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Review

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The cognitive reserve model: origins, main factors of development, and clinical applicability

Modelo de reserva cognitiva: orígenes, principales factores de desarrollo y aplicabilidad clínica

Abstract

The Cognitive Reserve Model has been proposed as a way of explaining the interindividual variability in cognitive performance in young normal subjects, as well as in aging subjects, and in pathologies that affect cognitive function. The model proposes that the brain is able to compensate for deficiencies by means of resources formed through life experiences. This article reviews some historical antecedents of the Cognitive Reserve Model and its basic principles. The three main factors associated with the development of Cognitive Reserve are analyzed: education level, occupation, and leisure activities. Findings are presented in which the important role of Cognitive Reserve in old age and in diseases such as head trauma, AIDS, multiple sclerosis, schizophrenia, and others, are reviewed. We conclude with some implications of the Cognitive Reserve Model for clinical practice.

Keywords

Cognitive reserve model; cognitive impairment; cognitive function.

Resumen

El modelo de reserva cognitiva se propone como una forma de explicar la variabilidad interindividual en el desempeño cognitivo tanto en sujetos normales jóvenes, como en el envejecimiento y en algunas otras patologías que generan afectaciones de la función cognitiva. Desde este modelo el cerebro es capaz de compensar deficiencias gracias a los recursos que se han ido formando a través de experiencias de vida. En este artículo se revisan algunos antecedentes históricos del modelo de reserva cognitiva y sus principios básicos. Se analizan los tres principales factores asociados al desarrollo de la reserva cognitiva: el nivel educativo, la ocupación y la realización de actividades de ocio; y se presentan algunos hallazgos en los que se expone el papel que juega la reserva cognitiva en la vejez y en padecimientos como traumatismos craneoencefálicos, el SIDA, esclerosis múltiple, esquizofrenia, entre otros. Se concluye con algunas implicaciones del modelo de reserva para la práctica clínica.

Palabras clave

Modelo de reserva cognitiva, deterioro cognitivo, función cognitiva.

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Introduction

In the 1970s, some authors questioned the prevalent stance that cognitive decline was “inevitable” and “normal” due to aging. For example, Barton et al.¹ proposed that the older adult’s intellectual function exhibits a great interindividual variability that is dependent on factors of their personal experience and life history such as their educational trajectory, family life, work, and recreational activities, and postulated that cognitive deterioration is neither inevitable nor generalized. In this context, the interest in understanding the factors that promote optimal cognitive functioning during aging was reaffirmed, originating the concept of a reserve,² which later led to the cognitive reserve model proposed by Stern.³

The cognitive reserve model was developed mainly in the context of aging and dementia, although the literature also reported applications in pathologies that affect cognitive function, which expanded its applicability and increased the clinical, theoretical, and methodological relevance of its understanding. Therefore, the analysis of the literature we present here seeks to be a reference point to understand the origins of the cognitive reserve model, its main postulates, some factors related to its development, and relevant aspects of its applicability in clinical practice.

Background

In 1988, Katzman et al. conducted a study in a community of elderly residents of an asylum. They collected evaluations of the mental and functional state of 137 subjects, the clinical manifestations suffered, and, finally, postmortem studies. Dementia was present in 78% of the patients. However, the most interesting thing was that anatomopathological studies of the brains of the elderly subjects without clinical features of dementia (control subjects) revealed neuropathological characteristics similar to those of patients with clinical manifestations of Alzheimer’s disease. It was observed that

these brains had a greater weight and a greater neuronal density compared to the control subjects (residents of the same center and of the same age). The authors proposed two explanations: 1) these people might have had Alzheimer’s Disease in an incipient state, but somehow their brain had prevented the loss of a large number of neurons; 2) the patients had started with a larger brain and a larger number of neurons than the rest, so it could be said that they had a greater reserve.⁴

