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Title

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Permalink

<https://escholarship.org/uc/item/4301q5rd>

Journal

Berkeley Scientific Journal, 30(1)

ISSN

1097-0967

Author

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Publication Date

2026-10-30

DOI

10.5070/BS3.69886

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Impacts of Traumatic Brain Injury on Synaptic Plasticity and Cognitive Function

BY: BENJAMIN HARTMAN

BAD BRAIN BLUDGEON

Why can a person walk away from a concussion with no visible injuries yet struggle to remember conversations or develop new skills? The answer lies in the connections between neurons and in how brain injuries trigger a cascade of changes that affect memory and learning.

Traumatic brain injury (TBI) is a type of injury caused by an external force to the head. TBI often stems from high-impact events, including sports collisions, vehicle accidents, and falls. TBI ranges in severity from mild concussions to severe permanent brain damage. Regardless of the severity, however, TBI disrupts brain processes and commonly damages the areas of the brain involved in memory—namely, the hippocampus, a small part of the brain that mediates the formation and retrieval of memories. In the hippocampus, memory formation results from changes in synaptic strength in response to stimuli.

Synaptic strength corresponds to the importance of the receiving signal. For example, an important memory, such as a topic taught in school or a significant event, results in increased synaptic strength, producing a stronger, longer-lasting memory. The opposite is also true—a less relevant thought or idea will be lost as the synaptic strength decreases. This dynamic process of strengthening and weakening synapses, called synaptic plasticity, provides the basis for how a healthy brain creates and stores memories and information. To understand how TBI disrupts these mechanisms, it is first important to examine how these pro-

cesses normally function in an uninjured brain.^{1,2}

NEURAL CONNECTIONS AT WORK

In a normally functioning brain, synaptic plasticity results in memory formation in the hippocampus, contributing to a cognitively healthy individual. There are several different ways in which synaptic plasticity regulates memory.

Synaptogenesis and synaptic pruning are opposite processes that determine which memories are kept and destroyed through regulating synapses. Synapses are the junctions between neurons where chemicals called neurotransmitters allow information to travel throughout the nervous system. Synaptogenesis is the process of forming synapses between neurons, and while it mostly occurs during early brain development, it also contributes to synaptic plasticity in a mature brain. As more synaptic connections form, the strength of a signal sent between neurons increases, corresponding to a more “important” memory. Synaptic pruning is the opposite of synaptogenesis, where unnecessary or weak synapses are eliminated, reducing connections and increasing the overall efficiency of the brain’s neural network. The overall purpose of these two processes is to strengthen important memories while getting rid of unnecessary or old ones to improve productivity within the brain.³ This ties into synaptic plasticity, as the strength of synapses is varied to manage memories and improve learning, while simultaneously removing unnecessary or old memories.

Long-term potentiation (LTP) and

long-term depression (LTD) are long-term processes that regulate synaptic plasticity, where synaptic strength is increased and decreased, respectively. During LTP, receptor sensitivity on the post-synaptic neuron is increased, allowing neural circuits to store information more effectively over long periods of time. LTD is the opposite—a prolonged decrease in synaptic strength by reducing receptor sensitivity. LTD is essential in removing outdated or unnecessary connections that slow the brain’s neural network. By balancing these two processes, the brain is able to adapt to new information, retain essential memories, and remove in-

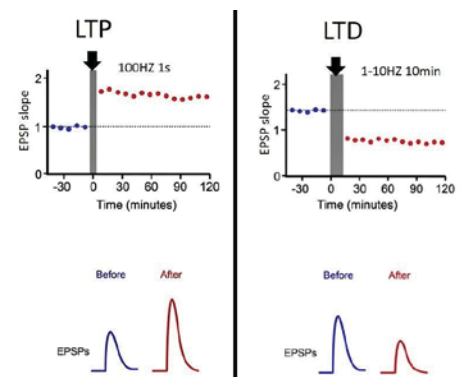


Figure 1: Excitatory Postsynaptic Potential (EPSP) in LTP and LTD. Diagrams showing changes in synaptic strength with LTP and LTD over time, indicated by the increase in EPSP after LTP and the decrease after LTD. The bottom images show changes in the strength of the action potential during signalling, with LTP similarly increasing synaptic strength and LTD decreasing strength.

