

THE ROLE OF COGNITIVE RESERVE IN PREDICTING COGNITIVE EFFICIENCY**DOI: <https://doi.org/10.26758/13.1.3>**

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Abstract

Objectives. The objective of the study is to assess cognitive reserve and to investigate the role of age and educational instruction level in cognitive efficiency.

Material and methods. All 146 participants, 105 women (72%), 41 men (28%), aged 60-96 years ($M = 74.61$, $SD = 7.12$), with primary to postgraduate studies ($M = 3.08$, $SD = 1.54$) completed the following test battery: questionnaire "Cognitive Reserve Index" (R-IRCq), Minimal Assessment of Cognitive Status-2 (MMSE-2) and Montreal Cognitive Assessment (MoCA).

Results. The educational level as well as the total cognitive reserve index are significant predictors of cognitive efficiency measures. Age and total R-IRCq score cover 32% of MoCA variance. Age and educational level cover 36% of the MoCA variance (adjusted $R^2 = 0.36$, $F(2.143) = 42.05$, $p < .001$), age ($B = -0.08$, $\beta = -0.27$, $t = -3.77$) and educational level ($B = 0.62$, $\beta = 0.43$, $t = 5.90$).

Conclusions. An inverse correlation between age and cognitive efficiency has been identified: the older the age of participants, the lower the cognitive efficiency, the stronger the correlation when evaluated by MoCA. Both educational levels and total R-IRCq index partially mediated the effect of age on cognitive performance (MoCA). The assessment of cognitive reserve in older people could be a useful additional measure to integrate existing protocols for the neuropsychological assessment of cognitive decline. Cognitive reserve should also be recognized as a factor, which will influence the rate of cognitive decline after diagnosis.

Keywords: MMSE-2, MoCA, cognitive reserve, education, cognitive decline.

Introduction

It is estimated that the number of people with dementia will rise to 152.8 million cases in 2050 (Nichols et al., 2022), being among the major public health problems.

Age-related dementia and normal cognitive decline can be explained by several theories, all of which have alternative but interrelated etiological hypotheses.

Over time, accumulation of predisposing factors may increase the risk of developing dementia, making longitudinal studies, as well as studies analyzing protective factors, necessary to fully understand the models of dementia analysis (Kivipelto et al., 2006).

Qiu and Fratiglioni (2011) consider that a model of dementia analysis throughout life can be a stronger method of studying this chronic disorder, often characterized by long latency periods.

The brain reserve hypothesis has been discussed in detail by Christensen et al. (2007, pp. 82-83), explaining that "the high level of pre-morbid intelligence, education, active lifestyle, or brain volume provides a spare capacity that protects the individual from the negative effects of aging and disease on brain function".

Many researchers proposed **passive models**, including Katzman (1993; brain reserve) and Mortimer, Schuman and French (1981; neural reserve, pp. 3-23). This type of model has also long **been implicitly adopted by most clinicians**. In passive models, reserve is defined in terms of **the sum of the damage** that may be incurred before reaching a threshold for clinical expression of the disease and refers to the positive correlation between brain size and the ability to resist pathological processes without showing clinical signs.

Passive models postulate the presence of a neuropathological threshold that is higher for high reserve people, and only beyond which cognitive impairment begins to take place. Therefore, they predict that the *rate of cognitive decline* will be slower, or delayed, for high reserve individuals who have not yet reached their neuropathological threshold, even if their *rate of neurobiological degradation* is comparable to that of low reserve individuals. However, this view is problematic for examining normal age-associated declines because it is very clear that age-related effects on cognitive performance begin in early adulthood and are continuous rather than abrupt (Salthouse, 2004). A continuum-based passive model might instead predict that high reserve individuals respond to neurobiological degradation to a lesser extent than do lower reserve individuals (i.e., differential preservation), although some passive models (Stern, 2002), are difficult to distinguish from functional threshold models.

The threshold model, revised critically by Satz (1993), is one of the best argued passive models of "brain reserve" (BR). There are many reasons why the threshold model can be considered a passive model. First, this type of model assumes that there is a fixed limit or threshold from which functional impairment will occur. In the case of Alzheimer's disease, this threshold could be the exhaustion of synapses until a minimum number is reached. Second, the threshold models are essentially quantitative models. Although the model recognizes that there are individual differences in the brain reserve, it does not consider them and how the brain processes cognitively or functionally tasks in the conditions of disruption caused by damage. It also does not address potential qualitative differences between different types of brain lesions.