In the 1980s, Snowdon⁵ began a longitudinal study in a group of 678 members of a congregation of nuns, aged between 75 and 102 years. The postmortem brains revealed neuropathological characteristics compatible with Alzheimer’s disease in a significant number of participants, even though several of them showed only mild cognitive alterations that could not be considered dementia. Given that there were clear cognitive differences between the participants (some had symptoms of dementia and others enjoyed a healthy old age), Snowdon described two factors to explain them: 1) the degree of the pathology presented in the brain, and 2) the resistance level of its clinical expression (some participants presented a high level of pathology at necropsy, but few or no symptoms in life). The clinical manifestations of dementia were related to the education level and intellectual abilities of the participants. In the group with the highest education level and the highest intellectual level, there were greater discrepancies between the clinical manifestations and the postmortem neuropathological findings. Snowdon proposed that the clinical expression of brain alterations of Alzheimer’s disease can be modulated by a greater reserve, formed throughout life. Since then, several risk factors for dementia have been found in the patients’ backgrounds, such as social, education, cognitive, and health histories.

The cognitive reserve model

The concept of cognitive reserve was proposed by Baltes *et al.*⁶ who defined it as the capacity of a person’s cognitive system exhibited by a general

learning potential or plasticity. The cognitive reserve is associated with the system's capacity to increase the maximum potential through the optimization of the surrounding conditions.

Stern^{3,7} defines the cognitive reserve as a set of brain resources that allow the recruitment of additional neural networks or the execution of cognitive strategies for processing, thereby optimizing functioning through compensation mechanisms. These resources can be tapped under normal conditions such as ordinary aging or in situations in which a pathology affects functioning.⁸

Stern^{3,7} proposes two subcomponents to explain how the compensation of the system occurs: the neural reserve and the neural compensation. The neural reserve accounts for the differences in the healthy brain in the execution of cognitive processes. This implies that the system has the ability (or flexibility) to properly execute a cognitive scheme underlying a specific task, even when the application of the scheme is difficult. The difficulty in applying the scheme may be due to the increase in demands. Neural compensation, on the other hand, implies that the effectiveness of the system depends on the use of alternative cognitive schemes that make it possible to correct the difficulties caused by the breakdown of the standard cognitive scheme for that task, which has stopped functioning due to deterioration. The compensation is not a simple response to the difficulty, it implies an attempt to maximize functioning, confronting the brain damage by using structures or brain networks that are not involved in this processing when the brain is healthy. This compensation can maintain or improve the execution.

Individual differences in cognitive processing would reflect differences in the level of reserve, allowing some people to compensate for deterioration better than others. Stern⁷ observes there is no direct relationship between the severity of brain pathologies and the degree of impairments evidenced by patients, so he postulates that the reserve allows explaining these discrepancies. Thus, the level of reserve allows the brain to better

tolerate the effects of a certain pathology, which delays the appearance of cognitive symptoms.

The cognitive reserve is considered a dynamic factor that results from exposure to its environment,^{7,8} in which an active mental and physical life induces its development,⁹ so it is considered that there is a close relationship between neuronal plasticity and cognitive reserve. Life experience can influence brain anatomy through neurogenesis (generation of central nervous system cells), angiogenesis (the process in which new blood vessels are formed), promoting resistance to apoptosis (programmed cell death), and regulation of compounds that promote neuroplasticity.⁷

The life experiences linked to the cognitive reserve are education level, the degree of complexity involved in the work activity, the performance of physical and of leisure activities, some factors related to lifestyle, bilingualism, and some cognitive stimulation activities.¹⁰⁻¹² The following is a description of the factors most frequently linked to the cognitive reserve.

Education

Nucci *et al.*⁸ define the education level as the years of formal education. This factor proves to be protective of the normal cognitive decline of aging. Many studies have shown that adults with higher education levels have a better performance in cognitive tests and have a lower risk of suffering cognitive losses related to age and dementia, so it is a highly relevant factor for the formation of a high level of cognitive reserve.¹³⁻¹⁵

