



The protective role of cognitive reserve on sleeping disorders on an aging population. A cross-sectional study

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ABSTRACT

Sleep disorders and depression have been identified as risk factors for aging-related problems. This cross-sectional study explored the protective role of the cognitive reserve on a sample of 377 healthy aging adults. After assessing participants' cognitive reserve (CR) in an extensive and comprehensive way, participants also filled out self-report scales aimed at measuring their sleep quality (PSQI - Pittsburgh Sleep Quality Index) and depressive symptoms. A series of regression models and path analyses showed that higher CR levels predicted lower PSQI scores overall, even after controlling for depressive symptoms, age, and gender. Depressive symptoms had significant role as a moderators of the effect of CR on sleep disorders. The possible effects of interventions aimed at increasing the CR in preventing the incidence of sleep disorders, cognitive decline, and other aging-related disorders are discussed.

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1. Introduction

We live in an aging world: a recent estimate hypothesized that by 2030, 1 in 5 people in the United States will be over the age of 65 [1]. With the increase of the aging population, and the associated increase in aged-related physical and mental problems, research has focused on possible comorbidities and risk factors that can help predict the occurrence of age-related clinical problems.

Considering the fact that almost half of the aging population reports some form of sleeping disturbance [2], quality of sleep and sleep problems seems to present relevant variables to consider when focusing on aging-related problems. Sleep-related problems have been shown to increase the chances of developing several health (e.g., cardiovascular diseases [3], headaches [4], memory loss and cognitive decline [5,6], and diabetes [7]) and mental health (e.g., depression [8] and anxiety [9]) conditions in the aging population. Overall, sleep disturbance in older adults has been reported to be associated with increased morbidity and mortality [10–12].

Sleep disorders and depression are two factors worth considering together when examining effects on age-related cognitive decline: sleep disturbances and depressive symptoms have been

reported to have a reciprocal and bidirectional relationship [13,14], and both sleep disturbances and depression have been flagged as a source of significant health problems within community dwelling older adults [15]. To be more specific, a growing body of research has highlighted how cognitive decline is impacted by poor sleep and depression [16–21]. Specifically, recent findings show that sleep disturbances may predict the incidence of dementia, as reported in a recent meta-analysis [22], which found that depression and higher risk of dementia or MCI (mild cognitive impairment) suggested a prodromal role for depression [23].

This evidence suggests that exploring this relationship more in depth and trying to understand any protective factors that could decrease the chances of aging individuals developing sleep disturbances, while controlling for the influence of depressive symptoms could be a relevant focus for new areas of preventive interventions to reduce the incidence of dementia and MCI. This is the aim of the present study.

When looking for possible protective factors, we decided to explore the role of cognitive reserve (CR). The cognitive reserve model [24–26] has been introduced to explain discrepancies that have been reported between an individual's level of brain pathology, and their actual function on neuropsychological testing and activities of daily living. Being exposed more frequently to CR proxies (which include educational activities, strong social

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network, physical, cognitive and leisure activities) has been reported to reduce the chances of developing cognitive decline or dementia. CR has also been reported to be a protective factor for physical and cognitive symptoms of Parkinson's disease [27], HIV related dementia [28], and to promote recovery after a traumatic brain injury [29] or a stroke [30].

The relationship between CR and sleep has been explored to some extent with studies reporting how lower levels of CR affect specific cognitive abilities (i.e. verbal fluency) in individuals who reported sleep onset/maintenance difficulties [31]. CR has also been reported as a protective factor in older adults against obstructive sleep apnea syndrome [32] and as a factor that can help to counter the impact of disturbed sleep on executive functions [33]. Regarding the protective role of CR, it has been recently reported that higher CR may reduce the negative influence of sleep disturbances on perceived fatigability during daily life activities in the early stages of dementia [34]. These studies lay a promising background to support our hypothesis regarding the protective role that CR can have in preventing or reducing sleep disturbances and its consequent detrimental role on physical and mental health. Yet, these studies present some limitations that our study aims to address. Specifically, most of the studies did not assess CR in a comprehensive way, but used only education level [31,33] or intelligence [32] as an indicator of CR. The only study that uses a more comprehensive assessment, taking into consideration other proxies of the CR had a very limited sample, 37 participants [34]. Moreover, this study did not consider the possible role of depressive symptoms that, since depression has been reported to be related to both CR and sleep disturbances, might play a significant role in the target relationship between CR and sleep.

