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Lasting Effects of Estrogen and Bisphenol-A Exposure During Adolescent Development on Dendritic Spines and Behaviors in Adulthood

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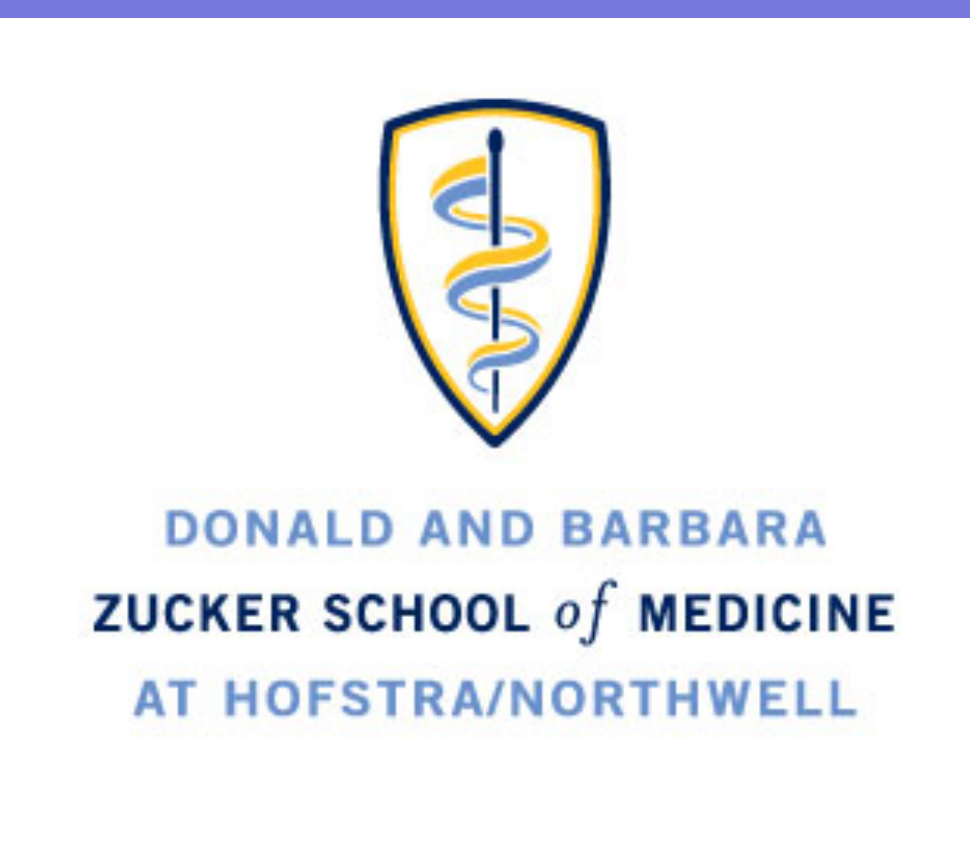
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Lasting effects of Estrogen and Bisphenol-A exposure during adolescent development on dendritic spines and behavior in adulthood.

