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Current Views on Neuroplasticity: What is New and What is Old?

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SUMMARY

The main aim of the paper is to show that many previously forgotten discoveries within the field of neuroscience own their rediscovery and renaissance to the refinement of tools provided by the technological advances. Most spectacular is the advancement of brain imaging techniques, which provide hard data that support for evidence for previously neglected presumptions and ideas. Neuroplasticity is an example of such a long ignored historical discovery. One reason for that neglect is that it stood in contradiction to beliefs and theories prevailing at the first half of the twenties century. The idea of neuronal plasticity is not disputed any longer since it has found confirmation not only in a dramatic development of neuroimaging but also in the advancement of neurobiology. Most authors concentrate upon neuronal plasticity, recent studies, however, have produced a wealth of information regarding neurogenesis, in which astrocytes have proved to play a significant role. The significance of adult neurogenesis for learning and memory and for treatment of depression is outlined. Moreover, it was observed that neuroplasticity benefits patients suffering from obsessive-compulsive disorder (OCD) who undergo effective, evidence-based treatment. Convincing examples of brain plasticity brings also clinical practice, which often unveils the appearance of hitherto hidden artistic abilities in people who have suffered from brain damage. In addition, the possibilities of altering the brain functions by mental force alone are discussed. Thus, the paper reveals that many "controversial" ideas were confirmed by contemporary studies forcing changes in a traditional view on brain works.

Key words: research paradigms, neuroplasticity, glial plasticity, mental force, neurogenesis

The present paper concentrates on previously forgotten discoveries within the field of neuroscience. It is stressed that their rediscovery and renaissance have been due to the technological advances of brain imaging techniques over the past decades. Most spectacular is the advancement of brain imaging techniques, which provide hard data that support evidence for previously neglected presumptions and ideas. Neuroplasticity is an example of such a long-ignored historical discovery. One reason for that neglect is that it stood in contradiction to theories and research paradigms prevailing in the first half of the twenties century. It is worth to remind that research paradigms are built upon our mental schemas or models of objects and events that surround us. Since our senses are pervaded by an abundance of stimuli the brain selects only some of them to create a coherent and comprehensible image of the reality. The world's image consists of mental models, which in fact are simplified schemas encompassing the most important features of a given object, event or action. Contrary to a general belief, we do not perceive ready-made information. Indeed, our perception is an act of creation and not a synthesis of particular features. Put differently, the images or models (percepts) are created in a process that strongly resembles sculpting: delineating distinctive features of an object or event (Brown, 2010, 2015; Dehaene, 2013; Pąchalska, Kaczmarek, and Kropotov, 2014). Those models play an important role both in perception and in action. Moreover, they provide stability and make the world safe and comprehensible.

Research paradigms are specific types of mental models, and, consequently, they provoke the scientific community to reject ideas that stand in contradiction to existing theories. Kuhn (1996) argues that research paradigms provide a stiff intellectual framework, or a "disciplinary matrix", imposing the ways of data gathering and interpreting the results. In consequence, any discrepancies are considered to follow from experimental errors or anomalies. Over time more and more anomalies and unsolved questions accumulate, and it turns difficult to solve them within a currently existing paradigm. As a consequence a paradigm shift takes place and a new model of thought aimed at explaining the existing puzzles is created. An example of such a shift is an approach to the idea of neuroplasticity.

Neuronal Plasticity

Neuroplasticity is extensively discussed in the contemporary literature, and it is often viewed as a recent discovery. Therefore, it might be worthy to remind experiments performed already at the beginning of the twentieth century. Two prominent physiologists, Graham Brown and Charles Sherrington, stimulated the motor cortex of experimental animals and found that the stimulation of the identical place in a particular monkey produced twitches of different muscles. Skeptics argued, however, that differences among movement maps of animals might well be inborn and not acquired (Schwartz, and Begley, 2003). This assumption was dispelled by Karl Lashley in 1923, who derived different maps in the brain motor area of one particular rhesus monkey. Lashley was able to show that those maps underwent considerable changes over the course of one month, in the wake of different motor tasks the monkey had performed. The idea was further supported by Donald Hebb (1949), who argued that learning and memory depend upon the strengthening of the activity of synapses in order for two events or stimuli to be bound together.

