

Jacobs Journal of Community Medicine

Review Article

Brain Plasticity and the Enhancement of Brain Functioning

Robert Perna*¹

¹Texas Institute of Rehabilitation Research, Houston, Texas

*Corresponding author: Dr. Robert Perna, 1Texas Institute of Rehabilitation Research, Houston, Texas Email: dr.perna@juno.com

Received: 08-23-2015

Accepted: 10-06-2015

Published: 11-23-2015

Copyright: © 2015 Perna

Abstract

Neuroscience methods and knowledge base are evolving quickly and there is a growing understanding of brain plasticity. There is now growing empirical evidence for specific and disparate mechanisms of plasticity. There appears great promise for future brain rehabilitation treatments, however, other than stimulation and forced use paradigms there is limited understanding of how to drive plasticity mechanisms.

There's even some empirical research that demonstrates that glial cells are also responsive to stimulation and may help remodel synapse and promote motor and cognitive functioning. Glutamate, the main excitatory neurotransmitter, and catecholamines have been shown to play crucial roles in brain reorganization [1]. After brain injury Nerve growth factors (NGF) and various neurotropic factors have been reported to improve memory and motor functions and reduce dendritic atrophy in the remaining pyramidal neurons [2] and appear to play a role in many aspects of recovery and brain functioning. Many of these mechanisms are potential treatment targets to facilitate improved brain functioning. Given the quick evolution of neuroscience research methods, massive increase in cellular neuroscience knowledge, and the growing insights into the mind body reciprocal interactions, it is likely that modern translational science will promote a plethora of new treatment approaches. The gene and stem cell therapy science is evolving so fast it is difficult to imagine what will be conceivable treatments over the next few years. Moreover the Connectome project (where the brain is currently being mapped) and cutting edge imaging strategies such as diffuse tensor imaging and high definition fiber tracking may hold great promise for understanding white matter and the connections and integration of many brain systems. Translational science has shown that the bench to bedside evolution of potential treatments can take many years to translate from experimental findings to evidenced based clinical treatments. Despite this, new findings in fundamental, integrative and cognitive neuroscience are

already changing the therapeutic landscape for professionals treating neurological and psychiatric disorders. Though mass marketing promising brain change on use of consumer brain training products is often not based on empirical evidence, there is growing evidence for a variety of methods to improve brain functioning.

Brain plasticity was a term that originally and simply referred to the brain's ability to change and adapt as a result of experience. There are a plethora of brain plasticity mechanisms. Recent developmental research is allowing us a far more in-depth understanding of the effects of pre, peri and postnatal pathology and the evolution of any resultant symptoms throughout the lifespan. Developmental brain plasticity causes our personality and other brain systems to be shaped during our youth. Recent developments in the understanding of injury induced plasticity are helping to clarify early and late mechanisms of brain plasticity and more accurate time frames for brain injury recovery. The currently expanding body of knowledge is helping allow clinicians to devise treatments that maximize recovery potential. However despite the fact that we now know the brain is more malleable than ever before, it seems fair to say that the development and testing of clinical interventions lags behind the cutting edge neuroscience findings. There is growing evidence that focused stimulation to certain brain areas can enhance brain functioning. A prime example of this is the forced-use paradigm. This approach has led to considerable evidence supporting Constraint Induced Movement Therapy

and Constraint Induced Language Therapy. Forced use helps maximize the stimulation of adjacent and even more distal connected neurons to help recruit these neurons to resume the function of dead or damaged tissue (neurons or glial cells). At the cellular level, the brain's plasticity can be seen in individual neurons, including growth of axons, dendritic spines, and synapse [3].

Modern research has demonstrated that the brain continues to create new neural pathways and alter existing ones in order to adapt to new experiences. Functional imaging has allowed current researchers to collect evidence that physical and cognitive exercise, and even talk therapies (when performed appropriately and consistently) can change brain functioning. Brain injury rehabilitation research suggests that generally there is a dose response relationship between the amount of treatment and amount of recovery [4].

This is an important detail in that a considerable dose of exercise or stimulation may be needed to change the brain and improve functioning. The unfortunate, but likely reality may be that many individuals may not receive a large enough dose of brain injury rehabilitation to maximize their recovery. The demonstrated neuronal need for stimulation clearly supports the "Use it or lose it paradigm" and "use it and it gets better." Considerable empirical research shows that physical and cognitive activity and certainly the combination of both can improve certain cognitive skills and boost brain metabolism [5] and functioning.

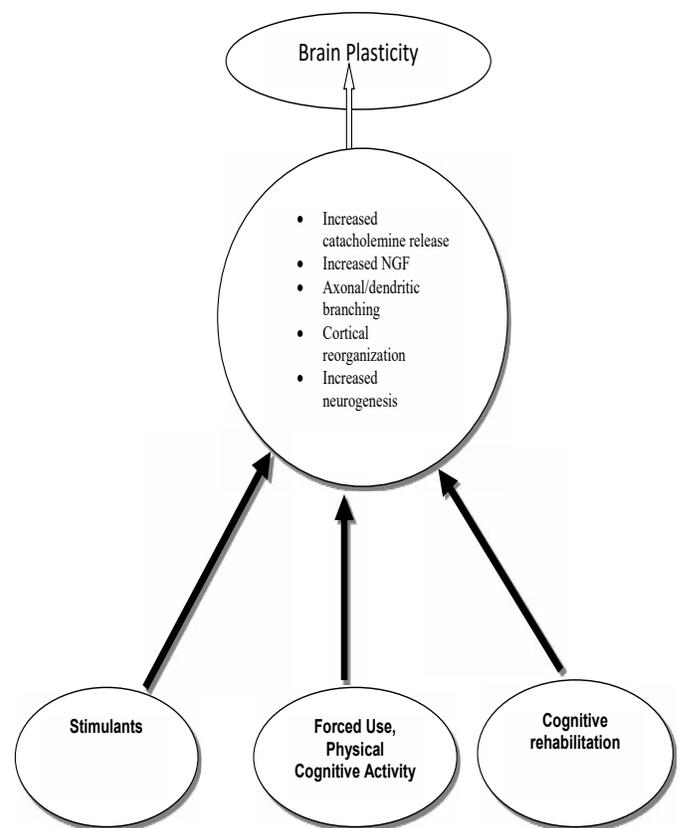
How Physical and Cognitive Activity Change the Brain

