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Cognitive reserve and cortical plasticity

Cristian Vasile[a]*

[a] *Education Sciences Dept., Faculty of Letters and Sciences, Petroleum-Gas University, Bd. Bucuresti 39, Ploiesti 100680 Romania*

Abstract

The term "cognitive reserve" describes resistance to the deterioration of cognitive functions in the dementia and other brain degradation of brain substance. Cognitive resistance is measured behaviorally, while neuropathological damage is assessed histologically and by imagistic methods or based on markers.
The study of cognitive reserve suggests that innate intelligence or life experiences (such as educational and professional achievements) may increase this reserve in the form of sets of behavioral skills that allow subjects to manage better than others cognitive pathology of a certain kind (e.g., Alzheimer disease). In these circumstances the psychological intervention for cognitive practice since the beginning of the pathology could contribute substantially to establishing or increasing the cognitive reserve.

Keywords: autonomy; behavior; teacher

1. Introduction

Cognitive dysfunction is a major problem becoming more common in modern society, not only in the third age population, but also in middle-aged adults. Attention and memory problems usually occur first, leading in their turn to the beginning of thinking disorders. If the first symptoms of these disorders are rejected by the patient (which they consider temporary imbalances due to fatigue, stress, reducing sleep period and so on) in time they can become serious problems, eventually becoming pathological and irreversible. The rejection of the symptoms by the patient's often occur as defense mechanism such as denying, or, according to cognitive psychotherapeutic approach, in the form of reasoning distortions such rationalization. On this background in recent years has appeared the concept of "cognitive reserve".

* Corresponding author.
E-mail address: clinical_psycho@yahoo.com

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The idea of a reserve of nervous tissue that remains unaffected when brain damage occurs, result from the repeated observation that there seems to always be a direct relationship between the degree of brain damage or brain lesions and clinical manifestations of such damage.

Katzman et al. (1989) described 10 cases of elderly who had normal cognitive level, but post-mortem revealed that they had an advanced stage of Alzheimer's disease. It has been speculated that these subjects have not had clinical manifestations of Alzheimer's dementia because their brains were larger than average. Similarly, a stroke can cause a certain profound functional impairment on a patient while the same stroke has minimal effect on another patient, in terms of its functionality. Given this evidence has been introduced the concept of cognitive reserve, which could explain the mentioned differences between people who have suffered the same degree of destruction of brain tissue.

2. Studies on cognitive reserve

In the late 80s, a study published in the Annals of Neurology, which reported results from post-mortem examination of over 100 elderly people, indicated that there is a discrepancy between the neuropathological damage caused by Alzheimer's disease and the clinical manifestation in the social area. These results show that some subjects, whose brain tissue underwent extensive degradation due to the pathology, had very little or no clinical symptoms. In addition, the study showed that these individuals had a larger brain weight and a greater number of neurons compared with the control group. The researchers set out two possible explanations for this phenomenon: subjects may have had Alzheimer's disease at an early stage, but somehow managed to avoid the loss of large numbers of neurons, or alternatively, have larger brains and more neurons and thus might say that they had a greater "reserve".

The study of cognitive reserve suggests that innate intelligence or life experiences (such as educational and professional achievements) may increase this reserve in the form of sets of behavioral skills that allow subjects to manage better than others cognitive pathology of a certain kind (e.g., Alzheimer disease).

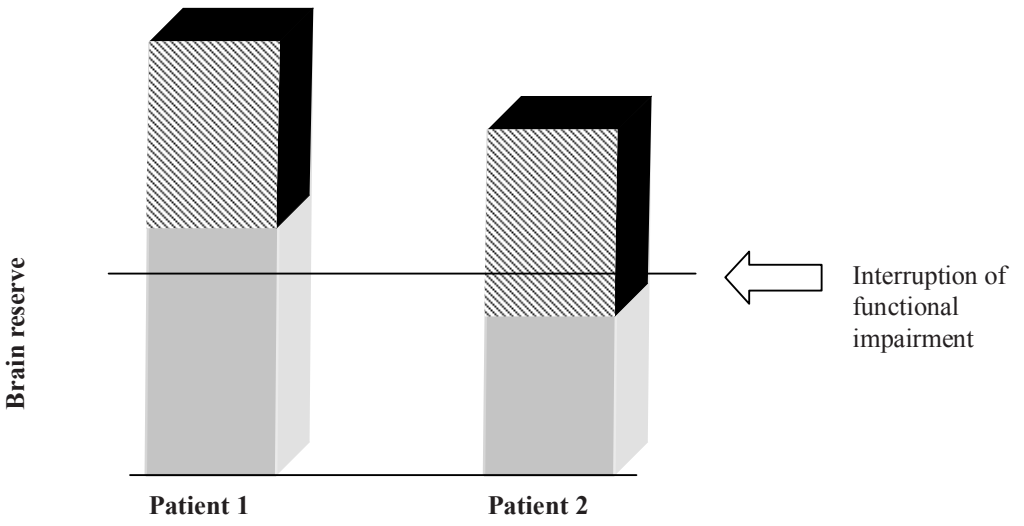

Fig. 1. Brain reserve threshold (Stern, 2002)

