International Journal Of Public Mental Health And Neurosciences
ISSN: 2394-4668
(An Official publication of Sarvasumana Association)

Neuroplasticity: Structural and Functional How the adult human brain rewires itself

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***Abstract*: This paper introduces the concept of neuroplasticity. It tries to show the evolution of the concept from its inception in psychology to a completely different meaning it has been able to acquire in modern neuroscience after the discovery by Paul-Bach-y-Rita that damaged nerve cells can regenerate again in humans. It shows the different types of neuroplasticity. Neuroplasticity can be broadly divided into two parts*: structural neuroplasticity* and *functional neuroplasticity*. In structural neuroplasticity, there are, based on experience of the subject, actual structural changes in the organization of neural networks of the brain. It includes neurogenesis, synaptogenesis and changes in the strength of synapses. In *neurogenesis* new neurons are formed. It takes place throughout our lives. Studies suggest its usefulness in maintenance of memory and learning of new tasks. *Synaptogenesis* is the formation of new synapses. It too takes place throughout the lifetime of individuals. Studies suggest it is majorly associated with learning. *Changes in strength of synapse* is the change in the frequency of firing in the pre-synaptic neuron. According to studies, the strength of synapses alter according to increase in dexterity in acquired skills like learning musical instruments, etc. The second type of neuroplasticity is called *functional neuroplasticity*. In functional neuroplasticity, different regions of the brain that perform different functions are modified according to the needs of the brain and its relation to the environment. There are four types of functional neuroplasticity: *Homologous area adaptation*: The substitution of a particular cognitive process by a homologous region in the opposite hemisphere. It occurs when one particular part of one hemisphere of the cerebrum is injured. *Map expansion*: It is the enlargement of a functional brain region on the basis of performance. This type of neuroplasticity, entails the flexibility of local brain regions that are dedicated to performing one type of function or storing a particular form of information. *Cross-modal reassignment*: It occurs when structures previously devoted to processing a particular kind of sensory input now accepts input from a new sensory method. *Compensatory masquerade*: It is a novel allocation of a particular cognitive process to perform a task. Most people, to a greater or lesser extent, have an intuitive sense of direction and distance that they employ for navigation. However, a person who suffers some form of brain trauma and impaired spatial sense will resort to another strategy for spatial navigation, such as memorizing landmarks.**

This paper deals with all the above mentioned types of neuroplasticity by providing pieces of evidence of each type of neuroplasticity. It is of relevance in modern day medical practices especially those that deal with neurodegenerative disease and mental health conditions that come as a result of neuronal disturbances.

***Keywords*: Neuroscience, Neuroplasticity, Types of Neuroplasticity, History of Neuroplasticity, Psychology, Neuroscience, Neuronal Adaptations,**

Neuroplasticity: Historical Overview

The word plasticity with reference to the human brain was first used by psychologist William James in the year 1890. He used the term to explain functional aspects of neuroplasticity in formation of new habits. [1] Polish neuroscientist Jerzy Konorski was the first to define theterm 'neuroplasticity' in 1948. According to Konorsky neurons which have been activated by closeness of an active neuronal circuit, alter and incorporate themselves into that circuit[2]. Paul Bach-y-Rita in the year 2003 demonstrated neuroplasticity on actual cases. He showed that healthy regions of the brain can take over the functions of injured parts of the brain.[3]

Neuroplasticity: Definition

Neuroplasticity can be defined as brain's ability to change, remodel and reorganize for purpose of better ability to adapt to new situations. Neural networks

arenot fixed, but occurring and disappearing dynamically throughout our wholelife, depending on experiences. While we repeatedly practice one activity or another such as a sequence of movements or a mathematical problem, neuronal circuits are being formed, leading to better ability to perform the practiced task withless waste of energy. Once we stop practicing a certain activity, the brain will redirect these neuronal circuits by a 'use it or lose it'principle. According to the 'use it or lose it' principle, the neural circuits that are used improve over a period of time. This shows in the increase of dexterity or skill. If certain neural circuits are not used, they will be lost. Neuroplasticity leads to many different occurrences, such as habituation, sensitization to a certain position, medication tolerance and recovery following brain injury.[4]