The present study aims at exploring more in depth the possible protective role that CR levels might play against sleep disturbances, by using a more extensive assessment of CR that consider not only the education level, but also type and frequency of leisure activities, as well as different measures of flexibility of thought [35–37]. We also assessed depressive symptoms in our participants in order to be able to assess the role that depression plays within our model.

We expect that individuals with higher CR will report less sleep disturbances, and we also expect to be able to highlight a significant protective role of CR levels in specific sleep-related problems.

2. Methods

This study was approved by Champlain College IRB (COA: IRB000187).

2.1. Participants

Three-hundred-seventy-seven healthy participants joined the study. Age of the sample ranged between 65 and 86 (Mean = 69.55; SD = 5.19). The sample was not balanced by gender (F = 57.3%). More detailed demographic info is reported in Table 1.

To be included in the study participants had to self-attest that they had no diagnosis of cognitive decline, dementia, Alzheimer or any other neurodegenerative disorder.

Table 1
Demographic info.

	Min-Max	Mean	SD
Age	65–86	69.55	5.19
Level of education	6–22	16.21	3.21
	N (%)	N (%)	
Gender	F = 216 (57.3%)	M = 161(42.7%)	

2.2. Measures

After signing a consent form, participants filled out three self-report measures online, using the platform Qualtrics.

The Cognitive Reserve Test - CoRe-T [35]. This self-report measure has two parts. The first section assess all the proxies traditionally used to assess the CR (see Introduction). Data include information about education level (years of completed education, including vocational training), type and frequency of leisure activities (both frequencies of performing teach specific selected activity and numbers of years they have been practicing it), and occupation history. The second section measures the flexibility of thought, which has been reported to be positively correlated with the CR both in healthy [37–39] and clinical populations [27,40] Two tasks are used in this section: the “Acronyms” task, where participants are given 5 min to list all the terms that can fit into the three given acronyms (the terms had to make sense together), and the “Alternative uses task” where participants, in 5 min, are asked to list as many different, interesting or unusual usages for an empty plastic bottle as they can. Scoring is described in detail in Colombo et al. [35]. The Co-Re-T also includes a demographic section where participants record age and gender.

Pittsburgh Sleep Quality Index (PSQI) [41]. This self-report questionnaire assesses sleep quality and disturbances over the previous month. It is composed of 19 items that allow us to compute 7 sub-scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The sum of scores for these seven components yields one global score.

Beck Depression Inventory (BDI) [42]. This is a 21-question multiple-choice self-report questionnaire that measures characteristics, attitudes, and symptoms of depression. Each item can be rated based on four response choices according to the severity of the symptoms, ranging from the absence of a symptom to an intense level.

2.3. Analyses

First analyses were performed using SPSS 28: We first ran multiple regression models, to assess if CR scores could negatively predict PSQI scores and sub scores. By adding BDI, age, and gender to the model we controlled for their possible confounding effects.

As a second step, we ran a path model (using Mplus 8.4 [43]) to explore more in depth the possible moderation role that BDI might have in influencing the relations between CR scores and PSQI scores.

3. Results

Our first analysis aimed at exploring possible predictors of the total PSQI score. After running 0-order correlation (Table 2), we ran a regression analysis, using total PSQI score as an outcome, and the CR score, BDI score, age and gender (to control for their possible confounding effect) as predictors. Results can be seen in Table 3.

As it can be derived from data reported in the tables, CR appears to be a protective factor (higher CR scores significantly predict lower PSQI scores). On the other hand, both age and the presence of symptoms of depression tend to increase the risks of sleep problems as assessed by the PSQI. Women also seem to be at a high risk for higher PSQI scores.

To further clarify the associations between CR and depression and the sleep problem we ran a path model (Mplus 8.4), in which we considered the subscales of the PSQI as separate outcomes, with age, gender, CR, BDI, and the interaction term CR by BDI as predictors. The scores of CR and BDI were centered in the sample.

Table 2
Correlation matrix of the study variables (N = 377).

