**Introduction**

- Psychological stress is known to increase neuroinflammation.
- Neuroinflammatory pathways are contributing factors to various stress-related psychopathologies.
- Exposure to chronic stress in rodents results in elevated markers of immune activity, such as activation of microglia, as measured by ionized calcium binding adaptor protein-1 (Iba-1) expression.
- There has been little research investigating the effects of acute social defeat stress on central immune activity.
- Acute stress can prime microglia to exhibit an enhanced proinflammatory response to a subsequent immune challenge (i.e. Lipopolysaccharide).
- The prefrontal cortex (PFC) is a critical brain region for coping with stress.
- Stress on central immune activity.
- Activation of microglia, as measured by ionized calcium binding adaptor protein-1 (Iba-1).
- Chronic stress significantly alters neuronal morphology in the medial PFC.

**Methods**

**Animals**: Adult Male Syrian hamsters (*Mesocricetus auratus*)

**Experiment 1:**
- **Social Defeat/No Stress**
- **LPS/Saline Injection**
- **Euthanasia & Perfusion**

**Experiment 2:**
- **Social Defeat/No Stress**
- **LPS/Saline Injection**
- **Euthanasia & Perfusion**

1. **Acute Social Defeat**: Subjects were exposed to three, 5-minute aggressive encounters in the home cage of three, separate, larger, aggressive hamsters. Encounters occurred at 5-minute intervals. Control animals were exposed to an empty aggressor’s cage.
2. **LPS Injection**: 24-hours following social defeat, the effects of stress-induced priming of microglia was assessed by exposure to an endotoxin immune challenge via intraperitoneal (i.p.) injection of 0, 20, 100, and 500 µg/kg lipopolysaccharide (LPS).
3. **Euthanasia**: 4-hours following LPS injection (Exp. 1) or 7 days following defeat (Exp. 2), hamsters were euthanized and transcardially perfused and brains extracted.
4. **Immunolabeling**: Microglial activation was measured by immunolabeling of ionized calcium binding adaptor protein-1 (Iba-1) through immunohistochemistry in the prefrontal cortex (PFC) using Rabbit monoclonal anti-Iba-1 antibody (1:10,000; ab178846).

**Figure 1**

- Iba-1 immunoreactivity in the infralimbic cortex.
- Optic density.
- Microglial morphology.
- Activation state.
- 7 days after stress.

**Figure 2**

- The morphological transformation that occurs in progressive activation states of microglia (Figure adapted from Hinwood et al., 2012).

**Figure 3**

- Iba-1 immunoreactivity in the infralimbic cortex.
- Optic density.

**Conclusions**

- These findings indicate that acute social defeat primes microglial activity in the vmPFC following subsequent LPS exposure.
- Acute social defeat appears to “activate” microglia in the vmPFC, as indicated by increased Iba-1 expression and acute changes in morphology.
- Acute social defeat does not appear to contribute to an arrested hyper-ramified state, as discussed in Hinwood et al., 2012.

**Discussion & Future Directions**

- The greatest differences were observed at 20µg/kg, which may indicate a point at which a greater percentage of cells display hyper-ramification.
- The disparity between OD and morphology data at 20µg/kg may be due to low sample size (n=4/group); current efforts are increasing the power of these samples.
- Future work will aim for phagocytic microglial activation state marker, CD68.
- Determine the effects of an antibiotic, minocycline, that selectively inactivates microglia on defeat-induced changes in behavior.

**Acknowledgements and References**

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