The Relationship between sleep and cognitive function in old age

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INTRODUCTION

Sleep is one of the few biological phenomena that is common to almost all living organisms. It is a fundamental aspect of human life, being a mechanism that leads to the restoration and maintenance of our genetic balance, responsible for promoting quality of life. Sleep is mainly characterised by a reversible change in the state of consciousness and by a reduction in sensitivity to exogenous stimuli, associated with typical postural characteristics, although not always present, such as inactivity and closed eyes (Guyton & Hall, 2006; Paiva & Penzel, 2011).

Sleep quality is understood as a multidimensional sleep-wake pattern adapted to individual, social and environmental demands, providing physical and mental well-being. It includes quantitative aspects of sleep, such as its duration, latency or number of awakenings, as well as purer subjective aspects, such as "depth" or "peacefulness" of sleep (Akerstedt et al., 1994; Buysse et al., 1989). Studies suggest that six to eight hours of sleep per night may be the optimal amount of sleep for maximum health benefits (Kripke, 2004). The literature associates poor sleep quality with higher mortality rates, and a higher prevalence of diseases such as diabetes, depression, hypertension and coronary heart disease.

Sleep deprivation and cognitive impairment are highly prevalent in older people (Bishop et al., 2010; Rapp & Heindel,1994; Glisky & Riddle, 2007).

Ageing brings numerous physiological and environmental changes in sleep quality and quantity that directly interfere with sleep complaints. These changes in sleep patterns, affected with advancing age, alter the homeostatic balance, with repercussions on the immune system, behavioural responses, mood and performance. Poor sleep quality and changes in brain activity during sleep are complex health problems in ageing populations, becoming potential risk factors for cognitive decline (Ebersole, 2001).

Ageing is known to be accompanied by profound changes in sleep that together affect neuropsychological performance (Reisberg & Gauthier, 2008; Hoogenhout et al., 2011; Diekelmann & Born, 2010). Sleep disturbances can interfere with the normal functioning of neuronal pathways (GABA and cAMP) which in turn can affect synaptic plasticity (Havekes et al., 2012). Non-restorative sleep may contribute to brain degeneration by promoting neuroinflammation, and impair neurogenesis, especially in areas of the hippocampus, a key neuroanatomical region for learning and memory, suggesting that this brain region may be especially sensitive to the consequences of sleep loss. Furthermore, reduced concentrations of acetylcholine (which helps maintain normal sleep patterns and is implicated in memory consolidation) resulting from the loss of cholinergic cells in the basal forebrain, which occurs during ageing, may contribute to cognitive decline by disrupting sleep and memory processes (Kang et al., 2009).

A study analysing the relationship between sleep quality and cognitive performance in older adults (65-80 years) found that differences in sleep quality were associated with poorer performance on tests of working memory, divided attention and problem solving, but not on tests of processing speed, inhibitory function and episodic memory (Nebes et al., 2009). Another study found lower performance on measures of verbal cognition, long-term memory and visuospatial reasoning, associated with self-reported sleep onset and latency (Schmutte et al., 2007).

METHODS

We used a sample of 86 Portuguese people (48F; 38M) elderly (X=69anos) and middle-aged (X=54anos).
The instruments used were:
Pittsburgh scale for the assessment of sleep quality (Del Rio João et al., 2017);
MOCA- Montreal Cognitive Assessment (Freitas et al., 2011);
Verbal fluency (Cavaco et al., 2013);
HADS- anxiety and depression scale (Pais-Ribeiro et al., 2007);
Digit Span from WAIS (Young et al., 2012);
AVLT- auditory verbal learning test (Cavaco et al., 2015);
TMT- trail making test (Cavaco et al., 2013);
Epworth sleepiness scale (Santos, 2001);
Sociodemographic and clinical questionnaire.

RESULTS

Pearson's correlation coefficients (r) for the sample population of middle-aged adults (aged 50-59 years)

	MOCA test total score	Anxiety component of the HADS questionnaire
PSQI - sleep quality (component 1)		
Pearson Correlation	-0.055	0.523*
Sig. (2-tailed)	0.765	0.002
PSQI - sleepiness (component 7)		
Pearson Correlation	-0.392**	0.598*
Sig. (2-tailed)	0.026	<0.001

*The correlation is significant at α=0.01 (2-tailed)
**The correlation is significant at α=0.05 (2-tailed)

Pearson correlation coefficients (r) for the population sample of older adults (age 60+)

	AVLT - Standardised total learning	AVLT - Standardised Trial Learning	HADS - Anxiety
PSQI - Latency (component 2)			
Pearson Correlation	0.070	0.221	0.337**
Sig. (2-tailed)	0.670	0.175	0.027
PSQI - Sleep duration (component 3)			
Pearson Correlation	0.082	0.478*	0.236
Sig. (2-tailed)	0.622	0.002	0.123
PSQI - Sleepiness (component 7)			
Pearson Correlation	0.339**	0.186	0.214
Sig. (2-tailed)	0.035	0.258	0.163

*The correlation is significant at α=0.01 (2-tailed)
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RESULTS

The main statistically significant results observed, assessed using Pearson's correlation coefficient, were, in the sample population of older adults (aged over 60 years), the moderate positive correlation (r = 0.478) significant at the level of α = 0.01 (p = 0.002, 2-tailed) between component 3 (sleep duration) of the PSQI questionnaire and the AVLT - Standardised Trial Learning; the weak positive correlation (r = 0.339) significant at the α = 0.05 level (p = 0.035, 2-tailed) between component 7 (sleepiness) of the PSQI questionnaire and the AVLT - Standardised Total Learning; the weak positive correlation (r = 0.337) significant at the α = 0.05 level (p = 0.027, 2-tailed) between component 2 (latency) of the PSQI questionnaire and the anxiety component of the HADS questionnaire.

In the sample population of middle-aged adults (aged 50 to 59 years), a weak negative correlation (r = -.392), significant at α = 0.05 (p = 0.026, 2-tailed) between component 7 (sleepiness) of the PSQI questionnaire and the total score of the MOCA test; the moderate positive correlation (r = 0.523) significant at α = 0.01 (p = 0.002, 2-tailed) between component 1 (sleep quality) of the PSQI questionnaire and the anxiety component of the HADS questionnaire; the moderate positive correlation (r = 0.598) significant at α = 0.01 (p < 0.001, 2-tailed) between component 7 (sleepiness) of the PSQI questionnaire and the anxiety component of the HADS questionnaire.

DISCUSSION

In the sample of older adults (aged over 60 years), it was found that subjects with longer sleep duration had better results in the AVLT test, in the Standardised Trial Learning component.

In both the elderly and middle-aged population samples, anxiety was found to be a variable that impairs sleep, with anxiety negatively influencing latency (time to sleep onset) in the elderly sample and greater daytime sleepiness in the middle-aged sample.

There was a peculiar result between component 7 (sleepiness) of the PSQI questionnaire and the AVLT - Standardised Total Learning, in that in the sample of the population of older adults (aged over 60 years) it was found that subjects with sleep disorders had better executive functions, this being an antagonistic result, as it would be expected that these individuals would have signs of cognitive impairment. In the middle-aged population sample, we observed that subjects with greater sleepiness had worse results in cognitive functions. Such results may be due to the presence of outliers. For future research, stricter inclusion and exclusion criteria should be applied in order to reduce the existence of outliers and increase the effectiveness of the research.

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