According to cognitive scientist Norman Doidge, neuroplasticity is the ability of the brain to compensate cognitive components in spite of degeneration of certain components of the brain. [5] This, as we shall see later falls under functional neuroplasticity. Neuroplasticity is the flexibility of the neural system. It is the ability of the brain to adapt to the lack or deterioration of sense and the capability of the neural system to modify itself through changing shape and function. Studies have revealed that neuroplasticity does not end in childhood. They have proven that it continues till the end of life. "It not limited to the neural system and covers the cognitive system as well. "In the field of cognitive science, neuroplasticity is defined as the ability to change old thoughts according to new conditions and the individuals' differences in using various styles of cognitive regulation inducing several social, emotional and cognitive outcomes."[6]

Neuroplasticity: Types

Neuroplasticity is classified on whether there are actual structural changes occurring in the human brain or not. There are two types of neuroplasticity based on the above mentioned classification. The first is called structural neuroplasticity and the second is called functional neuroplasticity.[4]

Structural Neuroplasticity:

The phenomenon where there are actual changes in the brain, for example, formation of new neurons, formation of new synapses and alterations in the strength of synapses in the brain is called structural plasticity. Sometimes neurons migrate to the brain, differentiate extend axons and express neuronal marker proteins.[7] Instructural neuroplasticity, based on experience of the subject, there are structural changes in the organization of neural networks of the brain. Structural neuroplasticity is of three types.

a) ***Neurogenesis***: When new neurons ore formed in the adult human brain, new neural networks are formed or previously existing neural networks are modified. This brings about a change in the overall organization of the brain. Studies suggest that neurogenesis and hence alteration in the orgainsation of the brain takes place throughout the human life.[8]Listed below are Evidences of studies that show that neurogenesis occurs throughout our lives.

   Evidence 1:

Study shows adult neurogenesis can be observed in the newly born cells of the subventricular zone migrate and differentiate into neurons within the olfactory bulb. The newly generated neuronal cells in the subventricular zone migrate over a long distance to the olfactory bulbs through the rostral migratory stream and differentiate into inter neurons at their final destination. These newly generated neuronal cells in the olfactory bulbs establish synaptic contacts and functional connections with neighboring cells.[9]

Evidence 2:

Study shows that the newly formed cells integrate into the granular layer of the sub granular zone of the dentate gyrus and start to extend their axons and dendrites into their target areas.[9]

Evidence 3:

Study shows neurogenesis in the neocortex, the piriform cortex, the subcallosal zone, the amygdala, the striatum, and the substantia nigra. Neurogenesis in these areas might be induced under non-physiological condition.[9]

Evidence 4:

A study shows that sustained visual and auditory environment enrichment could induce neurogenesis in adult hippocampus. [10]

Evidence 5:

A study on chronic depression patients shows that sustained anti-depressant administration induce neurogenesis in adult human brains. [11]

b) ***Synaptogenesis***:A synapse is the junction between two nerve cells. It consists of a presynaptic neuron ending, postsynaptic neuron ending and a synaptic cleft. The transmission of information from one neuron to another goes through synapses. The process of formation of new synapses is called synaptogenesis. It is a structural change at a subcellular level that takes place in response to synaptic activity, and provides a mechanism for processing and incorporating new information that can be used to make the appropriate, future adaptive response.[12] The formation of new synapses late in life make the human brain dynamic.

Evidence 1:

Molecular and cellular studies have demonstrated actions of antidepressant treatment on the expression of neurotrophic factors, particularly brain-derived neurotrophic factor, inlimbic structures of the brain. These changes in neurotrophic factor expression and function result in structural alterations, including regulation of synaptogenesis, dendrite length and spine density inhippocampus and prefrontal cortex. [12]

Evidence 2:

Study shows that in adults there is cholesterol induced synaptogenesis. Cholesterol enhances directly presynaptic differentiation and that it is essential for continuous synaptogenesis.[13]

Evidence 3:

Long-term memory (LTM) formation has been linked with functional strengthening of existing synapses and other processes including de novo synaptogenesis. (Radwanska et al., 2011)

Evidence 4:

A study on temporal lobe epilepsy shows that there is synaptogenesis after epileptic attack in order to compensate for the damages that might have taken place due to the epileptic attack. [14][15]

Evidence 5:

A study on hippocampal epilepsy suggests synaptic reorganization and synaptogenesis in the hippocampal region of the brain in adults after apileptic attack. [16]

c) ***Change in strength of synapses***: A synapse is the junction between presynaptic and post synaptic neurons. The space between them is called synaptic cleft. The strength of a synapse depends upon 'firing'(opening of Ca^{++} ion channels and modulation of neurotransmitters in the synaptic cleft) of the presynaptic neuron. It can be widely seen that the strength of synapses of neural networks associated with particular tasks alter with the alteration of the intensity with which that task is done. The process of strengthening of weakening of synapses is reversible and takes place throughout our lives. [17]

