
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

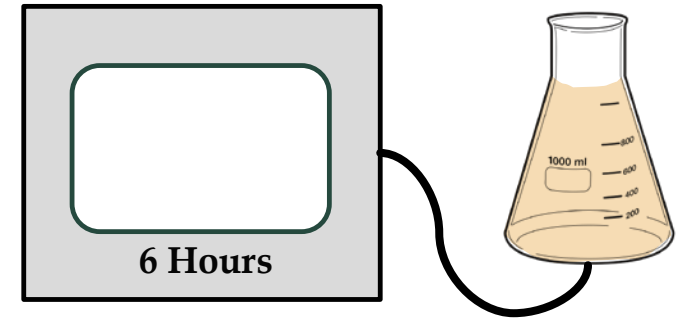
■ Our Model of PAE

- Vapor ethanol chambers – inhalation
 - Gradually increasing blood alcohol levels (mimicking drinking in humans)



12th Day of Pregnancy (G12)

Vapor Exposure



Our moderate exposure: 60-80mg/dL

Heavy exposures: 300+ mg/dL

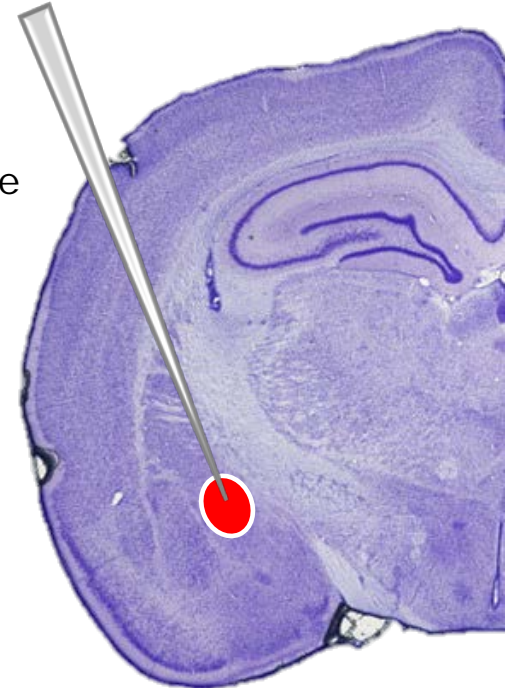
WHAT IS THE EFFECT OF MODERATE G12 PAE?

- **Does this PAE model increase anxiety?**
 - YES! (Rouzer et al., 2017)
- **Does this PAE model change neurotransmission (cell communication)?**

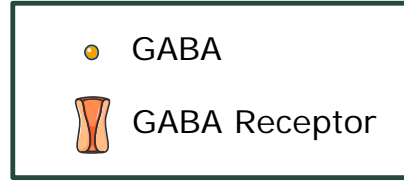
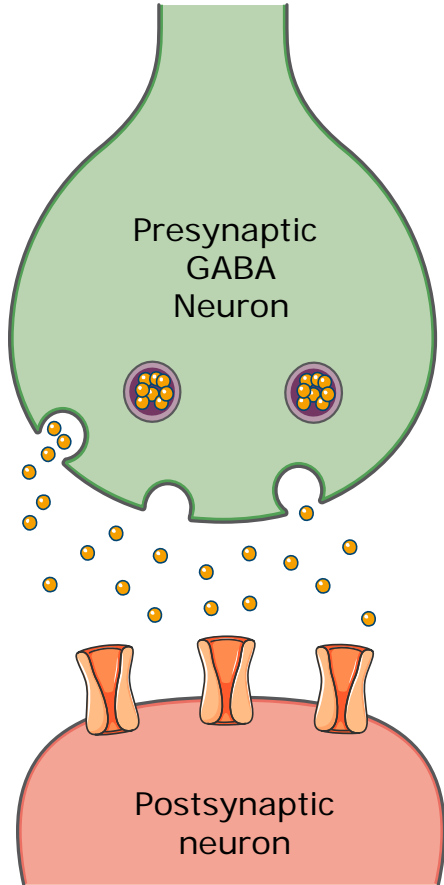
CENTRAL AMYGDALA