	1	2	3	4	5	6	7	8	9
1. Cognitive Reserve (total)	–								
2. BDI	-.25**	–							
3. PSQI (total)	-.63**	.68**	–						
4. Sleep quality	-.12*	.47**	.37**	–					
5. Sleep latency	-.36**	.63**	.70**	.24**	–				
6. Sleep duration	-.21**	.52**	.61**	.11*	.32**	–			
7. Sleep disturbances	-.64**	.25**	.71**	.01	.27**	.30**	–		
8. Daytime disturbances	-.45**	.55**	.66**	.11*	.38**	.28**	.59**	–	
9. Use of sleep medication	-.39**	-.05	.38**	-.17**	.05	.05	.46**	.33**	–

**p < .01 (2-tailed).
*p < .05 (2-tailed).

Table 3
Regression analysis predicting effects on PSQI scores.

	B	SE	β	t	p
<i>Outcome: PSQI Total Score</i>					
(Constant)	-3.33	1.50		-2.22	.03
Cognitive Reserve (total score)	-.06	.003	-.45	-15.92	<.001
BDI	.10	.01	.39	12.22	<.001
Age	.19	.02	.28	8.54	<.001
Gender	.58	.19	.08	3.10	.002

R²(4;372) = 0.74; p < .001.

We obtained a saturated model, but the interactions term CR by BDI was not significantly associated with the subscales *Sleep latency* and *Sleep duration*. Therefore, we fixed these two paths to 0, gaining 2° of freedom to estimate the model. The final model fitted the data well: $Chi^2(2) = 2.123$, $p = .346$, $CFI = 1.00$, $TLI = 0.998$, $RMSEA = 0.013$, $CI [0.000 0.104]$, $SRMR = 0.008$. The model is displayed in Fig. 1. BDI was associated positively with *Subjective sleep quality*, *Sleep latency*, *Sleep duration* and *Daytime disfunction*, but negatively with the *Use of sleep medication*. CR was not associated significantly with *Subjective sleep quality* and *Subjective duration* but was associated negatively with all the other subscales. This highlights how the CR alone can improve sleep by reducing the need to take sleeping aids (use of medication), helping individuals to fall asleep faster (sleep latency), have a more restful sleep (without

Table 4
Strengths of the associations between CR and the PSQI sub-scores, for low, average and high levels of BDI.

	Association indices of CR with PDSQI scores: β (p)		
	Low BDI (-1 SD)	Average BDI	High BDI (+1 SD)
<i>Subjective sleep quality</i>	0.116 (0.049)	0.023 ns	-0.071 ns
<i>Use of sleep medication</i>	-0.652 (<.001)	-0.409 (<.001)	-0.162 (0.017)
<i>Sleep disturbances</i>	-0.652 (<.001)	-0.584 (<.001)	-0.516 (<.001)
<i>Daytime disfunction</i>	-0.560 (<.001)	-0.318 (<.001)	-0.066 ns

disturbances) and hence being less tired during the day (daytime dysfunctions).

The interaction term CR by BDI was associated negatively with *Subjective sleep quality* and positively with *Use of sleep medication* and *Daytime disfunction*. The meaning of this interaction has been explored with follow-up analyses presented below.

In order to interpret the moderation effects, we ran follow-up analyses to estimate the strength of the associations between CR and the subscales of PSQI for high (+1 SD) and low (-1 SD) levels of BDI [44]. The results of these analyses are reported in Table 4. Results highlight how the protective role of CR decreases as the severity of depression increases. Yet, CR seems still to be effective, even with high levels of depression, in reducing the need for sleep medications and reducing self-reported sleep disturbances.

