical Sciences, University of Arizona, Tucson, AZ, USA; [2]Department of Endocrinology, University of Arizona, Tucson, USA; [3]Department of Pediatrics, University of Colorado Anschutz Medical Campus, Aurora, CO, USA

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SPEAKER BIOS

Teresa Davis, PhD

Texas A&M University
2025 KEYNOTE ADDRESS
Impact of Prematurity on the Nutritional Regulation of Growth

Teresa Ann Davis, PhD is the Director of the Institute for Advancing Health Through Agriculture at Texas A&M University and formerly a Professor of Pediatrics at the USDA/ARS Children's Nutrition Research Center at Baylor College of Medicine in Houston, Texas. She received her doctorate from the University of Tennessee and her postdoctoral training from Washington University School of Medicine in St. Louis, Missouri. Dr. Davis is recognized internationally for her studies on nutritional regulation of protein and amino acid metabolism and growth in humans and animals. She is an established expert on amino acid and insulin signaling and the metabolic effects of nutrients, hormones, and growth factors on muscle growth. Dr. Davis received the Research Mentor Award from Baylor College of Medicine, the Centennial Leader Award from the University of Tennessee, the Stockstad Award from the American Society for Nutrition, and the Animal Growth and Development Award and Morrison Award from the American Society of Animal Science. Dr. Davis was elected President of the American Society for Nutrition and the American Society of Animal Science and is a Fellow of both societies. Dr. Davis served as Editor-in-Chief of *The Journal of Nutrition* for 10 years and is currently Editor-in-Chief of *Critical Reviews in Food Science and Nutrition.* She was a member of the USDA/HHS 2020 Dietary Guidelines Advisory Committee and is an elected member of the National Academy of Medicine.

Laura Brown, MD

University of Colorado Anschutz Medical Campus
DJP BARKER MEMORIAL LECTURE
Reduced Myogenesis in Fetal Growth Restriction: Basic Mechanisms & Future Therapies

Laura D. Brown, MD is a Professor of Pediatrics at the University of Colorado Anschutz Medical Campus and a clinical neonatologist. She holds the Giacomo Meschia, MD Endowed Distinguished Professorship in Neonatal-Perinatal Research. Her overall research goal is to understand the basic biology of fetal muscle development and protein metabolism to optimize body composition and growth in infants born after exposure to pathological conditions in pregnancy, including fetal growth restriction. She made the novel discovery that lower muscle mass in the growth-restricted fetus is due to a marked reduction in muscle protein synthesis rates, as opposed to increased protein breakdown rates. She performs fetal physiological studies using large animal models and uses stable isotopes and metabolomics to assess muscle-specific protein metabolism. She blends her basic science/translational research in fetal growth and metabolism with clinical nutritional management of the preterm and IUGR neonate. She is a former K12 Building Interdisciplinary Research Careers in Women's Health (BIRCWH) Scholar and her work is currently R01 funded.

Jay Ramadoss, PhD

Wayne State University

SESSION I – EARLY INFLUENCES ON GROWTH & REPRODUCTIVE COMPETENCE

Sexual Dimorphism in E-cigarette-Induced Developmental Lung Immune Adaptations

Dr. Jay Ramadoss is a Professor of Obstetrics & Gynecology at Wayne State University. The emphasis of his current research is to identify the etiology of developmental disorders, with the goal of translating these findings clinically to reach the children affected by these disorders. Employing a whole animal physiological and cellular/molecular approach, his lab investigates the effects of pharmacological and nutraceutical agents for the prevention and treatment of gestational/developmental disorders. He is a member of the Research Society on Alcoholism, the Fetal Alcohol Spectrum Disorders Study Group, the Society for Reproductive Investigation, and the Perinatal Research Honor Society. Widely published, he has secured numerous research grants and academic awards. Dr. Ramadoss' lab has been consistently funded by NIH. Dr. Ramadoss has been an invited speaker at reputed institutions in the US, Europe, India, and Singapore. Dr. Ramadoss has served on over 30 NIH Study Sections. He has reviewed grants for Singapore National Medical Research Council and Republic of France - French National Research Agency. Dr. Ramadoss has reviewed for 25 peer-reviewed journals and serves on editorial boards and as associate editor of several journals in his field.

Caitlin Cadaret, PhD

Colorado State University

SESSION I – EARLY INFLUENCES ON GROWTH & REPRODUCTIVE COMPETENCE

Investigating the Impacts of Maternal Environment on Lamb Performance & Reproductive Dynamics

*****Early Career Speaker*****

Dr. Caitlin Cadaret is an Associate Professor of Stress Physiology in the Department of Animal Sciences at Colorado State University. She started out in agriculture from birth, growing up on a cow-calf operation and showing western pleasure horses in Northern California. She received her BS in Animal Science at California State University, Chico and her MS and PhD at the University of Nebraska-Lincoln studying developmental programming of skeletal muscle growth and metabolism under IUGR conditions for human and animal health. Upon joining the faculty at CSU in late 2019, her research shifted primarily to livestock industry-focused approaches aimed at determining how the environment in which we manage our gestating animals impacts their progeny and subsequent generations. Her lab collaborates across disciplines to elucidate how common stressors that livestock experience in extensive and intensive management systems impact animal welfare, performance, and reproductive competence. This research aims to address common challenges by understanding mechanisms that precede between animal variation in order to develop better selection and management strategies to improve animal productivity and producer profitability. Dr. Cadaret also has a large teaching appointment, instructing courses in reproductive physiology and neonatal management, which she has leveraged to perform pedagogical research and develop a substantial undergraduate research program to complement her graduate program.

