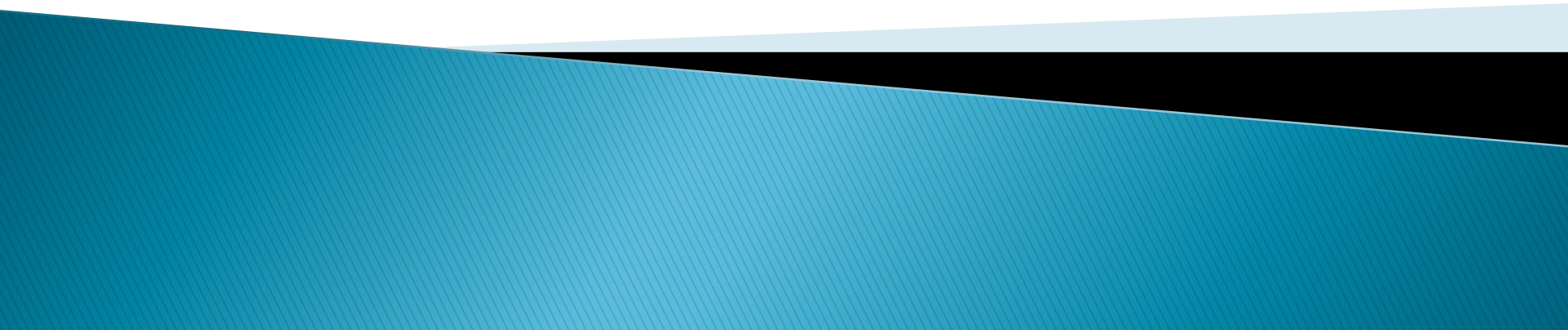
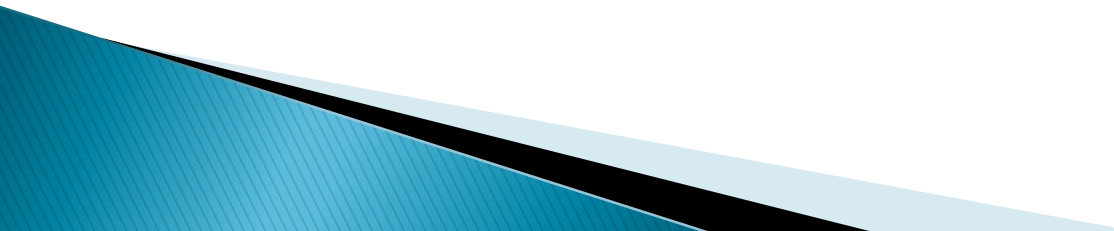


Exploring the Role of Bif-1 in Neural Connectivity

Adam Uppendahl



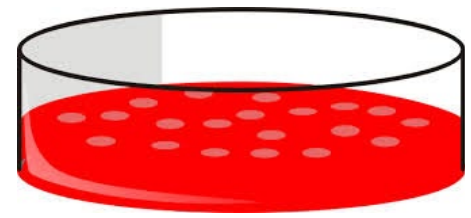
Background

- ▶ 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
- 

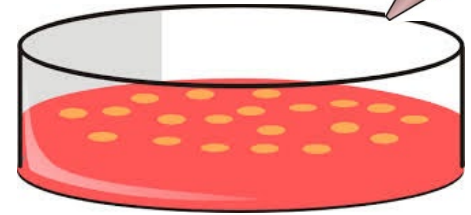
My Project

- ▶ 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

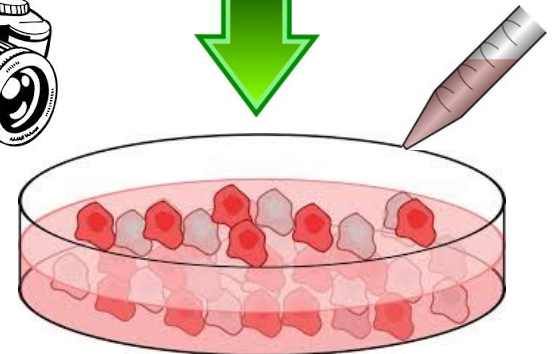
Day 0: Cells plated



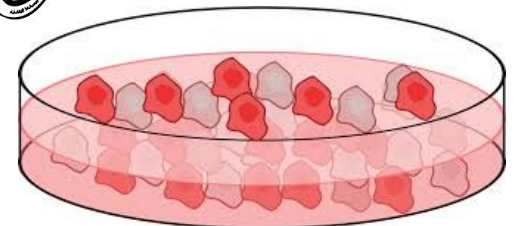
Day 1: Added Retinoic Acid and infected with lentivirus

