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Neuroplasticity: A Fresh Beginning for Life

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***Abstract*—**Neuroplasticity or the flexibility of the neural system 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. Not only have studies revealed that neuroplasticity does not end in childhood, but also they have proven that it continues till the end of life and is not limited to the neural system and covers the cognitive system as well. In the field of cognition, 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. This paper attempts to discuss and define major theories and principles of neuroplasticity and elaborate on nature or nurture

***Keywords*—**Neuroplasticity, Cognitive plasticity, Plasticity theories, Plasticity mechanisms.

I. Introduction

THE term neuroplasticity derived from Greek work Plastikos meaning to form and refers to structural and functional changes in brain that are brought about by training and experience during injuries. Neuroplasticity mechanisms include neuron connections and synopsis plasticity for learning and memory. Information on plasticity routes are of crucial importance in the pathologic understanding and essential treatment [1]. The history of plasticity started with Rita-y Bach studies in spite of the common idea that brain's development stops on maturity. His studies in 1960's and 1970's showed that brain has the ability to change. This experiments included substituting visual sense in blind patients through images reflection using touch signals in 1969.the core of this experiment was based on the fact that impaired sense can be replaced by a healthy one [2]. Major types of neuroplasticity in early stage of life: Adaptive neuroplasticity refers to changes in neural circuitry that enhances a special skill with practice allowing the brain to adopt or compensate for injuries or changes in sensory output. Excessive neuroplasticity in a developing brain can result in disability through reorganization of maladaptive neural circuits that cause neurological disorders as partial seizures following mesial temporal sclerosis or focal dystonia. Impair neuroplasticity refers to situation in which genetic or acquired disorders disrupt molecular neuroplasticity pathways.Plasticity becomes Achilles' heel in situations like status epileptics when excitatory mechanism becomes over stimulated resulting in excite toxic neuronal damage [3]. Neuroplasticity can work in two directions; it is responsible for deleting old connections as frequently as it enables the creation of new ones. Through this process, called ―synaptic pruning, connections that are inefficient or infrequently used are allowed to fade away, while neurons that are highly routed with information will be preserved, strengthened, made even more synoptically dense. Closely tied in with the pruning process, then, is our ability to learn and to remember. While each neuron acts independently, learning new skills may require large collections of neurons to be active simultaneously to process neural information; the more neurons activated, the better we learn [4].

Neuroplasticity, heredity and environmentwhile genetics certainly play a role in establishing the brain's plasticity, the environment also exerts heavy influence in maintaining it. Take, for example, the newborn's brain, which every day is flooded with new information. When the infant body receives input through its many different sensory organs, neurons are responsible for sending that input back to the part of the brain best equipped to handle it – and this requires each neuron to ―know‖ something about the proper neural pathways through which to send its bits and pieces of information. To make this mental roadmap work, each neuron develops an axon to send information to other brain cells via electrical impulses, and also develops many dendrites that connect it to other neurons so that it can receive information from them. Each point of connection between two neurons is termed a ―synapse.‖our genes have, at birth, laid down the basic directions for neurons to follow along this roadmap, and have built its major ―highways‖ between the basic functional areas of the brain. Environmental influence then plays the key role in forging a much denser, more complex network of interconnections. These smaller avenues and side roads, always under construction, can make the transfer of information between neurons more efficient and rich with situation-specific detail. This is clearly evidenced by the rapid increase in synaptic density that can be seen in a normally developing human. Genetics form a neural framework that, at birth, starts each neuron off with roughly 2,500 connections. By age two or three, however, sensory stimulation and environmental experience have taken full advantage of the brain's plasticity; each neuron now boasts around 15,000 synapses. This number will have declined somewhat by the time we enter adulthood, as many of the more ineffective or rarely used connections – formed during the early years, when neuroplasticity is at its peak — are done away with [5].