In active models, the reserve is constituted by differentiating the modes of load processing (Stern, 2009). In **active backup models** the emphasis is on the ability of the brain to compensate brain damage. Thus, at least two concepts are described: **cognitive reserve** and **compensation**.

The first is **cognitive reserve (CR)**. Stern (2002) proposes that this type of reserve should be considered a normal process, used by healthy people when coping with daily cognitive tasks. The second concept is **compensation**: it involves the use of brain structures or networks that are not normally used by people without brain changes to compensate for the occurrence of damage.

The concept of cognitive reserve is parallel to the concept of cerebral reserve, being a potential mechanism of resilience to brain damage.

Passive models, the brain reserve, take anatomical brain differences into account to determine who has more or less brain reserve.

Therefore, there is no explanation of these models for how the educational level, the professional achievement level, or the intelligence quotient, influences the reserve, other than to

assume that these experiences must somehow alter the anatomy of the brain. Instead, in active models, it's not based on gross differences in the anatomy of the brain.

Cognitive reserve is defined as the residual variation in cognitive performance between individuals that is not explained by brain volume and does not manifest by any directly measured cognitive or cerebral performance (Nillson & Lövdén, 2018). An example would be that, of two individuals with the same level of structural capacity of the brain, the one with a higher level of CR can tolerate a larger brain lesion and thus maintain a better cognitive level than the other (Nillson & Lövdén, 2018).

One of the most remarkable discoveries regarding the cognitive reserve hypotheses was that of Snowdon et al. (1996), a study in which he demonstrated that the level of language proficiency among nuns at the mean age of 22 was a predictor of their cognitive performance and the risk of developing Alzheimer's disease around 58 years later. A similar study of Whalley et al. (2000) found that, compared with the control group, people with dementia above the age of 72 had significantly lower performance when tested for cognitive ability at 11 years of age.

Brain reserve and cognitive reserve are not mutually exclusive, in that the brain reserve does not protect against the accumulation of pathology but protects against its negative effects (Stern et al. 2020). Instead, they influence each other - life experiences and involvement in stimulating cognitive activities can alter brain anatomy (i.e., neurogenesis, angiogenesis and apoptosis resistance) and positively regulate compounds that promote neural plasticity (Stern, 2009).

The concept of cognitive reserve has progressively evolved so that it occupies a central place in the literature on normal and pathological aging, despite the theoretical pitfalls and the methodological controversy generated by years of studies and concepts associated with reserves.

Several studies have shown that CR indicators (proxies) can reduce the rate of conversion to dementia in subjects with identical rates of pathological burden of Alzheimer's disease (Stern et al., 1995; Brayne et al., 2010) and even have a protective role against cognitive deterioration associated with changes in the white matter of the brain or changes in the ventricles of the brain (their enlargement) (Brickman, Muraskin, & Zimmerman, 2022; Schmidt et al., 2011), delay in the occurrence of clinical symptoms (Sunderman, et al., 2016). Understanding the role of these indicators in predicting cognitive trajectories serves a twofold goal, either through prevention or by diagnosing the disease.

Different indicators of cognitive reserve have been identified (Farina, Paloski, de Oliveira, de Lima Argimon, & Irigaray, 2018), but recent systematic reviews indicate that education, work, leisure activities and social involvement are the most common and most often used in research (Chapko, McCormack, Black, Staff, & Murray, 2018; Chen et al., 2019; Harrison, Maas, Baker, & August, 2018). Education appears to have a profound, protective effect on long-term cognition and is one of the most widely studied factors (Qiu, Bäckman, Winblad, Agüero-Torres, & Fratiglioni, 2001).

The number of years of formal education is the most constantly used in studies. A protective effect of education for age-related cognitive decline appears to lead to higher levels of CR (Pavão Martins et al., 2020; Boots et al., 2015).

Moreover, it should be borne in mind that the educational experience is not the same for all individuals, which can influence its potential impact as a proxy of the cognitive reserve.

