Research Article

Brain Disorders: Correlation between Cognitive Impairment and Complex Combination

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ABSTRACT

Introduction: Most of neurodegenerative diseases have cognitive impairment as early signs or consequence of the evolution of these diseases. Early detection appears unavoidable if, clinicians and researchers expect to win this war.

Methods: Demographic and clinical data, well-being, drugs usage associated with cognitive impairments, gender, level of education, family history disorders, memory deficiency and cognitive disorders were collected. Mc Nair score was calculated using the short version with 15 items.

Results: Simultaneous combination of complex medical history associated with family's medical disease with cognitive problems, exhibited a specific profile of subject at risk of future brain disorders

Introduction

Cognitive performance is mainly improved during delicate stage of development that is, between youth and adolescence. This important step is characterised by growth of the brain structures and development of the nervous system. Indeed most of the brain disorders are created by a failure in one or many of these natural processing. As a consequence of these imbalances, cognitive impairments in the brain, memory and central nervous system can appear. Regular good duration of sleep allows human brain to recover from the physical activities like football, and which is also known for his prevention skill against brain's disorders, or cognitive deficiency [1-3]. Literature has showed that nervous system are primarily influenced by environmental interactions beginning at the birth until the midlife, and this interactions are related to the probability of developing neurodegenerative diseases and associated diseases later in life [4-6]. The common symptoms are cognitive impairments. A change or imbalances in timing of the brain maturity timeline increase the risk of brain disorders and modulate the development of neurodegenerative diseases, dementia and psychiatric disorders in unknown rates for young adults (people aged between eighteen years old until fourty). Until now, no final therapy exists for brain's disorders but, prevention of risk factors and promotions of good lifestyle are important because, mental illness and brain failure are not simple to cure; when they are already diagnosed. Physical activity and sleep are potential tools able to decrease cognitive impairments for young adult's population (name YA in the rest of the article), compared with midlife and elderly adults [4,7-10]. Many others risk factors like cardiovascular diseases or brain injuries are known for their impact on cognitive performance, mental disorders and **Discussion:** Monitoring the mental state contributes both to prevention and; monitoring initial symptoms of cognitive decline will decrease considerably memory complaints and prevent the onset of neurodegenerative diseases. We also strongly state early detection of cognitive impairment will delay dementia and associated illness.

MeSh Headings/Keywords: Brain disorders, cognitive impairment, early detection, neurodegenerative diseases, young adults, McNair, prevention, early detection, mental health.

Abbreviations: YA = Young Adults

associated diseases. But few specific studies showed an accurate relation between combination of these two factors and cognitive impairments for YA. The aim of this study is to explore how cognitive impairments are linked in simultaneous with duration of sleep and quality, psychological stress, depression, anxiety, well-being, addiction and global health of participants; and if it is also influenced by the sociodemographic profile of each subject.

Methods

Ethics committee

This research article was approved beforehand, by the ethic committee of research of the faculty of arts and science, University of Montreal, Canada. All our subjects were volunteers and signed a consenting form.

Sample characteristics

Demographic and clinical data of age, drugs associated with cognitive impairments, gender, level of education, family history disorders; memory deficiency and cognitive disorders were collected with a questionnaire. Current and history of medications were classified as medications of musculoskeletal, neurological, respiratory or cardiovascular disease. Other treatments were categorized as antibiotics, antidepressants, vitamins or energetic drinks, acupuncture, hypnosis, anxiolytics, sleeping pills and anti-inflammatory. Mc Nair score was calculated using the short version with 15 items.

Sleep parameters

Quality sleep complaints and sleep disorders were identified with seven items: duration of sleep, use of sleeping pills, history of medication, duration of medication, beginning of sleeping disorders, sleep quality ranged from 1 "very bad" to 5 "very well" and the difficulty of falling asleep from 1"None" to 4 "very difficult".

Cognitive parameters

1344 respondents were assessed in subjective cognitive difficulties using an integrate version of Mc Nair's test. The self-report for assessing cognitive difficulties was composed of 15 items (scored from 0 "never or not applicable" to 4 "very often"). This version helped to evaluate cognitive complaints over the total score ranging from 0 (no difficulty) to 45 (high cognitive difficulties). Scores more than or equal to 15 points were considered indicative of cognitive impairment, with a maximum score of 45 points.

