

Introduction

Neuroplasticity, also known as brain plasticity or neural plasticity, is the ability of nervous system cells to adapt and change in response to interactions of the living organism with the environment, not only in normal condition but also in conditions that apply stress on the brain tissue such as infection, emotional stress, and trauma. This process includes the recovery from brain injury by inducing changes in the physiology and connectivity of individual neurons. This project aims to summarize the research literature about neuroplasticity in humans in response to sports-related traumatic brain injury.

Aims and Objectives

- Describe the axonal and synaptic morphological changes in the cerebral cortex secondary to traumatic brain injury related to sports.
- Describe the molecular and biochemical changes in the cerebral cortex secondary to traumatic brain injury related to sports.

Methods

This project designed as a systematic review and we conducted the steps of the projects as follows:

Databases searched: PubMed, Embase, Cochrane Library, Scopus, SportDiscus, Web of Science, Google Scholar, Northern Lights Conference Abstracts, Dissertations & Theses (Proquest).

Search terms: brain plastic, trauma, injury, neuroplastic, neuronal-plasticity, athletic injuries, accidental injuries, nervous system trauma, and their synonyms/variations.

Stages: Title/Abstract Screen → Full-text Screen → Quality Appraisal & Data Extraction

Inclusion criteria: Primary research articles pertaining to neuroplasticity in response to sports-related trauma. Publication must be written in English and published dates between 1980 and 2020.

Exclusion criteria: Studies on patients with preexisting brain injury/trauma. Non-scientific studies (reviews, editorials, comments, news items, etc.) and non-English studies were excluded as well, books and book chapters. Research articles on non-human subjects

Reliability and Blinding: At least 20% of the articles were double-reviewed for reliability. Reliability for each stage was greater than 80%. Reviewers were blind to the others' decisions.

Results

Most of the focus in the published articles is on BDNF, and NMDAR. Neuroplasticity response and the outcome of tissue regeneration as well as the level of response are important factors the determine the overall response. Traumatic brain injury research trends are toward understanding the main player in both acute and chronic TBI or CTE as shown in the figures below.

PubMed 542, Embase 684, Cochrane Library 4, Scopus 175, SportDiscus 188, Web of Science 268, Google Scholar 55, Northern Lights Conference Abstracts 51, Dissertations & Theses (Proquest) 23	1990 Initial search articles	1496 Title/Abstract screen	52 Articles/ Full text screen	19 Articles/ data extraction	Reasons of exclusion 10 articles-different study objectives 9 articles-different study designs 6 articles-methods used is not applicable 5 articles-study different TBI markers 2 articles-study on animals not human 1 article-summary not a full text
	494 Duplicates articles removed	1444 Studies excluded	33 Studies excluded	9 articles/good quality design and objectives 11 articles/lesser quality design or objective	

Table 1: Systematic review PRISMA analysis and list of databases searched in this study