Upasna Sharma, PhD

University of California-Santa Cruz

SESSION I – EARLY INFLUENCES ON GROWTH & REPRODUCTIVE COMPETENCE

Intergenerational Transmission of Paternal Environmental Effects via Sperm Small RNAs in Mice

*****Early Career Speaker*****

Dr. Upasna Sharma is an Assistant Professor in the Department of Molecular, Cell, and Developmental Biology at the University of California, Santa Cruz. She obtained her PhD from Wesleyan University in Connecticut and trained as a Charles H. Hood Postdoctoral Fellow in the laboratory of Dr. Oliver Rando at the University of Massachusetts Medical School. Dr. Sharma's lab investigates how the paternal environment influences offspring health. During her postdoctoral studies, Dr. Sharma utilized genomic approaches to elucidate the mechanism of intergenerational epigenetic inheritance of paternal dietary effects. Her work revealed a role of sperm small RNAs in such inheritance and provided evidence of RNA-mediated soma-germline communication in mammals. Her lab at UCSC is utilizing a unique and powerful combination of genomics, genetics, and assisted reproduction approaches to investigate the mechanistic basis of RNA-mediated soma-germline communication, its influence on sperm epigenome, and the consequences for offspring health. She is a recipient of the NIH Director's Innovator Award and the Searle Scholars Award. Lab website: https://sharmalab.sites.ucsc.edu/

Amy Desaulniers, PhD

University of Nebraska-Lincoln

SESSION I – EARLY INFLUENCES ON GROWTH & REPRODUCTIVE COMPETENCE

Developmental Origins of Boar Fertility: Consequences of In Utero Heat Stress

*****Early Career Speaker*****

Amy T. Desaulniers, PhD is an Assistant Professor in the School of Veterinary Medicine and Biomedical Sciences at UNL. Dr. Desaulniers is a reproductive physiologist specializing in testis biology and endocrinology. She received her BS in Animal Science from the University of Missouri-Columbia and her MS and PhD from UNL in swine physiology. She then served as an Assistant Professor of Biology at the University of Central Missouri before returning to UNL in 2021. The Desaulniers lab studies developmental programming of the mammalian testis, including how perinatal insults disrupt normal gonadal development and lead to long-term fertility consequences. Current projects are investigating how early life events (e.g., *in utero* heat stress, agrichemical exposure, milk deprivation) predispose a lifetime of reproductive dysfunction. The overall goal of Dr. Desaulniers' research program is to improve livestock fertility in order to enhance the productivity of animal agriculture. In addition, her work serves a dual purpose by utilizing swine as a biomedical model to enhance reproductive function in humans. To date, her research has been funded by USDA-NIFA-AFRI, the US Pork Center of Excellence, the USDA/ARD Hatch Multistate Enhanced Program, the Nebraska Center for the Prevention of Obesity Diseases (NIH COBRE), the NU Collaboration Initiative, the Great Plains IDeA-CTR Early Career Investigator Program (NIH COBRE), and the UNL Research Council.

Emilyn Alejandro, PhD

University of Minnesota

SESSION II – HEALTH CONSEQUENCES OF PLACENTAL INSUFFICIENCY

Unlocking Metabolic Fate: How Placental Insulin/IGF-1 & mTOR Signaling Shape Offspring Health

Emilyn U. Alejandro is a Professor of Integrative Biology and Physiology at the University of Minnesota. She received her BS from the University of Washington, before embarking on postbaccalaureate research training at NIH. She earned her PhD at the University of British Columbia in JD Johnson's lab, where she investigated autocrine insulin signaling in β-cells. Her postdoctoral training with Ernesto Bernal-Mizrachi at the University of Michigan deepened her expertise in β-cell development, metabolic phenotyping, and the impact of intrauterine environment on health outcomes. At UMN, Dr. Alejandro's lab studies the fetal origins of obesity and diabetes, focusing on how placental insufficiency driven by maternal obesity, inflammation, and diabetes affects β-cell function and susceptibility to diabetes and cardiovascular disease. Her lab investigates the roles of key nutrient-sensing proteins (mTOR, AMPK, OGT) in β-cell development, function, and regeneration using epigenetics, biochemistry, and *in vivo* metabolic phenotyping. Her long-term goal is to break the intergenerational cycle of diabetes through prevention and cure. The International DOHaD Society and Midwest Islet Club have recognized Dr. Alejandro's work with the Young Investigator and Early Career Investigator Awards. She received the American Physiological Society's Henry Pickering Bowditch Award for original and outstanding accomplishments in physiology. Dr. Alejandro is committed to providing exceptional training and mentorship to the next generation of leaders in medical research. She serves as Co-Director of the T32 Research Training Program in Systems Biology of Cardiovascular Inflammation and Director of the R25 Postbac Readiness in Metabolism, Endocrinology, and Diabetes program. She received the Outstanding Advisor Award from the Council of Graduate Students. Dr. Alejandro is currently funded by NIDDK. She is also honored to be supported by the UMN Medical School Land-Grant Professorship in Diabetes, the McKnight Land-Grant Professorship, and the McKnight Presidential Fellowship.

Larry Reynolds, PhD

North Dakota State University

SESSION II – HEALTH CONSEQUENCES OF PLACENTAL INSUFFICIENCY

Maternal Nutrition & Developmental Programming – Lessons from Livestock Models

During his 47-year career, Dr. Lawrence P. (Larry) Reynolds has focused on "Problems of Pregnancy," which have major socioeconomic and health implications for livestock and humans. These include infertility, poor pregnancy outcomes reflected by low birthweight, and premature birth. Dr. Reynolds is a founding Director of the Center for Nutrition and Pregnancy at NDSU. He has received the ASAS Animal Physiology and Endocrinology Award, Animal Growth and Development Award, and Research Fellow designation. He is ranked in the upper 2% of top-cited researchers in the world for all STEM fields and in the upper 0.3% in his primary field of Dairy & Animal Sciences. Dr. Reynolds helped establish that placental (uterine and umbilical) blood flows are key to normal placental function like nutrient, gas, and waste transport throughout gestation. They showed that the placenta produces angiogenic factors to promote its dramatic vascular development, which is key to sustained increases in placental blood flow. Recognizing that placental growth is key, they were among the first to evaluate rates of cell turnover *in vivo*, including in the placenta. Dr. Reynolds has shown the effects of maternal stressors (e.g., malnutrition, age, environment, ART) on placental development begin very early, altering placental function and affecting fetal growth and development. They were among the first to characterize the long-term consequences of developmental programming in livestock. Currently, Dr. Reynolds investigates strategies to improve fertility, pregnancy outcomes, and prevent premature birth.

