Undergraduate Research Symposium MAY 21, 2010 Mary Gates Hall

Online Proceedings

POSTER SESSION 1

MGH 241, Easel 156

12:00 PM to 1:00 PM

Fatty Acids as Neurogenesis Proliferation Factors

Behnum (Benny) Habibi, Senior, Philosophy, Neurobiology
Mary Gates Scholar
Mentor: Philip Horner, Neurological Surgery
Mentor: Elizabeth Stoll, Neurobiology & Behavior

One of the most important recent findings in neuroscience has been the discovery of neurogenesis in the adult mammalian brain. However, neurogenesis declines with age and this loss is correlated with cognitive decline. Recently, it has been shown that both exercise and a high-fat diet increase neurogenesis in adult mice. I hypothesize that a mechanism for these increases in neurogenesis is an increase in fatty acid metabolism in neural stem cells (NSC). For instance, fatty acids are a primary fuel source for neonatal brains, when neurogenesis occurs at its greatest rate. Additionally, neurogenic regions in the brain are closely associated with vasculature and thus have unique access to blood-borne factors. Therefore, it is plausible that NSC proliferation could be limited by fatty acid availability in the environment. We have found that application of a fatty acid transport inhibitor decreases proliferation of cultured NSC without negative effects on cell viability, supporting this hypothesis. My current work further examines the role of fatty acids in neurogenesis, for example, by determining if the application of these fuels is sufficient to increase NSC proliferation. Also, we are examining how blocking fatty acid transport affects neurogenesis in live, exercising mice. This study not only provides insights into the molecular mechanisms of adult neurogenesis but also improves our understanding of cellular metabolism and the regulation of regeneration in the adult mammalian brain.

SESSION 1F

PLANT, BACTERIAL AND FUNGAL EVOLUTION

Session Moderator: Benjamin Hall, Biology

Mary Gates Hall Room 234

1:00 PM to 2:30 PM

* Note: Titles in order of presentation.

Methylobacterium Extorquens AM1 Metabolic Integration

Alexander (Alex) Palmer, Junior, Microbiology
Mentor: Elizabeth Skovran, Microbiology

Methylobacterium extorquens AM1 is a methylotrophic bacterium ubiquitous in the environment and in particular on the undersides of leaves. This organism is able to metabolize both single carbon compounds like methanol and multi carbon compounds such as succinate and pyruvate. M. extorquens has been studied for decades because of its potential for creating value added compounds from methanol, yet genes required for methylotrophic growth are still being discovered. The first step in methylotrophic growth is the oxidation of methanol by methanol dehydrogenase encoded by the mxaF genes. The M. extorquens genome contains two homologs of the mxaF gene, xoxF1 and xoxF2. Here we show that single mutations in the xoxF1 gene lead to both a decrease in growth on methanol and decreased methanol dehydrogenase activity. Surprisingly, when both xoxF1 and xoxF2 are mutated, AM1 is unable to grow on methanol and has no methanol dehydrogenase activity, identical to the phenotype of the mxaF mutant. Further studies revealed that the level of MxaF protein in the cell is greatly reduced in this xoxF1 xoxF2 mutant suggesting that XoxF1 and XoxF2 are required for either expression of mxaF or the stability of the MxaF protein.