

# Neuroimmune Modulation of SSRI Response in a Corticosterone-Induced PPD Model

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- Postpartum depression (PPD) is common and poorly treated by SSRIs (e.g., fluoxetine [FLX]) alone.
- Key Mechanisms: HPA-axis imbalance → ↑ IL-1β
  neuroinflammation → ↓ hippocampal plasticity.
- IL-1β, a key pro-inflammatory cytokine, blocks SSRI-induced neuroplasticity.

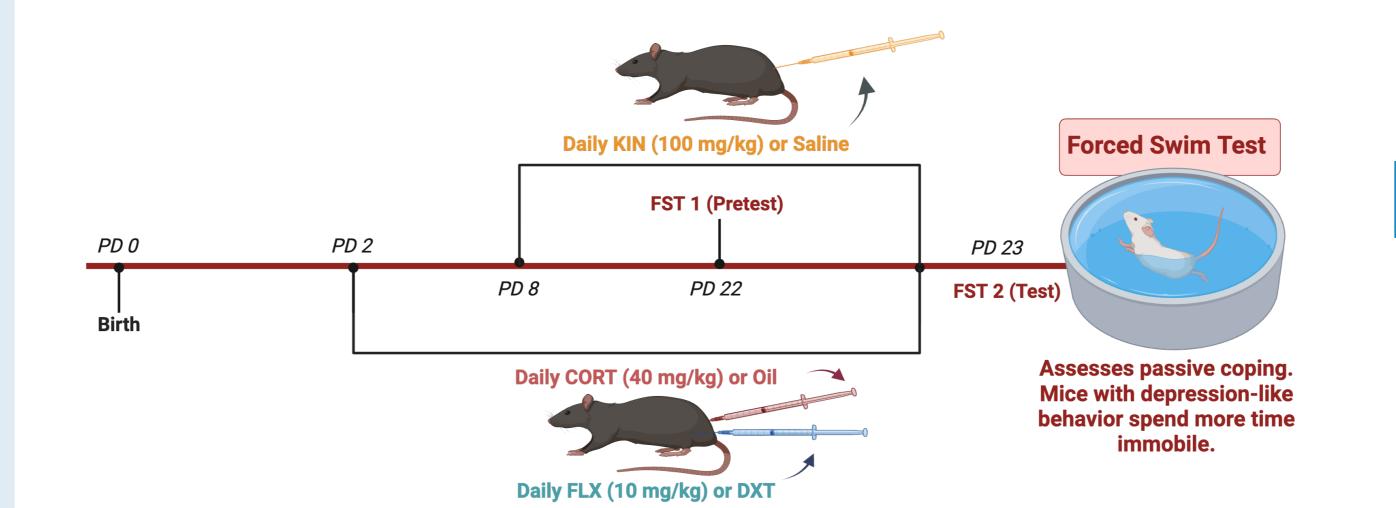
#### Behavioural Triggers and **Biological Factors Manifestations** Persistent Sadness **Hormonal Fluctuations** and Anhedonia **HPA Axis** Fatigue and Cognitive Dysregulation Fog Life Stressors and **Social Context** Impaired Maternal Care Postpartum Neuroimmune depression Activation (PPD) Increased Passive Coping 1 in 5 new mothers Reduced Hippocampal affected Neurogenesis

