

Undergraduate Research Symposium May 17, 2013 Mary Gates Hall

Online Proceedings

SESSION 1I

DEVELOPMENTAL NEUROPLASTICITY

Session Moderator: Sheri Mizumori, Psychology
251 MGH

1:15 PM to 2:45 PM

* Note: Titles in order of presentation.

Seasonal Plasticity in an Avian Song Control System: An Examination of Neuronal Recruitment and Apoptosis During Transition from Breeding to Nonbreeding Seasons

Nivretta Murlidharan (Nivi) Thatra, Senior, Neurobiology
Mentor: Eliot Brenowitz, Psychology
Mentor: Tracy Larson, Biology, University of Virginia

Ongoing neurogenesis in the adult brain is a fundamental process of neural plasticity. The songbird is an established model for studying neuroprotection, neurogenesis, and neuronal turnover due to dramatic plastic changes in morphology and function in the nuclei that control avian song production. HVC, a region of the brain that times the production of song, doubles in size at the beginning of the breeding season, largely as a result of an increase in new neuron incorporation. In the nonbreeding season the song control circuit rapidly regresses in size. Within 7 days HVC regresses to non-breeding condition volume, and neuron number decreases by around 25% (> 68,000 neurons) via neuronal apoptosis. We tested the hypothesis that regression of HVC via neuronal apoptosis upon transition into nonbreeding conditions is tightly linked to proliferation of neural stem cells (NSC) in the nearby ventricular zone and changes in singing behavior. We also asked whether new neurons recently incorporated into the song control system were retained or lost through transition into non-breeding conditions. We rapidly transitioned birds from breeding to nonbreeding conditions and measured neuronal death and survival, NSC proliferation, and song behavior over a time-course of 28 days. We found that some but not all neurons incorporated into HVC during the previous breeding season persist at least 28 days into the nonbreeding season, suggesting that both new and mature neurons must undergo apoptosis during transition into nonbreeding conditions. Interestingly, we found that proliferation in the VZ was tightly linked to the amount of cell death occurring within

HVC. We also quantified song degradation following rapid transition into nonbreeding conditions and correlated the observed changes in behavior to the cellular changes occurring within HVC. These findings demonstrate a relationship between cell death and neural stem cell proliferation.

POSTER SESSION 3

Commons West, Easel 9

2:30 PM to 4:00 PM

Testosterone Effects on Neuroplasticity in Gambler White-Crowned Sparrows

Elizabeth Marie (Elizabeth) Emau, Senior, Biology
(Molecular, Cellular & Developmental)
McNair Scholar

Mentor: Eliot Brenowitz, Psychology

Mentor: Ralf Luche, Psychology Department

Learning on a cellular level actively guides the stabilization or elimination of neuron connections, selecting the optimal pathways that are intrinsically and extrinsically stimulated. The strength and resilience of these neuron networks is what defines neuroplasticity, which can be inhibited by many factors, such as stress, ageing, or developmental disorders. Evidence shows rapid seasonal morphological changes in the High Vocal Center (HVC) and acropallium (RA) regions of the brain, defining regions for song-voice production in vertebrates. Testosterone (T) plays a cyclic role facilitating these changes and can act locally on the brain to prevent neuron loss and regression yet the T mechanistic pathway acting on neuron death and neurogenesis remains grossly uncertain. We address this issue by injecting eight cohorts of Gambler White-Crowned Sparrows (GWCS), that have been exposed to breeding and non-breeding conditions, with T and or/ a potent inhibitor of P13K (an intercellular molecule that indirectly regulates cell growth and apoptosis), Ly294002 (Ly). Information obtained will allow for the determination, if regulation of P13K is required in T mediated signal transduction. We evaluate the data by extracting the bird brain and staining the tissue with Nissl stain to examine HVC and RA regions. With the aid of camera imaging and a volumetric algorithm we then determine HVC and RA volumes with the expectation that Ly inhibits T-stimulated HVC and RA growth. Our results will provide insight to the complex molecular pathway that underlines adult neural plasticity contributing to specifying clinical treatments for neurodegenerative injuries.