

A Time Course of Traumatic Optic Neuropathy after Mild Traumatic Brain Injury in Adolescent Male Mice.

Emily M. Shalosky¹, Shelby M. Cansler², and Nathan K. Evanson^{1,3}
1. Cincinnati Children's Hospital, Division of Pediatric Rehabilitation Medicine. 2. University of Cincinnati, Department of Psychiatry

Figure 1. Time course of injury in the OPTIC TRACT

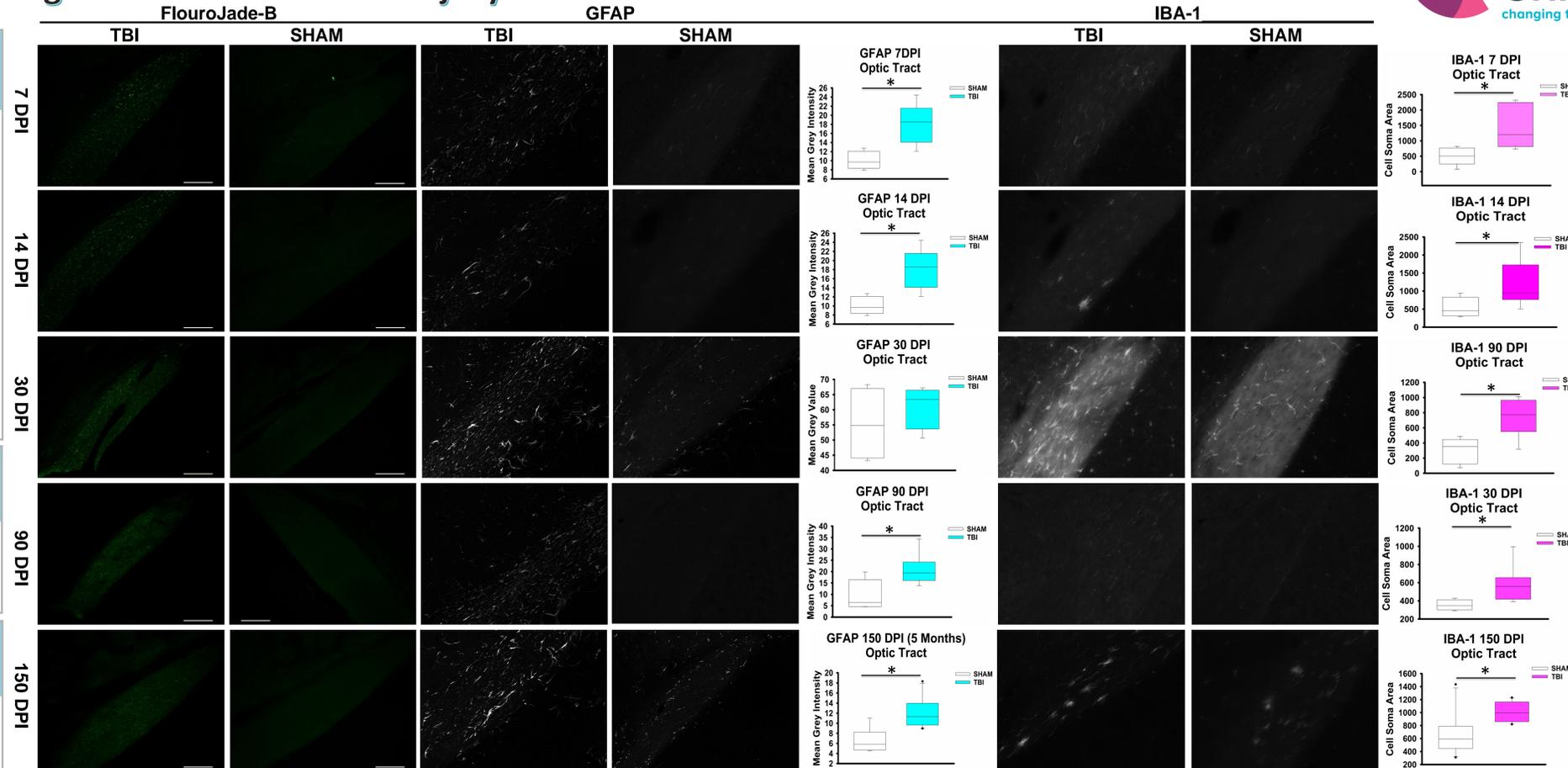


Figure 3. Neurodegeneration Across Time

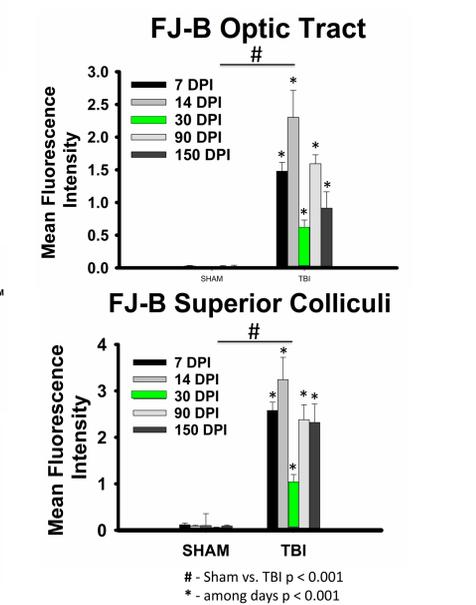
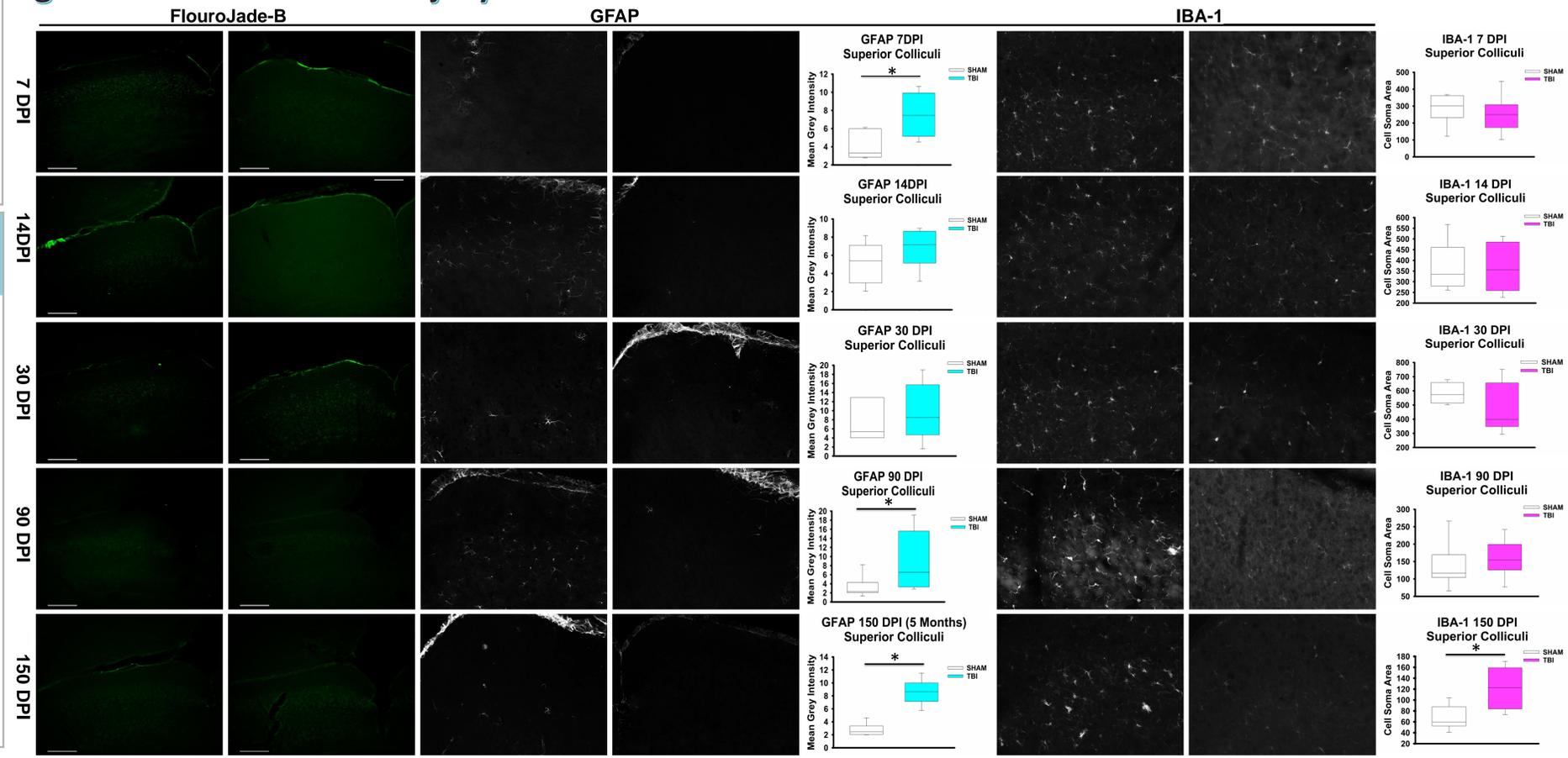