Helen Jones, PhD

University of Florida

SESSION II – HEALTH CONSEQUENCES OF PLACENTAL INSUFFICIENCY

Sexual Dimorphism in Placental Insufficiency

Helen Jones received her BS degree in Biochemistry from the University of St. Andrews and her PhD in Biomedical Sciences & Physiology from the University of Aberdeen in Scotland, UK. After postdoctoral fellowships at the University of Cincinnati and the Cincinnati Children's Hospital Medical Center, Helen joined the Cincinnati faculty as an Assistant Professor. In 2020, she relocated to the University of Florida where she is currently the William W. & Virginia Leach Professor of Obstetrics and Gynecology at the College of Medicine and the Director of the Center for Research in Perinatal Outcomes. Since completing her PhD projects investigating nutrient transfer in the placenta, Helen's research has focused primarily on understanding the placenta in a broad range of complicated pregnancies as well as the development of placental-targeted interventions.

Theresa Powell, PhD

University of Colorado Anschutz Medical Campus

SESSION III – MATERNAL HEALTH/DIET & PLACENTAL FUNCTION

Maternal-Placental-Fetal Cross Communication: Impact on Pregnancy Outcomes & Life-long Health

Dr. Powell is a Professor in the Department of Pediatrics - Division of Neonatology at the University of Colorado Anschutz Medical Campus. She graduated with her PhD from the University of Kentucky and completed postdoctoral fellowships at the University of California San Diego and the University of California San Francisco before moving to Sahlgrenska Academy at Gothenburg University, Sweden where she advanced to Associate Professor in the Department of Physiology. She was Associate Professor at the University of Cincinnati and the University of Texas, San Antonio before moving to University of Colorado in 2014. Dr. Powell is internationally recognized for her work in determining the molecular mechanisms regulating nutrient transport in the human placenta and in characterizing changes in placental function associated with pregnancy complications. Dr Powell's primary research focus is to better understand how the abnormal maternal metabolic environments associated with obesity and gestational diabetes affect placental function and the long-term health of the baby. Specifically, Dr. Powell is interested in identifying signals linking maternal adipose tissue and circulating lipids to placental function and fetal growth, and in developing novel intervention paradigms for improving the maternal metabolic environment and pregnancy outcomes.

Sathish Kumar Natarajan, PhD

University of Nebraska-Lincoln

SESSION III – MATERNAL HEALTH/DIET & PLACENTAL FUNCTION

Palmitoleate Protects against Zika Virus-Induced Endoplasmic Reticulum Stress & Apoptosis in Neurons

Dr. Sathish Kumar Natarajan is an Associate Professor in the Department of Nutrition & Health Sciences at the University of Nebraska-Lincoln. He obtained his BS and MS in Biochemistry at the University of Madras, India and his PhD in Biomedical Sciences from the Dr. MGR Medical University, India. His current research focus is to improve the outcome of newborns in mothers afflicted with Zika virus infection, maternal obesity, or preeclampsia and to establish dietary nutrient interventions to prevent adverse maternofetal outcomes. He has a broad background and expertise in the mechanisms of cell injury, placental Zika virus infection, protective nutrient signaling and apoptotic signaling pathways, mitochondrial function, and mitochondrial fatty acid oxidation disorders. The long-term goal of his research program is to develop a nutraceutical approach to mitigate liver and placental lipotoxicity that occurs during acute fatty liver of pregnancy and maternal obesity, respectively. He has 62 peer-reviewed publications and 1 book chapter, including 20 first-author and 15 corresponding author publications. These articles have been placed in journals that include *Hepatology*, *Cell Death & Disease*, *Cell Death & Discovery*, *Journal of Lipid Research*, *Journal of Biological Chemistry*, *International Journal of Molecular Sciences*, and *Journal of Virology*. His work has been cited more than 3,700 times. He is currently the President for Midlands Society for Physiological Sciences, a local chapter of the American Physiological Society, and is a faculty representative for the US DOHaD Society.

Jennifer Thompson, PhD

University of Calgary

SESSION III – MATERNAL HEALTH/DIET & PLACENTAL FUNCTION

Revisiting the Adipocyte Number Hypothesis & Its Implications for Developmental Programming

*****Early Career Speaker*****

Dr. Jennifer Thompson is an Associate Professor in the Cumming School of Medicine at the University of Calgary in Canada and is a member of the Libin Cardiovascular Institute and the Alberta Children's Hospital Research Institute. After receiving her PhD at the University of Western Ontario, she completed a postdoctoral fellowship at the Medical College of Georgia, in Augusta, Georgia. The Thompson laboratory aims to understand how key events along the life course such as perinatal development and menopause influence adipose plasticity and metabolic health. Jennifer's research program is currently supported by grants from the Canadian Institutes of Health Research, the Natural Sciences and Engineering Research Council of Canada, the Heart and Stroke Foundation of Canada, and Diabetes Canada. She is a past National Heart and Stroke Foundation New Investigator. Web: jenniferthompsonlab.com

Monica Mainigi, MD

University of Pennsylvania

SESSION IV – ART, REPRODUCTIVE ENDOCRINOLOGY, & OFFSPRING HEALTH

Assisted Reproductive Technologies & Adverse Perinatal Outcomes: The What, Why, & How?