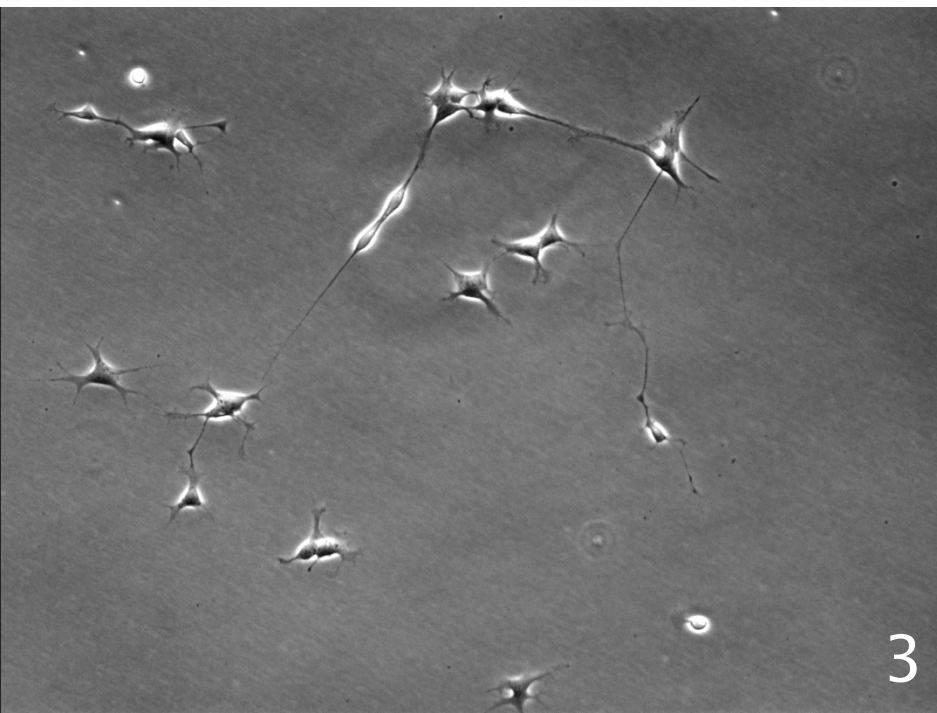
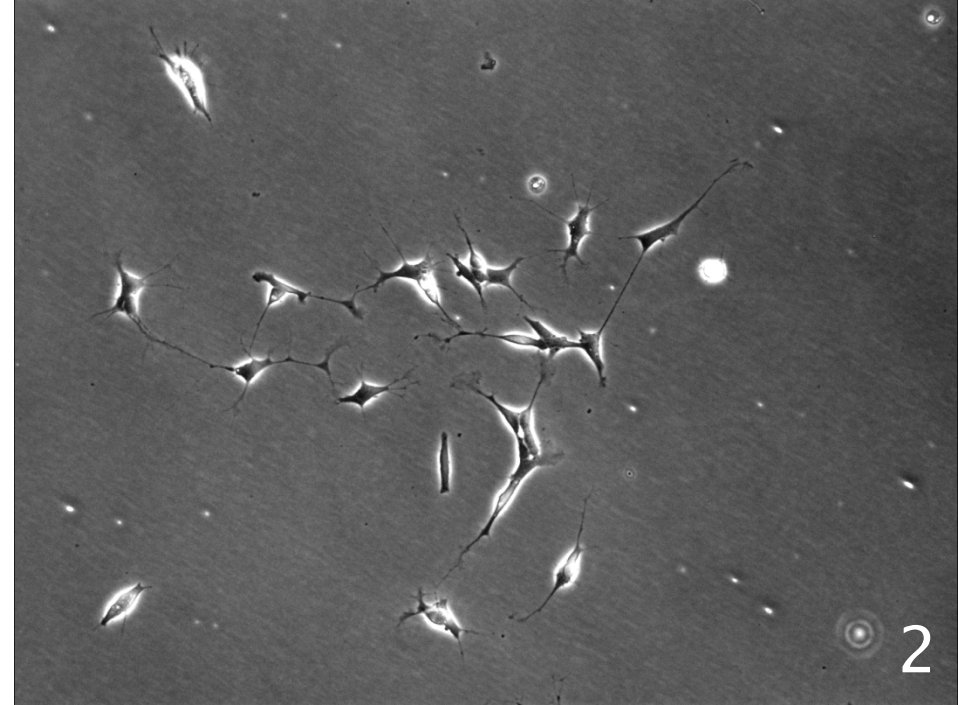