Those findings were neglected due to the prevailing belief in the immutability of the central nervous system. Researchers promoting a possibility of changes in neuronal circuits were as a rule attacked by the scientific establishment. It suffices to mention Walther Spielmeyer and Max Bielchowsky two widely recognized German neuropathologists (Stahnisch, and Nitsch,_2012). Spielmeyer collaborated with renowned scholars as Alois Alzheimer, Korbinian Broadmann, Emil Kraepelin, and Franz Nissl, while Bielschowsky is best known as the inventor of a derivative silver staining technique (Stahnisch, 2015). This might explain the ambiguous position of Santiago Ramón y Cajal, the great Spanish anatomist and Nobel laureate. In his Nobel lecture delivered in 1906, he firmly stated that the pathways in he firmly stated that the pathways in the mature brain are fixed and immutable (Kolb, 1995). In his later works, however, he assumed a possibility to elicit new connections between neurons in the process of learning (Stahnisch, and Nitsch, 2012; Pąchalska et al., 2014).

The revival of the notion of neuroplasticity took place thanks to experiments of Merzenich and his co-workers (1983) on changes within cortical somatosensory fields. As is often the case, Merzenich's initial observations met with strong opposition. It was argued that the expansion of cortical areas of the hairy back of the hand, which followed cutting the nerves of the palm, might be a result of unmasking already existing connections. To convince their critics, Merzenich at al. (1984) decided to take a drastic step amputating an owl monkey's finger to cut off all sensory input to it. After a couple of months, they then recorded the electrical activity of the monkey's somatosensory area. The recording showed that cortical representation of the amputated finger was taken over by the adjacent digits of the palm. It confirmed the possibility of changing the cortical representation of fingers of the hand. In 1991 Allard, Jenkins, and Merzenich created artificial syndactyly by sewing together two adjacent digits of adult owl monkeys. After two to three months they compared the previously recorded somatosensory brain maps of the monkey's hands with the recordings following the surgery and found that the two sewn digits had now a single overlapping representation.

At the same time, Taub (1980) showed that the deafferentation of one of a monkey's arm resulted in an inability to perform voluntary skillful movements using the limb. He then immobilized the monkey's healthy hand forcing it to use the injured one. Such training resulted in a significant improvement in the efficiency of injured hand movements. It is notable that Taub drew on observations published by German physiologist Hermann Munk already in 1909 (see Doidge, 2007).

Clinical Significance of Brain Plasticity

In humans, neuroplasticity also benefits patients suffering from obsessive-compulsive disorder (OCD) who undergo effective, evidence-based treatment.

Schwartz and Begley (2003) reported metabolism alternation in the OCD brain circuit after cognitive-behavior therapy had been applied. Positron emission tomography (PET) revealed a decrease of abnormally high hyperactivity of brain structures of that network (mainly the caudate, the orbital frontal cortex, and the cingulate gyrus).

Yet – as we all know – the cognitive-behavior therapy requires a long, effortful, and distressing work of both the patient and the therapist. Besides, the results are not always satisfactory. It raises one important question. Why is it so difficult to change our habits if the brain is believed to undergo constant changes? It needs to be noted that plasticity does not mean instability. Indeed, our everyday actions require stability to be performed in an efficient way without having to think about each individual movement. Repeating a particular action brings about the strengthening of connections among groups of neurons responsible for the action's performance. In consequence, those inter-neuronal connections become stable just like ruts in an old road. It means that repetition leads to rigidity by forming some kind of mental (and neuronal) "tracks", from which are difficult to extricate (Doidge, 2007; Schwartz and Begley, 2003).

Nonetheless, improvement of brain-damaged patients who were given carefully planned treatment including art therapy confirms the possibility of overcoming those difficulties. Moreover, the hitherto hidden artistic abilities are quite often revealed in the course of treatment (Pąchalska et al., 2014). It is worth pointing out that some of the patients reached a high level of their art of painting as was the case in a patient diagnosed with schizophrenia (Pąchalska et al., 2008). He was run over by a car after rushing out into traffic while actively hallucinating that he could fly. In effect, he suffered a brain contusion and remained unconscious for about 5 hours. After recovery he presented with disturbances of working memory, hemispatial neglect, and perseveration (Pachalska 2007; 2020). The tendency to perseverate was reflected in a series of his early paintings, which were composed of three overlapping human figures (see Fig. 1).