Dr. Monica Mainigi is an Associate Professor of Obstetrics and Gynecology at the University of Pennsylvania, in the Division of Reproductive Endocrinology and Infertility. Dr. Mainigi completed her residency in Ob/Gyn and her fellowship in Reproductive Endocrinology at the University of Pennsylvania. She completed postdoctoral training in the laboratory of Dr. Richard Schultz. She currently has a research laboratory in the Center for Women's Health and Reproductive Medicine at the University of Pennsylvania. Her research examines the interactions between peri-implantation exposures, such as ART, and early placentation by utilizing animal models, *in vitro* culture systems, and human tissues. Studying early implantation in humans is challenging. To address these challenges, her laboratory has partnered with bioengineers to develop an organ-on-a-chip model to study early placentation. Using this implantation-on-a-chip device and primary human cells, they have found that uterine immune cells play a critical role in regulating early trophoblast invasion and spiral artery remodeling. Moreover, her work has shown that patients at risk for abnormal placentation may have changes in these immune cell populations. She is currently using this model to further understand the different cells and factors that play a critical role in normal and abnormal placentation.

Lisa Vrooman, PhD

Oregon Health Science University

SESSION IV – ART, REPRODUCTIVE ENDOCRINOLOGY, & OFFSPRING HEALTH

Investigating Placental Protein Changes Associated with ART in a Mouse Model

*****Early Career Speaker*****

Dr. Lisa Vrooman is an Assistant Professor in the Division of Reproductive and Developmental Sciences at the Oregon National Primate Research Center, Oregon Health and Science University. Dr. Vrooman obtained her PhD with Patricia Hunt at Washington State University. Her dissertation work demonstrated how early low-dose postnatal exposure to environmental estrogens could alter spermatogenesis, potentially affecting fertility and inheritance in offspring. She then conducted postdoctoral work with Marisa Bartolomei at the University of Pennsylvania determining how epigenetic changes incurred during assisted reproductive technologies impact placentation and fetal growth. A priority research question in the Vrooman lab is determining the molecular mechanisms by which ART procedures contribute to pregnancy complications, with the goal of improving maternal and fetal outcomes in humans. More recently, the lab is utilizing their expertise in ART techniques to understand the basic genetic control of placental development in mouse and rhesus macaque models. Her lab is funded by NICHD and grants from The Maya's Wings Foundation/American Society of Reproductive Medicine, the Medical Research Foundation of Oregon, the Pacific Northwest National Laboratory, OHSU Center for Developmental Health, and ONPRC. She also teaches the long-standing Frontiers in Reproduction course at the Marine Biological Laboratory in Woods Hole, MA. Email: vrooman@ohsu.edu Twitter: @la_vrooman Bluesky: @lavrooman.bsky.social

Colin Conine, PhD

University of Pennsylvania

SESSION IV – ART, REPRODUCTIVE ENDOCRINOLOGY, & OFFSPRING HEALTH

The Immune System Regulates Sperm RNAs to Drive Transgenerational Epigenetic Inheritance in Mice

*****Early Career Speaker*****

Colin C. Conine, PhD is an Assistant Professor of Genetics and Pediatrics at the University of Pennsylvania Perelman School of Medicine and Division of Neonatology at the Children's Hospital of Philadelphia. He is a faculty member of the Penn Epigenetics Institute, the Institute of Regenerative Medicine, and the Center for Research on Reproduction and Women's Health, as well as an Investigator of the Center of Excellence in Environmental Toxicology. Dr. Conine received his BS in Biochemistry from the University of Rochester and his PhD from the University of Massachusetts Medical School. During his PhD in Craig Mello's lab, he found that small non-coding RNAs in sperm can transmit epigenetically inherited phenotypes to offspring. During his postdoc in Oliver Rando's lab, at UMass Medical, he found that sperm small RNAs in mammals can also transmit non-genetically inherited information to offspring. Dr. Conine started his lab at UPenn and CHOP in 2020. His research focuses on how RNAs function in male fertility, inheritance, and development. He was named a 2021 Pew Biomedical Scholar and in 2023 was awarded an NIH NIGM Maximizing Investigators Research Award (R35).

Maria Gracia Gervasi, PhD

University of Connecticut

SESSION IV – ART, REPRODUCTIVE ENDOCRINOLOGY, & OFFSPRING HEALTH

Metabolic Manipulation of Spermatozoa Improves Sperm Function with Implications for ART

*****Early Career Speaker*****

Dr. Maria Gracia Gervasi is an Assistant Professor of Reproductive Physiology in the Department of Animal Science at the University of Connecticut. Originally from Argentina, she earned her PhD in 2013 from the University of Buenos Aires, where she investigated sperm capacitation and its interaction with the oviductal epithelium using a bovine model. She then completed postdoctoral training at the University of Massachusetts Amherst, focusing on the molecular pathways involved in sperm maturation and capacitation in rodents. Following her postdoctoral work, Dr. Gervasi was appointed Research Assistant Professor at the University of Massachusetts Amherst, where she studied the application of sperm treatments to enhance assisted reproductive technologies and early embryo development in cattle. From 2021 to 2023, she also served as an Assistant Professor at the Bedford Research Foundation in Massachusetts, conducting research on human oocyte parthenogenetic activation for the generation of human parthenogenetic stem cells. In August 2023, Dr. Gervasi joined the University of Connecticut. The Gervasi Lab investigates the molecular mechanisms regulating sperm function and their influence on early embryo development in mice and cattle. A central focus of her lab is the development of sperm treatments aimed at improving *in vitro* fertilization outcomes by optimizing sperm function and embryo production. Her research extends beyond fertilization to assess the impact of sperm treatments on early embryo development.