In the longitudinal study of the nuns by Mortimer *et al.*¹⁶ it was observed that the risk of suffering dementia increased in the participants with low education levels. The relationship between education level and cognitive deterioration is also observed in the pattern of deterioration suffered by some patients. For example, Stern¹³ indicated that patients with a higher education level show a faster cognitive deterioration in memory scores than those with a low education level, and Hall *et al.*¹⁴ reported that patients with Alzheimer's disease with higher education levels died more quickly than

those with lower education levels. This does not imply that high education levels harm the cognitive capacity, on the contrary. Patients with a higher education level have a greater cognitive reserve, present a higher threshold, and can tolerate more the clinical manifestations of the disease, so that the necessary pathology to affect them must be very severe. Considering that the progression of Alzheimer's disease is independent of the cognitive reserve, when the severity increases, the reserve stops being sufficient to correct the damage and the deterioration occurs quickly.

A low education level has also been related to a low cognitive performance in patients with HIV,¹⁷ hepatitis C,¹⁸ autosomal dominant cerebral arteriopathy with subcortical infarcts and leukoencephalopathy,¹⁵ multiple sclerosis,^{19,20} Parkinson's disease,^{21,22} Huntington's disease,²³ schizophrenia,²⁴ bipolar disorder,²⁵ and epilepsy.²⁶ Additionally, it is reported that those patients with a higher education level who have suffered head trauma have a better recovery prognosis than those with a low education level.²⁷⁻²⁹

Mortimer *et al.*¹⁶ propose three plausible explanations on how education modifies the expression of cognitive deterioration: 1) low education levels can increase the exposure to factors that increase the risk of dementia; 2) high levels of education can contribute to greater neuronal connectivity in the early stages of life, which persist into adulthood; and 3) high levels of education can be related to a cognitively stimulating life that promotes neuronal development.

Work demands

The factor of work-related demands refers to the social and cognitive requirements necessary for a person to face their work environment.⁸ Being exposed to high cognitive and social demands in their occupation is a factor that promotes a higher level of cognitive reserve.^{30,31} Stern *et al.*¹³ found that a lower level of working life is a factor related to the increased risk of suffering from dementia. A higher level of work is related to better cognitive performance, a relationship that has been established in patients with HIV,^{17,32,33} hepatitis

C,¹⁸ Huntington's disease,²³ multiple sclerosis,³⁴ cranioencephalic traumatism,²⁷ epilepsy,²⁶ bipolar disorder,²⁵ and schizophrenia.²⁴

Recreational activities

Recreational activities are an important part of daily life. They refer to the pursuits that generally take place during free time and, within these, we find leisurely activities that may involve cognitive, physical, or social demands. The level of participation in these activities is also relevant to the cognitive reserve level,^{11,30,35} since it has been reported that participating in activities such as reading, socializing with friends, traveling, learning new things, practicing sports, etc., is associated with a higher level of cognitive reserve and with a lower risk of suffering from dementia.

Scarce engagement in leisure activities has also been associated with cognitive deterioration in patients with multiple sclerosis,³⁶ schizophrenia,²⁴ bipolar disorder,²⁵ HIV,³² and Down syndrome.³⁷

Aging and cognitive reserve

The demographic transition is generating a global profile oriented to the aging population worldwide. The WHO³⁸ postulates that, faced with the increase of older adults, there is a need to develop measures to help them remain healthy and active.

Aging by itself is a process that profoundly affects cognition and the brain of individuals; however, one of the most notable characteristics of cognition in adulthood is that as age increases, the variability between subjects in cognitive abilities is greater. It has been proposed that this variability could be mediated by the cognitive reserve formed throughout life that, to a greater or lesser extent, allows to compensate through the efficient use of neural networks the brain's deterioration caused by aging—especially the presence of dementia. The cognitive reserve seems to be an important factor that determines who ages with grace and who suffers cognitive deterioration.³⁹ A low education level, low work level, and low participation in leisure activities during life will increase the risk of suffering from dementia in people over 60 years,^{13,39} while high levels in these three variables

are associated with a slowing down in the decline of memory skills, executive functions, and language skills.¹³ Also, it is reported that older people who continue to work or who are involved in different activities, even when these are not professional, have a better cognitive performance than those who do not.⁴⁰ Thus, although cognitive deterioration is associated with some cerebral structural changes related to aging, one can attempt to slow down its appearance through various protective factors.