There are several mechanisms for improving traumatic brain injuries during several weeks after trauma as follows:

A. Synaptic functional recovery
B. Increased synaptic efficacy
C. Increased sensitivity after neuron abscission

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D. Continuance of neurogenesis
E. Reorganizing and utilization of silent synapses
F. Rehabilitating and cultural regeneration
G. Treatment of brain lesions
H. Treatment of learning disorders
I. Action-dependent plasticity
J. Rehabilitating stroke patients
K. Sensory replacement
L. Imaginary organ [2].

II. Cognitive Plasticity

Cognitive plasticity has often been defined in terms ofthe individuals latent cognitive potential under certaincontextual conditions. Plasticity has been defined in terms of the capacity to acquire cognitive skills, Cognitive skills are here defined as the abilities that an organism can improve through practice or observational learning and that involvejudgment or processing beyond perceptual motor skills. [6],[7].

A. Cognitive Flexibility Definition

Cognitive flexibility is described as the capacity to shift or switch one's thinking and attention between different tasks or operations typically in response to a change in rules or demands [8].

B. Cognitive Flexibility and Brain

Functional Magnetic Resonance Imaging (fMRI) research has shown that specific brain regions are activated when a person engages in cognitive flexibility tasks. These regions include the prefrontal cortex (PFC), basal ganglia, anterior cingulate cortex (ACC), and posterior parietal cortex (PPC) [9].

III. The Theory of Cognitive Flexibility

Cognitive flexibility theory is a conceptual model for developing learning environments based on cognitive learning theory. Cognitive flexibility theory focuses on the nature of learning in complex and ill-structured domains. Spiro &Jehng (1990) state it as the ability to spontaneously restructure one's knowledge, in many ways, in adaptive response to radically changing situational demands. Cognitive flexibility is the ability to restructure knowledge in multiple ways depending on the changing situational demands [10], [11].

IV. Principles of Cognitive Flexibility Theory

A. Learning activities must provide multiple representations of content.
B. Instructional materials should avoid oversimplifying the content domain.
C. Support context-dependent knowledge.
D. Instruction should be case-based.
E. Emphasize should be on knowledge construction, not transmission of information.
F. Knowledge sources should be highly interconnected rather than compartmentalized.

Although this theory is applicable to lower levels of learning, in general it focuses on advanced knowledge learning (especially in the case of hypertext).Researchers have argued that cognitive flexibility is also a component of multiple classifications, as originally described by psychologist Jean Piaget. In multiple classification tasks, participants (primarily children, who have alreadydeveloped or are in the process of developing this skill) must classify objects in several different ways at once, thereby thinking flexibly about them [12].

Research has suggested that cognitive flexibility is related to other cognitive abilities, such as fluid intelligence, reading fluency, and reading comprehension [12], [13]. Cognitive flexibility has also been shown to be related to one's ability to cope in particular situations. For example, when individuals are better able to shift their thinking from situation to situation they will focus less on stressors within these situations [14].

In general, researchers in the field focus on development of cognitive flexibility between the ages of three and five [15]. However, cognitive flexibility has been shown to be a broad concept that can be studied with all different ages and situations[16].Diminished cognitive flexibility has been noted in a variety of neuropsychiatric disorders such as Anorexia nervosa, Obsessive Compulsive Disorder, Schizophrenia, Autism, and in a subset of people with Attention deficit hyperactivity disorder [17],[18].

Learning as a new behavior and information acquirement from environment is a specific pattern of neural function activated through experience and education and depends on the flexibility of memorized changesduration through flexible neural webs engaged in endurance and acquirement of information. Information endurance as a form of synaptic flexibility including flexibility in synapse structure occurs in a specific set of neurons in long term memory [19].

Neuralsystem change often combined with correlational change in behavioral and cognitive change. This change in behavior is known as learning, addiction, maturity and rehabilitation. Thus, for example, when individuals learn a new motion skill, say, playing with instrument, a change in cognitive plasticity occurs; suggest that neural system specifically sensitive to experiment is developing [7].