The protective effect of education not only mediates the transition between normal and pathological aging, but also between stages of cognitive deterioration.

Different trades and leisure activities can also have protective effects on cognitive decline and dementia (Serra et al., 2017). In fact, it is known that involvement in lifelong mental stimulation activities can promote brain neuroplasticity (Then et al., 2014). In terms of work, cognitively demanding working conditions are associated with a low risk of cognitive decline in older adults (Dekhtyar et al., 2015).

Middle-aged individuals at risk of developing Alzheimer's disease (decreased volume of the hippocampus and increased cerebral atrophy) who had occupations requiring complex cognitive activity maintained a similar level of cognitive performance as those with lower pathology (Karp et al., 2009; Baldivia, Andrade, & Bueno, 2008). However, since higher levels of education are usually associated with jobs that are more cognitively demanding, there is still controversy surrounding the hypothesis that the protective effect of education is or is not independent of the levels of complexity of work at middle age (Dekhtyar et al., 2015; Karp et al., 2009). Furthermore, a synergistic effect of education and work on the risk of developing Alzheimer's disease has been described by Stern et al. (1994), when combined with cognitively demanding work in adulthood.

As regards involvement in leisure activities, it was also associated with a low risk of developing Alzheimer's disease (Scarmeas, Levy, Tang, Manly, & Stern, 2001; Verghese et al., 2003) and protective effects against cognitive decline (Ribeiro, Monteiro, & Pereira, 2017; Wang et al., 2013). At the same time, studies show that elderly with aims in social and cultural life have a better cognitive and emotional status (Rada, 2018, 2020).

As a result of the above, it becomes important to identify the set of factors, which differentiate some individuals from others because promoting lifelong protection factors can help to combat the negative consequences of pathology through resilience mechanisms, such as brain and cognitive reserve.

This study aims to evaluate cognitive reserve in normal elderly people, to examine and assess the impact of the education on building cognitive reserve and to investigate the role of age and educational instruction level in cognitive efficiency.

Based on the model proposed by Nucci, Mapelli, and Mondini (2011) using as parameters of the cognitive reserve: education, the level of professional achievement and involvement in social and leisure activities, the literature review and foregoing discussion, the following hypotheses, stated in formal fashion, are proposed:

H1: there is a positive relationship between CR and education;

H2: cognitive reserve and high level of education are predictors of cognitive efficiency, with a slowing effect on cognitive decline.

Material and Methods

Tools

In this study, the CRI-q questionnaire was used to assess cognitive reserve used in most research studies, Mini-Mental State Examination, 2nd Edition™ (MMSE-2) and Cognitive Assessment-Montreal Cognitive Assessment (MoCA) as cognitive efficiency tests.

A. The **CRI-q questionnaire** includes some demographic data (date and place of birth, sex, place of residence, nationality, marital status) and 20 items grouped in three sections: education, work and leisure, each of which creates a sub-score (Nucci, Mapelli & Mondini, 2012).

The fidelity of the R-IRCq questionnaire was assessed in an earlier study from the two previous perspectives: the internal consistency, calculated by the fidelity coefficient Cronbach alfa (0.78), and stability of the results over time, assessed through test-retest on a panel of 40 subjects, was 0.87[0.78% IC: 0.74-0.93].

B. **MMSE-2** (Mini-Mental State Examination, 2nd Edition™) - MMSE-2® is a standardized clinical examination for cognitive impairment (Test Central, 2013).

C. **Montreal Cognitive Assessment (MoCA)** was validated as a highly sensitive instrument for early detection of mild cognitive disorder (MCI) in 2000. MoCA has subsequently been adopted in clinical settings around the world and is widely used in academic and non-academic research. MoCA sensitivity for MCI detection is 90% compared to 18% for MMSE (Nasreddine et al., 2005).

MoCA accurately and rapidly evaluates: short-duration memory, visual spatial skills, executive functions, attention, concentration and working memory, language, and language,

D. Clinical Dementia Rating (CDR) scale

It distinguishes five stages of disease severity: CDR. = 0: Healthy Subject, CDR. = 0,5: Uncertain Dementia, CDR. = 1: Mild Dementia, CDR. = 2: Moderate Dementia, CDR. = 3: Severe Dementia. The information is obtained through a semi-structured interview with the patient and a caregiver. Six areas are evaluated: memory; orientation; judgment and problem solving; social behavior; socio-professional behavior and personal care (Hughes, Berg, Danziger, Coben, & Martin, 1982, as cited in Psychology Monitor, n.d.).