The relationship between cognitive reserve and various pathologies

As in aging, the cognitive reserve can help explain individual differences in the cognitive effects of various diseases. Barnett *et al.*⁴¹ suggest that the hypothesis of cognitive reserve is important to reduce symptoms of neuropsychiatric disorders such as schizophrenia, bipolar disorder, and depression. In addition, the reserve is now considered as a factor to be studied in other diseases such as hepatitis C, HIV, Parkinson's disease, Huntington's disease, multiple sclerosis, epilepsy, cancer, heart disease, and cranioencephalic trauma. According to Barnett,⁴¹ the cognitive reserve impacts neuropsychiatric disorders in three ways: a) by increasing the risk of developing the disorder, b) in the expression of symptoms within the disorders, and c) in the functional outcome of the recovery process. In the review of the literature, we find that these three forms proposed by Barnett also occur in other non-neuropsychiatric pathologies. We summarize some of these findings here.

The Bronx Aging Study,³⁵ a prospective study of a group of older adults, analyzed how the levels of accomplishment of leisure activities were associated to the risk of presenting cognitive impairment as a consequence of vascular factors. Participation in recreational activities involving cognitive demands—such as reading, writing, performing crossword puzzles, playing cards, or playing a musical instrument, among others—was associated with a lower incidence of vascular cognitive deterioration. In a period of 21 years, 71 of the 401 participants who were initially free of cognitive impairment developed vascular

cognitive impairment. The participants with the highest number of leisure activities with cognitive demands reduced the risk of suffering vascular cognitive deterioration in 55% compared with those with a lower level of activities. This persisted even after controlling for variables such as age, gender, education, and chronic diseases. This study highlights the increased risk of developing this disorder due to a low cognitive reserve.

This has also been reported in multiple sclerosis where it was found that a high level of cognitive reserve protects and delays the onset of the disease. Mestas, Salvador, and Gordillo⁴² studied a group of patients with this pathology and analyzed their schooling and occupation. They found a positive correlation between the level of schooling and the onset of the disease, so they concluded that these factors somehow determine symptoms to either manifest early or to slow down.

The evidence suggests that cognitive reserve can be a factor of resilience in various pathologies modulating the expression of cognitive symptoms associated with various disorders. In patients with autosomal dominant cerebral arteriopathy with subcortical infarctions and leukoencephalopathy, Zieren *et al.*¹⁵ analyzed the relationship between levels of cognitive reserve, levels of brain pathology (using magnetic resonance imaging), and cognitive performance. They found that the education level modulated the cognitive deterioration in patients who had mild or moderate cerebral pathology levels. Patients with high education had better cognitive performance in the evaluated functions than those with low education level. Adell-Serrano *et al.*⁴³ obtained similar results in patients who had suffered a stroke. They assessed the level of cognitive reserve (parents' schooling as well as their own, participation in training courses, work activity performed, level of musical training, ability with languages, reading activity, and participation in intellectual games) and the level of cognitive deficit. They observed that in patients with high cognitive reserve the level of cognitive deterioration was mild. Alladi *et al.*¹² analyzed the correlation between being bilingual (considering it a cognitive reserve measure) and suffering

cognitive deterioration after a non-disabling cerebrovascular accident. They observed that the group of bilingual patients had a lower proportion of cognitive deterioration than the monolingual group. Thus, bilingualism is considered a protective factor of cognitive deterioration after a stroke.

In psychiatric conditions, Forcada *et al.*²⁵ investigated the utility of the level of cognitive reserve (education level, occupation, participation in leisure activities, and premorbid IQ) to predict the psychosocial and cognitive performance of patients with bipolar disorder. They found that reserve levels are predictors of psychosocial and cognitive performance. Anaya *et al.*⁴⁴ confirmed that the level of reserve (premorbid IQ, education level, and occupation) correlated positively with levels of cognitive and psychosocial function as well as the quality of life in euthymic bipolar patients. On the other hand, de la Serna *et al.*²⁴ analyzed the relationship between levels of cognitive reserve (premorbid IQ, education level, and participation in leisure activities) and the neuropsychological performance of a sample of children and adolescents with first-episode schizophrenia. The level of the cognitive reserve was able to predict the scores in working memory and attention in a follow-up of two years.