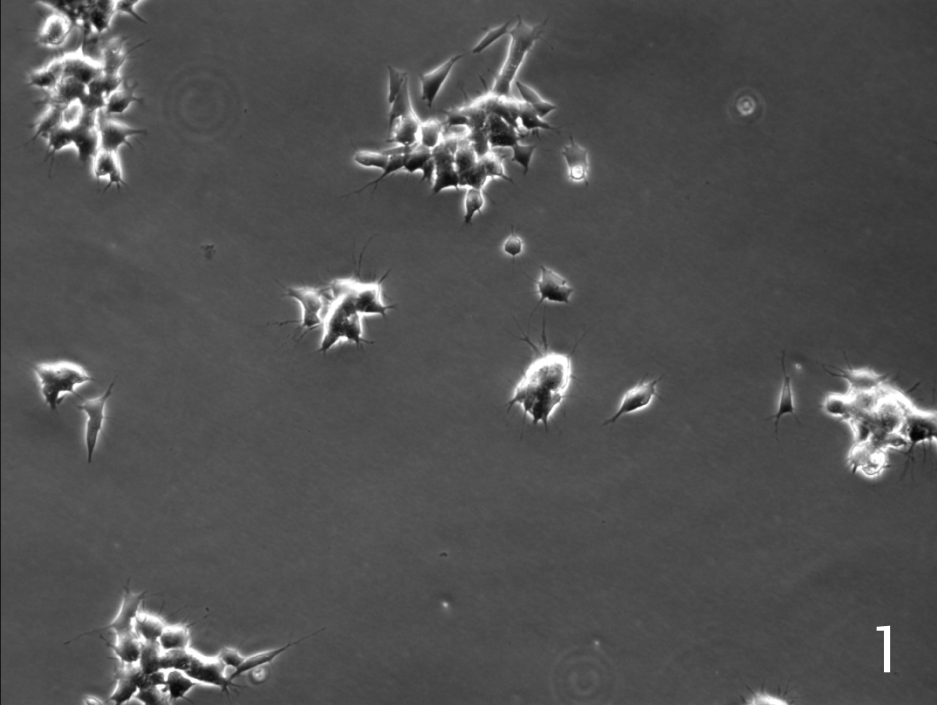