Statistical analysis

The normal distribution of cognitive measures was tested using the Kolmogorov-Smirnov's test. To analyse Mc Nair test responses, scores were converted to a dichotomous variable; and participants with a score fewer than 15 were scored "No cognitive complaints "vs whose score is more than or equal to 15 were scored "Presence of cognitive complaints". Spearman rank was used to analyse the relationship between the continuous variables general health, stress, dependency, wellbeing and Mc Nair score. Mann-Whitney's non-parametric test for independent samples was employed to compare Mc Nair score as a continuous variable between two groups. Kruskal Wallis test was used for comparing Mc Nair score for more than two groups. Logistic regression was carried out to assess the relationships between Mc Nair's score as a dependent variable and sleep parameters as independent variables. Statistical tests used an alpha of 0.05 as a level of significance. Odds ratios (OR) and risk ratios (RR) were estimated with the logistic regression model for sleep parameters. Multiple correspondence analysis was realized; to identify relationships between the variables most associated with Mc Nair score. Data analysis was performed using SPSS Statistics-version 23 for windows 10.64 bits- (IBM Corporation, Armonk, NY, USA).

Results

1344 subjects were used in the current investigation. The response to the questionnaire was 100%.

Population characteristics

In the survey population, 71.7% were in the YA group aged between 18 and 24 years, a record high proportion. One of the distinguishing characteristics of this research is the fact that a relatively large sample was recruited to observe the effect of associated factors on cognitive impairment in adults, especially younger adults. Women represented 76.2% of the sample. Most of the respondents are in the first cycle of education (62%). Table 1 represented socio-demographic and clinical parameters of the total cohort.

Subjective cognitive complaints and clinical parameters

In the analysis of family history's disease, 38.1% (n = 512) suffered from cardiovascular disease and 14.3% (n = 192) suffered from neurologic disease. 52.4% (n = 704) of family

members have a cognitive impairment and 21.4% (n = 288) suffered from Alzheimer. 2.4% (n = 32) of respondents were treated from cardiovascular disease; 2.4% (n = 32) suffered from musculoskeletal disease; 4.8% (n = 64) used medication for neurologic disease and 7.1% (n = 96) has a respiratory problem. All clinical parameters were associated with Mc Nair score (p < 0.0001, Kruskal Wallis test) except for the use of cognitive or memory impairment drugs (p = 0.735, U Mann-Whitney test). The analysis of depression and anxiety showed that 88.8% (n = 1184) of the participants have a normal depression but 33.3% (n = 448) has a moderate anxiety. The mean well-being score was 10.88 ± 1.38 (SD) with a good correlation with Mc Nair score (p < 0.0001, Spearman rank). The mean dependency score was 10.64 ± 1.04 (SD), based on Spearman rank it's associated with Mc Nair score (p = 0.0008). However, there was no correlation between general health score, stress score and Mc Nair score (p = 0.380, p = 0.419 respectively with Spearman rank test).

Relations among cognitive complaints and sleep parameters

Table 2 showed a good association between cognitive complaints and sleep parameters except for the beginning of sleep disturbances (p = 0.439, U Mann Whitney test). Table 3 shows that this association would persist even when we take into account all sleep parameters as independent variables and Mc Nair score as dependent variable. The logistic regression was performed on uncorrelated variables to identify the best predictors for Mc Nair score. Multicollinearity was detected between the parameters: sleeping pills, medication, and beginning of sleep disturbances. Four variables were included: duration of sleep, duration of medication, sleep quality and difficulty falling asleep. Logistic regression analysis revealed that 24.6% of the variance in cognitive impairments was explained by duration of sleep, duration of medication, sleep quality and difficulty falling asleep (Table 3). The model was significant (p < 0.05). The variable sleep quality was the least significant factor in the model (Wald statistic = 32.66, p-value < 0.0001) and duration of medication the most significant (Wald statistic = 100.97, p-value < 0.0001). Sleep time (Wald statistic= 67.18, p-value < 0.0001), duration of medication of one month or between one month and six months (p-value < 0.000 vs 6 months-1 year, p = 0.998), no difficulty of falling asleep (Wald statistic = 77.7, p-value = 0.001) or have a difficulty to fall asleep (p-value < 0.000 vs a little difficulty to fall asleep, p-value = 0.436), mild subjective sleep satisfaction (p-value < 0.000) were associated with cognitive problems. The odds ratios were ranged from 40.8 for the duration of medication (less than one month) to 0.069 for sleep time (5 hours).