Jessye Wojtusik, PhD

Omaha's Henry Doorly Zoo & Aquarium

SESSION IV – ART, REPRODUCTIVE ENDOCRINOLOGY, & OFFSPRING HEALTH

From Rhinos to Sea Stars: Innovative Applications of ART to Support Wildlife Conservation

*****Early Career Speaker*****

Dr. Jessye Wojtusik is the Lead Scientist for the Department of Reproductive Sciences at Omaha's Henry Doorly Zoo and Aquarium, where she leads interdisciplinary research at the intersection of reproductive physiology, conservation biology, and genetics. Her work integrates assisted reproductive technologies with genetic and endocrine strategies to support species recovery and the long-term preservation of biodiversity. Her current research portfolio spans a diverse array of taxa, including rhinoceros, polar bears, and the sunflower sea star. Her emphasis is on developing scalable, accessible reproductive-based techniques such as semen collection methods, cryopreservation protocols, and diagnostic hormone assays. Dr. Wojtusik earned her BS and MS in Animal Science from Cornell University and her PhD in Environmental Science and Policy from George Mason University. She completed two postdoctoral fellowships at the Cincinnati Zoo & Botanical Garden: one focused on identifying serum biomarkers for iron overload disorder in rhinos, and the other on advancing reproductive monitoring tools and ART techniques in polar bears. At OHDZA, her research bridges laboratory science with applied conservation practices through active collaborations with institutions around the world. In addition to her scientific endeavors, Dr. Wojtusik contributes her expertise through several volunteer leadership roles, serving as Vice-Chair Elect of the Conservation Centers for Species Survival, an advisor to the AZA SAFE Sunflower Sea Star program, and co-advisor to the Rhino Taxon Advisory Group's Research Council and the Ungulate Resource Group. Her efforts advance collaborative, data-driven strategies to improve reproductive outcomes, inform population management, and enhance the impact of conservation science.

Soumen Paul, PhD

University of Kansas Medical Center

SESSION V – REGULATION OF IMPLANTATION & PLACENTATION

Arginine Methyl Transferase & Epigenetic Equilibrium in Trophoblast Development to Prevent Early Pregnancy Loss

Dr. Soumen Paul is a Professor of Pathology & Laboratory Medicine at the University of Kansas Medical Center. He completed his MS in Biochemistry in 1995 from the University of Kalyani and his PhD in Molecular Biology from the University of Calcutta, India. In 2000, Dr. Paul joined his Alma Mater, Kalyani, as a Lecturer of Biochemistry. In 2002, he joined the University of Wisconsin as a postdoc, where he focused on the transcriptional mechanisms of hematopoietic development. He joined KUMC in 2007 and was promoted to full Professor in 2018. Since 2020, Dr. Paul has served as Director of the Center for Organogenesis, Vascular and Perinatal Research. His lab focuses on delineating transcriptional mechanisms for self-renewal, differentiation, and function of trophoblast stem/progenitor cells in rodents and humans. His long-term goal is to understand how changes in transcriptional and epigenetic mechanisms pre- and post-implantation lead to defective pregnancy, perinatal pathology, and death. Dr. Paul was awarded an American Heart Association Postdoctoral Fellowship, the Leukemia Research Foundation Promising Junior Investigator Award and fellowship, the KUMC Outstanding Faculty Researcher Award, and the KU Scholarly Achievement Award. His research has been consistently funded by the NIH and is currently supported by multiple awards from NICHD. He was recognized with an appointment to the NIH Scientific Advisory Panel on Pregnancy and Neonatology and other NIH study sections. He has served on international grant review panels for the MRC UK, Austrian Science Foundation, and Israeli Science Foundation.

Jane Cleal, PhD

University of Southampton

SESSION V – REGULATION OF IMPLANTATION & PLACENTATION

Maternal Exposures & Molecular Regulation of the Human Placenta & Endometrium

Dr. Jane Cleal is an Associate Professor in Reproductive Cell Biology at the University of Southampton, UK. She specializes in the integration of whole systems biology with cellular and molecular mechanisms in relation to developmental physiology, reproductive disorders, and pregnancy-related conditions. Dr. Cleal completed her PhD in 2005 and was appointed Lecturer at the University of Southampton in 2010. Her group investigates the molecular mechanisms that regulate the placenta and maternal tissues during pregnancy to translate the findings into interventions for improving pregnancy outcomes and fetal growth. Her pioneering work on placental nutrient transfer has significantly advanced our understanding of the underlying transport mechanisms. These include establishing how amino acids reach the fetus and the groundbreaking discovery of active placental control of vitamin D transfer during pregnancy. Her current projects investigate the endometrium in reproductive disorders, with a focus on the glands, extracellular vesicles, and cilia action. These studies identify novel factors associated with subfertility and recurrent pregnancy loss, which may be biomarkers for at-risk women. This work involves endometrial organoid culture, 3D imaging, and single cell gene expression analysis. Dr. Cleal's work has been recognized with the prestigious Andrée Gruslin Award for work in placental biology from the International Federation of Placenta Associations. She sits on core BBSRC panels and is on the leadership team of the Southampton widening access medical program.

Mike Soares, PhD

University of Kansas Medical Center

SESSION V – REGULATION OF IMPLANTATION & PLACENTATION

Trophoblast Cells at the Uterine-Placental Interface

Dr. Soares received his undergraduate degree from California State University, Chico and his doctoral degree from the University of Hawaii. He received postdoctoral training at the University of California, Santa Cruz and at Baylor College of Medicine. Dr. Soares is currently a University Distinguished Professor at the University of Kansas Medical Center, with appointments in the Department of Pathology and Laboratory Medicine and the Department of Obstetrics and Gynecology. He also serves as Director of the Institute for Reproductive and Developmental Sciences at the University of Kansas. Dr. Soares' research program focuses on regulatory processes associated with pregnancy, especially events controlling trophoblast cell differentiation and development of the hemochorial placenta.