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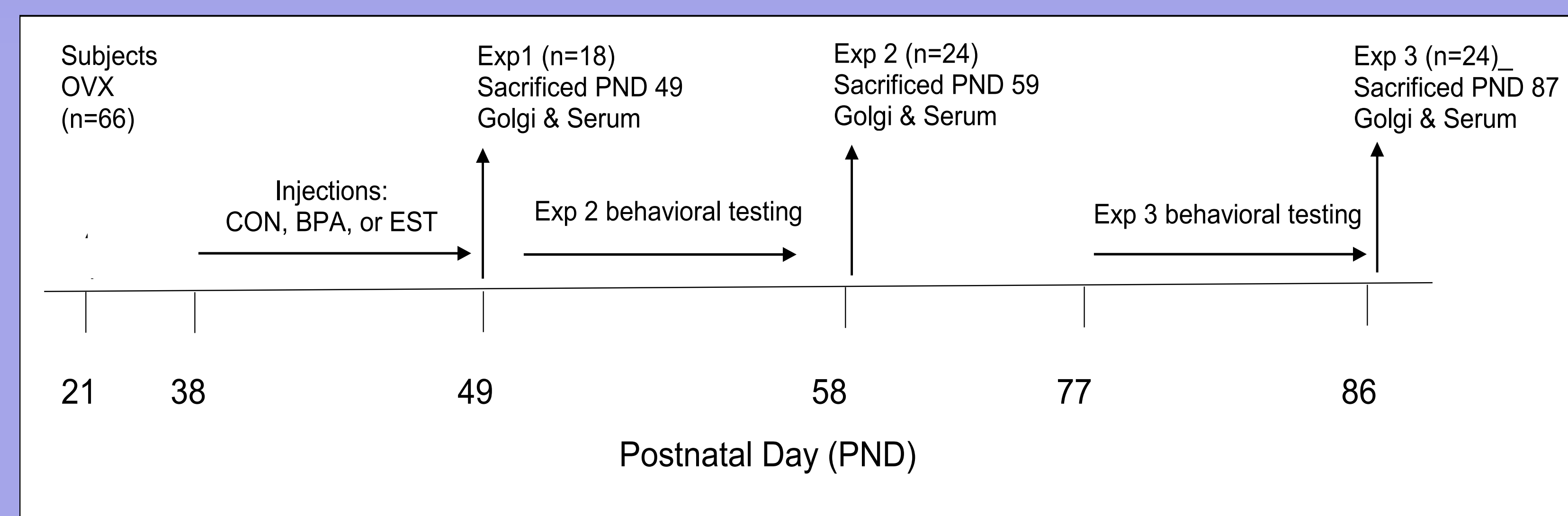
INTRODUCTION:

Bisphenol-A (BPA), a common environmental endocrine disruptor, modulates estrogenic, androgenic, and antiandrogenic effects throughout the lifespan. Most research investigating the effects of BPA in animal models has focused on exposure during early prenatal and neonatal periods which results in behavioral, structural and physiological alterations; however, adolescence is another important development period that is characterized by profound hormonal changes. Our laboratory has been investigating BPA exposure during the juvenile period of development and have found that both cognition function and neuronal morphology are all altered.

Our previous studies have all been conducted in gonadally intact male and female adolescent rats; this leads to the difficult interpretation of the extent to which observed effects may be due to the BPA exposure versus natural fluctuations in gonadal hormones. Additionally, estrogen is known to be neuroprotective, enhance memory, and increase dendritic spine density. However, while estrogen has been widely studied in adults and aging rats, estrogen replacement studies in juvenile rats has only recently begun to be investigated. Thus, this series of experiments examined the effects of adolescent BPA or estrogen exposure in adolescent, ovariectomized (OVX) female rats on cognitive functioning and neuronal architecture in both adolescence and adulthood.