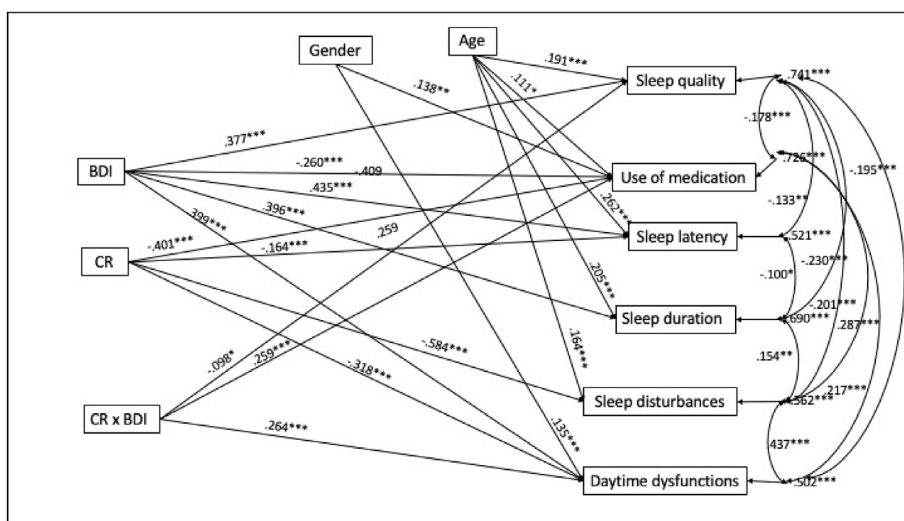


Fig. 1. Path model of the associations between BDR (total score), CR (total score) and the subscales of PSQI: *Sleep quality*, *Use of sleep medication*, *Sleep latency*, *Sleep duration*, *Sleep disturbances*, *Daytime dysfunctions*. CR X BDI = interaction term of CR by BDI.
Note. *p < .05, **p < .01, ***p < .001.

4. Discussion and conclusions

The main aim of this study was to explore in depth the possible protective effect of CR towards sleep disturbances after controlling for the possible confounding effect of depressive symptoms.

Our main hypothesis was confirmed: higher CR levels predicted lower PSQI scores overall, even after controlling for depressive symptoms, age, and gender. We were also able to explore more in depth this relationship by reporting a significant role of depression as a moderator of the effect of CR on sleep disorders. This result confirms preliminary results reported in the literature, using a more comprehensive assessment of the CR and a larger sample than some of these previous studies [31–33].

We were also expecting that the CR would be protective specifically towards individual sleep problems – and the data supported this hypothesis as well. Results from our path model and the follow up analyses, highlighted a specific role of the CR in reducing the need to take sleeping aids, helping older individuals to fall asleep faster, reduce the occurrence of sleep disturbances (like frequent wake up, nightmares, etc.), and leading individuals to be more rested during the day. The interaction between the levels of CR and the severity of depressive symptoms was also interesting. As it could be expected, more severe symptoms of depression reduce the protective effect of the CR. Yet, higher levels of CR were still effective reducing the need for sleep medications and reducing self-reported sleep disturbances even in participants with higher scores in the BDI scale.

Considering what we already know from literature, these results make sense. For example, both longer sleep latency [45] and self-reported sleep disturbances [46,47] have been reported to be associated with higher risk for cognitive decline onset or to be more common among populations with dementia or MCI. The same association was reported herein for the use of sleep medications [16,48] and incidence of daytime dysfunction derived from poor sleep [16,45].

Reading together our results and the evidence reported in the literature seem to imply that not only there is a relation between CR and cognitive decline and between certain sleep-related problems and CR, but that this relation involves all these three variables at the same time. CR can predict these specific sleep problems that have been reported to be linked to cognitive decline and dementia. More specifically, CR seem to play a mediation role between the well-known relation between depression and sleep disorders.

These results are not surprising since it is well known that some of the CR proxies are also linked to better sleep quality, like physical exercise [49] as well as engagement in social [11,50] and leisure activities [51,52]. Yet, this study is the first one to directly explore this complex relationship assessing the CR in a comprehensive way, on a relatively large sample, and controlling for possible confounding effects of depressive symptoms.

What are the meaning and the implications of these findings? First of all, since the CR is not fixed but can be improved at any point in life [53–56], our results suggests that by increasing CR it may be possible to decrease the chances of sleep disorders, and hence reduce the incidence of cognitive decline. But not only this finding supports the role of CR in sleep disorder. As discussed in the introduction, the same sleep disorders are also associated with other health related problems in the elderly: for example, cardiovascular diseases [3], headaches [4], memory loss and cognitive decline [5,6], and diabetes [7]) and mental health (e.g., depression [8] and anxiety [9]). It is reasonable to assume that interventions aimed at increasing CR might reduce the occurrence of these problems as well. Future cohort and longitudinal studies should test this hypothesis.

This study also presents some limitations, including the fact that

is not perfectly balanced by gender and is a cross sectional study where the absence of any form of cognitive decline was only self-reported by participants. Moreover, there is no cross-cultural comparison: something that future studies should address.

Declaration of competing interest

The authors whose names are listed immediately below certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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