V. Activity-Dependent Plasticity

A defining feature of the brain is its capacity to undergo changes based on activity-dependent functions, also called activity-dependent plasticity. Its ability to remodel itself forms the basis of the brain's capacity to retain memories, improve motor function, and enhance comprehension and speech amongst other things. It is this trait to retain and form memories that is functionally linked to plasticity and therefore many of the functions individuals perform on a daily basis [20]. This plasticity is the result of changed gene expression that occurs because of organized cellular mechanisms [21]. The brain's ability to adapt toward active functions has allowed humans to specialize in specific processes based on relative use and activity. For example, a right-handed person may perform any movement poorly with his/her left hand but continuous practice with the less dominant hand can make both hands just as able. Another example is if someone was

born with a neurological disorder such as autism or had a stroke that resulted in a disorder, then they are capable of retrieving much of their lost function by practicing and "rewiring" the brain in order to incorporate these lost manners [22].Researchers have begun examining ways to harness neuroplasticity to promote healing and recovery. Although these efforts are still in the beginning stages, there is promising evidence that the dynamic qualities of the brain may play a pivotal role in how one copes with stress and mental illness[23], [24].Medications have been shown to affect neuroplasticity in animal models and a few human studies. As noted previously, antidepressant medications can reverse the effects of various types of chronic stress on both behavior and brain structurealthough animal studies differ on which aspects of neuroplasticity (e.g., neurogenesis, dendritic remodeling, BDNF levels) are critical for therapeutic efficacy[25], [26].Induced stimulation of the brain focally or generally also effects neuroplasticity. Studies in multiple species, including non-human primates, have shown that electroconvulsive shock increases hippocampal BDNF levels, synaptic density, and neurogenesis [27].Reviews of the evidence indicate that exercise can beassociated with reduced psychiatric symptoms (particularlyof depression [MDD]) and cognitive deficits inmultiple conditions (e.g.MDD, schizophrenia, Alzheimer'sdementia) [28], [29]. There is evidence that exercise, used as a supportive treatment, may delay or even prevent disease-onset and progression[29].The cross-sectional design utilized in most studies makes it difficult to determine whether these structural differences were present before the psychiatric illness developed (possible risk factors) or if they are a result of the conditions themselves. This has continued to be an area of debate. For a period of time, it was assumed that stress and mental illness directly caused the observed differences in brain volume. However, a seminal study in pairs of identical twins suggested that smaller hippocampal volume served as a risk factor for developing PTSD, rather than anacquired trait [30], [31]. Given the conflicting results regarding acquired versus predispositional differences, the field seems to be moving toward a more nuanced, multifaceted understanding of these variables, recognizing that both genetics and environment likely play a role in the etiology and course of mental illness, as well as the associated differences and changes in brain function and structure [32]Moreresearch is needed, particularly twin and longitudinalstudies, in order to clarify the relationship betweengenetic (i.e., predisposing traits) and environmental (i.e., experience, mental illness, stress) factors[33].A common, yet simplistic, assumption regarding volumetric changes is that they are directly due to changes in cell quantity (increase or decrease); however, this has not necessarily been well-supported by postmortem studies [34].

VI. Conclusions

Central nervous system (CNS) has the ability to adjust with or rehabilitation traumatic brain injuries. The flexibility characteristic of CNS is well known and described as CNS ability to adaptation with changes in order to improve its function. Neuroplasticity is an endowment for maintaining brain function, without it damaged functions never rehabilitate. Plasticity enables brain to repair the dysfunctions induced by injuries or genetic disorders. Thanks to this ability, we can compensate unrecoverable injuries or dysfunctional neural paths through preserving or pruning remained connections. In cognitive neuroplasticity, individual differencesin applying different styles of cognitive adjustment results in variant emotional, cognitive and social consequences. On the other hand, everyday life complexities increase the need for cognitive flexibility for a better adjustment with induced changes. Research capabilities for human studies are limited, so most questions must be addressed by study of animal models. This makes disentangling genetic, environmental, and experiential influences much more challenging. Although there is not yet consensus, it appears the field is moving toward a more multifaceted, nuanced understanding that recognizes the likely contribution of multiple factors, rather than a single explanation. Future research and advances in technology will continue toincrease understanding of the human brain and its fascinating abilities and potential. The brain, once considered to be a fixed and stable organ, is now viewed as dynamic, flexible, and adaptive. Efforts are beginning to focus on ways to harness the plastic qualities of the brain for treatment and recovery. There is much that is still unclear about the relationship between neuroplasticity and mental health. Research capabilities for human studies are limited, so most questions must be addressed by study of animal models. This makes disentangling genetic, environmental, and experiential influences much more challenging. Although there is not yet consensus, it appears the field is moving toward a more multifaceted, nuanced understanding that recognizes the likely contribution of multiple factors, rather than a single explanation. Future research and advances in technology will continue to increase understanding of the human brain and its fascinating abilities and potential.

