THE INFLUENCE OF MODERATE PRENATAL ALCOHOL EXPOSURE ON THE CENTRAL AMYGDALA AND ANXIETY-LIKE BEHAVIOR IN ADOLESCENTS.

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Disclosure Statement: I do not have a relationship with any commercial interests, or affiliations which would impose bias.
Prenatal Alcohol Exposure (PAE) and Anxiety

- **Comorbidity**
  - 21% of school-aged children diagnosed with FASD meet the criteria for anxiety disorders (O’Connor and Paley, 2009)
  - Repeatedly observed in rodent models

- **Our Model of PAE**
  - Vapor ethanol chambers – inhalation
  - Gradually increasing blood alcohol levels (mimicking drinking in humans)

**12th Day of Pregnancy (G12)**
Vapor Exposure

- Heavy exposures: 300+ mg/dL
- Our moderate exposure: 60-80 mg/dL
WHAT IS THE EFFECT OF MODERATE G12 PAE?

- Does this PAE model increase anxiety?
  - YES! (Rouzer et al., 2017)

- Does this PAE model change neurotransmission (cell communication)?

  CENTRAL AMYGDALA
  - Develops during G12 in rodent models (Soma et al., 2009)
  - Regulates anxiety-like behavior (Agoglia & Herman, 2018)
  - Composed of GABAergic neurons
AIR-EXPOSED

Presynaptic GABA Neuron

Postsynaptic neuron

GABA

GABA Receptor

GABA

GABA Receptor

Presynaptic GABA Neuron

Postsynaptic neuron

Basal GABA transmission

Frequency (Hz)

Air

PAE

(16)

(22)

* p < .05

G12 PAE

Presynaptic GABA Neuron

Postsynaptic neuron
**AIR-EXPOSED**

- Presynaptic GABA Neuron
- Postsynaptic neuron

**G12 PAE**

- Presynaptic GABA Neuron
- Postsynaptic neuron

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% Change in Frequency

- (8) (8)

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- CRFR1 agonist
- CRFR1 Receptor
- GABA
- GABA Receptor

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* p < .05

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CONCLUSION

- A single instance of moderate PAE is sufficient to increase anxiety and change neuronal communication in the central amygdala of developing adolescents
  - Does not require “binge-like” ethanol exposure

Long Term Goal:
Provide support to the FASD population by understanding PAE-induced neurobiological impairments as targets of future pharmacological attention
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