Susan Carlson, PhD

University of Kansas School of Medicine

SESSION VI – THE ROLE OF NUTRITION IN THE DEVELOPMENT OF THE NEWBORN

DHA & Preterm Birth: What We've Learned Since the 2018 Cochrane Review

Dr. Susan E. Carlson, PhD is the AJ Rice Professor of Nutrition and University Distinguished Professor at the University of Kansas Medical Center. Dr. Carlson received her PhD in Nutrition, Biochemistry and Physiology from Iowa State University and did postdoctoral fellowships in Pathology at the University of Wisconsin, Madison and in Pediatrics at the University of South Florida. She has a long-term research interest in the long chain omega-3 fatty acid, docosahexaenoic acid that began with her observation in 1982 that infants fed formula had lower DHA status than those fed human milk. Subsequent clinical trials conducted by her lab and others led to the addition of DHA and arachidonic acid to US infant formulas in 2002. From 1983 until 1997 she conducted the first trials of DHA supplementation in preterm infants while on the Pediatric faculty at the University of Mississippi Medical Center and the University of Tennessee, Memphis. NIH-supported trials showed DHA supplementation improved visual acuity and cognitive outcomes of preterm infants. At the University of Kansas, she has continued to study the effect of DHA on neurodevelopment of infants and on preterm and early preterm birth with randomized clinical trials funded by the NIH and supported by industry. She recently revisited a chance finding of lower necrotizing enterocolitis in preterm infants fed egg phospholipid that was published in Pediatric Research in 1998 with a review in Pediatric Research 2024 that concludes phosphatidylcholine deficiency is plausibly linked to NEC.

Paula Meier, PhD

Rush University School of Medicine

SESSION VI – THE ROLE OF NUTRITION IN THE DEVELOPMENT OF THE NEWBORN

Mothers' Own Milk Provides Personalized Nutrition & Protection to Optimize Short- & Long-Term Health Outcomes in Term & Preterm Infants

Paula Meier, PhD, RN, is a Professor of Pediatrics and Nursing at Rush University Medical Center in Chicago and a Courtesy Professor of Biobehavioral Nursing at the University of Florida. Dr. Meier has worked as a practitioner, researcher, and educator in human milk, lactation, and breastfeeding for premature infants and their mothers since 1975. She spearheaded the multidisciplinary Rush University NICU Human Milk Research Team that has conducted substantial externally-funded translational research and demonstration projects focused on removing barriers to high-dose, long-exposure mothers' own milk feedings for NICU infants. Dr. Meier has published over 350 peer-reviewed manuscripts and parent educational materials and has mentored graduate students from multitudes of disciplines. She is the former President of the International Society for Research in Human Milk and Lactation and served for over 20 years on the Health Advisory Council for La Leche League. She has received Distinguished Alumna Awards from the University of Illinois and Rush University, and in 2013 received the Audrey Hepburn Award for Contributions to the Health and Welfare of Children from Sigma Theta Tau, International. She was an invited member of the WHO Task Force on donor human milk and the NICHD BEGIN (Breastmilk Ecology: Genesis of Infant Nutrition) Task Force. She has served as a reviewer for human milk research applications on multiple NIH review panels. Most recently, she was the recipient of the 2022 Macy-Gyorgy Award from ISRHML, a biennial award that recognizes outstanding lifetime research contributions to human milk, lactation, and breastfeeding.

Hannah Hollinger, PhD

University of Wyoming

SESSION VI – THE ROLE OF NUTRITION IN THE DEVELOPMENT OF THE NEWBORN

The Pre-Ruminant Microbiome: Why Do We Care & Where Does It Come From?

*****Early Career Speaker*****

Dr. Hannah Cunningham-Hollinger is an Associate Professor of Animal Genetics in the Department of Animal Science at the University of Wyoming. She grew up on a cow-calf operation in Kaycee, Wyoming and earned a BA in Biology from St. Olaf College in Minnesota. She earned her MS in Ruminant Nutrition and PhD in Animal Genetics from the University of Wyoming. She joined the faculty at the University of Wyoming in 2018 and has research and teaching appointments. Her overarching research objective focuses on understanding the physiological and genetic regulation of feed efficiency, predominantly in ruminant livestock. Her area of expertise is the interplay of host and microbial genetics as it pertains to ruminant livestock production at various stages of their production/life cycle. Ultimately, she aims to discover unique biomarkers that could be used for selecting more efficient livestock and to harness the programming potential of microbial niches to improve production outcomes. Hannah's efforts are supported by her husband Ben and their three children.

Clyde Wright, MD

University of Colorado Anschutz Medical Campus

SESSION VII – ENVIRONMENTAL EXPOSURE & TOXINS DURING PREGNANCY

Is the Developing Lung Susceptible to Acetaminophen Toxicity?

Dr. Wright is a Professor of Pediatrics at the Children's Hospital Colorado and the University of Colorado School of Medicine. He completed his medical school at the Johns Hopkins University School of Medicine, his residency and chief residency at the University of Wisconsin, and his fellowship training in Neonatal-Perinatal medicine at the University of Pennsylvania/Children's Hospital of Philadelphia. At UC, he leads an NIH-funded laboratory that studies unique aspects of perinatal innate immunity that contribute to neonatal morbidity and mortality. In addition to this research program, Dr. Wright studies the development of the innate immune system and the impact of perinatal acetaminophen exposures on the developing lung. Dr. Wright is on the editorial boards of *Neonatology*, *Acta Paediatrica*, and *NeoReviews*. He is a member of the American Board of Pediatrics Sub-board of Neonatal-Perinatal Medicine, and is on the Executive Board of the International Society for Evidence-Based Neonatology.

Carrie McCurdy, PhD

University of Oregon

SESSION VII – ENVIRONMENTAL EXPOSURE & TOXINS DURING PREGNANCY

Effect of Metformin Use during Pregnancy on Fetal Skeletal Muscle Growth & Metabolism in Rhesus Macaques

Carrie E. McCurdy, PhD is an Associate Professor at the University of Oregon in the Department of Human Physiology. Dr. McCurdy's research focuses on understanding how cells adapt their metabolism and insulin responsiveness in response to nutrient restriction or nutrient excess and on identifying where cellular systems break down in disease models (i.e., obesity, diabetes). This work extends to studies on the impact of maternal diet and health on growth and metabolism in fetal skeletal muscle as well as the lasting impact on risk of cardio-metabolic diseases in offspring. Current studies aim to understand how maternal metformin use during pregnancy influences fetal skeletal muscle growth and metabolism. Dr. McCurdy received her BS degree in Biochemistry from the University of Notre Dame in South Bend, Indiana and her PhD in Nutritional Science from the University of Wisconsin-Madison. Her postdoctoral training at the University of Colorado Anschutz Medical Campus in the Department of Pediatrics - Neonatology launched her interest in maternal-fetal physiology. At the University of Oregon, Professor McCurdy teaches undergraduate and graduate courses in Endocrine Physiology and Signal Transduction. Since 2022, she has been the Associate Department Head in Human Physiology.

