

**Exploring the Role of Neuroplasticity in Lifespan Learning:
A Neuro-Cognitive Perspective**

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Abstract

Plasticity and learning are closely related, as education aims to enhance learning, while neuroscience seeks to understand the neural mechanisms involved in it. This interactive relationship suggests a future in which educational practices can best support learning and sustainable human development. Some aspects facilitate a functional integration between both disciplines, and it can be argued that neuroscience has a crucial role in education reform for continuous improvement in individual teaching practices and education systems.

This contribution aims to explore the connection between neuroscience and education, emphasizing the significance of educational neuroscience approaches that could revamp the curriculum and innovative methods of learning and interpreting the world. The emerging field of educational neuroscience offers both opportunities and challenges for education. It provides a way to enhance mutual understanding and bridge the divide between educators, psychologists, and neuroscientists to uphold brain functionality for effective and satisfying learning, even in the later stages of life. To put it simply, learning can be a continuous process.

Keywords:

Neuroplasticity, Synapses, Astrocyte, Synaptogenesis, Lifespan Learning, Brain Aging.

Introduction

The field of brain and cognitive sciences has advanced to such a degree that it is now possible to objectively monitor the development of a person's brain using specialized measurements. This allows researchers to study the growth of cerebral and mental organization from childhood to advanced age and determine how this trajectory is influenced by parenting, education, and other environmental factors.

These advancements in neuroscience have provided new insights that could be of great value to educators and policy-makers. By deepening our understanding of how humans learn and how brain structure and function can be reconfigured to achieve new patterns of thought and behavior, we can explore the capacity of brain cells to adapt in response to intrinsic and extrinsic factors throughout a person's life. This plasticity through learning enables people of all ages to adapt to challenges and changes in their environment.

What is Neuroplasticity?

The concept of neuroplasticity stems from the Greek word "Plastikos," meaning "to form" (Mundkur M., 2005). In the realm of neuroscience, neuroplasticity refers to the modifications in the structure and operation of the brain that occur as a result of training and experience (Johanson M. V., Nishimura A., Harum K., Blue M. E., 2001).

The brain is an organ that is adaptable and can change in response to different experiences. Thanks to advancements in brain imaging technology, many previous assumptions about the brain have been disproven (Doidge N., 2007). It was once believed that the brain was "hard-wired," meaning that after the critical period of early development ended, a vast number of neurons and their connections remained fixed for life. However, recent research has revealed that the brain is a dynamic organism with synaptic connections that are constantly evolving both structurally and functionally. The brain is now viewed as a dynamic organism with synaptic connections that are fluid and constantly changing (Doidge N., 2007) both structurally and functionally. Structural plasticity means the brain can:

- Grow new neurons (neurogenesis);
- Alter the distribution of where neurons are located (somatotopic mapping);
- Promote new, extensive synaptic networks in response to virtually any stimulus; regardless of age, condition, or type of experience (Mahncke W.H.,2006).

Modern Neuro-Biological Definition of Neuroplasticity

Neuroplasticity is a term used to describe the brain's ability to adapt and change in response to internal and external factors. It refers to the permanent changes in the properties of nerve cells that occur as a result of environmental stimuli, such as learning, experience, or exposure to new situations, or from a break in the continuity or damage to the nervous system (Kolb B. & Gribb R., 2011).

The concept of neuroplasticity has revolutionized our understanding of the brain and its capacity to change and reorganize itself throughout life. A brain can form new neural connections throughout life, which are influenced by intrinsic or extrinsic stimuli, such as physical exercise, cognitive training, or exposure to new environments. or the capacity of neurons and neural networks in the brain to change their connections and behavior in response to new information, sensory stimulation, development, damage, or dysfunction (Malhotra M., 213). These changes can occur at the level of individual neurons, neural circuits, or entire brain regions, and can lead to improvements in cognitive function, emotional regulation, and sensory perception.

This ability is due to the inherent capacity of nerve tissue to form new interneuronal connections or synapses (synaptogenesis) or replace nonfunctional neurons (neurodegeneration) in the brain with new neurons (neurogenesis) (Bogdan F.K., Danuta W. & Dorota Z., 2017).

Neuroplasticity is the ability of neurons and neural networks in the brain to change their behavior and connections in response to various factors such as genetics, age, lifestyle, environment, sensory stimulation, development, damage, or dysfunction. This ability is crucial for learning, memory, and recovery from injury or illness.

Understanding the mechanisms of neuroplasticity is essential for developing effective treatments for a wide range of neurological and psychiatric disorders, including stroke, traumatic brain injury, and neurodegenerative diseases such as Alzheimer's and Parkinson's. The brain is not a fixed and static organ, but a dynamic and adaptable system that has the potential for lifelong learning and growth.

Systemic neuroplasticity is a natural feature of the nervous system that allows it to adjust to changing environmental conditions. This adaptation mainly affects learning and memory processes, as well as the self-repair capacity. These characteristics apply to neurons at all levels of the nervous system, and there are different types of neuroplasticity recognized. These types include developmental plasticity, compensatory plasticity (post-injury plasticity of a fully developed brain), and neuroplasticity caused by repeated sensory (inputs) or motor (outputs) experiences. (Ziemiańska K. & al, 2012). It refers to the ability of the brain to structurally change input from the environment (Shaw & McEachern, 2012).

Neuroplasticity refers to the brain's ability to adapt and reorganize itself by forming new neural connections throughout life. However, in certain conditions like addiction, epilepsy (epileptogenesis), or neuropathic pain, the changes in the strength of the interneuronal and the number of synaptic connections (nerve synapses) become pathological, leading to abnormal nervous system function. These changes are characterized by modifications in the efficacy and number of interneuronal connections (Ziemiańska K., Konopka A. & Wilczyński G.M., 2012).