For two patients with different levels of brain reserve, an injury with some extent lead to clinically manifested deficiency to the person with a smaller brain reserve (Fig. 1, Patient 2), because it exceeds the threshold of brain damage sufficient to cause an functional impairment. However a person with greater brain reserve may remain unaffected in terms of cognitive and functional. (Stern, 2002).

3. Epigenetic resources for cognitive reserve

Deep neural pathways formed by education (formal - in the school, or informal - the individual life experiences) can constitute cognitive reserve in case of brain damage.

Until two decades ago it was believed that the central nervous system degenerate cells cannot be replaced. Failure for this to happen was attributed to the lack of neurotrophic factors that support the survival and reproduction/cell growth and neurotoxic factors that prevent remyelinization.

Neuroplasticity was explained by the partial recovery of the function of a system due to takeover of other nerve structures and the activation of the secondary circuit (which is actually also a total or partial function takeover) by adaptive cellular mechanisms (e.g., neural sprouting) or by relearn.

The epigenetic mechanisms have major roles in neurogenesis and in increasing the cognitive reserve. They are widely used for the formation and storage of cell information as response to transitional environmental signals. Cellular information storage is thus in some ways similar to the mechanisms of memory storage in the adult nervous system.

In addition, sustainable changes at the cellular level are triggered by a transient signal in each case. The mechanism is analogous to the behavioral memory formation in the central nervous system (Levenson and Sweatt, 2005).

A considerable amount of study, although indirect, suggest that disruption of epigenetic mechanisms is a cause for human the cognitive dysfunctions (Roberson and Sweatt, 1999; Levenson et al., 2004).

Some studies have found a relationship between educational level and prevalence of dementia (Katzman, 1993; Mortimer & Graves, 1993). Other studies on the incidence of dementia show that low educational level increases the risk of Alzheimer's disease and other types of dementia, and this phenomenon is explained by the concept of cortical reserve (Mortimer, 1988; Katzman, 1993; Satz, 1993). It is assumed that people with high levels of education have a higher encephalon, or a richer cognitive reserve than those with low education. As we have shown, taking into account the role of education, in certain pathological brain injury is more likely to occur cognitive symptoms in people with low cognitive reserve. According to Figure 1, the concept of cognitive reserve employs a threshold that must be exceeded before symptoms appear.

The concept of cognitive reserve is approached from other points of view in literature. While some authors refer to brain volume, others take into account the intensity of cerebral metabolism, and a third category of authors refers to the connectivity of neural networks - density of synapses and dendritic branching. All these approaches have the same ultimate objective - the efficiency of the brain functions - and can be integrated into one unified vision.

Consistent with the cognitive reserve theory, education is a derivation of this reserve. A person can attain a high educational level if that person's cognitive reserve is large enough. There are authors who suggest that intelligence could be a form of indirect measurement of cognitive reserve more valid than education (Satz, 1993), since it is determined by other factors than individual capabilities. For example, socio-economic downturn may limit an individual's opportunities to receive an appropriate education. Thus, cognitive reserve theory assumes a closer relationship between intelligence and dementia than between educational level and dementia.

When analyzing the cognitive reserve, we should take into consideration the patients' lifestyle. For instance, the cognitive processes are strongly influenced by substances consuming (e.g. caffeine, energy drinks - Anitei, Schuhfried & Chraif, 2011).

4. Conclusions

In either direction we go, studies show that cognitive reserve, closely related to neuroplasticity, is influenced by both genetic and epigenetic (by environment). Under these circumstances the emergence of new populations of neurons in the adult brain and their migration to other areas is determined by stimuli coming from the environment. The most important stimulus is probably an adaptogen one (social) and refers to education in general and learning in particular. Exercising the memory and the desire of understanding the environment, together with a proper relationship with it, is probably one of the strongest factors generating cognitive reserve. Education, in this context, refers not only to formal education, but also to other types of changing of some cognitive patterns, such as personal development, psychotherapy, counseling, learning and the usage of metacognitive strategies.

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