Participants and procedure

A sample of 208 participants, from Bucharest, recruited with the help of two specialized clinics (neurology and psychiatry) and the White-Yellow Cross Foundation, took part in the study on a voluntary basis, from October 2018 to December 2018.

The criteria for selecting participants were:

- (a) the absence of a diagnosis of dementia
- (b) the absence of psychiatric illnesses which could impair cognitive functioning,
- (c) the absence of pharmacologically uncompensated systemic neurological or chronic disorders that could impair cognitive function,
- (d) without a history of stroke, brain injury or head trauma
- (e) autonomy in everyday life.

The study excluded 62 participants because: 14 participants assessed by the psychiatrist had scores on clinical evaluation of dementia possibly indicating mild to moderate cognitive deterioration, 26 participants were diagnosed with major depressive disorder, and 22 participants had a history of stroke.

All participants recruited for the study received the above-mentioned tests and also conducted a semi-structured interview (performed by the neurologist): to examine and exclude the following pathologies with possible negative impact on cognition: known neurodegenerative diseases other than dementia, psychiatric, neurological and chronic syndromes, systemic disorders not compensated by pharmacological treatment, strokes, brain lesions and head trauma.

Each psychological evaluation for every participant took approximately 90 minutes, with breaks offered at the request of the participants.

The Clinical Dementia Rating Scale (CDR.) was used to distinguish between healthy participants, with no evidence of cognitive impairment (CDR. = 0), and those with mild cognitive impairment (CDR. = 0.5) or Alzheimer's disease (CDR. = 1)

This measure was preferred to be used as a classification criterion because MMSE-2 and MoCA scores were used in the statistical model as result variables.

Ethical considerations

To ensure that human rights are respected among all participants in all phases of research, the study followed the Helsinki Declaration. All participants signed a consent form declaring their voluntary participation in the research and were fully informed about the confidentiality, content and aim of the study.

Statistical analysis

The study involves the use of a package based on descriptive, inferential and correlation statistics. The use of specialized statistical packages was called for in this regard: JASP 0.16.2 and IBM SPSS 21. To analyze the data and verify the research hypothesis the following statistical procedures were performed: correlation analysis, regression analysis and mediation analysis. The normality condition of the data was verified by graphical analysis of histograms and calculation of asymmetry and bolting indicators. Their compliance was verified against the values set out in the literature (Brown & Greene, 2006; Blanca, Arnau, López-Montiel, Bono, & Bendayan, 2013).

Results

Of the remaining 146 participants, 105 were women (72%), 41 men (28%) aged 60-96 years ($M = 74.61$, $SD = 7.12$) and with primary to postgraduate studies ($M = 3.08$, $SD = 1.54$)

Educational attainment was reported according to the International Standard Classification of Education (ISCED): Primary (0-4) - Level 1, Secondary (5-8) - Level 2, Secondary (9-12) - Level 3, Post-secondary - Level 4, High Short-Term - Level 5, Bachelor's or equivalent - Level 6, Master's or equivalent - Level 7, Doctorate or equivalent - Level 8

The total scores obtained with the R-IRC-q questionnaire ranged from 67 (low cognitive reserve) to 161 (high cognitive reserve). Lowest scores were obtained on the leisure time sub score, even in those whose composite score indicated high cognitive reserve.

Table 1 shows the means and standard deviations of the R-IRCq questionnaire scores and the neuropsychological tests in the sample.

Table 1

Mean, Standard Deviations of R-IRCq Questionnaire and Neuropsychological Tests

	Age	Level of studies	Total R-IRCq	MMSE-2	MoCA
<i>M</i>	74.61	3.08	105.96	27.85	24.36
<i>SD</i>	7.12	1.54	21.03	1.87	2.21

Note. 146. R-IRCq - Cognitive Reserve Index Questionnaire, MMSE-2 - Cognitive Status Minimal Assessment 2. MoCA - Montreal Assessment Cognitive Assessment

All correlations between predictors and the two cognitive efficiency measures were significant with $p < .001$. Table 2 shows the correlations between the score obtained on the R-IRCq questionnaire and the MMSE-2 and MoCA neuropsychological test scores.