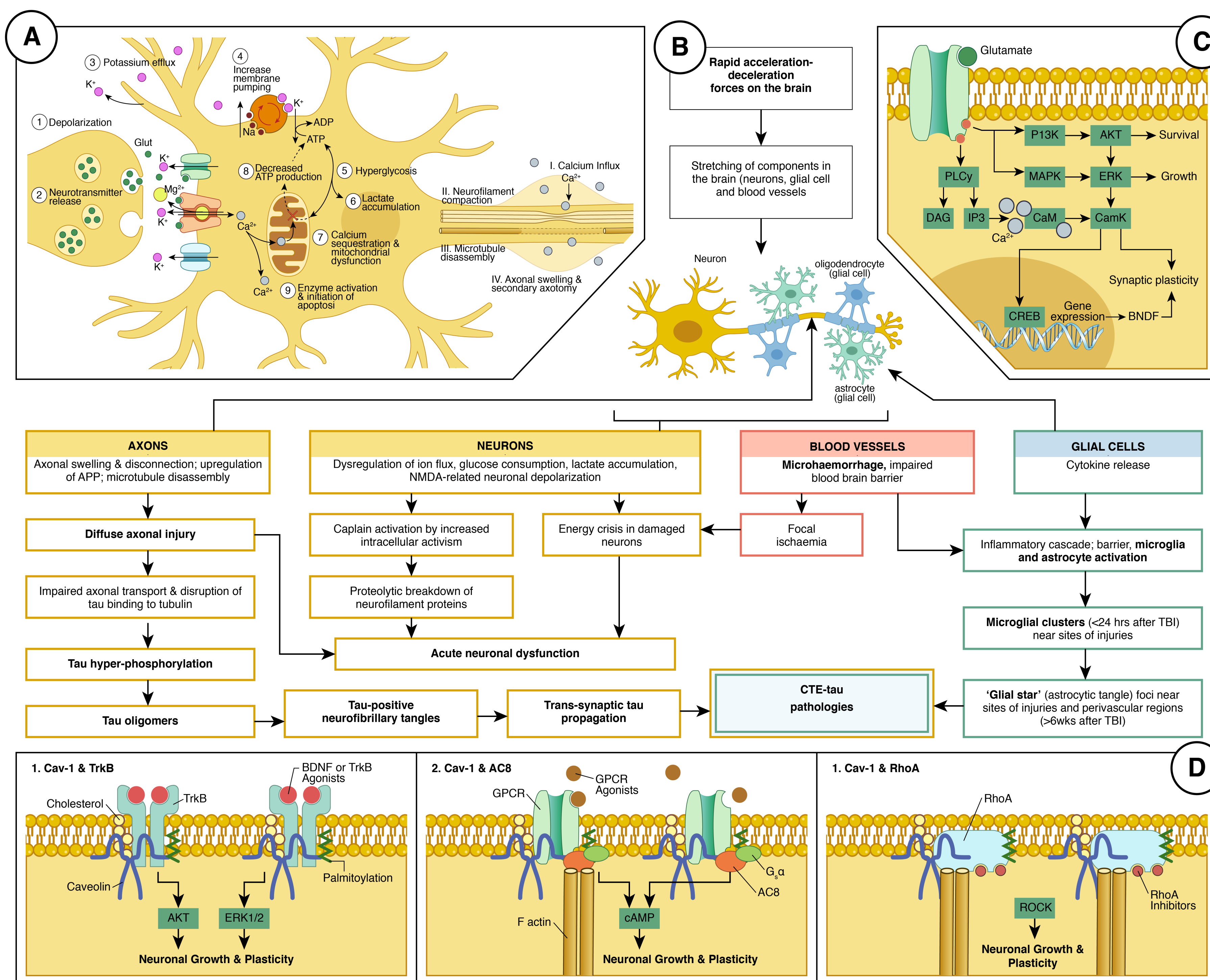


Figure 1: (A) Mechanism of metabolic cascade of mTBI, adapted from (Giza, et al, 2014) (B) Proposed schematic of the cascade events triggered by acute TBIs and its possible mechanistic links with the development of CTE pathology, cited from (Blennow, et al, 2012), (C) NMDR secondary effector and the transcription activation of BDNF, adapted from (Sta Maria, et al, 2019). (D) Proposed mechanism of neuronal growth at the tip of an injured axon BDNF, and TrkB are the main initiator, cited from (Pearn, et al, 2017). Figure courtesy of Audrey Bell

Conclusions

- The cognitive neuroscience shows an increased emphasis on understanding the malleability of distributed neural systems, Therefore, understanding the different consequences of TBI requires an understanding of the broader consequences of focal injury (frontal or otherwise) on neural networks, including the natural plasticity of those systems.
- Neuroplasticity researches are still in its early steps of the discovery of the complex and multilevel pathways, this process will take some time. We recommend more research that focus more on the recovery rather than the initial injury to the neuronal cortex.
- Studies on human brain cortex is limited because of the increased risk of adverse outcome due to intervention. We recommend conducting research on animals (specially primates) and use the animal model toward understanding the process of regeneration in the nervous system as well the ability to reverse the outcome of TBI.
- More research in humans can be conducted on the main player of the TBI such as BDNF, and NMDAR.

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Disclosure

The authors do not have any conflicts of interest to report. References, full search terms, and acronym lists available upon request.