

Abstract

- Traumatic brain injury causes long-term physical and neurological consequences to the body
- These consequences are caused by impacts of synaptic transmission and decreased neuroplasticity
- The study I will be conducting a literature review sees if there are any possible changes to neuron connectivity and any possible physiological and neurological changes after exercise is conducted
- This study was focused on the impacts of exercise on brain-derived neurotrophic factor (BDNF) signaling
- Male mice were utilized in the study as a portion of the mice had a fluid percussion injury as their TBI, and a portion of the mice was able to implement exercise through voluntary wheel running
- Behavior was measured through learning tasks (e.g. Morris Water Maze), and through molecular markers that measured BDNF signaling
- Voluntary exercise during the subacute phase (14-20 days after the injury) leads to an increase in BDNF signaling and proper recovery for the mouse

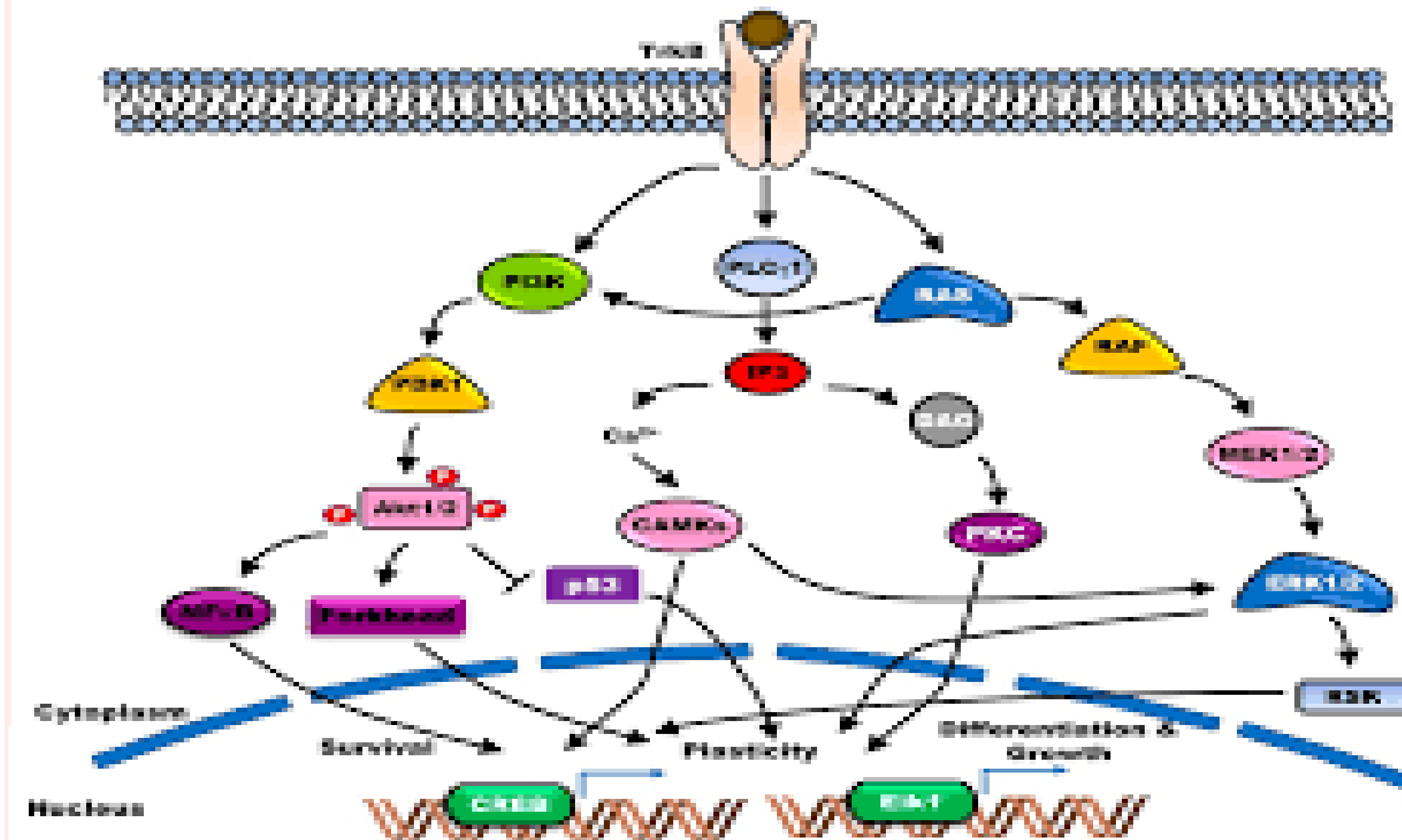


Figure: This represents the BDNF-TrkB signaling pathway which influences neuron connectivity, synaptic plasticity, proper recovery and proper function of the human with regards to the signaling cascades that are amplified