Michael Golding, PhD

Texas A&M University

SESSION VII – ENVIRONMENTAL EXPOSURE & TOXINS DURING PREGNANCY

Paternal drinking & the epigenetic influences on mitochondrial function, placental dysfunction, & structural birth defects

Dr. Michael C. Golding earned his PhD in Physiology from Texas A&M University in 2003, where his research investigated the developmental basis of structural and placental defects in bovine fetuses produced via somatic cell nuclear transfer or cloning. This work sparked his interest in embryonic DNA methylation and the role of altered epigenetic programming in birth defects and disease. He completed his postdoctoral training at Cold Spring Harbor Laboratory and the Children's Health Research Institute at the University of Western Ontario, examining the suppression of transposable elements in the placenta and how noncoding RNAs regulate imprinted genes. Dr. Golding joined Texas A&M's Department of Veterinary Physiology and Pharmacology in 2009, becoming a full professor in 2020. He teaches graduate and undergraduate courses in physiology, fetal physiology, and genetics, and he has developed specialized courses on human pregnancy and fetal development. His independent research focuses on the intersection of pregnancy and epigenetics, exploring how environmental exposures before conception or early in development contribute to structural birth defects and increase offspring susceptibilities to chronic disease. Using a preclinical mouse model, his lab has demonstrated that chronic paternal alcohol use can drive alcohol-related craniofacial birth defects and drive the early onset of age-related disease. His ongoing work aims to uncover the biochemical mechanisms of sperm-mediated epigenetic inheritance, assess the impact of alcohol and other toxicants on this process, and determine how these heritable changes affect physiological function and long-term health and contribute to fetal alcohol spectrum disorders. You can follow him at: x.com/GoldingLab vetmed.tamu.edu/golding-lab/

Almudena Veiga-Lopez, DVM, PhD

University of Illinois-Chicago

SESSION VII – ENVIRONMENTAL EXPOSURE & TOXINS DURING PREGNANCY

Placental Vulnerability at the Molecular Level: The EGFR Axis under Chemical Stress

Dr. Almudena Veiga-Lopez investigates how environmental exposures during pregnancy shape maternal and fetal health, placing the placenta at the center of her research. Her lab integrates *in vivo*, *in vitro*, and human cohort data to uncover how environmental chemicals affect placental function. She is especially interested in mechanistic insights that advance early-life risk assessment and reproductive health equity. Originally trained as a veterinarian in Madrid, Spain, Dr. Veiga-Lopez earned her PhD in reproductive physiology and completed postdoctoral training at the University of Michigan. She is now an Associate Professor in the Department of Pathology at the University of Illinois Chicago, where she directs the Reproductive and Developmental Research Program and co-leads the Career Development Program for the P30 Chicago Center for Health and Environment. Her research is supported by NIH funding, including awards from NIEHS and NIA, and she has authored over 95 peer-reviewed publications. She was recently honored with the 2024 Kenneth P. DuBois Award and the 2023 Stephen B. Harris Mid-Career Scientist Award from the Society of Toxicology. In addition to leading an interdisciplinary research program, she serves as Associate Editor of *Toxicology and Applied Pharmacology* and on the editorial boards of *Toxicological Sciences* and other journals. She continuously serves on NIH study sections and international grant panels in Canada, Spain, and the UK, helping shape the future of environmental reproductive health through science and service.

ABOUT THE PERINATAL BIOLOGY SYMPOSIUM

Core physiological mechanisms underlying maternal, fetal, and postnatal health outcomes are well-conserved among mammalian species. As such, the basic biological factors that determine lifelong wellness, metabolic function, growth, body composition, and disease predispositions are comparable among humans, livestock, and mammalian wildlife. Since its inception in 2000, organizers of the Perinatal Biology Symposium have recognized this broad relevance and have leveraged it into a uniquely integrated and inclusive meeting, reflected by the strong representation of expertise in agricultural sciences, biomedical sciences, and clinical/veterinary care. The meeting continues to be a powerful mechanism for effective transdisciplinary scientific interactions that advance perinatal science. Each Symposium includes scientific talks, poster presentations, and keynote addresses in these major areas of perinatal biology:

- *Maternal health*. Ensuring appropriate physiological changes and adjustments by the mother's body during pregnancy.
- *Placental function*. Optimal placental development, growth, & function to provide the fetus with necessary nutrients & O_2.
- *Fetal development*. Proper growth & maturation of the fetus, its tissues, & its organ systems.
- *Neonatal wellness*. Optimal outcomes in newborns, including navigating perinatal challenges resulting from intrauterine or postnatal environments.
- *Preventing pregnancy complications*. Understanding & resolving the biological factors for adverse conditions like pre-eclampsia, preterm delivery, & fetal growth restriction.
- *Improving early postnatal health*. Research-backed approaches for neonatal care following compromised pregnancies.
- *Understanding environmental impacts*. Determining how external conditions like climate, toxin exposure, & malnutrition affect pregnancy outcomes.
- *Developing new treatments*. Research-backed therapies & interventions that address pregnancy complications.