efficient connections, allowing for healthy cognitive function.^{3,4}

DISRUPTED SIGNALS

As mentioned earlier, TBI can majorly affect brain processes, especially those occurring in the hippocampus. The impairment of synaptic plasticity is usually caused by mechanical damage from the actual impact of TBI or through related chemical processes such as toxicity, neuroinflammation, and oxidative stress.⁵

TBI-induced mechanical damage can harm neurons through physical injury, causing signalling disruptions and cell death. It also triggers toxicity, in which neurotransmitters like glutamate overstimulate neurons and initiate a cascade that leads to excessive calcium influx. Calcium surplus overwhelms natural homeostasis and causes cell death, which often leads to tissue damage, inflammation, and disease. This chain of events damages neurons, affecting synaptic plasticity and memory. This neuronal death is the primary driver of many neurodegenerative diseases, including Alzheimer's and Parkinson's, highlighting the severity of neurotoxicity from TBI.⁶

Similar to getting sick, TBI also triggers an immune response from brain cells like microglia and astrocytes. In response to an external force, these cells release chemicals like cytokines and chemokines which protect the brain and promote tissue repair. However, this immune response serves as a double-edged sword, since cytokine and chemokine function causes inflammation in the brain. In the long run, this neuroinflammation disrupts synaptic plasticity by impairing the signalling required for LTP and LTD to take place, preventing learning and memory function.

Additionally, immune cells usually help regulate synapses by promoting synaptogenesis and pruning. However, after TBI, these cells can become dysregulated, leading to either too many or too few synapses being removed. This disruption also affects synaptic plasticity and results in cognitive decline.

Lastly, excessive cellular activity from toxicity and immune responses leads to an increase in cellular metabolism. Metabolism produces many molecules as by-products, including reactive oxygen species (ROS). With heightened metabolic activity, excess ROS accumulate and damage bio-

molecules such as DNA, proteins, and lipids. Because these molecules form the structural and functional foundation of cells, damage to them can severely disrupt important processes. Elevated ROS levels induce oxidative stress, which further activates immune responses and heightens inflammation; this, in turn, amplifies toxicity and further increases ROS production.

Altogether, these various effects of TBI reinforce one another in a self-perpetuating cycle, impairing synaptic plasticity and compromising neuronal function.⁵

THE SCIENCE OF BEING SIDELINED

From car crashes to sports concussions, TBI can be induced in many ways. However, a better understanding of TBI and synaptic plasticity can lead to better approaches for treating injuries and improving recovery.

A well-known form of TBI is concussion via sports accidents. Many high-impact sports, including American football, rugby, and wrestling, all have high risks of physical injuries and thus, higher risks of concussion. One survey from 2015 reported that approximately 37.9% of football players have had at least one concussion, underlining the relevance of this issue.⁸ Studies show drastic long-term effects from TBI, including a decrease in LTP, worsened synaptic strength over time, and a loss of hippocampal volume, which all contribute to reduced memory performance and learning capabilities. Even after an apparent short-term recovery from the body repairing minor damage, TBI can cause persistent impairments in the brain's ability to adapt and remodel synaptic connections, leaving people affected with permanent neurologi-

