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Neuroplasticity of the Brain: Neurophysiological Perspective

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ABSTRACT

The brain is the most important organ of human body. It is dynamic in terms of functional and structural aspects. Each human function is determined by brain. It was thought in the beginning that brain or its tissues do not regenerate once they are damaged. The recent research in neurosciences has shown that brain tissues have the ability to regenerate themselves. This phenomenon is known as neuronal plasticity. There have been multiple mechanisms which explain this phenomenon. Post injury experiences, neurochemical and neurophysiological aspects are some of the underlying mechanisms. The current paper attempts to explain the neurophysiological aspects of neuroplasticity. It has significant therapeutic implications.

Keywords: *Neuroplasticity, Neurophysiology*

Neuroplasticity, also known as brain plasticity describes lasting change to the brain throughout an individual's life course. The term become popular after the work of Livingston in 1966 which showed many aspects of the brain remain changeable (or "plastic") even into adulthood (Rakic, 2002). This work challenged the previous scientific consensus that the brain develops during a critical period in early childhood, then remains relatively unchangeable (or "static") afterward (Pascual-Leone,Amedi, Fregni & Merabet,2005).

Neuroplastic change can occur at small scales, such as physical changes to individual neurons, or at whole-brain scales, such as cortical remapping in response to injury; however cortical remapping only occurs during a certain time period meaning that if a child were injured and it resulted in brain damage then cortical remapping would most likely occur, however if an adult was injured and it resulted in brain damage, then cortical remapping would not occur since the brain has made the majority of its connections (Pascual-Leone et al, 2011). Behavior, environmental stimuli, thought, and emotions may also cause neuroplastic change, which has significant implications for healthy development, learning, memory, and recovery from brain

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damage. Synaptic plasticity refers to changes in how neurons connect to each other, from non-synaptic plasticity, which refers to changes in the neurons themselves.

The concept of neuronal plasticity has been extensively used to with the enduing change associated with brain's capacity to be shaped or moldered by experience, the capacity to learn and remember and the ability to reorganize and recover after injury. Synapse formation during development is thought to be depended upon both the genetic and environmental influences. This initial stage of synapse formation occurs independent of experience. Experience dependent refinement occurs during the critical period. A common view held that, after this critical period of fine turning, the resulting configuration of synaptic connections remained unaltered throughout the lifetime of the animal. However, research over the last two decades has provided evidence for tetensive experience dependent plasticity in the adult brain (Gilbert, 1998). On the early demonstrations of adult cortical plasticity in the primate somatosensory cortex has been carried out by Merzenich and co-workers in 1978. A wide range of neuronal response reconstruction studies conducted in animals and humans have shown that cortex reorganizes its effective local connections and responses following peripheral, central alterations of inputs and in response to behavior (Buonomano & Merzenich, 1998; Kasss, 1991). Subsequent studies have shown that environmental enrichment, standard learning tasks help to improve brain functions and promote brain plasticity in adults (Stewart & Rusakov, 1995; Turner & Greenough, 1985).

Understanding the neuronal basis of neural plasticity has been the goal of considerable research, the "Hebbian" theory attempts to provide much insight in to the mechanisms of neuronal plasticity. It postulates that the temporal correlation of pre and postsynaptic coactivity leads to synaptic strengthening, whereas lack of this correlation results in synaptic weakening (Hebb, 1949). After a decade, theories of structural plasticity changes in the pre-synaptic and post synaptic elements. The stage has been now set to accept the concept of neurogenesis in the adult neural system. Neuronal plasticity may be associated with

i) Activity dependent modification of the efficacy of existing synapses leading to long- term potentiating (LTP) or long – term depression (LTD),
ii) Morphological changes leading to enhanced dendrite branching and axon collaterals and
iii) Synaptogenesis leading generation of new synaptic contacts (Bear & Malenka,1994; Boroojerdi, Ziemann, Chen, Butefisch, & Cohen, 2001; Klintsova & Greenough, 1999; Zit, & Svoboda, 2002) and
iv) Neurogenesis leading to the incorporation of new neurons to influence subsequent behaviours.

It is widely believed that LTP holds the key for understanding how memories are formed in the brain. LTP is a long lasting enhancement of synaptic effectiveness that follows a brief, high frequency electrical stimulation in the hippocampus where it was first documented by Bliss and Lomo in 2003 and in other brain regions such as neocortex, brain stem and, amygdala . Recent

evidences suggest that induction of LTP may require, in addition to postsynaptic calcium entry, activation of metabotropic glutamate receptors and the generation.-if diffusible intercellular messengers. A new form of synaptic plasticity homosynaptic long- term depression (LTD) has also recently been documented, which like LTP requires Ca^{2+} entry through NMDA receptors. Studies suggest that LTD is a reversal of LTP, and vice versa, and the mechanisms of LTP and LTD may converge at levels of specific phcsphoproteins.

Some of the most detailed studies of experience dependent plasticity have been performed in the rodent barrel cortex. Manipulations of the sensory inputs such as, clipping the whiskers can change the receptive fields of cortical neurons. Robust experience dependent plasticity has been observed within 24 hrs of whisker clipping[13]. These models demonstrate the cellular basis of this experience dependent plasticity due to modifications of existing synapses such as long-term potentiation and depression.