During prenatal development of the nervous system, the number of developing neurons surpasses the number of neurons that eventually survive. The neurons compete with each other to form synaptic connections with other neurons through synaptogenesis. Those neurons that fail to make these connections undergo a programmed and controlled cell death known as apoptosis (Batson G., 2015).

That is to say, brain plasticity or neuroplasticity is the inherent capacity of nerve tissue to form new interneuronal connections or synapses (synaptogenesis) or replace useless, nonfunctional (neurodegeneration) neurons in the brain with new neurons (neurogenesis). The key factor in the formation of synapses at both the cellular and molecular levels is long-term potentiation (LTP) (Doroszevska J., 2008).

The brain is not a static organ, but a dynamic one that constantly changes throughout a person's life in response to both internal and external factors. This natural ability of the brain to shift its architecture in both positive and negative directions is called neuroplasticity (Shaffer J., 2012). Positive psychology aims to better understand and develop interventions that can help individuals, families, and communities thrive (Seligman & Csikszentmihalyi, 2000), offering hope for a brighter future.

Types of Neuroplasticity

As previously described, neuroplasticity is the brain's ability to continually change its structure and function in response to experience (Batson G., 2015). From this point, we identify two neuroplasticity types:

- **Structural neuroplasticity**

Structural plasticity is a common characteristic of fetal neurons during brain development, which is called developmental plasticity. This includes neurogenesis and neuronal migration (Demarin V., 2014), where millions of neurons migrate from their sites of origin in the ventricular and subventricular zones to their final destination within the Central Nervous System (CNS) (Poduri A. & Volpe J. J., 2018). This process is crucial for the proper development and functioning of the brain.

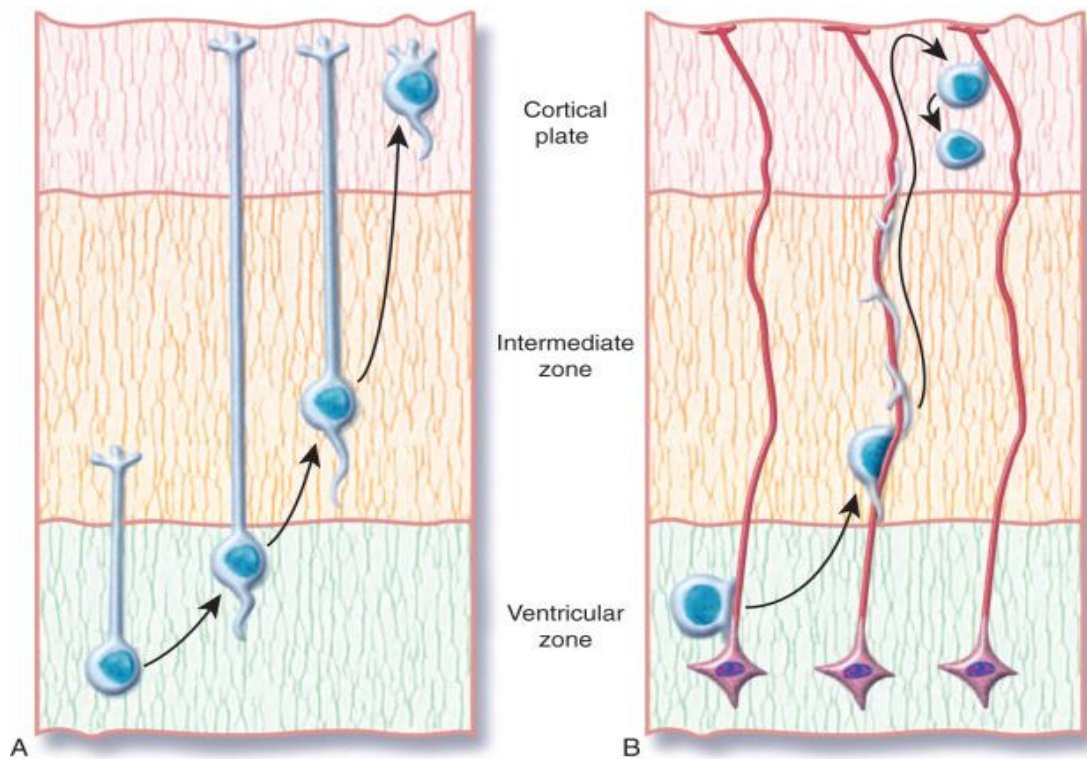


Figure (1). Two modes of radial neuronal migration. (Poduri A. & al., 2018).

Source: ScienceDirect (ELSEVIER)

For a better understanding, neuronal migration is a process in which neurons travel from their “place of birth” in the fetal ventricular or subventricular zone, towards their final position in the cortex (Demarin V. & al., 2014). It is necessary for proper brain architecture since most neurons are born in a position different from that in which they will reside (Reiner G. & Gerlitz G., 2013).

Another form of structural plasticity is “synaptogenesis”, which is one of the hallmarks of brain development (neurogenesis, migration, and pathfinding are others), and a major surge for synapse formation occurs during postnatal ages during which sensory pathways are sensitive to modification based-on neural activity (Aoki C. & Erisir A., 2014).

Synaptogenesis is guided by molecules, morphogens, and neuronal activity to develop and maintain synaptic contacts. (Jeanneteau F., Chao M.V., 2013). Additionally, other forms of structural neuroplasticity, such as changes in white or grey matter density, can be observed through magnetic resonance imaging (Demarin V. et al., 2014).

- **Functional neuroplasticity**

Two primary processes, learning, and memory, drive functional neuroplasticity by changing synaptic connections between neurons. (Pascual-Leone A., Amedi A. & Fregni F, 2005). These changes may result from structural adjustments or biochemical processes that lead to permanent alterations in the synaptic relationships between neurons. (Demarin V. & al., 2014). The capacity for this type of plasticity tends to increase during childhood and young adulthood but decreases later in life. The extent to which this capacity diminishes varies based on individual differences in genetics, personality, motivation, lifestyle, socio-cultural background, exercise opportunities, and learning experiences (Cai L. & al., 2014).