Evidence 1:

Prefrontal cortical working memory functions depend on pyramidal cell networks that inter connecton dendritic spines. Recent research has revealed that the strength of pre frontal cortex network connections can be rapidly and reversibly increased or decreased by molecular signaling events within slender, elongated spines: a process termed as Dynamic Network Connectivity.[18]

Evidence 2:

A study shows that cannabinoids modulate the strength and plasticity of pyramidal neurons of pre frontal cortex. [19]

Evidence 3:

A study shows that beta catenin regulates excitatory post synaptic strength at hippocampal synapses. [20]

Evidence 4:

Long-term memory (LTM) formation has been linked with functional strengthening of existing synapses and other processes including de novo synaptogenesis.[21]

Evidence 5:

Study shows motor skill training induces coordinated strengthening between neighbouring synapses. Motor skills are acquired through repetitive practice and persist without additional reinforcement. Purkinje cells are the sole efferent neurons of the cerebellar cortex. They possess elaborate dendritic arbors bearing numerous dendritic spines. These small protrusions represent the postsynaptic sites of most excitatory synapses. They exhibit

experience-dependent remodeling, i.e., strengthening and weakening of synapses. [22]

Functional Neuroplasticity:

In certain neuroplasticity, there is no actual dynamic reorganization of the structure of the brain. However, because of externally inflicted injuries certain portion of the brains might have to take up the task of certain other portions of the brain to keep the overall behavioral and cognitive functionality of the individual intact. This type of neuroplasticity is called functional neuroplasticity.[23] It is of four types.

a) ***Homologous Area Adaptation***: It appears most active during an early critical stage of human development. It underlies the notion that damage to a particular brain region and its cognitive operation(s) can be compensated for by shifting the individual (or set of) operation(s) to other brain areas that do not include the affected module.[23] The function is usually shifted to another module in the homologous region of the opposite hemisphere. Homologous area adaptation can be said to be the assignment of the task of a damaged region in one hemisphere to the homologous region in the other hemisphere of the brain.

Evidence 1:

An adolescent who had incurred a severe right parietal llobe brain injury as a young child was studied. Despite the severity and location of the injury, the evaluation showed that the subject had developed normal visuospatial skills. The inference is that at the time of the injury, the left parietal region assumed some of the responsibilities of the functions normally stored in the right parietal lobe. It is to be noted that this process may sometimes come at a cost of full functionality of the contralateral region that takes up the function.(Levin et al., 1996) [23]

Evidence 2:

Study shows that when there is damage of the left inferior frontal gyrus, there is adaptive plasticity in the right homologous areaduring speech production. As a result speech is not impaired. [24]

Evidence 3:

Studies using single photon emission computed tomography (SPECT) or positron emission tomography (PET) show increased glucose metabolism or regional cerebral blood flow in the right hemisphere as well as in undamaged portions of the language network in the left hemisphere in recovering aphasic individuals. [25]

Evidence 4:

After specific rehabilitative treatment patients showed varied patterns of fMRI changes

Related to improvement of upper limb motor function. Neurophysiological and neuroimaging studies suggest that neuroplasticity happens in the sensorimotor cortex of the homologous area of the

opposite hemisphere of affected hemisphere with task-specific training. [26]

Evidence 5:

In a study on working memory with right frontal lobe damage, it was seen that fully functional working memory was present with left frontal lobe activation in fMRI. [27]

b) ***Cross Modal Reassignment***: It involves the introduction of new inputs into a representational brain region that has been deprived of its main inputs. If a section in some pathway of signal processing in the brain has been damaged, the same signal reaches its target through another path bypassing the damaged region. This is called cross modal reassignment. [23]

Evidence 1:

Positron emission tomography and functional magnetic resonance imaging studies of tactile discrimination ability have shown that persons who became blind early in childhood, but tested as adults, have somato sensory input redirected into area V1 of the occipital cortex, whereas normal control participants do not show evidence of any V1 activation during the same task (Sadato et al., 1996)[23]

Evidence 2:

Study finds age-related hearing loss increases cross-modal reorganisation. Recent electrophysiological studies have provided evidence that changes in multisensory processing inauditory cortex cannot only be observed following extensive hearing loss, but also in moderatelyhearing-impaired subjects. [28]