There was little evidence for rapid synaptogenesis in the adult brain in response to sensory stimulation. Recent electron microscopic studies provide evidence that the adult cortex generates new synapses in response to sensory activity within 24 hrs of sensory stimulation. Knott et al[4] studied the effects of stimulating a single whisker on synapses in the barrel cortex of mice. Remarkably, after this relatively brief period of stimulation, they observed a significant increase in both synapse and spine density specifically in the barrel corresponding to the stimulated whisker. Their study suggests that new synapses form predominantly on spines, either through addition of a new synapse on the preexisting spine or by the growth of new spines. Whisker stimulation resulted in a transient increase in excitatory synapses; however, there was an absolute increase in total inhibitory synaptic density and a shift of inhibitory synapses from shaft to spines. This enhanced inhibitory synaptic density could account for the powerful homeostatic mechanisms that keep the neuronal activity in a reasonable operating range, may be to preserve the network stability. The observed increase in inhibition may act to reduce the excitation of layer IV neurons in response to sensory stimuli.

Activity dependent synaptogenesis is thought to be mediated by gene expression and protein translation. In adult animals, whisker stimulation causes upregulation of immediate early-genes and experience -dependent plasticity paradigms cause CRE -mediated gene expression such as brain derived neurotrophic factor (BDNF). Activity dependent up regulation of BDNF leads to spine growth and recruitment of new synapses and proposed to enhance dendritic morphogenesis. Enhanced synaptic density and changes in expression of for protein associated with a complex motor learning task has been reported. It is generally assumed that the global nature of the motor learning task may demand the integration of a variety of inputs. Similarly it is assumed that Fos proteins play a role in the memory process. Disrupting the functions of cAMP element response binding (CREB) protein which induce the transcription of c-fos gene has been reported to cause learning impairment in drosophila. Fos gene may act to promote the

transcription of various proteins (necessary for changes in neuronal structure and function) such as nerve growth factor, deregulate cytoskeletal proteins to promote morphological transformation. It is most likely involved in cellular process associated with cell function that may be up regulated, during periods of plastic change. Animals lacking functional c-fos gene are impaired on some learning task and may be attributed to a gross behavioural impairment rather than specific learning deficit.

The molecular mechanisms involved in neuronal plasticity have been a topic of intensive research in recent years. The gene knock out technology further enhanced the understanding of the role of genes and proteins in synaptic plasticity. The occurrence of experience dependent, CaMK II dependent LTP phenomenon has been reported in the hippocampus and in many cortical areas. In contrast, CaMK-ll gene knockout mice fails to elicit both the behavioural plasticity and generate the potentiating phenomenon. Physiological examination of mouse knockouts has demonstrated roles for CaMK II and CREB in activity dependent barrel cortex plasticity.

There is an increasing evidence that neurotrophins (NTs) are involved in processes of neuronal plasticity besides their well established action in regulating the survival, differentiation and maintenance of functions of specific populations of neurons. The NTs and their presynaptic Trk receptor activation contribute to the activity dependent plasticity by various means; through modifications of pre synaptic machinery locally, regulation of synaptic protein levels and transcriptional regulations. There is increased evidence .of neurotrophins and their receptor signaling in the production of LTP and activity -dependent plasticity associated with learning. BDNF expression and TrkB signaling has been associated with dendritic and synaptic restructuring by means of regulating spine dynamics, functional maturation of presynaptic terminals, dendritic growth, triggering AMPA receptor proteins and in activity dependent conversion of silent synapses into functional ones. The cascade of cellular events associated with the changes in dendritic structure is a very complex process involving synthesis, targeting and transport of essential proteins. A combination of local regulation of trophic factor receptor activation and protein synthesis could be the principle mechanisms leading to the structural plasticity.

Recent advances in imaging technology permits the real time observation of dendritic opines, and therefore can detect the dynamic structural changes associated with synaptic plasticity. Sensory deprivation by unilateral whisker trimming for a short duration decreased the. Motility of dendritic spines in deprived regions and degradation in the tuning of layer II and III receptive fields. These studies suggest that sensory experience drives structural plasticity in dendrites, which may underlie reorganization of neural circuits during plasticity. Although short term changes in synaptic strength are attributed to changes in existing synapses, structural changes represent one of the key feature of the long-term memory process, either through formation or

elimination of synapses. Support for structural dynamics including synapse formation and elimination has been demonstrated following sensory experience in adult barrel cortex, learning and in other plasticity evoking paradigms.

Recent research has shown the existence of neural stem cells in the sub ventricular zone, olfactory bulb and in the dendate gyrus of hippocampus which give rise to new neurons and glial cells. It is postulated that the new cells undergo differentiation to be incorporated into the existing functional network and allow a strategic increase in network complexity may be to accommodate the continued modulation of input pathways. Thus, it appears that behavior can induce structural changes and changes in structure can subsequently change or at least affect subsequent behaviors.

Activity dependent synaptic plasticity has been implicated in a variety of physiologically and behaviorally induced changes in neuronal organization both during development and adulthood. Although extensive information is available about the mechanisms of synaptic plasticity in the adult mammalian brain, a coherent understanding has yet to be emerged.

CONCLUSION

Experimental and clinical work has shown that brain tissues have the ability to regenerate after the injury. Post-injury experiences in the form of cognitive and behavioral stimulation to the brain help damaged tissues or its adjacent parts to take over the function of the damaged portion. This dynamic property of the brain has significant therapeutic and educational implications.

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Conflict of Interests

The author declared no conflict of interests.

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