Table 2

Cognitive reserve correlations, MMSE-2, MoCA, age and studies

Variables	R-IRCq	Level of studies	Age/years	MMSE-2	MoCA
	total				
1. Total R-IRCq	—				
2. Level of studies	.75***	—			
3. Age/years	-.42***	-.43***	—		
4. MMSE-2	.28***	.31***	.20*	—	
5. MOCA	.49***	.55***	-.46***	.72***	—

Note. *** $p < .001$, * $p = 0.013$

Significant inter-predictor correlations were demonstrated across the sample: both total R-IRCq score and educational level are negative with age. Significant ($p < 0.001$) was covariance between age and educational level. This study also confirmed a significant positive correlation between total R-IRCq score and educational level ($r = .75, p < .001$).

Correlation between MMSE-2 and MoCA scores was also significant ($r = .72, p < .001$).

As expected, age-related cognitive decline is more evident by MoCA score ($r = -.46, p < .001$) than by MMSE-2 score ($r = -.20, p = 0.013$).

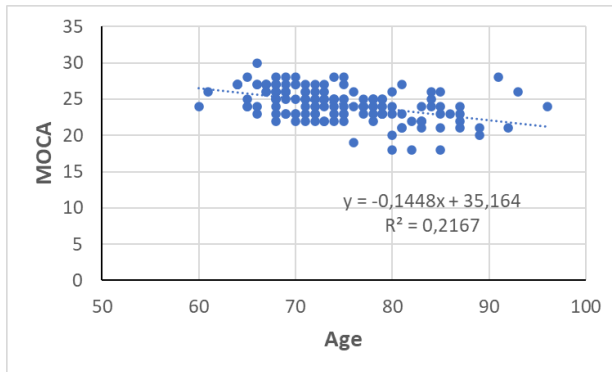
The first linear regression analysis investigated the correlation between age and dependent variables of the two cognitive efficiency measures (MMSE-2 and MoCA) (see Appendix A)

1. the regression equation was significant for MMSE-2 (adjusted $R^2 = 0.035, F(1.144) = 6.31, B = -0.05, \beta = -0.20, t = -2.512, p < 0.05$).

2. in relation to MoCA, the regression equation was significant (adjusted $R^2 = 0.21, F(1.144) = 39.83, B = -1.49, \beta = -0.46, t = -6.311, p < .001$) (figure 1).

Figure 1

Age dependence of MoCA across the sample



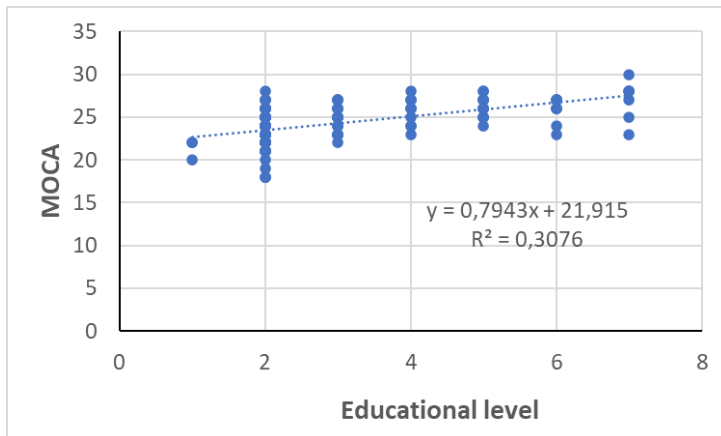
The predictive significance of the level of studies on the same measures of cognitive efficiency was evaluated in the second sandstone analysis.

1. the regression equation was significant for MMSE-2 (adjusted $R^2 = 0.094$, $F(1.144) = 15.98$, $B = 0.38$, $\beta = 0.31$, $t = 3.999$, $p < .001$).

2. the regression equation was significant for MoCA (adjusted $R^2 = 0.30$, $F(1.144) = 63.97$, $B = 0.79$, $\beta = 0.55$, $t = 7.999$, $p < .001$) (figure 2).