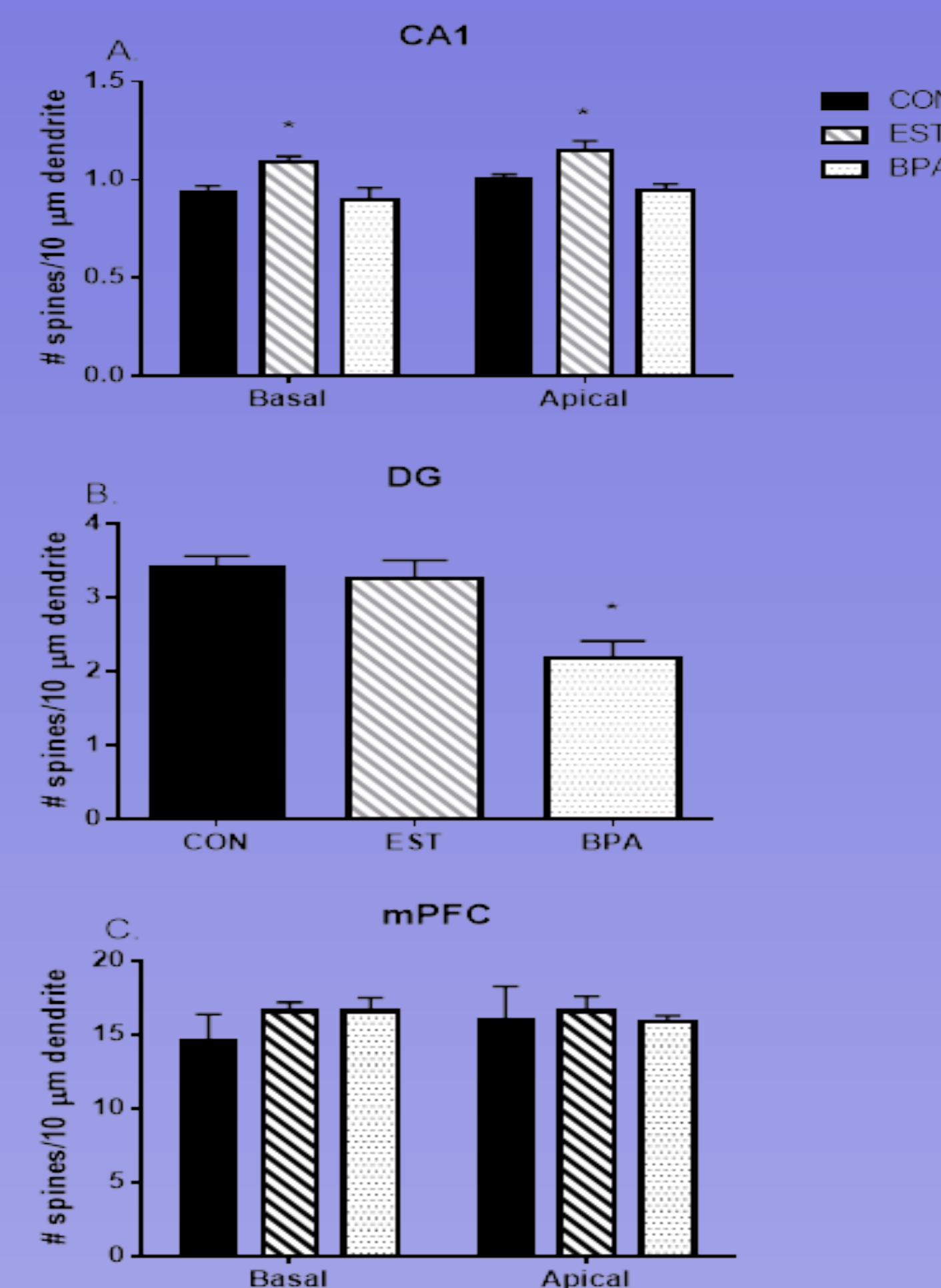
METHODS:

Figure 1. Overview of experimental research design timeline.