Risk ratios were reported in Table 3. The RR of suffering from cognitive complaints is 0.31 for participants who sleep four hours relative to those who sleep more than eight hours. The risk of suffering from cognitive complaints for respondents who are under medication between one month and six months or more than six months is 1.75 more than respondents under medication more than one year. The risk of a participant with a mild sleep quality getting cognitive problems is about 0.83 times the risk of a participant with a very well sleep quality getting Brain Disorders: Correlation between Cognitive Impairment and Complex Combination 217

Characteristics	Mean ± SD Or n (%)	Mcnair score Mean ± SD	Statistics
Demographics			
Age			0.01
18 - 24	960 (71.7%)	14.3 ± 0.21	
24 - 30	352 (26.2%)	14.45 ± 0.39	
30 - 36	32 (2.4%)		
Gender			
Male	320 (23.8%)	12.6 ± 0.309	0.000
Female	1024 (76.2%)	14.75 ± 0.219	
Level of education			0.000
First cycle	833 (62%)	14.18 ± 0.23	
Secondary cycle	160 (11.9%)	13.60 ± 0.238	
Third cycle	320 (23.8)	15.30 ± 0.458	
Else (certificat. AEC. DEP. microprogramme)	31 (2.3%)		
Medication History			
ognitive or memory impairment drugs			0.735
Yes	64 (4.8%)	13 ± 0.504	
No	1280 <mark>(95.2%)</mark>	14.30 ± 0.191	
Family history of neurological.			
musculoskeletal. respiratory or			0.000
cardiovascular disease (1-6)			
Cardiovascular disease	512 <mark>(38.1%</mark>)	12.88 ± 0.225	
Musculoskeletal disease	64 <mark>(4.8%)</mark>	22 ± 0.756	
Neurologic disease	192 <mark>(14.3%</mark>)	13 ± 0.494	
Respiratory disease	32 <mark>(2.4%)</mark>	20	
Other	192 <mark>(14.3%)</mark>	15.83 ± 0.493	
None	352 <mark>(26.2%)</mark>	14.09 ± 0.412	
mily's history for cognitive or memory impairments (1-6)			0.000
Memory deficiency	96 <mark>(7.1%)</mark>	14.33 ± 0.491	
Attention deficit disorders	128(9.5%)	23.75 ± 0.221	
Alzheimer	288(21.4%)	9 ± 0.233	
Cognitive impairments	704(52.4%)	14.86 ± 0.258	
Other	0	0	
None	128 <mark>(9.5%)</mark>	13 ± 0.226	
Medication. current			
Medication of neurological.			
musculoskeletal. respiratory or			0.000
cardiovascular disease (1-6)			
Cardiovascular disease	32(2.4%)	16	
Musculoskeletal disease	32(2.4%)	21	
Neurologic disease	64(4.8%)	12.5 ± 0.315	
Respiratory disease	96(7.1%)	14 ± 0.22	
Other	96(7.1%)	12 ± 0.825	
None	1024(76.2%)	14.34 ± 0.222	
Depression			0.000
Normal	1184 (88.1%)	14.62 ± 0.19	
Moderate	96(7.1%)	15 ± 0.8	
Mild	32(2.4%)	4	
Severe	32(2.4%)	8	
Anxiety			0.000

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Normal	448(33.3%)	15.5 ± 0.35	
Moderate	448(33.3%)	14.86 ± 0.26	
Mild	320(23.8%)	12.9 ± 0.31	
Severe	128(9.5%)	11 ± 0.8	
General health score	9.71 ± 0.45		0.380
Well-being score	10.88 ± 1.38		0.000
Stress score	37.81 ± 7.87		0.419
Dependency score	10.64 ± 1.04		0.008

Table 2: Relationships between sleep parameters and Mcnair score.