Evidence 3:

Study shows that visual and auditory speech perception after cochlear implantation makes use of cross modal plasticity. [29]

Evidence 4:

By using MRI whole brain voxel-based morphometry, a study shows that the visual pathways of the blind are atrophied. Their visual cortex however can be recruited in tasks involving the somatosensory system. (Ptito, 2006)

Evidence 5:

There is neurophysiological evidence of short-term cross-modal plasticity functional plasticity in the human primary somatosensory cortex following acute lesion of the anterior lateral spinal cord. [30]

c) ***Map Expansion***: demonstrates the flexibility of brain regions devoted to a particular kind of knowledge or cognitive operation. Recent work has indicated that the size of cortical maps devoted to a particular information processing function may enlarge with skilled practice or frequent exposure to a stimulus. This type of neuroplasticity is known as map expansion. [23]

Evidence 1:

Study shows that the cortical regions of temporal lobe of a person learning musical instruments increase in size. [31]

Evidence 2:

Study shows subjects learning driving at a later period of their lives have developed temporal and prefrontal cortices. [32]

Evidence 3:

If there is damage of the left inferior frontal gyrus, there is adaptive plasticity and map expansion in the right homologous area during speech production. As a result speech is not impaired. (Hartwigsen et al., 2013)

Evidence 4:

After specific rehabilitative treatment patients showed varied patterns of fMRI changes

related to improvement of upper limb motor function. Neurophysiological and neuroimaging studies suggest that neuroplasticity happens in the sensorimotor cortex of the homologous area of the opposite hemisphere of affected hemisphere with task-specific training. There is map expansion in this region so as not to affect the usual cognitive tasks attributed to these regions. (Kiper et al., 2016)

Evidence 5:

Map expansion and transference of language functions to homologue areas in the contralateral hemisphere are thought to be two main processes sustaining speech in recovery from acquired childhood aphasia patients.[33]

d) ***Compensatory Masquerade***: It means that the novel use of an established, but intact, cognitive process to perform a task previously dependent on an impaired cognitive process has occurred.[23]In other words a cognitive task is performed using different functional regions of the brain which was otherwise performed by a damaged portion of the brain.[23]

Evidence 1:

A study shows that people who have traumatic brain injuries affecting their sense of direction, develop the ability to remember landmarks so that ultimately their sense of direction is not lost. [23]

Evidence 2:

Using facial affect recognition (FAR) tests and functional magnetic resonance imaging tasks for FAR a study was conducted on autism spectrum disorder patients. After training it was seen that the amygdala, fusiform gyrus and other regions of the social brain were activated bilaterally. This can be seen as an example of training induced compensatory masquerade. [34]

Evidence 3:

Damage to certain left hemisphere regions leads to reading impairments, at least acutely, though some individuals eventually recover reading.A study (Representational Similarity Analysis Approach) on recovery of reading after damage shows reorganization of functions of the damaged brains by compensatory masquerade. [35]

Evidence 4:

Study shows that when there is damage of the left inferior frontal gyrus, there is

adaptive plasticity in the right homologous area during speech production. As a result speech is not impaired. This can be said to be both homologous area adaptation and compensatory masquerade. (Hartwigsen et al., 2013)

Evidence 5:

After specific rehabilitative treatment patients showed varied patterns of fMRI changes

related to improvement of upper limb motor function. Neurophysiological and neuroimaging studies suggest that neuroplasticity happens in the sensorimotor cortex of the homologous area of the opposite hemisphere of affected hemisphere with task-specific training. (Kiper et al., 2016)

Discussion:

Neuroplasticity is the brains ability to change with respect to environmental changes. Previously it was believed that neuroplasticity occurred only in development of the embryo to adolescent periods. It was believed that neurogenesis, synaptogenesis and alteration of strength of synapses (the three types of structural plasticity) stopped after the fully grown adult was formed. Later (Bach-y-Rita & W. Kercel, 2003) it was found out that these processes take place in fully grown adult brains also. Neuroplasticity is no understood as the brains ability to rewire itself according to internal or external needs of the whole body and; it takes place throughout the human life. It has also been found that neuroplasticity occurs in neurodegenerative diseases like Alzheimer's and Parkinson's disease so as to minimize if not resist the impact of neurodegeneration. Studies show that neurogenesis, though, in a low rate takes place in patients affected by Alzheimer's and Parkinson's disease. (Colangelo et al., 2019)

