

Submitter Name: Yudhishtar Bedi  
Submitted email: [yudibedi@tamu.edu](mailto:yudibedi@tamu.edu)  
PI Name: Dr. Michael Golding  
PI email: [mcgolding@tamu.edu](mailto:mcgolding@tamu.edu)

## **Paternal Alcohol Exposure Leads To Altered Sperm Epigenome And Fetal Growth Restriction.**

Yudhishtar S. Bedi, Tracy M. Clement, Michael C. Golding

Department of Veterinary Pharmacology and Physiology, College of Veterinary Medicine,  
Texas A&M University

To better understand the contribution of paternal preconception alcohol exposure in fetal growth and development, we gave male C57BL/6J mice access to 10% ethanol (w/v) during the first four hours of their dark cycle for ten weeks to recreate a chronic binge exposure model, also known as the Drinking-in-the-Dark model (DID). These mice were then mated to alcohol-naive female mice and pregnancies were terminated at gestational day 16.5 to measure fetal and placental growth parameters. Male reproductive physiology and effects on sperm small non-coding RNA profiles were also assessed. This exposure had no significant effect on male reproductive physiology, sperm count, sperm DNA damage or chromatin integrity. Preconception alcohol exposure did lead to late-term fetal growth restriction and a significant drop in placental efficiency. This was correlated with a shift in the proportion of transfer RNA-derived small RNA fragments (tRFs) and Piwi-interacting RNAs (piRNAs), two major classes of small non-coding RNAs present in sperm, as well as an altered enrichment of microRNAs 21, 30 and 142 in alcohol-exposed sperm. These findings suggest that environmental exposures can alter the sperm epigenetic landscape without effecting DNA damage and have robust contributions in fetal growth and development of the offspring.