Figure 2

Educational level dependence of MoCA



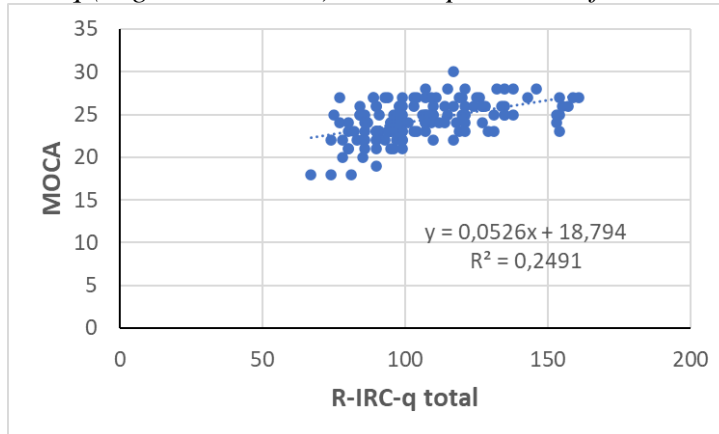
In the third s analysis, the predictive character of the total R-IRCq score on the same variables was assessed.

1. relative to MMSE-2, the regression equation was significant (adjusted $R^2 = 0.073$, $F(1.144) = 12.37$, $B = 0.025$, $\beta = 0.28$, $t = 3.518$, $p < .001$).

2. the regression equation was significant for MoCA (adjusted $R^2 = 0.24$, $F(1.144) = 47.75$, $B = 0.053$, $\beta = 0.49$, $t = 6.911$, $p < .001$) (figure 3).

Figure 3

Total R-IRCq (cognitive reserve) score dependence of MoCA scores in study sample



It is noted that the most significant predictor of MMSE-2 and MoCA scores is educational level.

The fourth regression analysis evaluated the combined predictive character of predictors (age and total R-IRCq score) associated with the dependent variable (MoCA scores), regression equation was significant. The two predictors account for 32% of the MoCA variance - model one, cognitive reserve (adjusted $R^2 = 0.24$, $F(2,143) = 47.75$, $B = 0.039$, $\beta = 0.36$, $t = 4.874$, $p < .001$) and model two, age (adjusted $R^2 = 0.31$, $F(2,143) = 34.94$, $B = -0.097$, $\beta = -0.31$, $t = -4.107$, $p < .001$) (see Appendix B).

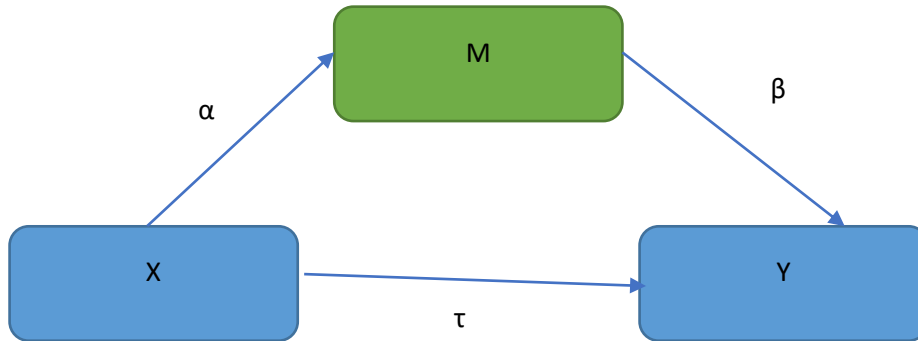
In the fifth regression analysis, the combined predictive character of predictors (age and educational level) associated with the dependent variable (MoCA scores) was evaluated, the regression equation was significant. The two predictors account for 36% of the MoCA variance (adjusted $R^2 = 0.36$, $F(2,143) = 42.05$, $p < .001$), age ($B = -0.08$, $\beta = -0.27$, $t = -3.77$), and educational level ($B = 0.62$, $\beta = 0.43$, $t = 5.90$) (see Appendix B).

Two mediation analyzes were conducted: with cognitive reserve as mediator of age effect on cognitive MoCA performance and level of education as mediator of age effect on cognitive performance (MoCA scores).

