Exploring the Role of Bif–1 in Neural Connectivity

Adam Uppendahl
Morrison lab discovered a novel protective function of Bax-interacting factor-1 (Bif-1) that may provide potential treatment for Alzheimer’s Disease (AD).

Neuron specific forms of Bif-1 decline in the AD brain and contribute to AD progression.

Morrison lab noted that Bif-1 is required for mitochondrial function and maintenance of neuronal health.
My Project

- Does Bif–1 regulate the elaboration of neural processes?
  - Overexpressed different forms of Bif–1 to determine if they effect neurite outgrowth
  - Knocked down Bif–1 to see if it is required for neurite outgrowth
Day 0: Cells plated

Day 1: Added Retinoic Acid and infected with lentivirus

Day 6: Took pictures of neurites and then added BDNF

Day 8: Took pictures of final growth
1) Cells prior to treatment
2) Cells after 5 day retinoic acid treatment
3) Cells after 5 day retinoic acid and 2 day BDNF treatment
Neurites >20um per Cell in 5 Day Retinoic Acid

- GFP
- Bif-1a
- Bif-1b
- Bif-1c
- Bif-1 shRNA
- Cont shRNA

Treatment

Significance:
- * indicates significance at the 0.05 level.
- # indicates significance at the 0.01 level.
Conclusions and Future Studies

- Bif–1 contributes to RA–induced neurite outgrowth
- Expression of neuron–specific Bif–1c significantly enhances RA–induced neurite outgrowth
- Loss of Bif–1c in AD might adversely influence the maintenance of neuronal connections
- Could Bif–1 be a therapeutic target for AD treatment?
Thank You

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