Sleep parameters		Mcnair score Mean ± SD	Kruskal ou mann whitney	p-value
	4h	16 ± 0.63	85.11	0.000
	5h	11.75 ± 0.58		
	6h	15.42 ± 0.42		
	7h	13.06 ± 0.26		
Sleep duration	8h			
	More than 8h	15.63 ± 0.30		
	Yes	23	7.57	0.000
Sleeping pills	No	14.02 ± 0.185		
	None	13.54 ± 0.23	283.91	0.000
	antibiotics	20		
	antidepressants	8		
	vitamins or energetic drinks	12.29 ± 0.415		
	Acupuncture or hypnosis	26		
Medication	Anxiolytics or sleeping pills	16 ± 0.88		
	anti-inflammatory	18.50 ± 0.183		
	None	13.52 ± 0.23	130.98	0.000
	Less than one month	17.5 ± 0.59		
	1 month - 6 months	14.25 ± 0.54		
uration of medication	6 months - 1 year	26		
	More than one year	13.29 ± 0.34		
Beginning of sleep disturbances	None	14.23 ± 0.19	-0.775	0.439 NS
	Before	14.29 ± 0.474		
	Very bad	4	102.37	0.000
Slear andlite	Bad	15 ± 0.71		
Sleep quality	Mild	14.75 ± 0.29		
	Well	13.62 ± 0.28		
	Very well	15.4 ± 0.34		
	None	17 ± 0.26	219.72	0.000
	little	12 ± 0.28		
Difficulty falling a sleep	Difficult	12.60 ± 0.48		
	Very difficult	12.33 ± 0.81		

cognitive problems. The risk to suffer from cognitive difficulties for participants who have no difficulty or a difficult of falling asleep is 1.83 and 1.2 respectively more than for participants who have a very difficult to fall asleep. This would suggest that levels of difficulty of falling asleep being considered are associated with an increase in risk.

Multiple Correspondence Analysis (MCA)

In order to explore more closely the relationships between the most pertinent variables (clinical parameters and sleep complaints) to predict Mc Nair score, a multiple correspondence analysis was established between these categorical variables. This method is a part of a family of descriptive methods (clustering, principal component analysis, multiple factor analysis) used for, modeling complex data sets as points in a multidimensional plane, when the data collected is qualitative. Generally it is the case in epidemiological, clinical and social studies [11].

MCA applied to the data of 1344 participants using sleep time, duration of medication, difficulty falling asleep, family history of musculoskeletal, neurological, respiratory or cardiovascular disease, current diseases and family history for Brain Disorders: Correlation between Cognitive Impairment and Complex Combination 219

	β	SE	OR (95% C.I.)	RR (Risk Ratio)	p-value
Sleep duration					
4h	-1.439	0.395	0.237(0.109/0.514)	0.31	0.000
5h	-2.667	0.352	0.069(0.035/0.138)	0.16	0.000
6h	-0.803	0.243	0.448(0.278/0.720)	0.31	0.001
7h	-1.017	0.200	0.362(0.244/0.535)	0.27	0.000
More than 8h*					
Duration of medication					
None	0.440	0.212	1.552(1.024/2.353)	0.89	0.038
Less than month	3.709	0.458	40.807(16.630/100.134)	1.75	0.000
1 month - 6 months	2.359	0.316	10.578(5.693/19.653)	1.75	0.000
6 months - 1 year	20.759	7105.18	1133096220.169	-	0.998
More than one year*					
Sleep quality					
Very bad	-20.052	7105.18	0	-	0.998
Bad	0.382	0.430	0	-	0.374
Mild	0.955	0.276	2.598(1.512/4.462)	0.83	0.001
Well	-0.085	0.244	0	-	0.728
Very well*					
Difficulty falling a sleep					
None	1.080	0.370	2.945(1.427/6.077)	1.83	0.003
little	0.153	0.384	0	-	0.690
Difficult	-1.731	0.395	0.177(0.082/0.384)	1.2	0.000
Very difficult*					

Table 3: Logistic regression analysis of the association between subjective cognitive complaints and sleep complaints

(*) Reference category. (-) Not Significant.

cognitive or memory impairments as imputed values, showed that the total inertia explained is equal to 75.8%. Two dimensions from MCA were retained as the best solution. Cronbach's alpha, 0.727 indicated a good internal consistency and it is acceptable in exploratory research [11]. Figure 1 presents the two-dimensional map of MCA with the co-ordinates of 1344 respondents. The variables duration of sleep (D1 = 58.9%, D2 = 55.2%), difficulty falling asleep (D1 = 47.5%, D 2= 42.7%), family history disease (D1 = 26.2%, D2 = 21.4%) and family history for cognitive or memory impairments (D1 = 30.6%, D2 = 32%) presented similar discrimination measures in both dimensions (correlations noted in parentheses as a percentage). The first dimension (eigenvalue = 2.917), explained 41.7% of the total inertia (Table 4 and Figure 1).