Neuroplasticity implies that the brain is always learning, meaning that it is constantly developing, changing, and learning throughout life. This is a critical neurochemical foundation for learning and memory. Neuroplasticity reorganizes the functions of neurons, allowing for rapid adaptation and self-repair, which can result in learning and memory processes at all levels of the nervous system. It also includes the intrinsic excitability of neurons, which affects information storage (Bogdan F.K., Danuta W. & Dorota Z., 2017). That is to say, If we can “train” the brain properly (direct it towards a positive learning experience), then perhaps deterioration (or negative behaviors) can be slowed, stopped, or even potentially reversed (Doidge N., 2007).

In recent decades, the constant dynamic movement of synapses and their components has been recognized as a crucial aspect of synaptic transmission and its plasticity. However, maintaining functional synaptic connections has proved to be a considerable challenge. To overcome this challenge, several recent studies have focused on identifying mechanisms that regulate the synaptic availability of mitochondria, as they play a critical role in synapse formation and function (Guo & al., 2005; Stowers &, 2002; Morris & Hollenbeck, 1993; Misgeld & Schwarz, 2017; Lin & Sheng, 2015; MacAskill & Kittler, 2010; Saxton & Hollenbeck, 2012; Sheng & Cai, 2012; Cai & Tammineni, 2017).

Badel and other researchers have discovered that the formation of functional synapses in the pre-synaptic sensory neurons of the *Aplysia* gill withdrawal reflex leads to a persistent improvement in the bi-directional transport of mitochondria. Even in the absence of a functional synapse, activation of cAMP signaling is sufficient to enhance bi-directional transport in sensory neurons. It is important to note that the persistent improvement in transport does not depend on NMDA and AMPA receptor signaling or signaling from the post-synaptic neuronal cell body. However, it does depend on transcription and protein synthesis in the pre-synaptic neuron. (Badel & al, 2019).

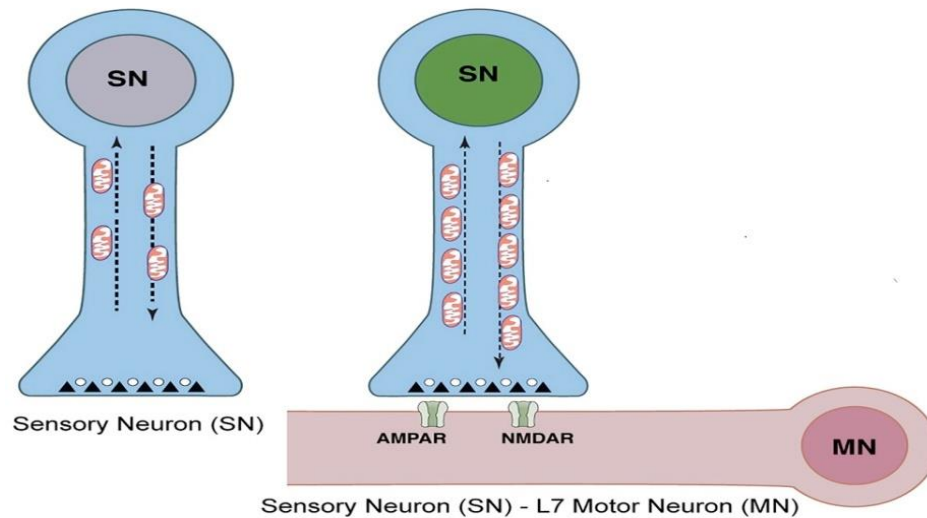


Figure 2. Mitochondrial transport in the presence of a functional synapse (Badal K. K. & al., 2019)¹.

Source: ScienceDirect (ELSEVIER)

According to Badel's summary, the neuron that receives the signal after the synapse creates a strong and lasting change in the pre-synaptic neuron's transcriptional program. This change results in the specific alteration of the transport of organelles. By understanding how intercellular signaling leads to persistent changes in the transcriptional program, thereby enabling specific changes in bi-directional transport, we can gain valuable insights into the functioning of neuronal networks (Badal K. K. & al., 2019).

Plasticity can be broadly classified into two main categories - functional and structural. Functional plasticity refers to changes in the brain that do not involve any anatomical modifications, such as alterations in synaptic strengths. On the other hand, structural plasticity refers to changes in the brain that involve anatomical modifications, such as changes in the number of synapses and neuronal cells, the density of axonal fibers, and the branching of axons and dendrites. (Butz, Wörgötter, & van Ooyen, 2009).

¹ the formation of a functional synapse between pre-synaptic sensory and post-synaptic motor neurons of the gill withdrawal reflex of Aplysia results in persistently enhanced anterograde and retrograde transport of mitochondria in sensory neurons. Once synapses are formed, transcription and translation in pre-synaptic neurons constrain mitochondrial transport (Badal K. K. & al., 2019).

Astrocyte-Synapse interactions

The concept of elasticity in brain map space was initially discovered by Canadian psychologist Hebb. He observed that if any two cells or systems of cells are repeatedly active at the same time, they tend to become "associated". As a result, activity in one cell or system facilitates activity in the other (Hebb D., 1949, p. 70). Later this theory was entitled Hebbian Plasticity and popularly paraphrased into "cells that fire together, wire together" (Doidge, 2007, p. 64; Schwartz & Begley, 2002, p. 107).

