Consumption of alcohol (ethanol) during pregnancy can lead to defects on the offspring. These defects have been described using the umbrella term fetal alcohol spectrum disorders (FASD), which includes fetal alcohol syndrome (FAS), partial FAS, and alcohol-related neurodevelopmental disorder (ARND). The most serious consequences of prenatal alcohol exposure occur in the developing central nervous system (CNS). Accordingly, FASD is associated with life-long CNS defects, which include motor incoordination and delayed motor skill acquisition, learning disabilities, memory deficits, hyperactivity, behavioral problems, attention deficits, impairment of judgment and reasoning, and psychiatric disorders. However, despite these serious consequences, there is currently no therapeutic intervention to prevent or treat the CNS deficits caused by maternal consumption of alcohol during pregnancy.

To identify potential therapeutic targets, rodent models of FASD have been utilized to probe the structural and functional pathology produced by ethanol in the developing CNS. These models have demonstrated multiple mechanisms of ethanol pathogenesis including neuronal apoptosis, inhibited neurogenesis, altered synaptic activity, impaired plasticity, disrupted neurotransmitter systems, disrupted neurotrophic support, oxidative stress, altered levels of micronutrients, and neuroimmune system activation. Rodents have also been used to explore the behavioral consequences of this alcohol-induced pathology. Neurobehavioral deficits associated with FASD in humans can be observed directly in rodent FASD models by utilizing a plethora of behavioral tests such as those that assess motor function, hyperactivity, anxiety, learning, and memory. This presentation will selectively highlight the utility of behavioral tests including balance beam walking, rotarod, elevated plus maze, Y-maze, open field activity, novel object recognition, and Morris water maze for assessment of CNS deficits associated with developmental ethanol exposure. Demonstration of ethanol-sensitivity in behavioral tests provides opportunities to increase our understanding of the molecular mechanisms that underlie FASD and the CNS pathology that generates FASD neurobehavioral deficits. In addition, rodent behavioral tests provide a critical opportunity to assess novel therapeutic strategies for FASD. Thus, persistence in exploring rodent FASD behavioral models may shed light on new translational approaches for the alleviation of behavioral consequences associated with FASD, leading to improved outcomes for patients affected by these disorders.