The mediation analysis investigates whether and to what extent the effect of an X variable on the Y variable is explained by the M variable. The theoretical mediation model is shown in Figure 4. The arrows indicate effects: τ is a direct effect of X on Y and $\alpha \cdot \beta$ is called an indirect effect. The total effect is the sum of the direct and indirect effect: analysis of mediation breaks down an existing effect in these two terms.

Figure 4

Theoretical Mediation Model



Note: X, Y, M – variable; τ – direct effect; $\alpha \cdot \beta$ – indirect effect

Results of the analysis (see tables 3 and 4) showed that there was a total effect of age on the MoCA score. Both educational levels and total R-IRCq index partially mediate the effect of age on cognitive performance (MoCA). The model of the mediation analysis in both cases is shown in Figure 5 and 6.

Table 3

Mediation analysis: direct, indirect and total effects Age-IRC-MoCA

	β	Z	p	95% CI for β
Direct effect				
Age - MoCA	0.044	4.15	<.001	-0.06, -0.02
Indirect Effect				
Age-R-IRCq-MoCA	0.022	3.70	<.001	-0.03, -0.01
Total effect				
Age - MoCA	0.065	6.33	<.001	0.08, 0.04

Note. MoCA - Montreal Assessment; R-IRCq - Cognitive Reserve Index; CI - confidence interval. β - standardized regression coefficient, z - Sobel test (Sobel, 1982)

Figure 5

Median Age Analysis Model - R-IRCq (CRI) - MoCA

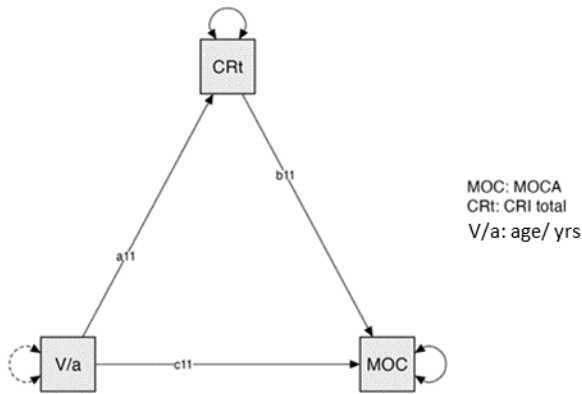


Table 4

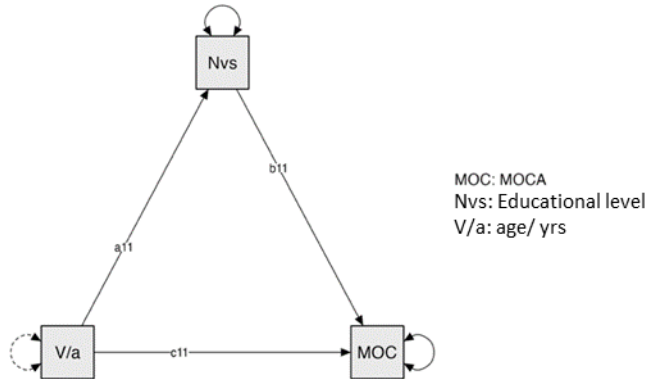
Mediation Analysis: Direct, Indirect and Total Effects Age-MoCA Educational Level

	β	z	p	95% CI for β
Direct effect				
Age - MoCA	-0.039	3.81	<.001	-0.05, -0.01
Indirect Effect				
Age-educational level -MoCA	-0.026	4.15	<.001	-0.03, -0.01
Total effect				
Age - MoCA	-0.065	6.35	<.001	0.08, 0.04

Note. MoCA - Montreal Assessment; CI - confidence interval, β - standardized regression coefficient, z - Sobel test (Sobel, 1982).

Figure 6

Mediation Age Analysis Model - Educational Level - MoCA



The mediating effect of the educational level is caused by the correlation with the age.

Discussions

This study analyzed the relationship between education and cognitive reserve recorded as a total R-IRCq score (education, occupational complexity, and leisure and social activities indicator).