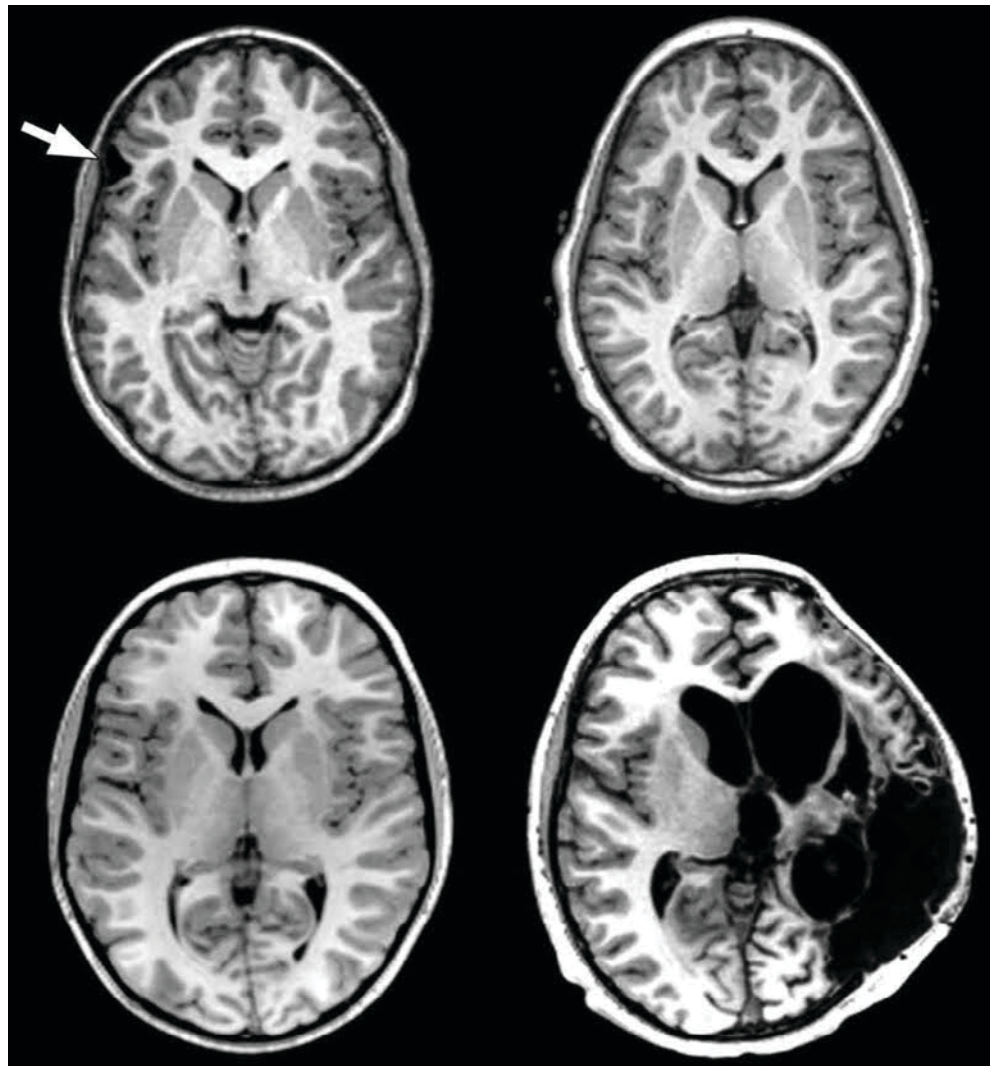


Figure 2: Healthy Brains versus TBI-affected Brains. (Top) MRI scans of healthy brains and brains affected with mild TBI (bottom left) and severe TBI (bottom right). Healthy brains appear regularly shaped and uniform in composition. Bottom left brain shows slight inflammation and slight deformations in brain tissue, indicating mild TBI. Bottom right brain shows major damage to severe TBI, a large amount of inflammation, deformation, and hematoma (bleeding).