He has since become a prolific artist, who exhibits his paintings all over the world. At the same time, his works receive very good opinions from art critics (see Pąchalska et al., 2013, 2014). The critics also point out that his recent paintings progress towards symbolic art as exemplified in Figure 2.

The above-mentioned case, as well as the artistic achievements of other brain-damaged patients, seem to support the idea of neural Darwinism proposed by Gerald Edelman (1987). He presumes that the brain neuronal networks compete with each other to perform a given task. In consequence, the job is done by the network that performs the work best. Hence, any function may be taken over by other brain networks if the one (or some) of them has been impaired. That idea finds additional support in an experiment conducted by Pascual-Leone and co-workers (2000). The authors disabled the occipital lobes of blind persons with electromagnetic coil (Transcranial Magnetic Stimulation), and found that they lost fluency in reading Braille texts. It shows that in the blind visual occipital cortex plays a significant role in reading the Braille alphabet.

Fig. 1. Perseverated paintings produced by a patient diagnosed with schizophrenia
Source: Pachalska, Kaczmarek, Kropotov (2014)

Fig. 2. A recent symbolic painting of the same painter
Source: clinical material of M. Pachalska

The Power of Mental Force

Pascual-Leone et al. (1995) performed also an experiment that displayed the possibility of reconstructing neuronal circuits with the use of mental exercise alone. They trained two groups of people in skills of playing the piano. The first group's participants were asked to imagine they played and listened to a one-handed, five-finger piano exercise for two hours, for five days. The other group was actually playing the same sequence during the same period. Changes in cortical areas targeting fingers involved in the task were observed in both groups. Furthermore, the changes were limited to the cortical representation of the hand used in the exercise. The researchers noted the improvement of playing skills in both groups, although, naturally, the improvement produced by physical exercise was better. Yet, a two hour physical exercise resulted in a considerable improvement of the playing skills of the mental practice group.

Some neuroimaging studies have shown that mental and motor training activate distinct neural networks (Graybiel et. al., 1994; Lacourse et al., 2005; Nyberg et al. 2006; Olsson et al., 2008), while others indicate that the two types of training can affect similar, but not identical, neural circuits (Decety, 1996; Decety et. al, 1989; Jeannerod, 1994, 1995; Kosslyn et al., 2007; Lotze et al., 1999; Munzert et al., 2009). Slagter et al. (2011) argue that motor imagery depends on cognitive higher-order aspects of action control, and report studies that found higher activation of midline and lateral motor as well as midline frontal areas (see also Nyberg et al., 2006; Ranganathan et al., 2004). This research attests to the possibility of direct influence on motor networks, which might explain the improvement of performance following mental exercise. It might be of interest to remind the reader that such a possibility was presumed by Raymon y Cajal over one hundred years ago (see Doidge, 2007).

Recent years brought additional examples of changes in the brain work induced by meditation practice. Neuroimaging studies using functional magnetic resonance (fMRI) and positron emission tomography (PET) scans have shown that the changes include both alterations in connectivity among the various brain networks as well as structural changes (Brefczynski-Lewis et al., 2007; Davidson and Lutz, 2008; Fan, 2011; Lazar et. al., 2005; Lutz et al. 2008, 2009; Slaghter et al., 2011; Telles et al. 2015).

Fox et al. (2014) performed a meta-analysis on 21 brain imaging studies examining 300 meditation practitioners. They found:

> …eight brain regions consistently altered in meditators, including areas key to meta-awareness (frontopolar cortex/BA 10), exteroceptive and interoceptive body awareness (sensory cortices and insula), memory consolidation and reconsolidation (hippocampus), self and emotion regulation (anterior and mid cingulate; orbitofrontal cortex), and intra- and interhemispheric communication (superior longitudinal fasciculus; corpus callosum). (p. 40)

A follow up by Fox et al. (2016) suggests that different meditation styles are reliably associated with distinct brain activity. Activations in some brain regions are usually accompanied by deactivation in others but convergence is the exception rather than the rule. They conclude that the results should be interpreted with caution since theoretical bias as well as methodological limitations are of concern.