References

[1] Stiles, Joan. (2008). the Fundamentals of Brain Development: Integrating Nature and Nurture. Cambridge, Mass: Harvard University Press.W.-K. Chen, Linear Networks and Systems (Book style). Belmont, CA: Wadsworth, 1993, pp. 123–135.

[2] Haghgoo,Hojjatallah (2011), Fundamentals of Neural Science,Welfare Science Publications.B. Smith, "An approach to graphs of linear forms (Unpublished work style)," unpublished.

[3] Johnston MV. (2004). Clinical disorders of brain plasticity. Brain & Development, 26:73-80.J. Wang, "Fundamentals of erbium-doped fiber amplifiers arrays (Periodical style—Submitted for publication)," IEEE J. Quantum Electron., submitted for publication.

[4] Michael S. Gazzaniga, Editor-in-Chief ‹2009 Massachusetts Institute of TechnologyY. Yorozu, M. Hirano, K. Oka, and Y. Tagawa, "Electron spectroscopy studies on magneto-optical media and plastic substrate interfaces(Translation Journals style)," IEEE Transl. J. Magn.Jpn., vol. 2, Aug. 1987, pp. 740–741 (Dig. 9th Annu. Conf. Magnetics Japan, 1982, p. 301).

[5] MichaelPluess& Jay Belsky the Nature (and Nurture?) of Plasticity in Early Human Development Perspectives on Psychological Science July 2009 vol. 4 no. 4 345-351J. U. Duncombe, "Infrared navigation—Part I: An assessment of feasibility (Periodical style)," IEEE Trans. Electron Devices, vol. ED-11, pp. 34–39, Jan. 1959.