The first axis included participants who suffered from musculoskeletal or other diseases and the lowest duration of medication (less than one month), at the positive pole. At the negative pole, dimension one encompasses duration of medication between one month and six months or more than one year and the participant suffered from cardiovascular or respiratory disease. The second dimension (eigenvalue = 2.387), explained 34.1% of the total inertia. The most discriminant parameter was Mc Nair score. This dimension bipolarity encompasses participants with no cognitive complaint at the negative pole and participants with cognitive complaints at the positive pole. The profile of respondents who suffered from

cognitive complaints is positioned in the top right quadrant of the co-ordinate graph of modalities, which is illustrated by the positive pole of the first axis and the positive pole of the second axis. This region is described by profiles with the lowest sleep time (4 hours), high difficulty to fall asleep (Difficult), family respondents suffered from other diseases than cardiovascular, neurologic, respiratory or musculoskeletal in addition to a memory deficiency, a duration of medication between one month and six months and the participant suffered from musculoskeletal disease. Contrariwise, the profile of participants with no cognitive impairment is characterized specifically by low difficulty of falling asleep (little) and duration of medication more than one year or none. All other parameters were weakly associated with both dimensions and

Table 4: Discrimination measures.

	Dimension	
	1	2
Sleep duration	0.589	0.552
Duration of medication	0.456	0.102
Difficulty falling asleep	0.475	0.427
Family's history disease	0.262	0.214
Participant's current disease	0.823	0.644
Family's history for cognitive or memory impairments	0.306	0.320
Mcnair score	0.005	0.130
Total active variables	2.917	2.387

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the interpretation was insufficient due to the proximity of the modalities to the center of the plot.

Discussion

MCA and logistic regression were used in this study to explore relationships between cognitive complaints, clinical and sleep parameters. The main result reported in Table 3 was that duration of sleep, duration of medication, sleep quality and difficulties falling asleep were significantly associated with cognitive problems. These findings are consistent with other studies in which cognitive complaints are shown, related to their impact on clinical outcomes [2-4,12-14]. However the RR reported in Table 3 showed that the risk to suffer from cognitive difficulties for participants who have no difficulty of falling asleep is more than for participants who have a very difficult to fall asleep. This would suggest that the fact to have no difficulty of falling asleep is associated with an increase in risk. This finding is not consistent with studies in which cognitive complaints were shown as associated with difficulty of falling asleep [15]. Further, the result reported in Figure 1 showed that respondents suffering from cognitive complaints reported low sleep time, high difficulty to fall asleep, family's participants suffered from other diseases than cardiovascular, neurologic, respiratory or musculoskeletal in addition to a memory deficiency and the participant suffered from musculoskeletal disease. These results agree with authors like Lucassen et al. and Zhu, who found a positive association between many physiological disorders (including metabolic syndromes and physiopathology illness) and cognitive complaints [15-20].

Our goal is to build the most complete model of detection and prediction of cognitive impairments for the YA population. So, this protocol was designed to test in simultaneous, all

the main risk factors known for their serious influence on the brain disorders. Subjects with responsibilities and people executing daily multitask professionals, stimulated regularly and intensively their brains. And if these individuals are anxious, not psychologically stable with a poor well-being and at least minor depression; cognitive performance is strongly affected. At the same time, our results suggest a specific profile of participants at risk of future brain failure; because we determined here the proportion of cognitive decline (according to the results of each participant in the McNair test). Many studies were focused on pathological factors such as stroke or cardiovascular diseases that could negatively impact cognitive function, by declining attention, reducing motor reflexes, and slow capacity of performing multitask action [12,21-26]. But there are just few of them which observed deeply the simultaneous actions of all these factors in the same time as us, in the present investigation. It has been established in the last decade that reduces psychological stress with associated illness such as depression and anxiety; decrease cognitive decline, while a good quality of sleep and a perfect well-being ensure brains stability and good neurocognitive function [2,4,14]. Issue is adequate data for YA brain disorders are not present actually, and our results evoke the possibility of a negative impact on the simultaneous combination of these 9 lifestyles and physiological factors; on cognitive impairment and brain disorders, if not properly prevent. Because people with less than eight hours of sleep and less physical exercise has a bad global score on McNair tests, and the four dimensions of the same scale. We are able to detect cognitive decline with our questionnaire, if it is employed as a continuous evaluation; even with people without a cognitive diagnostic.