The above described studies concentrated on neuronal circuits in accordance with the "neuron doctrine" supported by Ramón y Cajal in his disputes with Golgi (Koob, 2009; Stahnisch and Nitsch, 2012). Recent research, however, shows that it is not only restructuring the neuronal networks that plays a significant role in brain plasticity but also so far neglected glia.

Glial Plasticity

Preoccupation with neurons had its roots in discoveries of electric capacities of the human body and hence the supreme importance of electrical signaling in neurons. In the eighteenth century, the interest in electricity was comparable to the fascination with quantum physics today. The interest was beefed up by electro-stimulations provoking contraction of muscles in dead bodies. Those demonstrations, often applied in a highly theatrical way, were meant to show the power of galvanism. Suffice it to mention the experiments of Giovanni Aldini (see Parent, 2004) who triggered contractions of various muscles by applying currents at different points along the head and body. Remarkably, he was able to produce expressions of smiling or grimacing in this way.

Glial cells were found to be electrically nonexcitable; hence they were excluded from further research. Recent studies, however, revealed that neurons do not function in isolation. In fact, they are embedded in complex glial networks in which astrocytes appear to play a very important role. They modulate multiple aspects of synaptic plasticity by releasing gliotransmitters, calcium signaling, and oscillations, coupling via gap junctions. Moreover, glia stabilize new synapses and provide ensheathment of synapses, which restricts the spread of neurotransmitter to neighboring synapses (Barker and Ullian, 2010; Chao et al., 2002; De Pita et al., 2016; Kandel et. al., 2016; Pirttimaki and Parri, 2013). In addition, astrocytes maintain hemodynamic control, metabolic demand homeostasis, and energy supply (De Pitta, 2016; Giaume et al., 2010; He and Sun, 2007; Navarrete and Araque, 2011).

Above all, astrocytes have proven to be able to communicate among themselves via calcium waves, which results in a release of glutamate, and is linked to hemodynamic control. They form complex communication nets that can generate various regulatory signals and bridge structures and networks that are otherwise disconnected from each other (Fields, 2010; Volterra and Meldolesi, 2005). Barker and Ullian (2010) state that:

> Astrocytes are organized into discreet domains, with the territory of a single astrocyte estimated to contact between 300 to 600 dendrites and upwards of 105 synapses…This extensive synaptic interaction …positions astrocytes to directly influence the structure and function of the synapse. Indeed, there

> is increasing evidence that astrocytes play active roles in controlling the number and strength of a neuron's synapses and thus may contribute to mechanisms underlying synaptic plasticity. (p. 40)

It was the introduction of fluorescent calcium indicators that enabled revealing the communicative role of calcium (Fields, 2010). Recent studies unveiled also the role of sodium in neuron-glia interaction and neuro-metabolic coupling. This is an important finding since cellular sodium homeostasis is of the upmost functional significance for the brain. Indeed, most of the brain energy is consumed by the Na+/K+-ATPases (Carter, and Bean, 2009; Howarth et al., 2012). Accordingly, altered sodium homeostasis is observed in brain pathological conditions (Rose, and Chatton, 2016).

Neurogenesis

We also cannot pass over neurogenesis, in which astrocytes have proved to play a significant role. The neurogenesis (the ability to generate new brain cells) takes place in the subventricular zone (SVZ), and the subgranular zone (SGZ) (Altman, 1969; Doetsch at al., 1999; Luskin, 1993) as well as in hippocampus (Seneki, et. al., 2001, Wang, and Bordey, 2008; Woollett, and Maguire, 2011). Generation of new neurons is largely restricted to the adult SVZ and SGZ, whereas astrocytes and oligodendrocytes are continuously born throughout life (Duan et al., 2008, Bruel-Jungerman, Rampon, and Laroche, 2007). Those new cells show unique electrophysiological characteristics, which makes them prone to undergo plasticity. It is a multi-step process modulated to a considerable degree by genetic, behavioral and environmental factors. A significant role of those newborn cells is facilitation of learning and memory due to the ability to incorporate into existing neural circuits. Indeed, Woollett, and Maguire (2011) observed an increase in the hippocampus volume in London taxi drivers. Significantly, no such changes were observed in bus drivers who used to drive a constrained set of routes (Maguire, Woollett, and Spiers, 2006)