Astrocytes are a type of glial cell that is found in large quantities in the brain (Khakh & Sofroniew, 2015). These cells are primarily composed of processes that respond to neurotransmitters and help to regulate synapses. In terms of their physical structure, up to 95% of an astrocyte's volume consists of its various processes. (Shigetomi et al., 2013). They play an important role in establishing and maintaining proper synaptic connectivity. Astrocytes are the most abundant glial cells in the mammalian brain and interact dynamically with neuronal synapses to regulate synaptogenesis and physiology (Allen J. N. & Eroglu C., 2017). The number and size of astrocytes increase in proportion to brain size and cognitive capabilities (Allen, 2014, Stogsdill and Eroglu, 2017). For instance, a single mouse cortical astrocyte can connect to over 100,000 synapses, while a human astrocyte can connect to up to 2,000,000 synapses (Bushong & al., 2002; Oberheim & al., 2009).

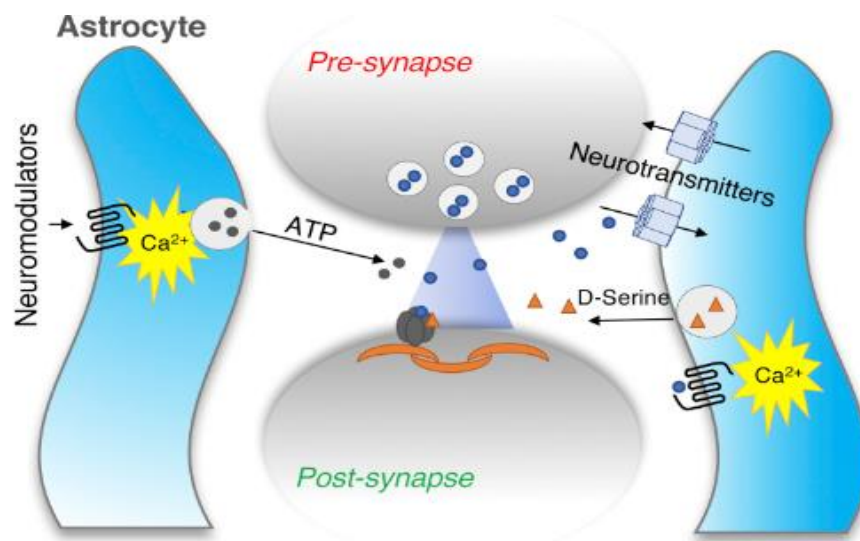


Figure 3. Perisynaptic astrocyte processes are structural and functional components of synapses (Allen J. N. & Eroglu C., 2017)². Source: ScienceDirect (ELSEVIER)

² Astrocytes are also functionally linked to synapses, as they possess the ability to sense synaptic activity and respond to it through intracellular Ca²⁺ transients and by releasing neuroactive molecules that can signal back to synapses (Allen j. N. & Eroglu C., 2017).

Significant strides have been made in comprehending how astrocytes influence the development of neurons. It has been suggested that the increase in astrocytic Ca^{2+} levels, which is induced by synaptic activity, leads to the secretion of neuroactive compounds called "gliotransmitters" from astrocytes. These compounds directly signal to synapses, regulating basal synaptic transmission and modifying neural plasticity. (Araque et al., 2014).

Studies have shown that astrocytes play a crucial role in the architecture of the nervous system by interacting with synapses in a dynamic and bi-directional manner. They are an important regulator of brain function and plasticity. Astrocytes respond to all kinds of brain trauma, infections, injuries, and diseases. These changes are often accompanied by changes in morphology. (Khakh and Sofroniew, 2015).

Synaptic plasticity is the ability to change the synaptic strength. Changes in the strength include neurotransmitters (Bogdan F.K., Danuta W. & Dorota Z., 2017). Synapses are morphologically distinct subcellular junctional structures, composed of a presynaptic terminal, a postsynaptic target, and the synaptic cleft aligning pre- and post-synaptic specializations (Cowan et al., 2001; Pappas and Purpura, 1972). Biological synapses act as unique dynamic signaling units, whose effects on the transmitted signal from one neuron to another can vary enormously depending on the activity history at either or both sides of the synapse (Abbott F. L. & Regehr G. W., 2004). The connection between neurons is through synapses, enabling communication between neurons.

Recent advancements in nanotechnology and communication engineering are making it possible to develop a new generation of nanoscale devices that can be implanted inside the human body (Jornet M. J. & Akyildiz F. I., 2013; Akyildiz F. I., Jornet M. J & Pierobon M., 2011). This is achieved by exploiting the similarity in size between the nanomachines and the nervous biological structures. The unique interaction between the pre- and postsynaptic neurons is what determines the synapse-specific signaling. This signaling is driven by the complex biophysical mechanisms activated at the presynaptic terminals by the electrical signals, also known as action potentials (APs) or spikes, fired by the neuron in response to a stimulus. When an AP reaches different presynaptic terminals, the synapse-specific signaling produces different patterns of neurotransmitter release. Consequently, a presynaptic neuron can transmit several different signals simultaneously to a targeted postsynaptic neuron (Cacciapuoti S. A., Caleffi M. & Piras A. 2015). It's important to note that synapse strength and number can change in the adult brain due to experience and learning.

Synaptogenesis

Synaptogenesis is a highly organized process that occurs when the dendrite of a postsynaptic neuron and an incoming axon communicate at specific sites to establish a stable synapse. This

complex process involves synapse formation, synapse maintenance, and activity-dependent synapse refinement and elimination. Synaptogenesis is crucial for the establishment of neuronal connections and the precise functioning of brain circuitry (Cory C., 2002). It is often described as a hierarchy of recognition, adhesion, and protein targeting, starting with axonal guidance to the appropriate brain region and ending with neurotransmitter release opposite the appropriate cluster of neurotransmitter receptors and signaling molecules. (Akins R. M. & Biederer T., 2005; Cory C., 2002; Zhang W. & Benson L. D., 2001).