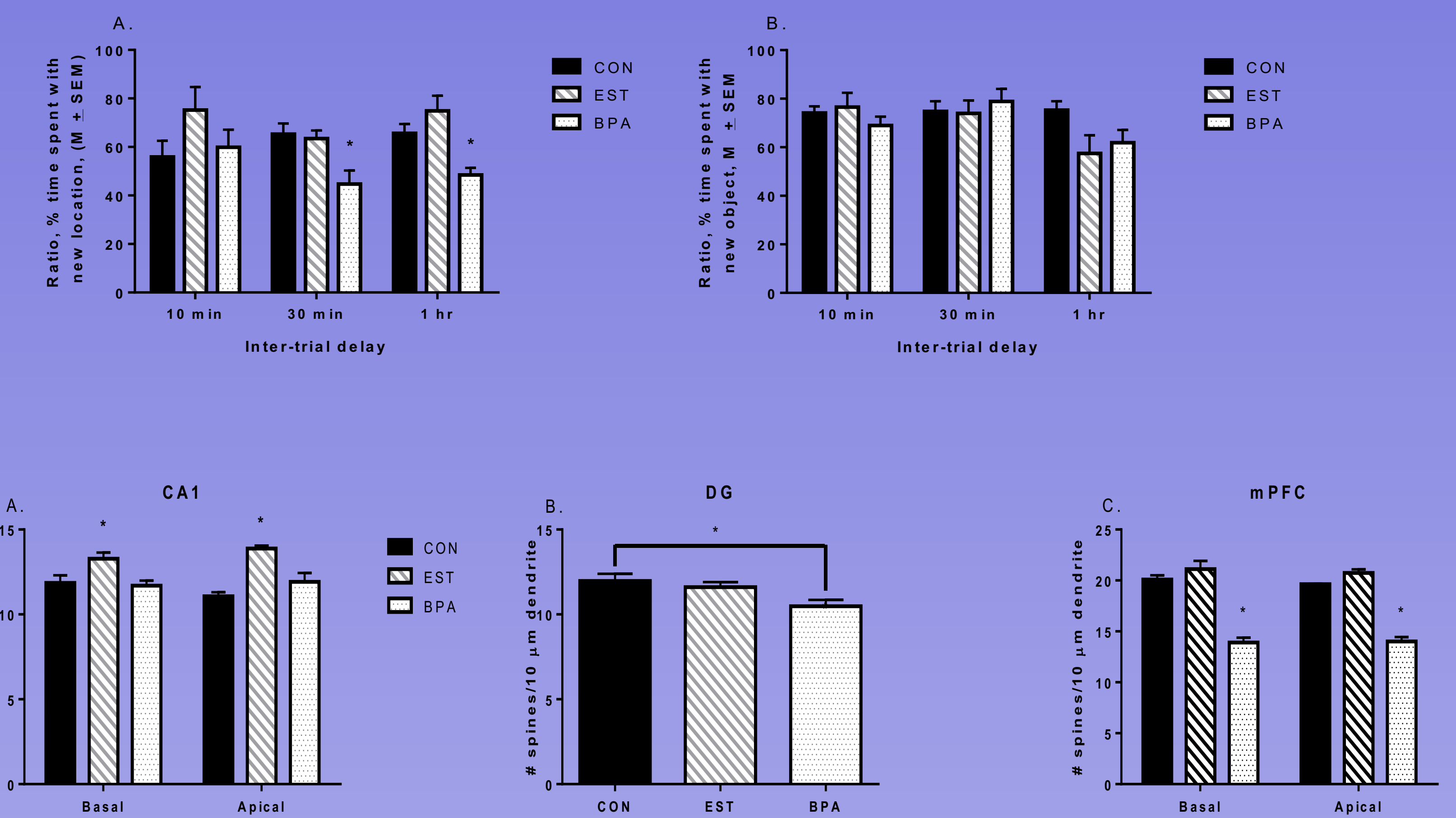


RESULTS:

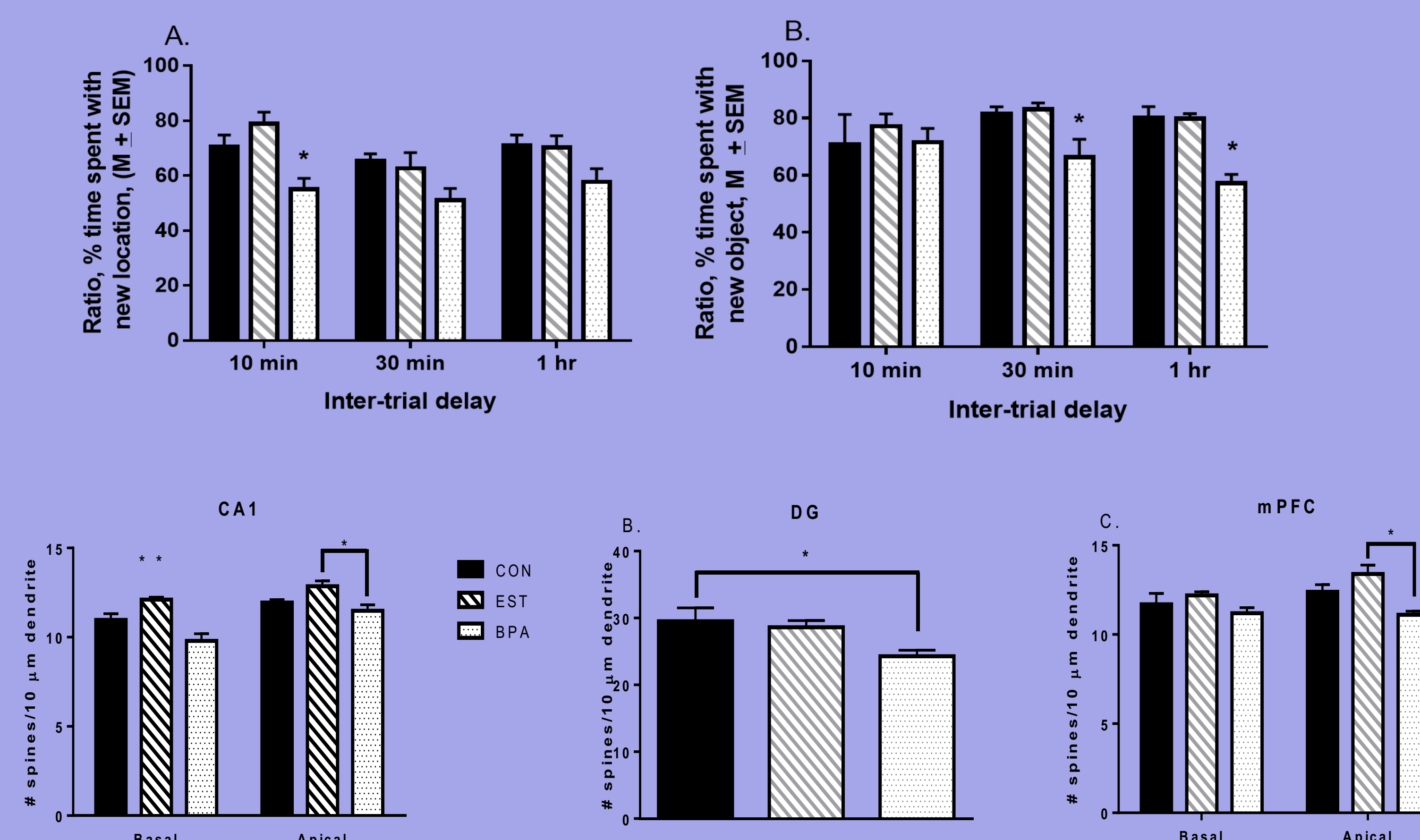
Experiment 1:



Experiment 2:



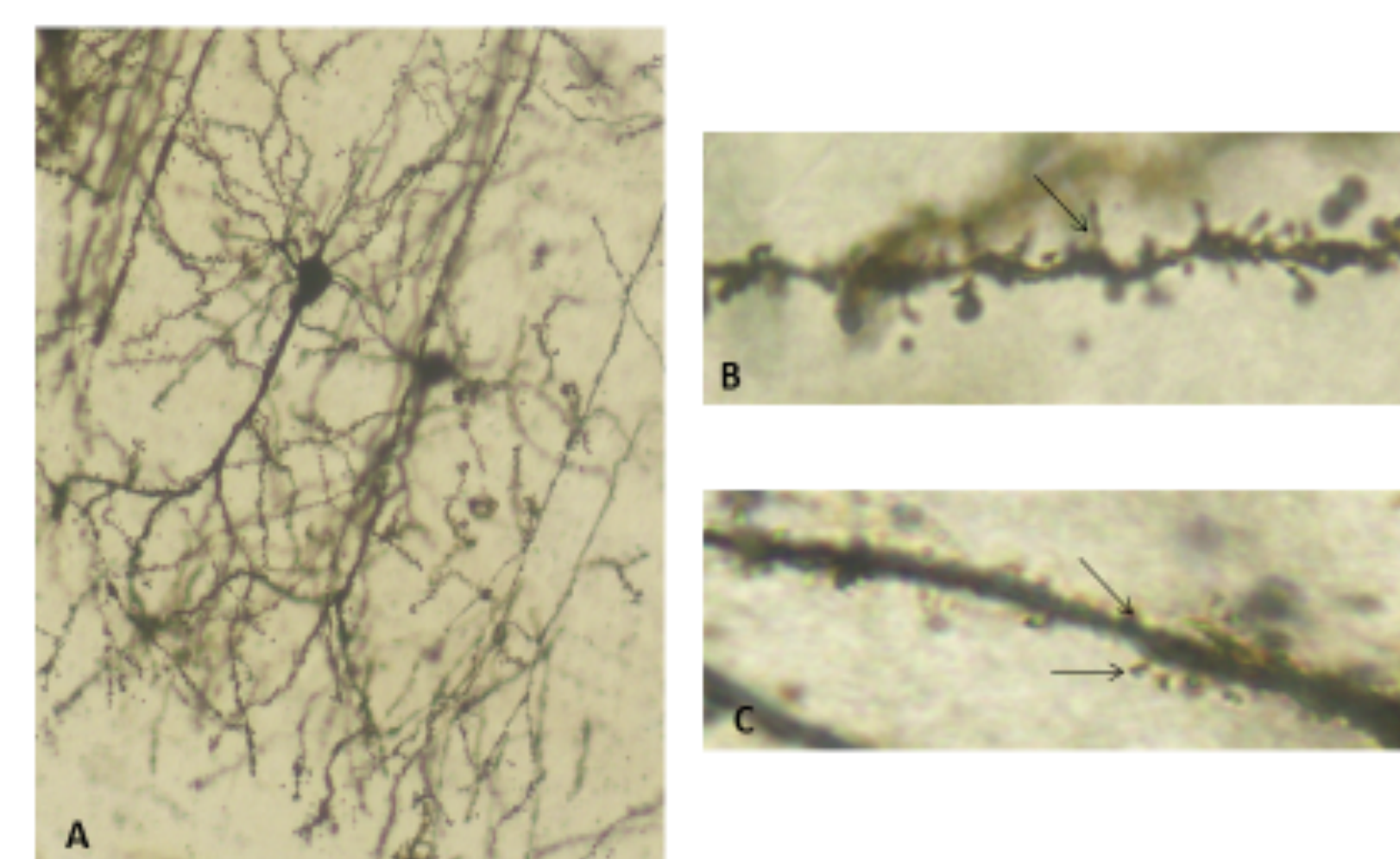
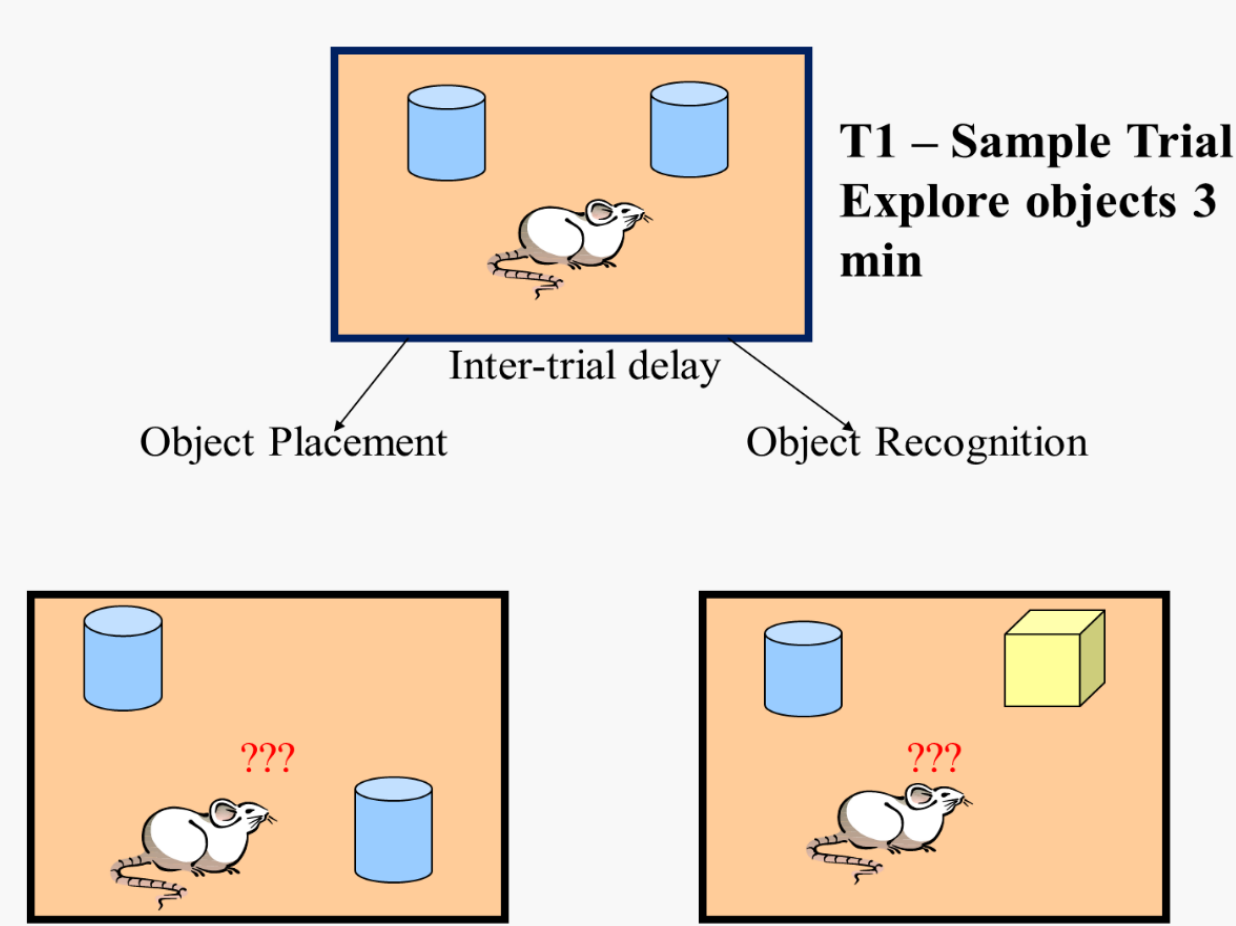
Experiment 3:



CONCLUSIONS:

- In experiment 1, adolescent hormonal manipulations altered dendritic spine density in a region-specific manner. In the hippocampus, CA1 dendritic spine density was increased by E-treatment, while BPA decreased spine density in the DG. No architectural changes were observed in the mPFC.
- In experiment 2, BPA decreased spatial memory long term. No behavioral differences were observed in non-spatial memory. In the hippocampus, CA1 dendritic spine density was increased by E-treatment, while BPA decreased spine density in DG and mPFC.
- In experiment 3, BPA decreased in spatial memory short term, and decreased non-spatial memory long term. In the hippocampus, CA1 basal dendritic density was increased by E-treatment, while BPA decreased spine density. There were less architectural changes observed in apical dendrites and mPFC with a decrease in spine density only between the E and BPA treatments. BPA decreased spine density in DG.
- Adolescent exposure to BPA shows lasting effects into adulthood in neuronal architecture and coinciding behavior.
- This experiment shows that the effects of BPA are maintained in the OVX model indicating that the endogenous model did not impact our previous results.
- The dosage used in this experiment was below what is approved by the EPA, and was found to have detrimental effects. This indicates that the EPA should reconsider the current restrictions on BPA use.

Object Placement & Recognition



A. Golgi impregnated pyramidal cells in the mPFC. 10x
B. Apical dendrite from estrogen treated rat. 100x, arrows denote spines.
C. Apical dendrite from BPA treated rat 100x, arrows denote spines.