On the other hand, environmental factors, stress in particular, result in a shrinkage of dendrites or even cell death (Sapolsky, 2004). Stress hormones deplete hippocampal neurons of glucose makings them sensitive to elevations of glutamate to which they have a toxic reaction. Moreover, stress hormones have been found to amplify the amygdala reaction to fear leading to a significant rise of hormones and neurotransmitters such as cortisol, noradrenaline, and epinephrine. It mobilizes body resources to deal with short-term dangers. In case of a prolonged stress, however, the hypothalamic-pituitary-adrenal axis (HPA axis) is pathologically activated leading to erosion of resilience and depletion of metabolic reserve (Le Doux, 2002, Sapolsky, 2004). The malfunction of HPA axis is believed to be linked to the development of depressive disorders (for review see Davey, Breakspear, Pujol, and Harrison, 2017; Gerhard, Wohleb, and Duman., 2016).

Accordingly, treatment of depression is directed to alter the abnormal level of cortisol. The therapeutic onset of antidepressant medication is delayed since the drugs used here activate the second messenger systems. It leads to increased gene activation and to a synthesis of proteins that enhance synaptic transmission. In effect, it enables new memory formations, learning new ways of coping with stressful situations, and enhancing future learning experiences (Duman, et al. 2001, 2004). In other words, the greater plasticity of the brain is acquired. Since such treatment takes time the research aiming at developing faster-acting therapeutic agents such as ketamine is being conducted (Duric, and Duman, 2013; Gerhard, Wohleb, and Duman, 2016; Medrihan, et. al., 2017). Yet the outcome of such procedures is still not sufficiently documented.

CONCLUSIONS

We remind the reader that new ideas do not suddenly appear out of a void in the mind of the individual. In the case of science, it is the refinement of research methods as well as an accumulation of the new data that make possible a new interpretation of the existing but so far neglected theories (see also Kaczmarek and Markiewicz, 2018). It also allows seeing the existing ideas in a new light as, for example, the possibility of changing the brain functions by mental effort alone. In addition, the emergence of new techniques created possibilities to determine the contribution of astrocytes to brain plasticity, and their role as integrators of neuronal network activity. Other significant manifestations of plasticity are alternations in the brain works induced by meditation practice. Neuroimaging studies using functional magnetic resonance (fMRI) and positron emission tomography (PET) scans have shown that the changes include both alterations in connectivity among the various brain networks as well as structural changes.

We also argue that brain plasticity is the rule and not exception. It enables humans as well as animals to deal effectively with the changes occurring in the environment, and, therefore, plays a significant role in learning. It can be best observed in the hippocampus where new cells are born in response to new experiences and training. Moreover, experimental evidence reveals a link between synaptic plasticity and the proliferation and survival of new hippocampal neurons. At the same time, sensitivity to environmental factors makes hippocampal cells prone to react adversely to harmful situational agents such as heavy trauma or stress leading to the hippocampus shrinkage. Hence, decreases in hippocampal volume can be also observed in people plagued with depression. Spectacular development of neurobiology made it possible to create new medications that are successfully used in the treatment of depression. Those antidepressants stimulate genes to create new proteins, and, consequently, cause alternations in the level hormones and neurotransmitters.

In sum, the data presented in the paper prompt to revise the idea of neuroplasticity. It is linked not only with alternations within neuronal circuits, and neurogenesis but with epigenetic changes as well. In fact, presently carried research has marked a new era not only in neuroscience but also in genetics. Current epigenetic research has thrown a new light upon factors that affect gene activity and expression. In effect, the contemporary idea of neuroplasticity includes not only cellular mechanisms but also environmental, and genetic factors. It also means that our brain is prewired and not hard-wired to a considerable extent (see Marcus, 2004; Merzenich, 2013).

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