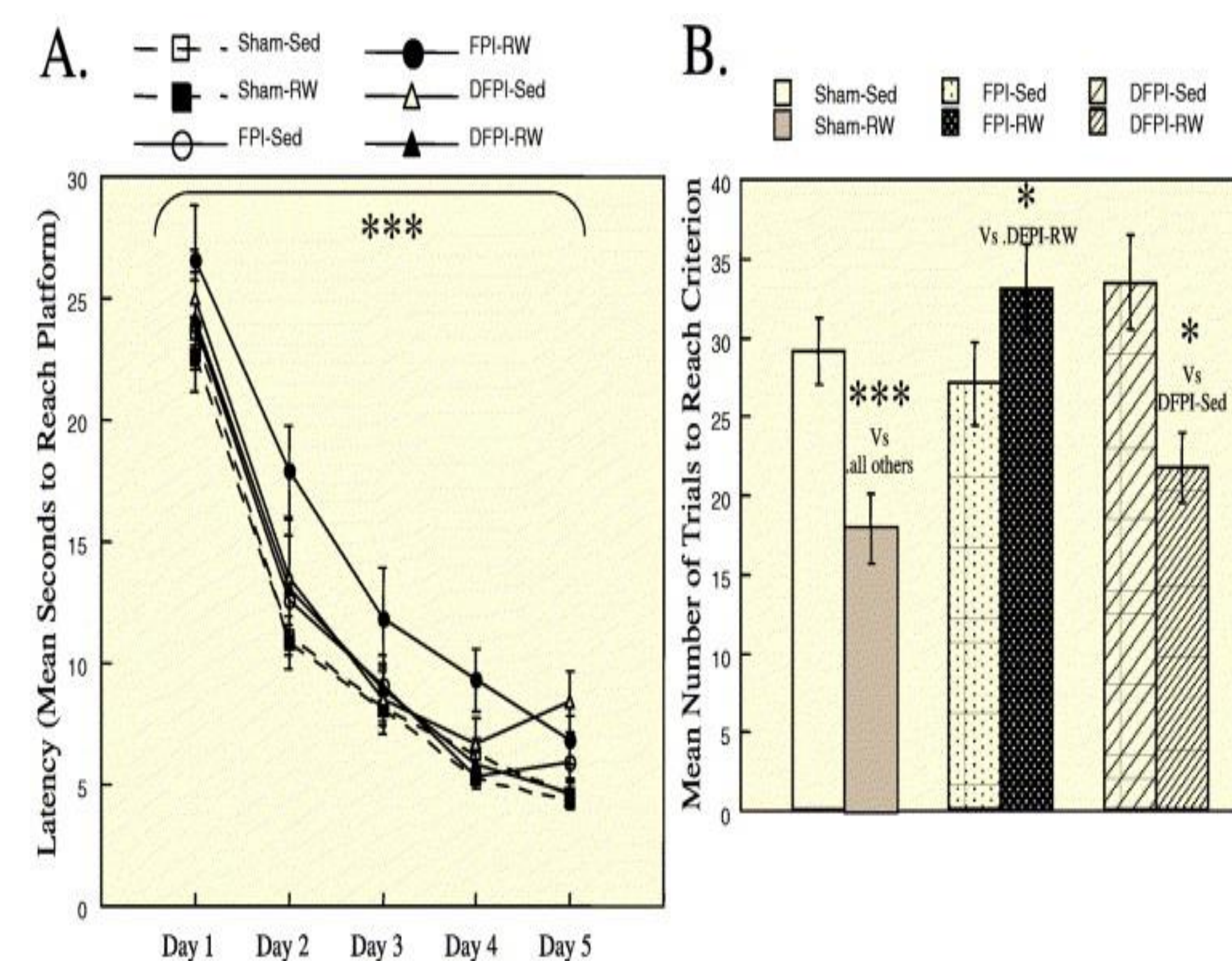
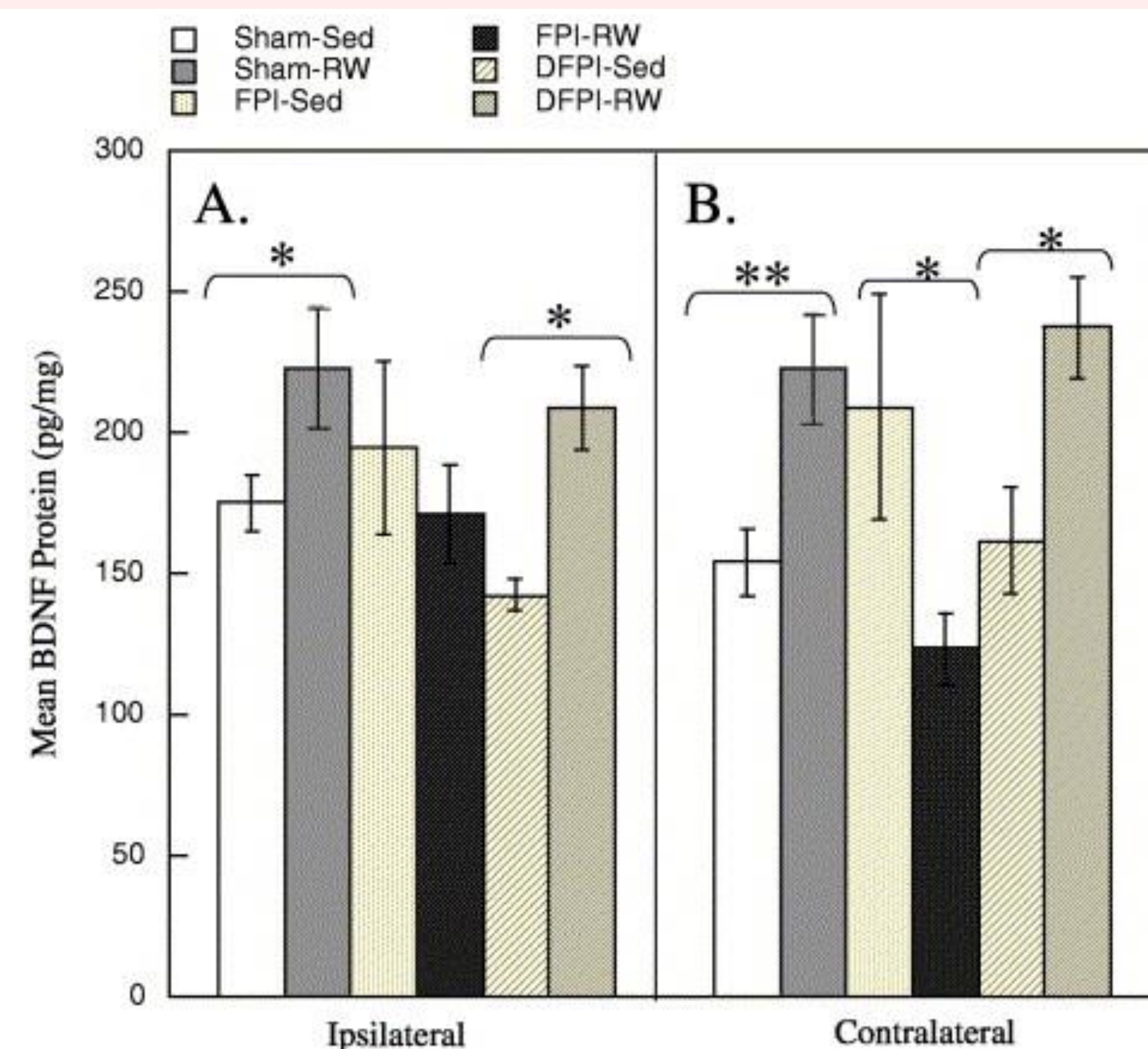
Next questions I would like to be answered:

- What possible TrkB agonists could improve BDNF expression and cognition?
- Is BDNF expression the only necessary signaling pathway that benefit one's recovery after TBI?
- What would happen when female mice were utilized in the study?

Data

Figure 1: The effect of exercise timing on hippocampal BDNF Expression after a TBI

Figure 2: The effect of The amount of exercise Implemented upon Cognition which was Analyzed from the Morris Water Maze



Discussion

- The timing of the exercise of TBI is essential
- Acute exercise is not beneficial for one's recovery due to metabolic stress and an immense amount of energy utilized when recovering
- The changes in BDNF, CREB, and Synapsin depict how neuroplasticity is affected based on exercise
- The lack of BDNF expression and signaling which was seen in the acute phase lead to decreased cognition which was seen in the Morris Water Maze
- Exercise can be beneficial, but the timing is also necessary in order for the needed impacted to be seen

Methods & Procedure

- 89 Sprague-Dawley rats (Fluid Percussion Injury)
- 72 Sham Sprague-Dawley rats (control)
- Sprague-Dawley rats either implemented voluntary exercise (running wheel), or implemented no voluntary exercise
- Timing of exercise also separated the group (acute- 0-6 days after injury,; subacute- 14-20 days after injury)
- Exercise was implemented with a running wheel
- The Morris Water Maze places stimuli (the hidden platform) that the rat would need to identify through a certain period of time
- Five days of training was implemented for analysis of the Morris Water Maze
- A probe test (the platform) was removed in order to analyze memory and retention for the rats
- Performance from the training was measured through the amount of time it takes to find the platform, and the amount of times it takes to reach a certain criteria implemented

- Researchers were able to utilize ELISA (analyze BDNF expression), Western blots (Synapsin and CREB signaling), and three-way ANOVA to analyze the results

Results

- Delayed exercise (14-20 days after injury) for mice led to increased BDNF expression, and improved neuron and functional recovery
- Acute exercise (0-6 days after the injury) for mice didn't increase BDNF expression and led to disruption of signaling pathways, such as the CREB pathway
- Cognitive performance (analyzed in the Morris Water Maze) improved for delayed exercise and control rats
- Acute exercise rats had worse learning and memory compared to its counterparts

Conclusion

In conclusion, this experiment was able to portray why exercise is beneficial for the functional recovery. The timing is also necessary for the proper benefit to be shown for BDNF expression, neuroplasticity and the proper recovery of the brain. To conclude, therapeutic interventions must align with the brain's state.

Acknowledgments

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Reference

- Griesbach, G. S., Hovda, D. A., Molteni, R., Wu, A., & Gomez-Pinilla, F. (2004). Voluntary exercise following traumatic brain injury: brain-derived neurotrophic factor upregulation and recovery of function. *Neuroscience*, 125(1), 129-139. <https://doi.org/10.1016/j.neuroscience.2004.01.030>