At birth, a human brain has trillions of neurons, but they are not connected. Between birth and the age of 3, a process called synaptogenesis takes place (Maher K.M., 2013), during which the brain constantly forms neuron connections or synapses. This results in three times more activity than that of an adult brain. The process continues until puberty, after which the excessive connections that are not used get pruned off (Kuhl, 2002), indicating that effective information transfer between neurons contributes to the formation of functional synapses while eliminating nonproductive or noncompetitive ones (Walsh & Lichtman, 2003).

Synapses represent a major functional unit of brain circuits, forming the basis of neuronal networks. The differentiation of synapses changes continually in response to the needs of circuits, and this dynamic process underlies the concept of synaptic plasticity (Jeanneau F., Chao V.M., 2013). In other words, synaptogenesis is a process that manages the formation of synaptic connections, helping to maintain and remove synapses over time.

Neuroplasticity: learning throughout the lifespan

Over more than 20 years, numerous studies have been conducted to explore brain plasticity in cognitive and motor development. These studies aim to understand the brain as an organ of learning and interpretation. Developmentalists propose that the human brain undergoes ongoing structural and functional changes in response to stimulation or training, regardless of age. It's important to note that lifetime cognitive and motor development are closely intertwined. (Salhouse & Davis, 2006; Johnston & al., 2001; Yan & al., 2000; Samuelson & Smith, 2000).

Correspondingly, to many researchers, brain plasticity (also known as neuroplasticity, cortical plasticity, cortical re-mapping), neural maturation, and cognitive development are lifetime developmental processes that play a crucial role in human brain maturity and a wide variety of daily functions (Cai L. & al., 2014; Barnett et al., 2009; Colcombe & al., 2003, 2006). Several brain functions including sense, motor, and association process stimuli in the environment that can lead to the change known as learning (Zull, 2006, p.3).

As previously explained, neuroplasticity is an innate characteristic that allows individuals to learn and relearn skills throughout their lives. Biologically, neural plasticity refers to the central nervous system's capacity to modify its existing cortical structures (anatomy and organization) and functions (physiological mechanisms or processes) in response to experiences, learning, training, or injury. (Cai L. & al., 2014; Ballantyne & al., 2008; Kolb & al., 2003; Wall & al., 2002; Kolb & Whishaw, 1998; Hubel & Wiesel, 1970) Learning is initiated by the nervous system's response to environmental stimuli. The information is then sent to the neocortex, the part of the cortex that evolved more recently, for processing. (Chung S.J., 2019). It is, therefore, a biological foundation of the learning brain (Taubert et al., 2010).

Appropriately to Cai and other studies, relevant neurons often “fire together [and] wire together” in skill learning or repeated exposure to stimulations and experiences (2014). The associated neurons of a given response will be activated simultaneously in response to similar stimuli in the future. Learning endeavors or experiences modify the existing cortical structures or mechanisms via neurogenesis, gliogenesis, or by changing the strength of inter-neuronal connections (synaptogenesis) (Ponti & al., 2008; Voelcker-Rehage & Willimczik, 2006; Dong & Greenough, 2004; Cotman & Berchtold, 2002; Buonomano & Merzenich, 1998). Ordinarily, structural and functional changes³ in the brain include neurogenesis⁴, gliogenesis⁵, strengthening of existing connections or synaptogenesis⁶, and the creation of new blood vessels in the brain (Pontiet al., 2008; Voelcker-Rehage & Willimczik, 2006; Mingand S., 2005; Dong & Greenough, 2004; Cotman & Berchtold, 2002; Buonomano & Merzenich, 1998).

The current body of literature presents compelling evidence that experience and training can induce functional and structural changes in the human brain. These changes include enhancing neural functions, increasing cortical volume, preventing natural volume loss, and improving cognitive abilities. (Abrahamsson S., 2017). According, to Doidge “If we stop exercising our mental skills, we do not just forget them, the brain map space for those skills is turned over to the skills we practice instead” (2007, p.59). Therefore, the more we use and practice the L2 we want to learn, the more brain map space is allocated. The less we use our L2, the more brain map space goes to other activities that we practice more (Maher K.M., 2013). That's why some researchers believe that aerobic exercise is an effective way to induce neuroplasticity in the human brain, which enhances cognitive and motor function by causing neural changes that can be detected and analyzed using molecular, cellular, and systems-level neuroscience techniques. (El-Sayes J. & al., 2019).

³ neural reorganizations

⁴ development of new neurons

⁵ generation of new glial cells

⁶ growth of new synapses

Neuroplasticity is closely related to the idea of competitiveness. If we stop exercising our brain, we not only forget what we have learned, but our brain automatically assigns that space to other functions we continue to use. This explains why it is difficult to quit bad habits, and emphasizes the importance of learning behavior during childhood when the brain maps are still being developed. (Guglielman E., 2012).

Early life events have a significant impact on both brain architecture patterns and behavior. The wiring diagram of a child's brain is influenced by both early and later experiences. However, experiences during critical periods establish the foundation for development beyond the early years (Leisman G., Mualem R., Mughrabi K. S., 2015). This process occurs through a continuous series of dynamic interactions between genetic influences, experiences, and environmental conditions. (Friederici, 2006; Majdan & Shatz, 2006). Enriched early experiences have been shown to have neuroprotective properties and can aid in functional compensation, which is therapeutic for a variety of different brain disorders (Gubert C., Hannan J. A., 2019). This means that the full growth of brain characteristics is significantly impacted by such experiences, and they directly affect the child's brain architecture by strengthening or eliminating neural connectivity. It is important to note that brain, cognitive, sensory, and perceptual development do not occur simultaneously, but rather at different developmental stages. (Leisman G., Mualem R., Mughrabi K. S., 2015), as represented below in Fig.4:

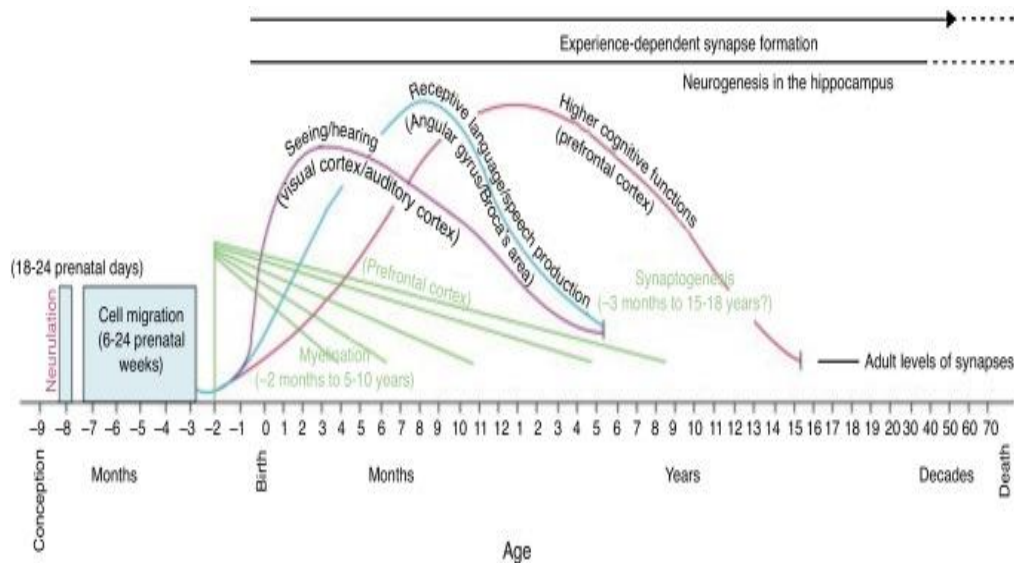


Figure 4: Human Brain Development: Neurogenesis in the Hippocampus⁷ through Experience-dependent⁸ Synapse Formation (Leisman G. & al., 2015).Source: ScienceDirect (ELSEVIER)

⁷ The hippocampus is a major component of the brains of humans and other mammals. It belongs to the limbic system and plays important roles in long-term memory and spatial navigation. The role of the hippocampus in relational (declarative) memory is in binding together multiple inputs to create and allow for the storage of representations of the associations among the constituent elements of scenes and events ().

⁸ The 'critical period' is defined as a period of time during which neuronal connections are susceptible to experience-dependent modifications (Hensch T. K., 2004).

Today research in neuroscience shows that Brains that have been trained are different from untrained brains (Kesselring J, Comi G, Thompson A J., 2010). Leisman and others suggest that if a second language is acquired early enough, it will have the same brain representation as the first language throughout one's lifetime. However, if a second language is learned later in development, even when spoken at a native level, it will be represented differently in the brain in comparison to the first language. (Leisman and Melillo, 2015, Leisman, 2012). For this reason, specific mental training can improve motor and sensory representations in the cortex, fostering the signal transmission restitute effectiveness to the neuronal connections (Guglielman E., 2012). To maximize human brain fitness and motor functions signaled by the quality of life and independence in daily activity, habitual cognitive and motor learning or practice is required across the lifespan, particularly for older adults (Cai L. & al, 2014). Furthermore, it is crucial to act promptly for such interventions to improve brain health throughout one's life and promote positive neuroplasticity by utilizing effective behavioral techniques. This requires measuring progress, motivating oneself, strategizing, and ensuring compliance with the latest neuroscience research. (Shaffer J., 2016).

Cognitive training is a commonly used method to slow down cognitive aging, which occurs due to changes in the brain leading to negative plasticity. This process of negative plasticity includes four components: disuse, noisy processes, weakening of neuromodulatory function, and negative learning.⁹ (Merzenich, M.M., 2005). Dr. Merzenich suggests that with proper exercise, brain structure and cognitive skills can be improved. Throughout our lifetime, our brain maps transform based on our activities, and they can continue to change even in adulthood. By following various strategies, we can improve our brain function and cognitive abilities. (Guglielman E., 2012):

- To combat disuse: engage the brain in new challenging tasks;
- To help the brain to order confusing signals: carry out activities that require attention and concentration;
- To regulate the production of neuromodulators: activities able to activate their production;
- To eliminate compensatory adaptive behaviors: engaging in activities that have become complicated to perform, rather than avoiding them.

As mentioned before, the brain undergoes constant changes throughout its lifetime. During fetal development, the primary changes are structural, such as neurogenesis and neuron migration. In the adult brain, functional neuroplasticity is the dominant type of change, allowing the brain to adapt to different environments and recover from injuries. (Demarin V. & al, 2014). Guglielmo suggests that to address the cognitive aging needs of adults, we need to develop pedagogical

⁹ People who begin to feel less mentally agile than once tend to implement mechanisms for compensation. If, for example, their hearing is impaired, they turn off the TV, or learn to read words on the lips (Merzenich, 2005).

strategies and approaches. This involves identifying the factors and variables that need to be monitored to make our training strategies effective. The training should focus on real-life themes and problems that are useful and applicable to daily life. (Guglielman E., 2012). Furthermore, we should suggest strategies and activities that are based on the principles of neuroplasticity, communication tools, and technologies that can enhance cognitive function at all stages of life with equal effectiveness. These strategies should be based on competencies, situated learning, and active knowledge construction.

Conclusion

Learning is not limited to younger generations or those with a fully functioning mind. It can be effective in all stages of life, from conception to death. A better understanding of the brain can help us develop more effective teaching and learning methods that are suitable for different ages. This can also help us keep people engaged and active throughout their lives. (Greenwood & Parasuraman, 2012; Lovat et al., 2011; Willis, Schaie & Martin, 2009).