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

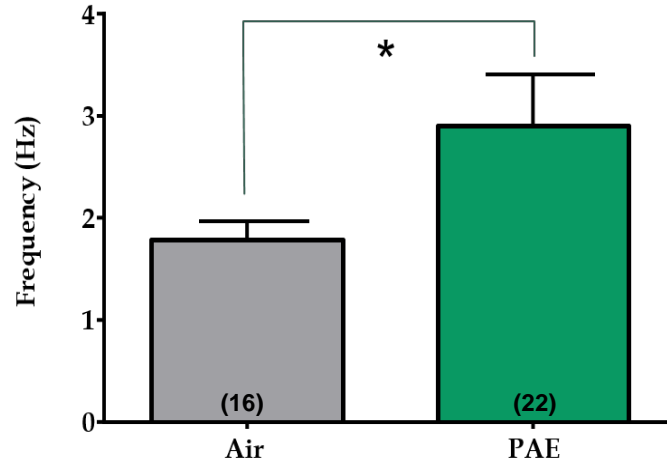
Recording pipette



AIR-EXPOSED

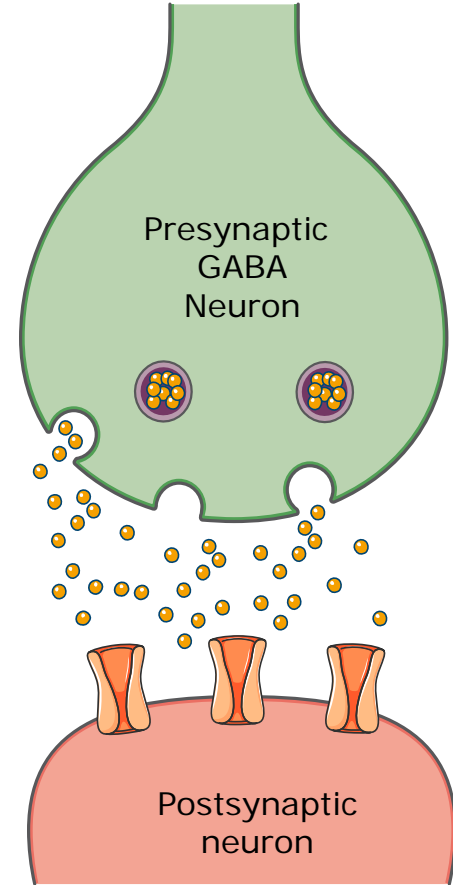


Basal GABA transmission

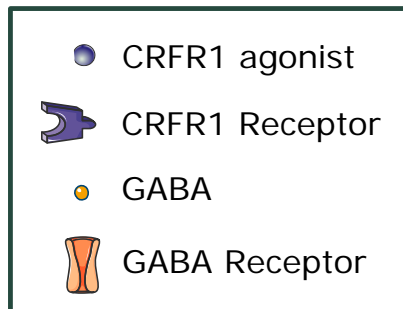
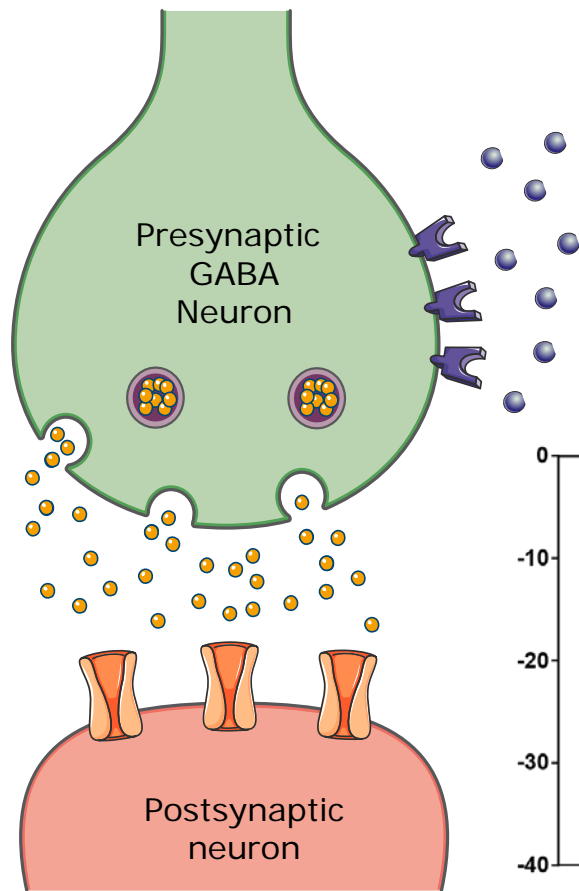