[6] Jones, S., Nyberg, L., Sandblom, J., Stigsdotter Neely, A., Ingvar,M., Petersson, K., et al. (2006). Cognitive and neuralplasticity in aging: General and task-specifi c limitations.Neuroscience and Biobehavioral Reviews, 30, 864-871.
[7] Mercado, E. (2008). Neural and cognitive plasticity: Frommaps to minds. Psychological Bulletin, 134, 109-137.
[8] Miyake, A; Friedman, N.P; Emerson, M. J;Witzki, A.H; Howerter, A; & Wagner, T. (2000).The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. Cognitive Psychology,41, 49–100.
[9] Leber, A. B; Turk-Browne, N. B, Chun, M. M.(9 September 2008). "Neural predictors of moment-to-moment fluctuations in cognitive flexibility".ProcNatlAcadSci U S A105 ,36,13592–7
[10] Chikatla, S; Rebecca Reese (2007)."Cognitive Flexibility Theory". University of South Alabama. Retrieved November,18, 2012 .
[11] Spiro, R. J,Jehng, J. C. (1990). Cognitive flexibility and hypertext: Theory and technology for the non-linear and mulit-dimensional traversal of complex subject matter. In D. Nix and R. J. Spiro (Eds.), Cognition, education, and multimedia: Exploration in high technology. Hillsdale, NJ: Lawrence Erlbaum
[12] Cartwright, K. B.(2002). "Cognitive development and reading: The relation of reading-specific multiple classification skill to reading comprehension in elementary school children".Journal of Educational Psychology, 94, 56–63.
[13] Colzanto, L .S; van Wouwe, N. C; Lavender, T. J; &Hommel, B. (2006). "Intelligence and cognitive flexibility: Fluid intelligence correlates with feature "unbinding" across perception and action." Psychonomic Bulletin &Review, 13, 1043–1048.
[14] Han, H. D; Park, H. W; Kee, B. S; Na, C; Na, D. E; &Zaichkowsky, L.(1998). "Performance enhancement with low stress and anxiety modulated by cognitive flexibility".Korean Neuropsychiatric Association, 7, 221–226.
[15] Zelazo, P. D; Frye, D. (1998). "Cognitive complexity and control: II. The development of executive function in childhood".Current Directions in Psychological Science, 7, 121–126.
[16] Chelune, G. J; Baer, R .A. (1986). "Developmental norms for the Wisconsin Card Sorting Test".Journal of Clinical and Experimental Neuropsychology, 8,219–228.
[17] Steinglass, J. E; Walsh B. T, Stern, Y. (May 2006). "Set shifting deficit in anorexia nervosa".JIntNeuropsycholSoc, 12, (3). 431–5.
[18] Etchepareborda, M. C; Mulas, F. (Feb 2004). "Cognitive flexibility, an additional symptom of attention deficit hyperactivity disorder. Is it a therapeutically predictive element?" (Article in Spanish)".Rev Neurol 38 (Suppl 1), S97–102.
[19] Lundqvist, A., Grundström, K., Samuelsson, K., &Rönneberg, J. (2010).Computerized training of working memory in a group of patients
[20] Samuels BA, Hen R: Neurogenesis and affective disorders. Eur J Neurosci 2011; 33:1152–1159.
[21] Bruel-Jungerman E, Davis S, Laroche S (2007). "Brain plasticity mechanisms and memory: A part of four". *Neuroscientist*13 (5): 492–505.
[22] Flavell S, Greenberg ME (2008). "Signaling Mechanisms Plasticity of the Nervous System". *Annu Rev Neurosci* 31: 563–90.
[23] Pascual-Leone A., Amedi A., Fregni F., Merabet L. B. (2005). "The plastic human brain cortex". Annual Review of Neuroscience 28: 377–401.
[24] DeCarolis NA, Eisch AJ: Hippocampal neurogenesis as a target for the treatment of mental illness: a critical evaluation. Neuropharmacology 2010; 58:884–893.
[25] Cramer SC, Sur M, Dobkin BH, et al: Harnessing neuroplasticity for clinical applications. Brain 2011; 134:1591–1609
[26] Hanson ND, Owens MJ, Nemeroff CB: Depression, antidepressants, and neurogenesis: a critical reappraisal. Neuropsychopharmacology2011; 36:2589–2602.
[27] Petrik D, Lagace DC, Eisch AJ: The neurogenesis hypothesis of affective and anxiety disorders: are we mistaking the scaffolding for the building? Neuropharmacology 2012; 62:21–34.
[28] Bolwig TG: How does electroconvulsive therapy work? theorieson its mechanism. Can J Psychiatry 2011; 56:13–18.
[29] Ernst C, Olson AK, Pinel JP, et al: Antidepressant effects of exercise: evidence for an adult-neurogenesis hypothesis? J Psychiatry Neurosci 2006; 31:84–92.
[30] Knöchel C, Oertel-Knöchel V, O'Dwyer L, et al: Cognitive and behavioural effects of physical exercise in psychiatric patients. ProgNeurobiol 2012; 96:46–68.
[31] Gilbertson MW, Shenton ME, Ciszewski A, et al: Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. Nat Neurosci 2002; 5:1242–1247.
[32] Kremen WS, Koenen KC, Afari N, et al: Twin studies of posttraumatic stress disorder: differentiating vulnerability factors from sequelae. Neuropharmacology 2012; 62: 647–653
[33] Couillard-Despres S, Aigner L: In-vivo imaging of adult neurogenesis. Eur J Neurosci 2011; 33:1037–1044.
[34] Bremner JD, Elzinga B, Schmahl C, et al: Structural and functional plasticity of the human brain in posttraumatic stress disorder. Prog Brain Res 2008; 167:171–186.

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