Currently, the challenge facing Educational Neuroscience is to improve the scientific dialogue and establish a common language among academic, neuroscientific, educational, and psychological circles. The bridge connecting education and neuroscience must be a two-way pathway, and Educational Psychology could provide the necessary support to connect neuroscience to education. (Berninger & Corina, 1998; Mason, 2009).

The use of neuroscience and brain plasticity in adult education is crucial for promoting lifelong learning, improving quality of life, and enhancing independence in daily activities. This can be achieved by creating learning environments that are focused on developing competencies, allowing for situated learning, and actively constructing knowledge. From this perspective, "Biological, sensory and neurological influences on learning must become equal partners with social, emotional and cultural influences to have a truly effective discipline of education" (Goswami, 2008).

There is a general agreement on the significant role of various neuroscience techniques in providing a complete understanding of the neural mechanisms responsible for exercise-induced neuroplasticity in both humans and animals, structurally and functionally. However, it should be noted that the brain plasticity induced by experience and training could leave gaps in knowledge.

References

- Abrahamsson, S. (2017). Neuroplasticity induced by exercise. University of Skovde.
- Abbott, L. F., & Regehr G.W. (2004) Synaptic computation. *Nature*, 431:796–803, October.
- Allen, N. j., & Eroglu, C. (2017, November). Cell Biology of Astrocyte-Synapse Interactions. (CelPress, Éd.) *Neuro*, 96, pp. 697-708. <https://doi.org/10.1016/j.neuron.2017.09.056>
- Allen, N.J. (2014). Astrocyte regulation of synaptic behavior. *Annu. Rev. Cell Dev. Biol.* 30, 439–463.
- Berninger, V. W., & Corina, D. (1998). Making cognitive neuroscience educationally relevant: Creating bidirectional collaborations between educational psychology and cognitive neuroscience. *Educational Psychology Review*, 10(3), 343-354.
- Bushong, E.A., Martone, M.E., Jones, Y.Z., and Ellisman, M.H. (2002). Protoplasmic astrocytes in the CA1 stratum radiatum occupy separate anatomical domains. *J. Neurosci.* 22, 183–192.
- Buonomano, D.V. & Merzenich, M.M. (1998). Cortical plasticity: from synapses to maps. *Annu. Rev. Neurosci.* 21, 149–186. doi: 10.1146/annurev.neuro.21.1.149.
- Cai, Q., and Tammineni, P. (2017). Mitochondrial Aspects of Synaptic Dysfunction in Alzheimer's Disease. *J. Alzheimers Dis.* 57, 1087–1103.
- Chung, J.S. (2019). Educational Neuroscience for Adult Education Students in the U.S. and Maine. MA TESOL Collection. 741. https://digitalcollections.sit.edu/ipp_collection/741
- Liuyang Cai, J. S. (2014, March 10). Brain plasticity and motor practice in cognitive aging. *Frontiers in Aging Neuroscience*, pp. 1-12.
- Colcombe, S., & Kramer, A. F. (2003). Fitness effects on the cognitive function of older adults: a meta-analytic study. *Psychological Science*, 14(2), 125-130.
- Cotman, C. W., & Berchtold, N. C. (2002). Exercise: a behavioral intervention to enhance brain health and plasticity. *Trends Neurosci.* 25, 295–301. Doi: 10. 1016/s0166-2236(02)02143-4.
- Demarin, V., Morovic, S., & Bene, R. (2014). Neuroplasticity. (M. C. Aviva, Éd.) *PERIODICUM BIOLOGORUM* (2), pp. 209-211.
- Doidge, N. (2007). *The brain that changes itself: stories of personal triumph from the frontiers of brain science*, New York: Viking.
- Doroszewska, J. (2008). Neurogenesis and Synaptic Plasticity of CNS. In: Kozubski, W. and Doroszewska, J., Eds., *Apoptosis in Central Nervous System Disorders* Czelej, Lublin, 45-64.
- El-Sayes J., Harasym D., Turco V. C., Locke B. M. & Nelson J. A. (2019, Feb 1). Exercise-Induced Neuroplasticity: A Mechanistic Model and Prospects for Promoting Plasticity. (SAGE, Éd.) *The Neuroscientist*, 25.
- Friederic A.D., (2006). The neural basis of language development and its impairment *Neuron*, 52 (2006), pp. 941-952.
- Greenwood, P.M., & Parasuraman, R. (2012). *Nurturing the Older Brain and Mind*, Cambridge: Massachusetts Institute of Technology.
- Goswami, U. (2008). *Cognitive development: The learning brain*. New York, NY, US: Psychology Press.
- Guglielman, E. (2012, June). *The Ageing Brain: Neuroplasticity and Lifelong Learning*. (S. P.A.U. Education, Éd.) *eLearning Papers* (29).