In patients with temporal lobe epilepsy, the relationship between cognitive reserve (premorbid IQ, education level, and occupation level) and cognitive functioning was analyzed. Patients with low levels of cognitive reserve had a lower performance than the group of patients with high reserve in various measures of cognitive function, which suggests that a higher level of cognitive reserve may be an important factor to reduce cognitive decline in temporal lobe epilepsy.²⁶

Lifshitz-Vahav, Shnitzer, and Mashal³⁷ reported that in a group of adults with intellectual disabilities, including participants with Down syndrome, participation in recreational activities was positively correlated with performance in cognitive tests.

Bonner-Jackson *et al.*²³ examined the relationship

between cognitive reserve and longitudinal change, both in brain volume and in cognitive changes, in patients in the prodromal phase of Huntington's disease. High reserve levels were associated with fewer changes in cognitive function and with a lower rate of volume loss in the putamen and caudate nucleus for patients who were close to the onset of the motor disorder. In patients with Parkinson's disease, the relationship between mild cognitive impairment and cognitive reserve (education level and IQ) was analyzed. It was found that higher levels of cognitive reserve reduced the chances of suffering mild cognitive impairment.²² Lucero *et al.*,⁴⁵ also in patients with Parkinson's, found that cognitive reserve (education level) modulated the correlation between cortical accumulation of Beta-amyloid and cognitive deterioration. In patients with fewer than 16 years of schooling, the accumulation of Beta-amyloid was associated with a higher incidence of cognitive impairment, while in patients with higher education there was no correlation. The high education levels allowed maintaining a good cognitive level despite the cortical accumulation of Beta-amyloid.

Some researchers who have analyzed the relationship of cognitive reserve with brain atrophy and cognitive efficiency in patients with multiple sclerosis have found that cognitive reserve can be a means to reduce or prevent cognitive problems in this disease. Cognitive reserve was measured through IQ,³⁶ education level,^{19,36} work activity,³⁶ and participation in leisure activities.^{36,46} Amato *et al.*³⁶ found, in the first evaluation, that both the reserve levels and the level of brain atrophy reliably predicted the cognitive efficiency of the participants. In a second evaluation, only the level of cerebral atrophy and age predicted cognitive deterioration. For their part, Pinter *et al.*¹⁹ found that the level of brain atrophy as much as the cognitive reserve can predict cognitive efficiency in patients, a finding similar to that reported by Sumowski *et al.*⁴⁶

In the context of some infectious diseases, for example, in patients with HIV, Stern *et al.*¹⁷ analyzed the influence of cognitive reserve on neuropsychological performance in asymptomatic

seropositive patients. Seropositive patients with lower levels of reserve (education level, occupation, and vocabulary) had greater cognitive deficits and earlier deterioration, compared with seropositive patients with high levels of reserve, similar to what was reported by Sheppard *et al.*³³ Fazeli *et al.*³² also found a relationship between the level of activity (physical exercise, social activities, and occupation) and the cognitive performance of participants infected with HIV. This same tendency occurs in patients with hepatitis C when relating the cognitive reserve (education level, occupation, and vocabulary)¹⁸ to cognitive deterioration.