Figure 2. Time course of injury in the SUPERIOR COLICULI



Future Directions

Future Directions

- Explore other optic system projections
- Examine other secondary mechanisms (cytokines/inflammation).
- Control oxygen intake (ie: amount of time in O2 chamber, concentration).

Conclusions

Emerging Patterns across Stains & Time - Recovery and Resurgence

Time After Injury	Neurodegeneration Severity*		Significant Astrogliosis		Significant Microglial activation	
	OT	SC	OT	SC	OT	SC
7 DPI	+++	++++	✓	✓	✓	
14 DPI	++++	+++	✓		✓	
30 DPI	+	+			✓	
90 DPI	+++	+	✓	✓	✓	
150 DPI	++	++	✓	✓	✓	✓

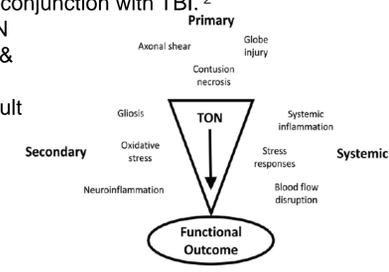
* The "+" is a representation of staining severity based on the 2 way RM ANOVA calculated across time points and a subjective scoring of the severity/presence of the staining. Highlighting indicates whether patterns agree across histological markers.

References

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Introduction

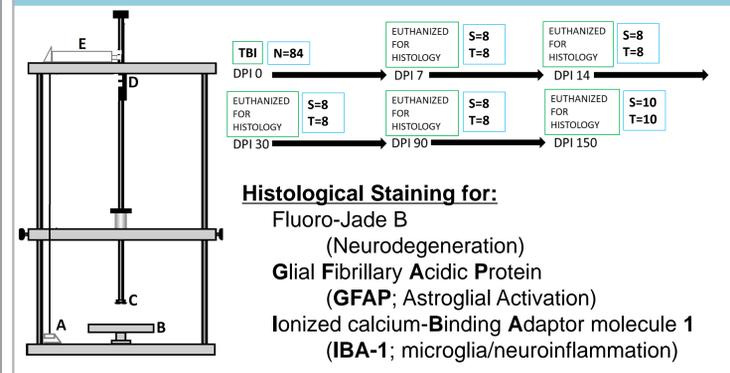
- Traumatic Brain Injury (TBI) affects 2.8 million people in the USA per year, of which about 1 million are children under 19.¹
- Traumatic Optic Neuropathy (TON) is an acute injury to the optic nerve and can be caused in conjunction with TBI.²
- Most common affects of TON are blurred or double vision & blindness.³
- Indirect TON may be the result of secondary injury.⁴
- These secondary cascades occur over time and require chronological analysis.



Hypothesis

Neurodegeneration will persist throughout the time course and be related to secondary injury through inflammation and gliosis.

Methods



Discussion

- In this model of indirect TON there is initial axonal degeneration, inflammation, and gliosis with a period of recovery then decline in the OT.
- Degeneration and gliosis also follow this pattern in the SC, but inflammation is delayed.
- Secondary cascades caused by TBI follow a pattern of **Wallerian Degeneration** as such: neuronal death → calcium dysregulation → apoptosis/necrosis → membrane failure/degradation of neuronal cytoskeleton/cytoplasm.
- Known time courses:
Somatic → neuronal death 30DPI → recovery over 2-12 months.⁵
Axonal → partial recovery at 2 weeks → decline up to 8 weeks.⁶

Limitations

- Each time point a different animal was used
- Location of injury is non-specific (i.e., "over bregma")
→ though this makes it more generalizable to humans
- Only two major optic tract projections were investigated.
- These mice received supplemental oxygen after injury.