across exposures. **Conclusions:** Overall, the current study found evidence of differential cardiac remodeling during pregnancy following a previous gestational insult, and alterations in placental development in male offspring. This highlights the strengths of the current subsequent pregnancy model for addressing other toxicological insults and their impacts on fetomaternal health.

ABSTRACT NUMBER: 5103 **Poster Board Number:** P204
TITLE: Heat Stress Alters Abundance of Ovarian Chemical Biotransformation Proteins

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KEYWORDS: Reproductive and Developmental Toxicology; Phase II Metabolism; Ovary

ABSTRACT: Background and Purpose: Global ambient temperatures have risen, and heat stress (HS) causes female infertility. During HS, blood flow is altered to facilitate cooling, and this circulatory event induces intestinal hypoxia, which causes hyperpermeability. Reduced food intake and hyperinsulinemia are two consistent observations during HS across species. We have discovered an altered ovarian response to ovotoxicant exposure during obesity (another hyperinsulinemia model), including changes to basal and induced abundance of ovarian chemical biotransformation proteins. Insulin regulates hepatic and ovarian chemical biotransformation and hyperinsulinemia can alter phosphatidylinositol-3 kinase signaling which regulates ovarian biotransformation enzymes, thus, the hypothesis that HS impacts ovarian biotransformation protein abundance during the follicular phase of the estrous cycle was tested. **Methods:** A porcine model was used in which eight post-pubertal females (126.0 ± 21.6 kg) were synchronized in estrus and allocated to either thermal neutral (TN; 20.3 ± 0.5°C; n = 4) or cyclical HS environmental conditions (25.4 - 31.9°C; n = 4) for 5 d in a 12 hr light-dark cycle. Ovaries were collected at the late follicular phase, prior to ovulation and ovarian protein isolated. **Results:** Western blotting and quantification, using the ImageJ software was performed to identify the abundance of chemical biotransformation proteins, epoxide hydrolase 1 (EPHX1), glutathione S-transferase pi (GSTP1), cytochrome P450 2EI (CYP2E1), ATP binding cassette subfamily C member 1 (ABCC1) and ATP binding cassette subfamily B member 1 (ABCB1). In HS females, ovarian abundance of EPHX1 and GSTP1 were increased (P = 0.02, P < 0.01, respectively), ABCC1 increased (P = 0.05) and ABCB1 tended to be increased (P = 0.07), and CYP2E1 was unaltered (P > 0.32) relative to TN pigs. **Conclusions:** This data supports that during the follicular phase of the estrous cycle, HS may affect the capacity of the ovary in post-pubertal pigs to metabolize ovotoxicants, potentially contributing to observed HS-induced infertility. This work was supported by the Iowa Pork Producers Association (AFK, JWR, LHB).

ABSTRACT NUMBER: 5104 **Poster Board Number:** P205
TITLE: The Transgenerational Effects of Maternal Nano-TiO2 Inhalation

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KEYWORDS: Reproductive and Developmental Toxicology; Endocrine; Estrogens; Nanoparticles

ABSTRACT: Background and Purpose: According to the developmental origins of health and disease, a compromised *in utero* environment is detrimental to fetal development and long-term adult health outcomes. Past research in our laboratory has shown maternal toxicant inhalation deteriorates placental

function and fetal health. The aim of this study was to determine the effects of nano-titanium dioxide (nano-TiO2) inhalation on endocrine and reproductive outcomes as well as transgenerational outcomes in F0-F4 progeny. **Methods:** Pregnant Sprague Dawley rats were exposed from gestational day (GD) 10-19 to nano-TiO2 (12.35±0.13 mg/m3/6h/6d) or filtered air (sham-control). Dam weight was recorded before each exposure as an indicator of pregnancy maintenance and health. F0-F3 dams were euthanized on GD 20. Dams and neonates were weighed for tissue collection. Endocrine disruption was analyzed by competitive binding enzyme-linked immunosorbent assay (ELISA) for maternal plasma for E2. Ovarian and uterine tissue were analyzed for mRNA and protein analysis of estrogen. Placentas and fetuses were processed for reverse-transcription polymerase chain reaction (RT- qPCR) for *3BHSD*, *17BHSD*, *StAR*, and *SRY* detection. **Results:** F1- F3 litter size was significantly reduced when born to exposed dams compared to sham- controls. F1 placental tissue was found to express *17BHSD* and was significantly lower when born to exposed dams ($P < 0.05$). Additionally, not only was circulating estrogen was significantly decreased at GD 20 in exposed F0 dams (11.08±3.0 pg/ml) versus sham-control dams (66.97±3.0 pg/ml), but also in F1 dams with exposed mothers (12.12±3.1 pg/ml) versus dams with sham-control mothers (29.81±8.8 pg/ml). F2-F3 statistical significance could not be determined. Pup wet weight and placental efficiency were significantly reduced in F2 and F3 litters born to exposed dams when compared to sham-control. Interestingly, there was no statistical significance between F4 litter size (13.1±0.88 vs. 12.67±0.60) or fetal wet weight (4.12±0.05 vs. 4.13±0.04) between groups. However, placental efficiency was significantly reduced in F4 litters born in exposed dams compared to sham-controls. **Conclusions:** These data lay the groundwork for future research focusing on estrogen signaling in F2-F4 generations and determining downstream signaling pathways. Identifying the mechanisms by which nanomaterial exposure adversely affects maternal and fetal health not only contributes to overall health, but also has direct implications for public health and workplace safety. With a particular focus on occupational settings, where exposure to toxicants may be prevalent, the project sheds light on the potential risks faced by individuals of reproductive age, advocating for informed decision-making, awareness, and the development of safer work environments.

ABSTRACT NUMBER: 5105 **Poster Board Number:** P206
TITLE: Modeling the Effects of Wildfire Smoke on Reproductive Toxicity Using Caenorhabditis Elegans

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KEYWORDS: Reproductive and Developmental Toxicology; *In Vitro* and Alternatives; Developmental/Teratology

ABSTRACT: Background and Purpose: Intensifying wildfires, driven by hotter and drier climates, subject us to increased smoke exposures year over year. While a large body of literature detailing cardiopulmonary effects of these inhaled toxicants exists, new evidence suggests that the reach of these pollutants extends the reproductive system. This poses significant public health burdens, specifically on pregnant persons, who may face having their children suffer from lower birth weight, preterm birth, and birth defects due to smoke exposure. By screening reproductive systems using the *C. elegans* model, we can understand the reproductive risks associated with exposure to wildfire smoke (WFS). **Methods:** Simulated WFS from Douglas Fir needles were generated in a combustion chamber at smoldering temperatures and collected onto PTFE filters, then methanol extracted into M9 buffer for experimentation. L4 stage *C. elegans* (n=10/group) were exposed to control medium, 200, or 1000

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Preface

This issue is devoted to the abstracts of the presentations for the Continuing Education courses and Scientific Sessions of the 63rd Annual Meeting of the Society of Toxicology, at the Salt Palace Convention Center, Salt Lake City, Utah March 10–14, 2024.

An alphabetical Author Index, cross-referencing the corresponding abstract number(s), begins on page 860.

The issue also contains a Keyword Index (by subject or chemical) of all the presentations, beginning on page 881.

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