### **BACKGROUND**

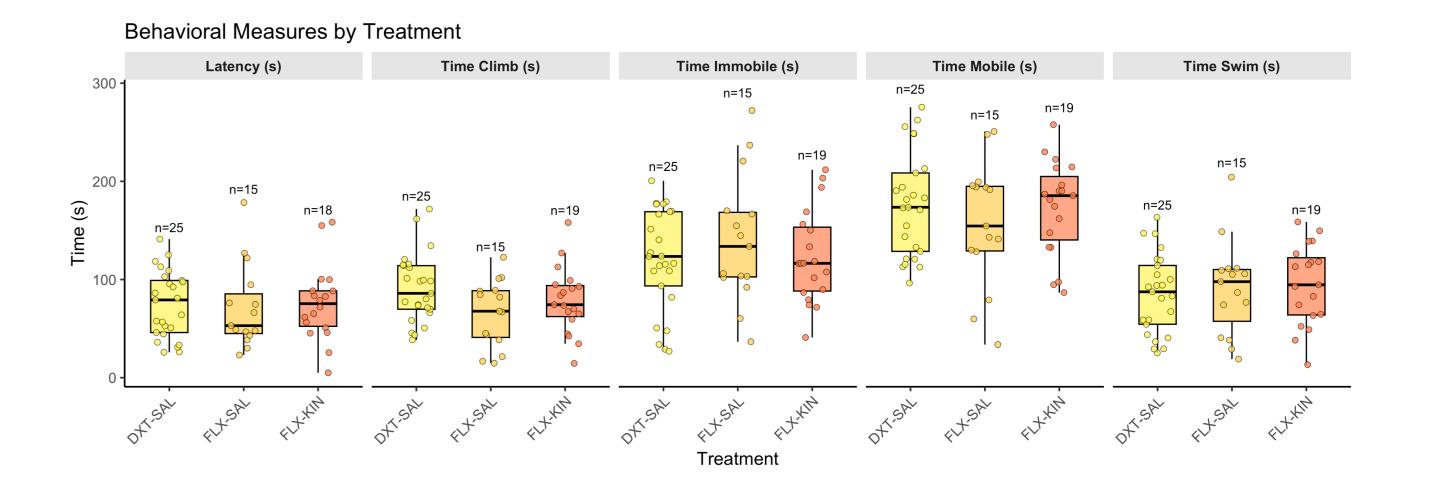
- Anakinra (KIN): A clinically used IL-1 receptor antagonist that crosses the bloodbrain barrier to block IL-1β signaling.
- Does combining FLX + KIN rescue active coping mechanisms in this PPD model?

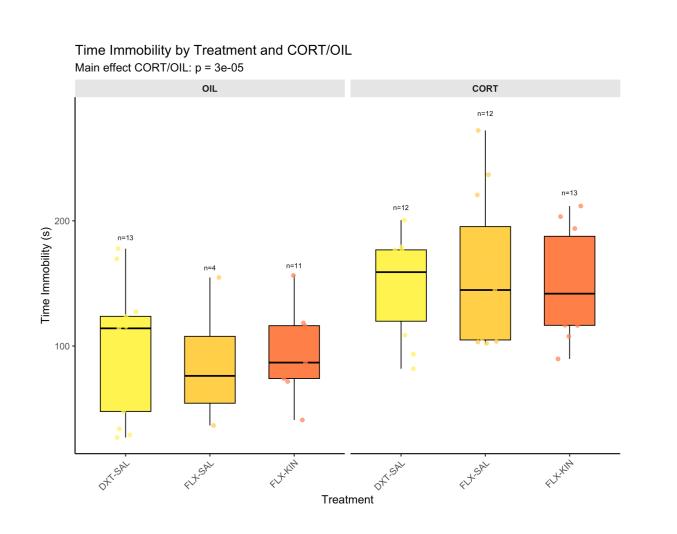
#### **METHODS**

- **CORT Model:** Daily corticosterone replicates PPD hallmarks:
  - ↑ stress, ↓ maternal care, impaired neurogenesis and passive coping in the forced swim test (FST).



#### RESULTS





- CORT: 1 immobility across all groups
- FLX & FLX + KIN: no rescue of passive coping
- FLX + KIN (OIL): ↑
   active coping
   (climbing & mobility)

# **CONCLUSION**

- Postpartum CORT model mimics human PPD, and increases passive coping.
- Next steps: conduct brain assays to assess neuroplasticity and inflammation, and explore female-specific brain-behavior dynamics.

## **REFERENCES**

Bloch et al. (2003); Gobinath et al. (2018); Qiu et al. (2020); Workman et al. (2016); Brummelte & Galea (2010); Syed et al. (2018); Anderson et al. (2013)

