That is, gut microbes impact growth by potentially altering the growth hormone (GH)/insulin-like growth factor-1 axis. Our previous research has also shown that GH - in states of absence and excess - is associated with altered gut microbial composition, maturity and predictive metabolic function in mice. Moreover, both GH and the gut microbiome are implicated in development and aging. Yet, it is unknown how GH impacts the longitudinal microbiome. This study thus aimed to characterize the longitudinal changes in the gut microbial profile of bovine GH transgenic mice (a model of chronic, excess GH action and accelerated aging). Microbial composition was quantified from fecal pellets of the same bGH and control mice at 3, 6 and 12 months of age through 16S rRNA gene sequencing and QIIME 2. Additional bioinformatic analyses assessed the unique signature and predictive metabolic function of the microbiome. The bGH mice had a distinct microbial profile compared to controls longitudinally. At 3 months, bGH mice had increased Firmicutes and Actinobacteria, decreased Bacteroidetes, Proteobacteria and Campylobacterota, and a significant reduction in microbial richness and evenness. By 6 months, all of the aforesaid phyla were increased with the exception of Firmicutes. By 12 months, bGH mice exhibited dysbiosis with increased Firmicutes and Proteobacteria and reduced Bacteroidetes, microbial richness and evenness. Moreover, abundance in Firmicutes, Bacteroidetes and Campylobacterota were significantly explained by the combined effect of genotype and age ($p = 0.006$, 0.005 and 0.02, respectively). Across all timepoints, bGH mice had a significantly different microbiome compared to controls ($p = 0.002$), and the development of microbial richness and evenness were also significantly different in bGH mice ($p = 0.034$ and 0.023). Bacterial genera *Lactobacillus*, *Ruminococcaceae* and *Lachnospiraceae* were identified as a unique candidates in bGH mice across all timepoints. Likewise, metabolic pathways involved in biosynthesis of heme b, menaquinol, acetate and butyrate differentiated the longitudinal bGH microbiome. Collectively, these results show that chronic, excess GH impacts the development and aging of the gut microbiome. Notably, several of the stated bacterial genera and metabolic pathways were associated with GH in our previous study, suggesting that GH may influence the longitudinal presence of certain gut microbes and metabolic functions. Additional studies will be performed to further explore the GH-associated gut microbiome and its impact on host health. *Research was partially funded by the John J. Kopchick MCB/TBS Fellowship, a fellowship from the Osteopathic Heritage Foundation and the MMPC at UC, Davis (NIH grant U240DK092993).*

Reproductive Endocrinology

FEMALE REPRODUCTION: BASIC MECHANISMS

Peripartum Sertraline (Zoloft®) Increases Pup Mortality Immediately Postpartum

Celeste Sheftel, BA[1], Luma C. Sartori, UG[1], Robbie Manuel, BA[1], Hannah Fricke, BS[2], Autumn Bell, UG[1], Elizabeth Mulvenna, UG[1], Laura L. Hernandez, PhD[3].

[1]UNIVERSITY OF WISCONSIN MADISON, Madison, WI, USA, [2]University of Wisconsin - Madison, Madison, WI, USA, [3]University of Wisconsin, Madison, WI, USA.

MON-001

Peripartum and postpartum depression can be detrimental to both the mother and the developing child. Use of antidepressants, such as selective serotonin reuptake inhibitors (SSRIs), is common during the peripartal period and SSRIs have been the leading prescribed antidepressant to treat maternal depression. One of the most commonly prescribed SSRIs is sertraline (Zoloft®) because of the limited fetal teratogenic effects observed, unlike maternal paroxetine (Paxil®) usage which can manifest in fetal cardiovascular defects. Fluoxetine (Prozac®), like sertraline, has previously been shown to have limited teratogenic effects, however, we have shown treatment with fluoxetine for the entire period of pregnancy and lactation in mice compromises pup bones at weaning resulting in decreased long bone length and head circumference. Furthermore, maternal fluoxetine usage results in a sustained reduction in maternal bone mineral density post weaning, which may lead to long-term osteopenia, putting the mother at risk for bone-related disorders later in life. We hypothesized sertraline, like fluoxetine, will compromise maternal bone postpartum and fetal bone development at weaning. Treatment with sertraline in C57BL/6 dams throughout pregnancy and lactation reduced litter size (5.4 pups/dam) and increased pup mortality during the first 24 hours postpartum (20% dead pups/litter) compared to controls (6.8 pups/dam, 5% dead pups/litter, respectively; $P < 0.018$). Maternal calcium transporters (Orai1 and Serca2) were downregulated in the mammary gland in sertraline-treated dams on day 21 of lactation ($P < 0.0032$). Together, our data suggests *in utero* pharmacological exposure to sertraline may induce a failure to thrive in the pups and alters calcium metabolism in the dam. SSRI exposure during pregnancy and lactation may adversely affect the developing neonate(s) as well as have lasting impacts on the mother.

Cardiovascular Endocrinology

PATHOPHYSIOLOGY OF CARDIOMETABOLIC DISEASE

Long-Term Mental Stress Implications to Cardiovascular Disease in an Aged Mouse Model

Natalie Grace Carroll, MS[1], Shripa Amatya, MS[1], Lilly Kamberov, BS[1], Oscar Gomez-Torres, PhD[2], A. Wayne Orr, PhD[1], Matthew D. Woolard, PhD[1], Steven Bailey, MD[1], Diana Cruz Topete, PhD[1].

[1]LSU HSC SHREVEPORT, Shreveport, LA, USA, [2]University Castillo-La Mancha, Toledo, Spain.

SUN-562

While clinical evidence indicates that exposure to mental stress is a linked to a two-fold increased risk for coronary heart disease, even independently from traditional risk factors, the underlying direct mechanisms between psychological stress and cardiovascular health status has not been determined. A growing aging population of adults 65 and older represents a particular patient population vulnerable to chronic mental stressors due to a decline in normal physiologic functions. The decrease in function of the cardiovascular system that occurs during aging leads to the activation of pathological processes associated with