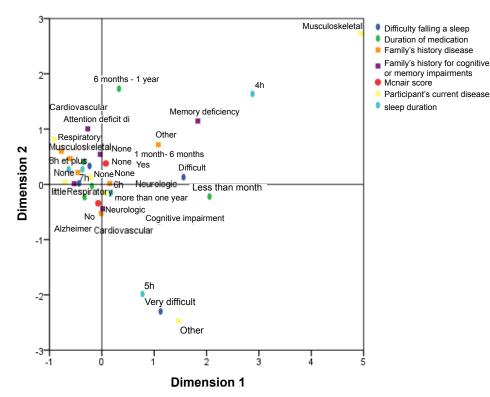


Figure 1: Multiple correspondence analysis: Coordinates graph of cognitive complaints. Clinical and sleep variable modalities.

According to our results, a regular evaluation of cognitive performance (with a classical cognitive test like McNair or Mini-mental state) while controlling behavioural components (anxiety, depression, well-being and psychological stress) and physiological components (duration of sleep and quality, addiction, global health) and physical activity, should lead to a sensitive and accurate model of prediction. A study is running currently in few hospitals and universities in Quebec, to validate our questionnaire and the findings of this present research. We also think it will be more accurate in our next data collection, to use this protocol with a continuous follow up of the evolution of cognitive impairments or mental status of our sample, during at least one year with a control every month, for example. This will allow us to draw a linear progression of cognitive impairment, and elaborate a midterm model of prediction relates to brain disorder.

Conclusion

Follow at the same time all listed parameters in this research, contributes both to a better brain disorders prevention and monitoring initial symptoms of cognitive decline. YA and midlife samples for both men and women have almost the same mental health profile but, cognitive parameters of men are more affected by this combination compared to women. Age is also a main factor because the majority of our sample was aged between eighteen and thirty, and the best score was obtained by people over thirty years old. This result suggests that monitoring the mental state of YA will decrease considerably memory complaints and prevent the onset of brain disorders; and we also suggest starting early detection.

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References

- Atherton KE, Nobre AC, Zeman AZ, Butler CR. Sleepdependent Memory Consolidation and Accelerated Forgetting. *Cortex.* 2014; 54: 92-105.
- 2. Burke TM, Scheer FA, Ronda JM, Czeisler CA, Wright KP. Sleep Inertia, Sleep Homeostatic and Circadian Influences on Higher-order Cognitive Functions. *J Sleep Res.* 2015; 24: 364-371.
- Ferini-Strambi L, Galbiati A, Marelli S. Sleep Microstructure and Memory Function. *Front Neurol.* 2013; 4: 159.
- Martin MS, Sforza E, Roche F, Barthélémy JC, Thomas-Anterion C. Sleep Breathing Disorders and Cognitive Function in the Elderly: An 8-year Follow-up Study. The Proof-synapse Cohort. *Sleep*. 2015; 38: 179-187.
- 5. Kimhy D, Vakhrusheva J, Bartels MN, Armstrong HF, Ballon JS, et al. Aerobic Fitness and Body Mass Index

in Individuals with Schizophrenia: Implications for Neurocognition and Daily Functioning. *Psychiatry Res.* 2014; 220: 784-791.