The results confirmed a significant correlation between educational level and cognitive efficiency measures, both educational level and total cognitive reserve index (R-IRCq) are significant predictors of cognitive efficiency measures. This was also observed by Perneckzy et al. (2006) in a neuroimaging study, in which a higher level of education was associated with a more depleted flux in the parietotemporal area, the location of PET changes in Alzheimer's disease. Perneckzy et al. (2006) found that education altered the association between the pathology of the disease and the levels of cognitive function measured before death. For each additional year of education, the relationship between pathology and cognition was reduced by 0.088 standard units. "Notably, early cognitive function is a major predictor of cognitive function and its rate of change in midlife and beyond as well as of educational and occupational attainment" (McCall, 1979 as cited in Kreitler, Weissler, & Barak, 2013, pp. 238–269).

The covariance between the age and the educational level can be attributed to the specific socio-historical conditions of Romania (the increase in the general level of education during the life of the participants in the study and the specific features of everyday life of the third age). This has also been observed by Nucci et al. (2011) at the time of the construction and validation of the questionnaire. Many older Italians did not have more than five years of study, due to social and/or historical reasons.

The older the age of the participants, the lower their cognitive efficiency. Higher levels of education lead to a greater accumulation of resources older adults can use to cope with cognitive decline, which in turn leads to better cognitive efficiency.

The results of this study are consistent with other literature data and the results of the study, Moglan, Boscaiu and Tudose (2021, pp. 159-187), for the analysis of cognitive reserve and

cerebral atrophy in patients with neurodegenerative disorders, who observed that cognitive reserve acted as a moderator of the relationship between brain changes and the clinical profile of neurodegenerative pathology.

Conclusions

Scientists describe the phenomenon of brain resistance and its function in connection with age-related diseases. Some authors indicate that a person's susceptibility to the development of brain pathology is the result of the interaction of multidirectional processes: damage to brain tissue and the ability of the brain to maintain high functional activity due to "brain reserve". Cognitive reserve parallels the concept of brain reserve in that it is a potential mechanism for coping with brain damage (Stern, 2002). Active cognitive reserve is formed in the process of educational and cognitive activity of the subject. Higher educational attainment is associated with later onset of cognitive decline.

Results from this study demonstrated that cognitive efficacy was predicted by age (as expected), educational level, and total R-IRCq score. An inverse correlation between age and cognitive efficiency has been confirmed: the older the age of participants, the lower the cognitive efficiency, the more significant the correlation when cognitive efficiency is measured using the MoCA cognitive assessment test.

Age and total R-IRCq score cover more than a quarter of the MoCA variance, and educational levels further cover this variance. Both educational levels and total R-IRCq index partially mediated the effect of age on cognitive performance (MoCA). The assessment of cognitive reserve in older people could be a useful additional measure to integrate existing protocols for the neuropsychological assessment of cognitive decline. It is important to assess this 'threshold effect' where people with a higher level of education may resist the effects of neurodegeneration for a longer period.

Cognitive reserve should also be recognized as a factor that will influence the rate of cognitive decline after diagnosis.

Limitations

The main limit of this study is the small sample, the non-homogeneous structure on socio-demographic variables, as well as the fact that it did not include in the assessment of cognitive efficiency other parameters such as occupation, leisure activities.

Acknowledgements

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Appendix A.

Results of Linear Regression Analysis

Variable	Beta	SE	95% CI		β	p
			LL	UL		
MMSE-2						
Age	-0.05	0.02	-0.096	-0.011	-0.20	0.013
Education	0.38	0.09	0.193	0.572	0.31	< .001
R-IRCq	0.02	0.007	0.011	0.039	0.28	< .001
MoCA						
Age	-0.14	0.02	-0.190	-0.099	-0.46	< .001
Education	0.79	0.09	0.598	0.991	0.55	< .001
R-IRCq	0.05	0.008	0.038	0.068	0.49	< .001

Appendix B.

Results of Linear Regression Analysis (combined predictors)

Variable	Beta	SE	95% CI		β	p
			LL	UL		
MoCA						
Age *	-0.08	0.02	-0.132	-0.041	-0.27	< .001
Education	0.62	0.10	0.414	0.831	0.43	
Education*	0.59	0.14	0.296	0.885	0.41	< .001
R-IRCq	0.02	0.01	-0.002	0.042	0.19	

Note. Age * Education, Education * R-IRCq as combined predictors to evaluate the combined predictive character associated with the dependent variable MoCA

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