Based on the study of structural components of the brain neuroplasticity is divided into three types under the heading of ***structural neuroplasticity***. These three types of structural neuroplasticity are a) ***neurogenesis***: When new neurons ore formed in the adult human brain, new neural networks are formed or previously existing neural networks are modified. Often neurons formed in one part of the brain migrate to other parts of the brain. These bring about a change in the overall organization of the brain. It occurs throughout our lives(Dokter& von Bohlen und Halbach, 2012) b) ***synaptogenesis***: A synapse is the junction between two nerve cells. It consists of a presynaptic neuron ending, postsynaptic neuron ending and a synaptic cleft. The transmission of information from one neuron to another goes through synapses. The process of formation of new synapses is called synaptogenesis. It is a structural change at a subcellular level that takes place in response to synaptic activity, and provides a mechanism for processing and incorporating new information that can be used to make the appropriate, future adaptive response. The formation of new synapses late in life make the human brain dynamic. (Duman & Li, 2012) c) ***Alteration of the strength*** of synapses: The strength of a synapse depends upon 'firing'(opening of Ca ++ ion channels and modulation of neurotransmitters in the synaptic cleft) of the presynaptic neuron. It can be widely seen that the strength of synapses of neural networks associated

with particular tasks alter with the alteration of the intensity with which that task is done. The process of strengthening of weakening of synapses is reversible and takes place throughout our lives. (Wilson, 1988) Neurogenesis in adults, Synaptogenesis in adults and alteration of strength of synapses in adults are together clubbed as structural plasticity. Structural plasticity takes place throughout human lives making the idea that the brain is not prone to change redundant. It also shows that our brains are much capable to attempt to resist neurodegenerative diseases by compensating for neurodegeneration through structural plasticity.

The second form of neuroplasticity is called ***functional neuroplasticity***. Functional neuroplasticity is the ability of the brain to respond to damage and trauma. If there is damage/trauma the brain takes up the task of allocating functions of the damaged/traumatized region of the brain to healthy parts of the brain so that the personality, cognitive abilities and behavior are preserved. Functional plasticity is of four types. a) ***Homologous area adaptation***: It underlies the notion that damage to a particular brain region and its cognitive operation(s) can be compensated for by shifting the individual (or set of) operation(s) to other brain areas that do not include the affected module.(Grafman, 2000) The function is usually shifted to another module in the homologous region of the opposite hemisphere. Homologous area adaptation can be said to be the assignment of the task of a damaged region in one hemisphere to the homologous region in the other hemisphere of the brain. b) ***Cross Modal Reassignment***: It involves the introduction of new inputs into a representational brain region that has been deprived of its main inputs. If a section in some pathway of signal processing in the brain has been damaged, the same signal reaches its target through another path bypassing the damaged region. This is called cross modal reassignment. (Grafman, 2000) c) ***Map Expansion***: demonstrates the flexibility of brain regions devoted to a particular kind of knowledge or cognitive operation. Recent work has indicated that the size of cortical maps devoted to a particular information processing function may enlarge with skilled practice or frequent exposure to a stimulus. This type of neuroplasticity is known as map expansion. (Grafman, 2000) d) ***Compensatory Masquerade***: It means that the novel use of an established, but intact, cognitive process to perform a task previously dependent on an impaired cognitive process has occurred.(Grafman, 2000) In other words a cognitive task is performed using different functional regions of the brain which was otherwise performed by a damaged portion of the brain.(Grafman, 2000) Homologous area adaptation, cross modal reassignment, map expansion and compensatory masquerade together constitute functional plasticity. As a result of trauma if any portion of the brain is affected, it is not necessary that the cognitive tasks performed by that portion of the brain will not be performed. Functional neuroplasticity tries to restore the personality, cognitive abilities and behavior (of the adult inflicted with damage or trauma to portions of the brain) intact.

It has been seen above that neuroplasticity takes place throughout our lives. It has also been seen that in spite of injuries to the brain, neuroplasticity keeps cognitive and behavioral functions of the personality

intact. Neuroplasticity also takes place in neurodegenerative diseases to minimize the effect of the disease on the personality. [8] Overall it can be said that neuroplasticity is a positive force inherent in humans which helps to adapt to environment changes and also to function in spite of brain injury or disease.

Acknowledgements:

This paper is a product of ongoing M.Phil research. Thanks to Swami Vivekananda Merit-cum-Means scholarship that was awarded to carry out this research.

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