The triennial Perinatal Biology Symposium was created with the primary objective of growing and strengthening perinatal research nationally and globally. The initial meetings were developed as FASEB summer conferences on perinatal health in 2000 and 2004. However, the long-followed FASEB conference criteria were not intentional about promoting early career investigator and trainees or their work beyond presentations in poster sessions. They also focused exclusively on the biomedical aspects of perinatal biology. For these and other reasons, the conference separated from FASEB after the 2004 meeting. The 1st stand-alone Perinatal Biology Symposium was held in 2007 in Aspen, CO, and the meetings have been held in the Aspen/Snowmass area every 3rd year since.

Perinatal biology is a preeminent example of the One-Health scientific approach, and the Symposium leverages this by highlighting the broad areas of intersection among human health, animal health, and environmental health factors. As one example, outcomes of environment-influenced fetal metabolic programming are strikingly similar between pregnant sheep/cattle exposed to summer heat and pregnant women from regions where socioeconomic status requires that they continue in laborious jobs throughout their gestation. As another example, changes in early nutritional needs following preterm birth are similar in human infants and piglets. For humans and animals alike, potentially harmful environmental elements as obvious as climate change and as inconspicuous as chemicals in car seat foam continue to threaten perinatal health.

Epidemiological studies have emerged in the last three decades that show the clear association between environment-altered fetal/early neonatal development and subsequent risk for metabolic dysfunction and other health pathologies. Consequences of these programmed pathologies worsen as offspring age and ultimately manifest in conditions like diabetes, obesity, immune dysfunction, behavioral and cognitive disorders, subfertility, and cardiovascular disease, to name but a few. In humans, such conditions shorten lifespan and reduce quality of life. In livestock, the very same underlying biological mechanisms increase early morbidity and mortality, reduce growth efficiency, and diminish the volume and quality of food produced from each animal. Understanding environmental and biological components of this perinatal phenomenon is necessary to improve outcomes for people and animals affected by adverse conditions.

Prenatal and early postnatal development in mammals depends on intricate interactions among maternal, placental, and fetal systems. Because of the expansive and complex nature of these components, it is generally necessary that each component be studied individually. However, periodic opportunities to consider a more holistically integrated view of the perinatal paradigm are important to maintain the effectiveness of research efforts that focus on discrete components and outcomes. The Perinatal Biology Symposium is widely recognized as a productive mechanism for facilitating such integration of scientific thought across these important areas. Unique integrative interactions are created by the meeting in the following areas:

- *Biomedical & agricultural biosciences*. Developmental mechanisms that affect acute and long-term health and welfare are common among mammalian livestock species and humans. Consequently, scientific discoveries related to perinatal health and pathologies are also relevant to both.
- *Clinical, basic, & applied research*. Advancing perinatal health requires a complex and diverse understanding of perinatal events and the conditions that affect them. This in turn requires strong interdisciplinary networks of clinicians, basic scientists, and applied scientists from the myriad disciplines and expertise areas across agricultural, biomedical, and environmental fields.
- *Medical centers, land-grant universities, & other research institutions*. Research institutions differ broadly in the resources they provide and in the resources they lack. Scientific meetings like the Perinatal Biology Symposium provide opportunities for scientists at these different types of institutions to connect and combine their assets and capabilities to increase the impact of research approaches.
- *Leading experts, early-career investigators, & trainees*. Sustainability of impactful perinatal biology research, education, and knowledge requires regular opportunities for established researchers, early-career scientists, and trainees to interact and learn from each other. This is prioritized in the design of the Perinatal Biology Symposium.

The aim of the Perinatal Biology Symposium is to bring together investigators from all career stages with wide-ranging expertise in clinical medicine, basic and applied research, and biomedical and agricultural sciences to disseminate and discuss research-based information under an overarching theme. Symposium priorities include:

- Invited speakers that are currently leading cutting-edge research and clinical/industry translation of new knowledge.
- Placing an emphasis on transdisciplinary science with strategically-chosen topics for the scientific sessions.
- Intentionality in the inclusion of early-career investigators as invited speakers alongside more established investigators.
- Ensuring that the scientific committee, workshop panels, and invited-speaker lineups appropriately reflect the ethnic, cultural, and gender diversity of the scientific community.
- Providing daily Trainee Workshops that focus on critical soft-skill development.
- Inviting trainees and very early-career investigators to serve as chairs of the scientific sessions as a way to increase their exposure among the meeting attendees.
- Including two keynote addresses by internationally-recognized experts that summarize their career contributions to perinatal biology, which disseminates high-impact information and creates an opportunity to inspire trainees.
- Creating extensive methods to reward productive trainees and recognize their work in the broad areas of perinatal biology.

The Perinatal Biology Symposium sets itself apart from other international meetings the with its prioritization of career development opportunities and broad One Health focus. The UK-based Fetal and Neonatal Physiological Society and the Perinatal Research Society each hold annual meetings, but these typically highlight only the work of established senior investigators and only in biomedical fields. Themes for each iteration of the Perinatal Biology Symposium are:

- PERINATAL REGULATION OF THE CARDIOVASCULAR SYSTEM (2000)
- MOLECULAR & CELLULAR SIGNALING IN THE PERINATAL CARDIOVASCULAR SYSTEM (2004)
- INTERACTION OF MATERNAL, PLACENTAL, & FETAL SYSTEMS IN PERINATAL DEVELOPMENT (2007)
- INTRAUTERINE STRESS & ADVERSE FETAL OUTCOMES: PERINATAL MECHANISMS OF ADAPTATION (2010)
- FETAL ADAPTATION TO MATERNAL & PLACENTAL DYSFUNCTION (2013)
- INTERCONNECTING ANIMAL & HUMAN SYSTEMS TO UNDERSTAND LIFE-LONG DISEASE (2016)
- PERINATAL EXPOSURES: INTERSECTING MECHANISMS LEADING TO DEVELOPMENTAL OUTCOMES (2019)
- DEVELOPMENTAL PROGRAMMING – PUTTING THE PIECES TOGETHER (2022)
- PERINATAL PROGRAMMING: CHALLENGES AND SOLUTIONS FOR OPTIMIZING LONG-TERM HEALTH (2025)

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