- Hebb, D. O. (1949). *The organization of behavior*. New York: Wiley.
- Hensch T.K. (2004). Critical period regulation. *Annu Rev Neurosci*. 2004; 27:549–579.
- Hubel, D.H., & Wiesel, T.N. (1970). The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J. Physiol*. 206, 419–436.
- Johanston, MV., Nishimura, A., Harum K., Peker, J., Blue, ME. (2001). Sculpting the developing brain. *Adv Pediatr* 2001; 48: 1-38.
- Khakh, B.S., Fisher, J.A., Nashmi, R., Bowser, D.N., and Lester, H.A. (2005). An angstrom scale interaction between plasma membrane ATP-gated P2X2 and alpha4beta2 nicotinic channels measured with fluorescence resonance energy transfer and total internal reflection fluorescence microscopy. *J. Neurosci*. 25, 6911–6920
- Kesselring J, Comi G, Thompson AJ, *Recovery of function and neurorehabilitation in multiple sclerosis*, Cambridge University Press, 2010.
- Kolb, B. & Gibb, R. (2011) *Brain Plasticity and Behavior in the Developing Brain*. *Journal Academy Child Adolescence Psychiatry*, 20, 265-276.
- Leisman, G., Mualem, R., & Mughrabi, S. K. (2015). The neurological development of the child with educational enrichment in mind. *Psicología Educativa*, 21
<http://dx.doi.org/10.1016/j.pse.2015.08.006>
- Leisman, G., & Melillo, R. (2012). *The Development of the Frontal Lobes in Infancy and Childhood: Asymmetry and the Nature of Temperament and Adjustment and Childhood: Asymmetry and the Nature of Temperament and Adjustment* NY: Nova Scientific Publishers.
- Lovat, T., et al. (2011). *Values Pedagogy and Student Achievement*. Contemporary Research Evidence, New York: Springer.
- Merzenich, M.M. (2005). Change minds for the better. *The Journal of Active Aging*, November-December, 22-30.
- Mason, L. (2009). Bridging neuroscience and education: A two-way path is possible. *Cortex*, 45(4), 548-549.
- MacAskill, A.F., and Kittler, J.T. (2010). Control of mitochondrial transport and localization in neurons. *Trends Cell Biol*. 20, 102–112.
- Malhotra, M. (2013) *Physical Exercise and Neuroplasticity. Examining the Role of Exercise on Long-Term Brain Function*. *Physical Therap@rehab Medicine*.
- Morris, R.L., and Hollenbeck, P.J. (1993). The regulation of bidirectional mitochondrial transport is coordinated with axonal outgrowth. *J. Cell Sci*. 104, 917–927.
- Misgeld, T., and Schwarz, T.L. (2017). Metastasis in Neurons: Maintaining Mitochondria in an Extended Cellular Architecture. *Neuron* 96, 651–666.
- Mahncke, H.W., Bronstone, A., & Merzenich, M.M. (2006). Brain Plasticity and Functional Losses in the Aged: Scientific Bases for a Novel Intervention. *Progress in Brain Research*, 157, 81-109.
- Oberheim, N.A., Takano, T., Han, X., He, W., Lin, J.H.C., Wang, F., Xu, Q., Wyatt, J.D., Pilcher, W., Ojemann, J.G., et al. (2009). Uniquely hominid features of adult human astrocytes. *J. Neurosci*. 29, 3276–3287.
- Shaffer, J. (2016, July). Neuroplasticity and Clinical Practice: *Frontiers in Psychology*, 7 (1118). doi: 10.3389/fpsyg.2016.01118

- Salthouse, T. A., and Davis, H. P. (2006). Organization of cognitive abilities and neuropsychological variables across the lifespan. *Dev.Rev.* 26, 31–54.doi:10. 1016/j.dr.2005.09.001.
- Schwarz, J. M. (2016). Sex and the Developing Brain. Dans R. M. Shansky, *Sex Differences in the Central Nervous System* (pp. 221-245). Academic Press. <https://doi.org/10.1016/C2014-0-01274-9>
- Shaw, C., & McEachern, J. (2012). *Toward a theory of neuroplasticity*. London: Psychology Press.
- Sheng, Z.-H., and Cai, Q. (2012). Mitochondrial transport in neurons: impact on synaptic homeostasis and neurodegeneration. *Nat. Rev. Neurosci.* 13, 77–93. Sheng, M., and Kim, E. (2011). The postsynaptic organization of synapses. *Cold Spring Harb. Perspect. Biol.* 3, a005678.
- Seligman, M. E. P., & Csikszentmihalyi, M. (2000). *Positive psychology: An introduction*. *American Psychologist*, 55, 5-14.
- Shigetomi, E., Bushong, E.A., Haustein, M.D., Tong, X., Jackson-Weaver, O., Kracun, S., Xu, J., Sofroniew, M.V., Ellisman, M.H., and Khakh, B.S. (2013). Imaging calcium microdomains within entire astrocyte territories and endfeet with GCaMPs expressed using adeno-associated viruses. *J. Gen. Physiol.* 141, 633–647.
- Stogsdill, J.A., and Eroglu, C. (2017). The interplay between neurons and glia in synapse development and plasticity. *Curr. Opin. Neurobiol.* 42, 1–8.
- Stowers, R.S., Megeath, L.J., Go´rska-Andrzejak, J., Meinertzhagen, I.A., and Schwarz, T.L. (2002). Axonal transport of mitochondria to synapses depends on Milton, a novel Drosophila protein. *Neuron* 36, 1063–1077.
- Voelcker-Rehage, C., and Willimczik, K. (2006). Motor plasticity in a juggling task in older adults—a developmental study. *Age and Ageing* 35, 422–427.doi:10. 1093/ageing/afl025
- Wall, J. T., Xu, J., and Wang, X. (2002). Human brain plasticity: an emerging view of the multiple substrates and mechanisms that cause cortical changes and related sensory dysfunctions after injuries of sensory inputs from the body. *BrainRes.BrainRes. Rev.* 39, 181–215.doi :10.1016/s0165-0173(02)00192-3.
- Yan, J.H. (2000). Effects of aging on linear and curvilinear raiming arm movements. *Exp.AgingRes.* 26, 393–407.doi:10.1080/03610730075001 5778.
- Ziemiańska, K., Konopka, A. and Wilczyński, G.M. (2012) The Role of Extracellular Proteolysis in Synaptic Plasticity of the Central Nervous System. *Postępy Higieny i Medycyny Doświadczalnej*, 66, 959-975. <https://doi.org/10.5604/17322693.1021851>.
- Zull, J.E. (2006). Key aspects of how the brain learns. In S. Johnson & K. Taylor (Eds.), *The Neuroscience of Adult Learning* (Number 110, pp. 3-10). San Francisco: CA: Jossey-Bass.