Effects of LPS Administration on Sickness Behavior and PVN Vasopressin Expression

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Introduction

- Chronic, low dose inflammation is an ethologically relevant model of sickness.
- Sickness causes broad behavioral changes, including anxiety and sociability. (Dantzer 2004)
- Vasopressin is a neuropeptide that regulates anxiety and social behaviors (Caldwell et al 2007).
- A major source of vasopressin is the paraventricular nucleus of the hypothalamus (PVN), which regulates stress responses.
- Chronic inflammation causes cFos expression in PVN (Tarr et al 2012)
- **Goal:** determine the effects of chronic inflammation on behavioral responses of sickness, and vasopressin expression in the PVN.

Methods

- Adult male and female mice were injected bi-weekly for four weeks with 0.25mg/kg LPS or Sterile saline.
- After treatment, all mice underwent tests for anxiety-like and social behaviors.
  - Anxiety behavior was evaluated using an open field test.
  - Sociability was quantified by the time interacting with a same-sex stimulus mouse.
- Vasopressin mRNA was labeled using fluorescent in situ hybridization.
  - Fluorescent intensity was quantified and used as a measure of relative mRNA expression

Results

- LPS did not cause behavioral sickness, but may have increased PVN vasopressin expression.
- Behavioral sex differences were observed.
  - Males were more anxious and sociable than females
  - LPS had no sex-specific effects
- LPS may increase PVN vasopressin expression in females.
- In the future, a larger dose of LPS or longer time-course will be needed to cause behavioral sickness and greater differences in PVN vasopressin expression.
- Other sources of vasopressin, such as the SON, BNST, and SCN, may also respond to inflammation

References