Several mechanisms are likely to be involved in brain plasticity [6]. Neural plasticity may also alter the function of the original neural substrate used to produce a behavior through neuronal sprouting and dendritic growth. Focused brain use and stimulation is thought to stimulate and enhance neural connections, unmasking latent neurons, and reducing surrounding or adjacent neuronal inhibition with reduced afferent input [7, 8, 9]. Some of the possible proposed mechanisms of activity-dependent modification of synaptic connections and reorganization of adult cortical areas are thought to involve long-term potentiation (which is the strengthening of a synapse) and long-term depression (which is the weakening of a connection or synapse), changes in dendritic spines, and other mechanisms by which information is stored in the mammalian central nervous system [10] and up and down regulation of neuronal connections. Synaptic plasticity in cortical horizontal connections has been proposed to underlie cortical map reorganization [11].

As neuroscience evolves it appears that more mechanisms are found that support neuroplasticity. There's even some empirical research that demonstrates that glial cells are also responsive to stimulation and may help remodel synapse and promote motor and cognitive functioning. Glutamate, the main

excitatory neurotransmitter, and catecholamines have been shown to play crucial roles in brain reorganization [1]. Nerve growth factors (NGF) and various neurotropic factors have been reported to improve memory and motor functions and reduce dendritic atrophy in the remaining pyramidal neurons [2] and appear to play a role in many aspects of recovery and brain functioning. Many of these mechanisms are potential treatment targets to facilitate improved brain functioning. It is important to understand that improved understanding of brain plasticity holds promise for effective treatment of diverse symptoms (physical, cognitive, psychological). Active cognitive reserve theory suggests that after injury, stimulation may help the brain differentially recruit neurons to perform tasks. This is similar to neural compensation, which is when residual neural substrate(s) are used to help perform impaired functions, as may occur at some point during recovery from aphasia [12] or when ipsilateral cortex works to help resolve hemiplegia. Figure 1(see below) provides a basic overview of the general schematic of the concepts discussed in this editorial.

Figure 1. Basic Diagram of Brain Plasticity Variables.



This review is far too brief to discuss all the latest findings in neuroplasticity research, but hopefully it is clear that the neurosciences and our understanding of neuroplasticity is evolving quickly. The increasingly advanced knowledge continues to shed light on the considerable malleability of the human brain.

Brain plasticity research suggests many current treatments have great potential in the appropriate dose.

Moreover, there may be great promise for numerous new brain injury rehabilitation treatments evolving over the next decade.

Unfortunately, many potential treatments for brain disorders are early in the translation process.

References

1. Garraghty PE, Muja N. NMDA receptors and plasticity in adult primate somatosensory cortex. *J Comp Neurol*. 1996, 367(2): 319–326.
2. Kolb B, Cote S, Ribeiro da Silva A, Cuello A C. Nerve growth factor treatment prevents dendritic atrophy and promotes recovery of function after cortical injury. *Neuro science*. 1997, 76(4): 1139–1151.
3. Duffae, H. Brain Plasticity: From pathophysiological mechanisms of therapeutic Application. *Journal of Clinical Neuroscience*. 2001, 13(9): 885-897.
4. Lohse K K, Lang C E, Boyd L A. Is more better? Using meat-data to explore dose-response relationship in stroke rehabilitation. *Stroke*. 2014, 45(7): 2053-2058.
5. Shah G, Verdile H, Sohrabi A, Campbell E, Putland C et al. combination of physical activity and computerized brain training improves verbal memory and increases cerebral glucose metabolism in the elderly. *Translational Psychiatry*. 2014, 4: e487.
6. Johansson BB. Brain Plasticity and Stroke Rehabilitation: The Willis Lecture. *Stroke*. 2000, 31(1): 223-230.
7. Tinazzi M, Zanette G, Volpato D, Testoni R, Bonato C et al. Neurophysiological evidence of neuroplasticity at multiple levels of the somatosensory system in patients with carpal tunnel syndrome. *Brain*. 1998, 121(Pt 9): 1785–1794.
8. Urasaki E, Genmoto T, Wada S, Yokota A, Akamatsu N. Dynamic changes in area 1 somatosensory cortex during transient sensory deprivation: a preliminary study. *Journal of Clinical Neurophysiology*. 2002, 19(3): 219–231.
9. Ziemann U, Hallett M, Cohen LG. Mechanisms of deafferentation-induced plasticity in human motor cortex. *Journal of Neuroscience*. 1998, 18(17): 7000–7007.
10. Bear MF, Malenka RC. Synaptic plasticity: LTP and LTD. *Current Opinions in Neurobiology*. 1994, 4: 389–399.
11. Hess G, Aizenman CD, Donoghue JP. Conditions for the induction of long-term potentiation in Layer II/III horizontal connections of the rat motor cortex. *Journal Neurophysiology*. 1996, 75(5): 1765–1777.
12. Saur D, Lange R, Baumgaertner A, Schraknepper V, Willmes K et al. Dynamics of language reorganization after stroke. *Brain*. 2006, 129(Pt 6):1371–1384.