Association of Alzheimer’s with NF-kb through LPS-induced inflammation in female rats

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Introduction

- AD is the most common neurodegenerative disease and is the third leading cause of death amongst older people.¹
- Hallmark of AD are neurotoxic Amyloid Beta plaques that activate microglia.²
  - Previous treatments blocking AB overproduction have been unsuccessful.
- NF-kb is a neuroinflammatory gene associated with the regulation of aging and proliferation, and its overactivation has been linked AD.¹
- Majority of the scientific literature uses male rats and its overactivation has been linked AD with the pathogenesis and therapeutics of Alzheimer Disease.³
- The objective of this study was to determine whether LPS-induced inflammation results in any change in microglial morphology in the DG, which would indicate the progression of microglia into an activated state.
- After running an independent sample t-test to assess the differences between LPS and saline groups, it was found that the mean microglial soma area for the saline group was larger on average than that of the LPS group.
- A large effect size with process lengths of the saline group was also found after running an unpaired sample t-test when assessing microglial process lengths. In addition to microglial morphology, this study sought to identify whether microglia induction is affected my LPS treatment.
- Cell counts following Iba-1 and NF-kb staining showed higher numbers of microglia in the saline group than that of the LPS group, however, although this difference is large, it is not statistically significant.

Methods

- 16 adult female rats
- 8 LPS
- 8 saline
- Sacrificed 24 hours after injection
- Transection perfume
- Brain was withdrawn and post-fixed with 4% paraformaldehyde for two hours
- Stained in cresyl violet

Results

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References

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