Day 6: Took pictures of neurites and then added BDNF



Day 8: Took pictures of final growth

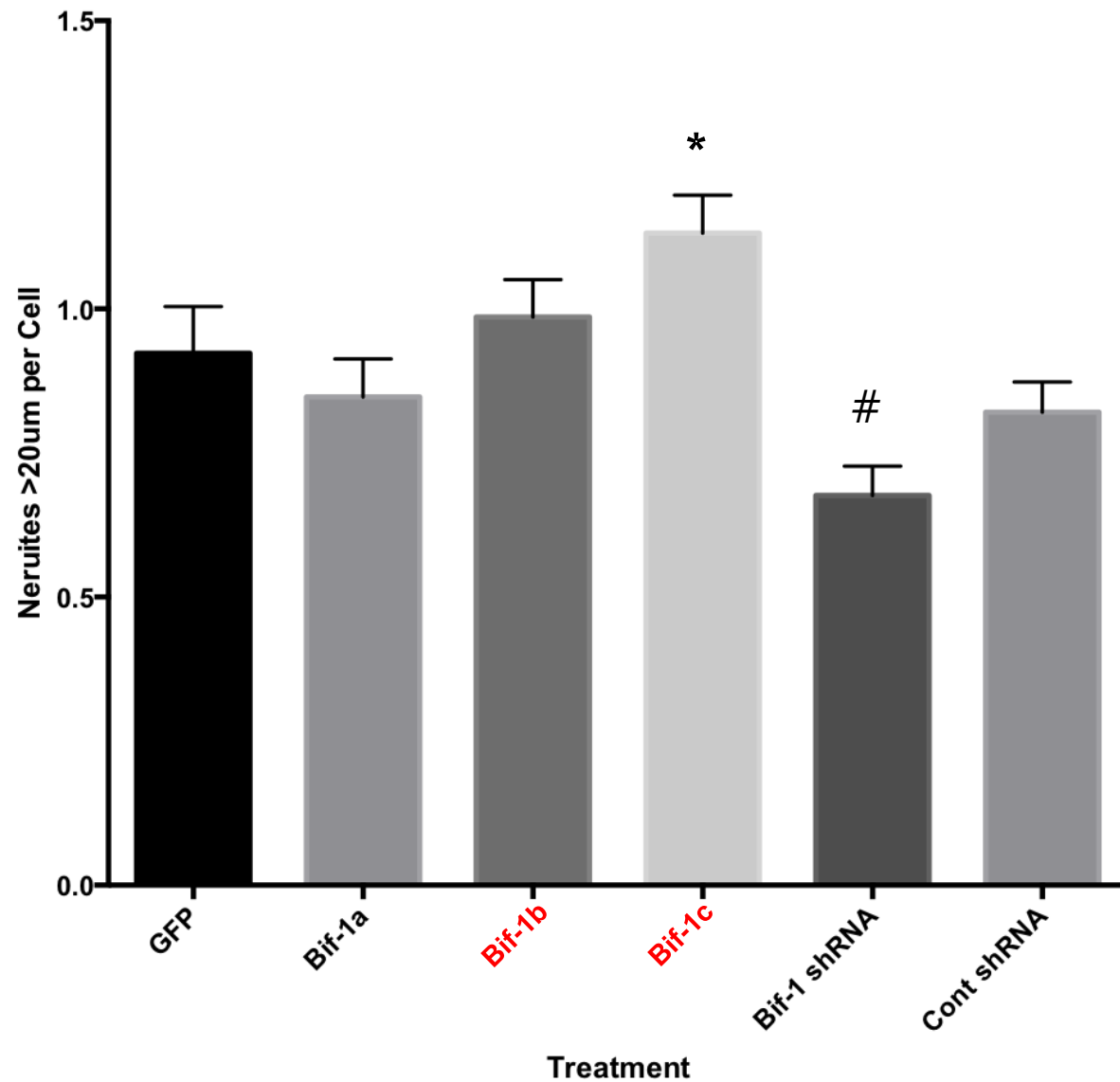




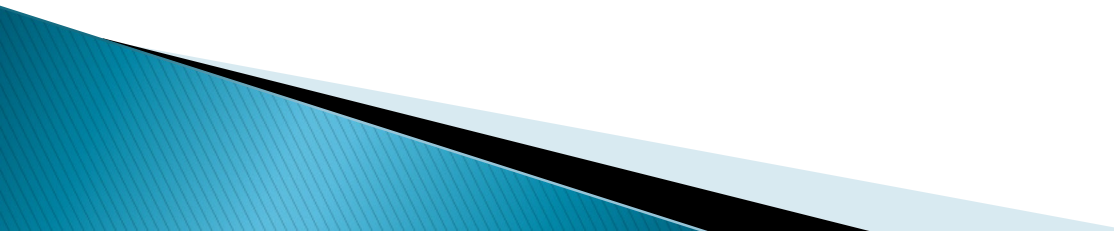
- 1) Cells prior to treatment
- 2) Cells after 5 day retinoic acid treatment
- 3) Cells after 5 day retinoic acid and 2 day BDNF treatment



Neurites >20um per Cell in 5 Day Retinoic Acid



Conclusions and Future Studies

- ▶ Bif-1 contributes to RA-induced neurite outgrowth
 - ▶ Expression of neuron-specific Bif-1 c significantly enhances RA-induced neurite outgrowth
 - ▶ Loss of Bif-1 c 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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