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

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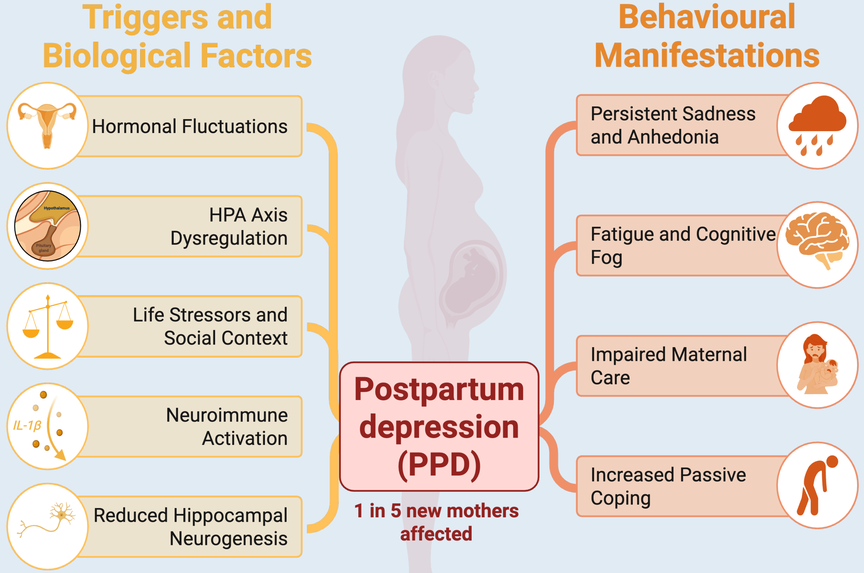
- Postpartum depression (PPD) is common and poorly treated by SSRIs (e.g., fluoxetine [FLX]) alone.
- **Key Mechanisms:** HPA-axis imbalance → ↑ IL-1β neuroinflammation → ↓ hippocampal plasticity.
- **IL-1β**, a key pro-inflammatory cytokine, blocks SSRI-induced neuroplasticity.

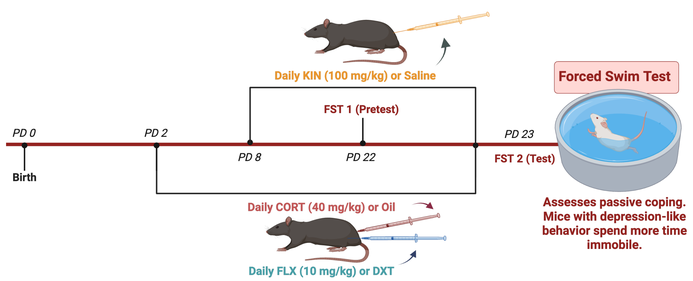


BACKGROUND

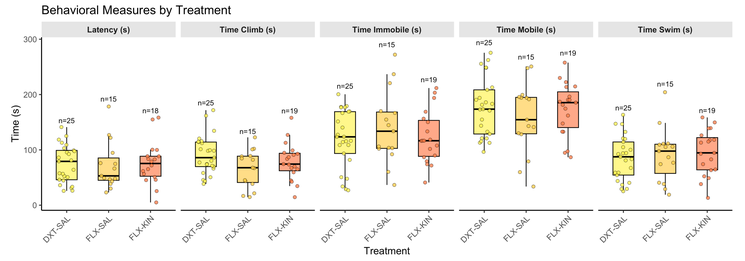
- **Anakinra (KIN):** A clinically used IL-1 receptor antagonist that crosses the blood–brain barrier to block IL-1β signaling.
- Does combining FLX + KIN rescue active coping mechanisms in this PPD model?

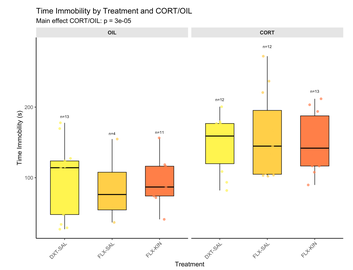
METHODS

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



RESULTS





- **CORT:** ↑ 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)

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