cal issues even years later.^{9, 10, 11}

Brain issues stemming from TBI, especially repeated cases of TBI, can develop into other issues, including diseases such as chronic traumatic encephalopathy.¹² TBI also shows a strong association with other neurological disorders, including Alzheimer's and Parkinson's. Even mild TBI, such as a concussion, has been shown to increase the risk of Parkinson's by up to 56%, with moderate to severe TBI increasing the risk by about 83%.¹³ Both Alzheimer's and Parkinson's are associated with decreased functioning of LTP and LTD, leading to degeneration of memory and dementia in Alzheimer's and impaired motor skills in Parkinson's.^{14, 15} While these diseases occur naturally, their increased incidence as a result of TBI shows how brain injuries can majorly affect cognitive functioning and synaptic plasticity. TBI can also lead to an earlier onset of these diseases, as cognitive decline may begin earlier due to decreased synaptic plasticity.^{13, 16}

Since these diseases directly affect cognition and memory, they clearly illustrate how TBI can disrupt the processes underlying synaptic plasticity, such as synaptogenesis, pruning, LTP, and LTD, and how these disruptions translate into real-world impacts on people's lives. This highlights not only the importance of preserving synaptic plasticity to support cognitive health, but also the need to better understand how TBI alters the brain in order to develop more effective treatments. Currently, Alzheimer's is one of the most extensively studied and clinically relevant diseases, and exploring its origins, including TBI-induced synaptic plasticity decline, can lead to improved solutions in the future.¹⁶

CONCLUSION

Traumatic brain injury has been shown to disrupt synaptic plasticity in many ways, decreasing the brain's ability to form and retain memories and learn new things, thus leading to long-term neurological impairments. Greater awareness of TBI and synaptic plasticity not only deepens our understanding of the brain's intricate processes but also highlights the importance of advancing research to promote recovery and prevention of neurodegenerative disease.

ACKNOWLEDGEMENTS

I would like to thank Professor Robert

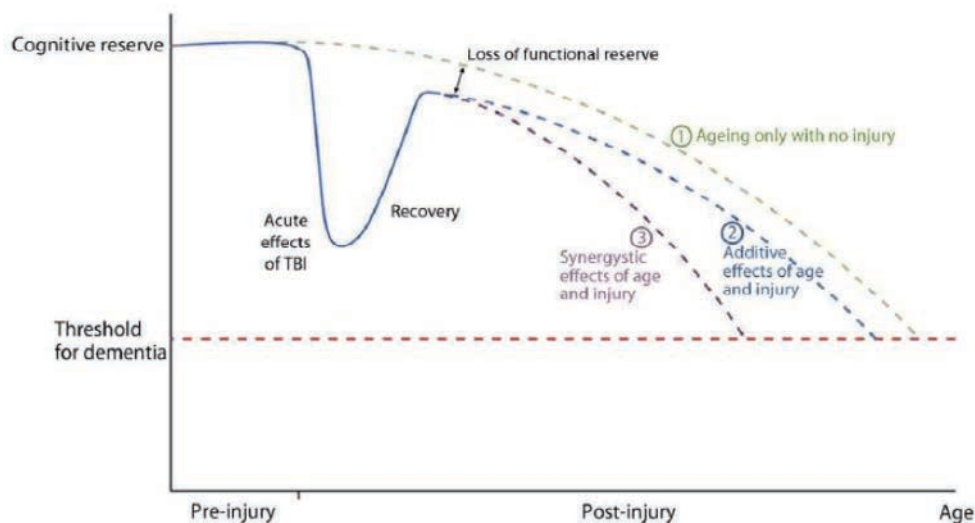


Figure 3: Visualisation of Cognitive Function over Time. A graph of cognitive functioning with and without TBI. The highest cognitive reserve (y-axis) is with line 1, aging with no injury, while the other lines show the impacts of TBI. Note the large negative effect of TBI in the short term, with relatively quick recovery, leading to a largely return to normal functioning. However, in the long term, TBI in combination with other factors, such as age, can lead to significant cognitive decline and early-onset dementia.

Zucker of the UC Berkeley Neuroscience department for his kind and helpful feedback on this piece and for reviewing its scientific accuracy.

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