* $p < .05$

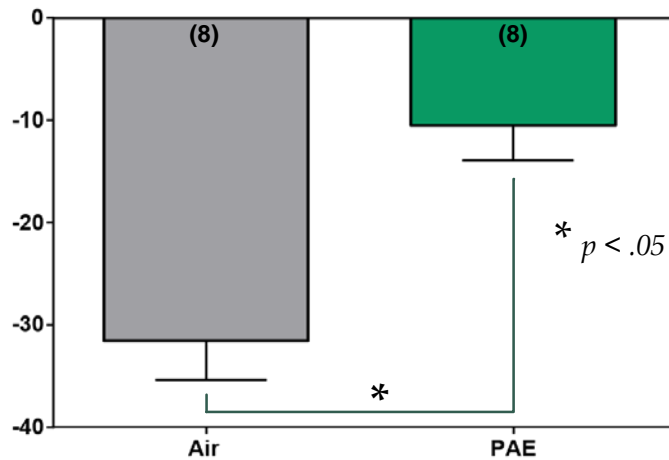
G12 PAE



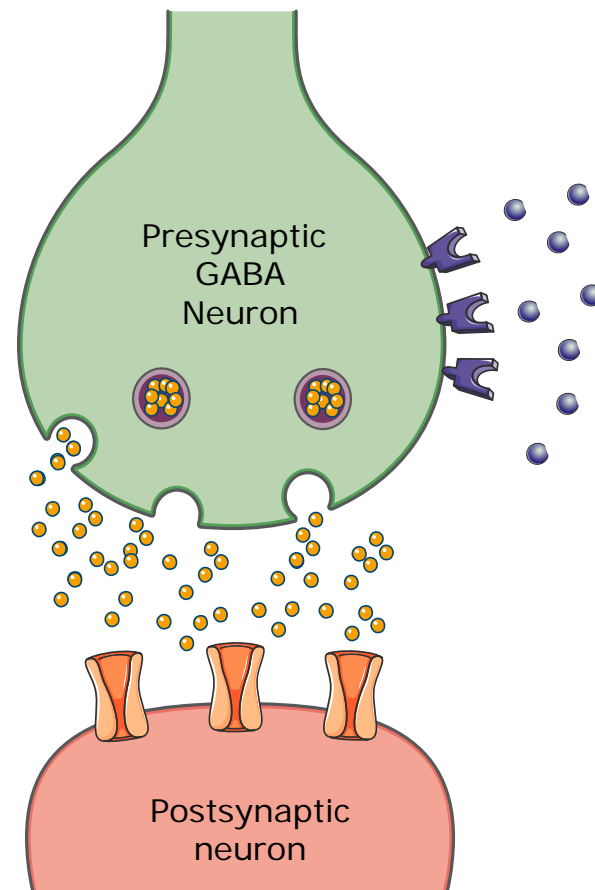
AIR-EXPOSED



% Change in Frequency



G12 PAE



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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SPECIAL THANKS

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