- Maesako M, Uemura K, Iwata A, Kubota M, Watanabe K, et al. Continuation of Exercise is Necessary to Inhibit High Fat Diet-induced Beta-amyloid Deposition and Memory Deficit in Amyloid Precursor Protein Transgenic Mice. *Plos One.* 2013; 8: E72796.
- Panza F, Solfrizzi V, Barulli MR, Santamato A, Seripa D, et al. Cognitive Frailty: A Systematic Review of Epidemiological and Neurobiological Evidence of an Age-related Clinical Condition. *Rejuvenation Res.* 2015; 18: 389-412.
- Gomes da Silva S, Unsain N, Mascó DH, Toscano-Silva M, de Amorim HA, et al. Early Exercise Promotes Positive Hippocampal Plasticity and Improves Spatial Memory in the Adult Life of Rats. *Hippocampus*. 2012; 22: 347-358.
- Atkinson HH, Cesari M, Kritchevsky SB, Penninx BW, Fried LP, et al. Predictors of Combined Cognitive and Physical Decline. J Am Geriatr Soc. 2005; 53: 1197-1202.
- 10. Thomas AG, Monahan KC, Lukowski AF, Cauffman E. Sleep Problems Across Development: A Pathway to Adolescent Risk Taking Through Working Memory. J Youth Adolesc. 2015; 44: 447-464.
- Costa PS, Santos NC, Cunha P, Cotter J, Sousa N. The Use of Multiple Correspondence Analysis to Explore Associations between Categories of Qualitative Variables in Healthy Ageing. J Aging Res. 2013; 2013: 12.
- Miller MA. The Role of Sleep and Sleep Disorders in the Development, Diagnosis and Management of Neurocognitive Disorders. *Front Neurol.* 2015; 6: 224.
- Urbain C, Galer S, Van Bogaert P, Peigneux P. Pathophysiology of Sleep-Dependent Memory Consolidation Processes in Children. *Int J Psychophysiol.* 2013; 89: 273-283.
- Villa C, Ferini-Strambi L, Combi R. The Synergistic Relationship between Alzheimer's Disease and Sleep Disorders: An Update. *J Alzheimers Dis.* 2015; 46: 571-580.
- Torelli F, Moscufo N, Garreffa G, Placidi F, Romigi A, et al. Cognitive Profile and Brain Morphological Changes in Obstructive Sleep Apnea. *Neuroimage*. 2011; 54: 787-793.
- 16. Joo EY, Kim H, Suh S, Hong SB. Hippocampal Substructural Vulnerability to Sleep Disturbance and Cognitive Impairment in Patients with Chronic Primary Insomnia: Magnetic Resonance Imaging Morphometry. *Sleep.* 2014; 37: 1189-1198.
- Lucassen EA, Piaggi P, Dsurney J, de Jonge L, Zhao XC, et al. Sleep Extension Improves Neurocognitive Functions in Chronically Sleep-deprived Obese Individuals. *Plos One.* 2014; 9: E84832.
- Parisi P, Bruni O, Pia Villa M, Verrotti A, Miano S, et al. The Relationship between Sleep and Epilepsy: The Effect on Cognitive Functioning in Children. *Dev Ned Child Neurol.* 2010; 52: 805-810.

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- Rolinski M, Zokaei N, Baig F, Giehl K, Quinnell T, et al. Visual Short-term Memory Deficits in REM Sleep Behaviour Disorder Mirror those in Parkinson's Disease. *Brain.* 2016; 139: 47-53.
- Zhu B, Dong Y, Xu Z, Gompf HS, Ward SA, et al. Sleep Disturbance Induces Neuroinflammation and Impairment of Learning and Memory. *Neurobiol Dis.* 2012; 48: 348-355.
- 21. Kwon KJ, Lee EJ, Kim MK, Jeon SJ, Choi YY, et al. The Potential Role of Melatonin on Sleep Deprivation-induced Cognitive Impairments: Implication of FMRP on Cognitive Function. *Neuroscience*. 2015; 301: 403-414.
- 22. Kreutzmann JC, Havekes R, Abel T, Meerlo P. Sleep Deprivation and Hippocampal Vulnerability: Changes in Neuronal Plasticity, Neurogenesis and Cognitive Function. *Neuroscience*. 2015; 309: 173-190.

- Hayes SM, Alosco ML, Forman DE. The Effects of Aerobic Exercise on Cognitive and Neural Decline in Aging and Cardiovascular Disease. *Curr Geriatr Rep.* 2014; 3: 282-290.
- 24. Yu F, Xu B, Song C, Ji L, Zhang X. Treadmill Exercise Slows Cognitive Deficits in Aging Rats by Antioxidation and Inhibition of Amyloid Production. *Neuroreport.* 2013; 24: 342-347.
- 25. Moreira EL, Aguiar AS Jr, de Carvalho CR, Santos DB, de Oliveira J, et al. Effects of Lifestyle Modifications on Cognitive Impairments in a Mouse Model of Hypercholesterolemia. *Neurosci Lett.* 2013; 541: 193-198.
- 26. Gomes da Silva S, Simões PS, Mortara RA, Scorza FA, Cavalheiro EA, et al. Exercise-induced Hippocampal Anti-Inflammatory Response in Aged Rats. *J Neuroinflammation*. 2013; 10: 61.

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