UNIVERSITY of WASHINGTON

Undergraduate Research Symposium May 18, 2018 Mary Gates Hall

Online Proceedings

SESSION 1R

POPULATION HEALTH

Session Moderator: Clarence Spigner, Health Services
JHN 026
12:30 PM to 2:15 PM

* Note: Titles in order of presentation.

Placentomal Conversion as Adaptive Response to Maternal Pregnancy Stress: Pilot Findings in Pregnant Sheep Model
Yael Leah (Leah) Frank, Junior, Pre-Major
Mentor: Martin Frasch, Obstetrics and Gynecology

Placentation, the formation of the placenta, is a dynamic process which is vital for the development of a fetus. A key structural feature of placenta in ruminants, such as sheep, is a placentome. Placentomes are thickenings in tissue that form along both the maternal and fetal sides of the placenta. During pregnancy, placentomes generally begin with the same type of structure, called Type A. Over the course of a gestation, Type A placentomes are found to convert to functionally more efficient forms (Types B, C, or D). Prenatal stress (PS) has been shown to reduce uterine blood flow in pregnant sheep model. This would influence placental blood flow, supply of nutrients, clearance of metabolites and exposure to stress hormones. Undernutrition accelerates the placentomal conversion in pregnant ewes of certain breeds as an adaptive response to maintain the fetal developmental growth curve, while other breeds are not able to adapt. We hypothesized that placentomal conversion is influenced by PS. We modeled chronic PS in the last trimester in pregnant sheep through reoccurring and unpredictable bouts of maternal isolation over a period of 30 days, a validated paradigm to model human PS. At ˜136 days of gestation (full term is 145 days), the placentomes were collected, weighed, and sorted according to their morphology. The weights of Type B, but not Type A, placentomes were found to be lower for the stressed sheep when compared to the control. These findings confirm our hypothesis that PS reduces the degree of the placentomal conversion from Type A to a more advanced form, Type B. This may be caused by a chronic reduction in placental perfusion (blood circulation) during PS. The found reduction in placentomal conversion may be one of the missing mediating factors linking PS to known postnatal developmental abnormalities in metabolism and phenotype.

POSTER SESSION 4

Balcony, Easel 101
4:00 PM to 6:00 PM

Impact of Prenatal Stress on Maternal and Fetal Brain Development and Metabolism: A Model Approach
Colin Michael Atwood Wakefield, Junior, Microbiology
Benjamin Carl (Ben) Janoschek, Sophomore, Pre-Sciences
Mentor: Martin Frasch, Obstetrics and Gynecology

Research in human cohorts and animal models has established a link between prenatal stress (PS) and alterations in development of the fetal hypothalamic-pituitary-adrenal (HPA) axis and autonomic nervous system (ANS). These alterations are physiological, cognitive and behavioral and reach into adulthood. One of the mechanisms is an increased transfer of stress hormones, glucocorticoids, to the fetus. Therefore, interest lies in identifying biological markers that indicate the onset of future developmental problems caused by PS on HPA and ANS pathways.This project is investigating how heart rate (HR) fluctuations, a proxy for ANS activity, can act as a biological marker. The animal model is chronically instrumented unanesthetized fetal sheep. We measure the impact of PS on maternal and fetal glucose, cortisol, ACTH and HR. PS is modeled by repetitive-intermittent-unpredictable three hour isolations during the last trimester. Sheep and humans have remarkably similar physiological responses to stress. Their endocrine system and gestation patterns mirror humans, causing them to be prone to the same developmental issues and diseases. These similarities and the ability to perform chronic fetal instrumentation and long-term monitoring, make sheep ideal for studying the effects of PS on fetal development. ECG and intra-arterial glucose levels are monitored continuously through the experiment to derive HR and quantify chronic-metabolic effects of elevated maternal stress. Maternal plasma samples are obtained pre- and post-isolation for both control and isolation animals. Postmortem, both maternal and fetal adrenals, fetal liver and fetal brain tissues are analyzed histologically. While the model is now established, the data is currently being analyzed. We expect HR patterns to correlate with hormone levels and tissue outcomes, rendering HR as a non-invasive method of identifying changes in ANS caused by PS. Such HR patterns can be used as a predictor for complications during pregnancy, and

neurodevelopmental issues post-partum due to PS in humans.