With respect to the role of the cognitive reserve as an intervening factor in recovery processes, García-Molina *et al.*²⁷ analyzed the recovery process in patients who had suffered moderate or severe head trauma. They divided the group into two, according to the level of premorbid cognitive reserve (education and occupation). After completing the treatment to which they were subjected, they observed that the group with the greater cognitive reserve (daily activities, cognitive function, social and emotional skills) had a greater functional capacity than the group with a lesser cognitive reserve. The results suggest that the level of cognitive reserve may be an important factor in the recovery processes of patients affected by moderate or severe cranioencephalic trauma. These results are similar to those observed by Rasseovsky *et al.*,²⁹ who also found a relationship between the performance of leisure activities and recovery with respect to long-term cognitive, social, and functional performance, regardless of the severity of the injury. Schneider *et al.*,²⁸ when doing a retrospective study of the database of the National Institute on Disability and Rehabilitation Research, also in patients with moderate or severe traumatic brain injury, found that the education level taken as index of cognitive reserve was a powerful predictor of achieving a full recovery without any disability one year after the injury. As the years of education increase, the probability of post-recovery disability decreases. This suggests that the cognitive reserve could be a factor that drives the neural adaptation during the recovery of traumatic brain injury.

Finally, it has also been observed that the cognitive reserve increases the resistance to the effect of some treatments. For example, Legendre, Stern, Solomon, Furman, and Smith⁴⁷ analyzed the difference in performance in a deferred verbal memory task between two groups of depressed patients undergoing electroconvulsive therapy. The groups were assigned according to the level of cognitive reserve (education and occupation). It was observed that memory loss was reduced in patients with greater cognitive reserve compared to patients with lower reserve when the information retrieval task was performed after electroconvulsive therapy.

Implications of cognitive reserve for clinical practice and future directions

The cognitive reserve model seems fitting when trying to understand the discrepancy in cognitive performance among patients with similar pathologies that affect cognition. Its inclusion in the clinical diagnosis is fundamental since it will allow having a better idea about the prognosis. It is also important to consider the level of cognitive reserve as a factor with an impact on treatment outcomes, so keeping it in mind can help develop more appropriate intervention strategies.⁴⁸ Considering the cognitive reserve can also allow the identification of patients at risk of suffering a cognitive deterioration before the symptoms appear.⁴⁹ Thus, the cognitive reserve model can provide information that favors better management of patients, facilitating the process of clinical diagnosis, and the implementation of more effective treatments.

The cognitive reserve should also be recognized as a factor that will influence the rate of cognitive deterioration after diagnosis. Thus, patients with different levels of reserve will have different deterioration patterns, which may result in differences in the manifestation of symptoms that can be mistakenly attributed to the treatments, especially when patients participate in clinical trials.⁴⁸

As a preventive measure, the cognitive reserve can be considered a factor capable of developing

at any stage of the life cycle, which can favor the delay or attenuation of deterioration, so strategies should be sought to promote its formation at all stages of life. An important point in this context is that the cognitive reserve, rather than being an entity pre-established during development, is a dynamic process during development as much as throughout aging, which has a crucial implication for cognitive function in the later stages of life.⁵⁰ In this sense, although the exact “recipe” to help create this reserve is still unknown,⁴⁸ the promotion of the formation of this reserve through intellectual enrichment could help to avoid, delay, or mitigate cognitive deterioration. It is important, however, to be careful not to present these activities as established treatments or fully tested preventive strategies against dementia.⁴⁸

Although in recent decades there is greater knowledge about the cognitive reserve, which has given rise to a model that integrates biological and psychological information, it is still necessary to conduct more research on the subject. Research should include longitudinal and epidemiological studies of diseases that affect cognitive function, using various study techniques such as functional neuroimaging, electrophysiological techniques, etc. Intervention studies are also needed to clarify how the causal links between life experiences, individual characteristics, and cognitive reserve are established⁴⁸ and how some neurodevelopmental disorders can affect cognitive reserve formation throughout life.⁴¹

Conclusions

The cognitive reserve model begins in the context of aging; however, it has proven to be a model applicable to various pathologies that affect cognitive function. Its applicability for diagnosis, treatment, and prevention has been established in the scientific literature. It is important to increase the body of knowledge in this regard and promote the application of the model in clinical practice, which favors better clinical management of patients suffering from ailments such as AIDS, hepatitis C, head trauma, heart attacks, multiple sclerosis, Parkinson's disease, Huntington's disease, depression, bipolar disorder, schizophrenia, and epilepsy, among others.

Conflicts of interest

In this scientific report, there are no potential conflicts of interest for any of the authors.

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