

# **Factors Underlying Cognitive Decline in Old Age and Alzheimer's disease: the Role of the Hippocampus**

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## **Abstract**

There are many factors that strongly influence the etiology, development, and progression of cognitive decline in old age, mild cognitive impairment (MCI), and Alzheimer's disease (AD). These factors include not only different personality traits and moods, but also lifestyle patterns (e.g., exercise and diet), and awareness levels that lead to cognitive decline in old age. In this review, we discuss how personality traits, mood states, and lifestyle impact brain and behaviour in older adults. Specifically, our review shows that these lifestyle and personality factors affect several brain regions, including the hippocampus, a region key for memory that is affected by cognitive decline in old age as well as AD. Accordingly, appropriate recommendations are presented in this review to assist individuals in decreasing chances of MCI, dementia, AD, and associated symptoms.

## **Introduction**

The occurrence of cognitive decline in old age depends on many differences among individuals. In this study, we review research addressing personality, mood, and lifestyle factors underlying cognitive decline in old age that may vary across individuals. Identifying these factors will help therapists address such factors at a younger age, thus reducing chances of developing old age related neurological disorders. Cognitive decline in later life commonly occurs as part of a range of neurological conditions that come under the umbrella term of dementia. Alzheimer's dementia (AD) is the most common form of dementia with no cure existing today. It has a higher occurrence rate in females than in males (approximately 56% and 31.4%, respectively), and often occurs in individuals aged 65 years and older ("Alzheimer's Association - 2014 Alzheimer's Disease Facts and Figures ", 2014). AD is also a common cause of death in elderly individuals ("National Institute on Aging- Alzheimer's Disease Fact Sheet," 2015). Like healthy ageing, various individual differences impact the progression of AD, including lifestyle (e.g., type of diet and amount of exercise) and personality (e.g., neuroticism and exposure to/coping with stress).

In this review, we explore the evidence for lifestyle and personality traits that influence individuals' rate of cognitive decline in old age as well as onset of Alzheimer's disease pathophysiology. In addition, we consider emotional states associated with sadness, stress, and loneliness, which also lead to MCI and AD symptoms. Moreover, older adults and AD patients' lifestyles such as diet and exercise patterns will be discussed, as well as how lifestyle can improve or worsen cognitive function and disease progression.

### **Personality effect on Cognitive Decline:**

#### **Neuroticism**

Personality traits such as neuroticism generally have negative impacts on normal functioning; they may be linked to increased risks of developing cognitive decline and dementia (Archer, 2009). Neuroticism is a personality trait characterised by moodiness, anger, fear, anxiety, and jealousy, among others. According to Chapman et al. (2012), older individuals scoring high on neuroticism scales but low on openness (e.g., being accepting of change) and conscientiousness (e.g., being careful, organised, and mindful) scales exhibit poor long-term cognitive functioning and steeper rates of cognitive decline (Chapman, 2012). A possible explanation for cognitive decline in old age is hippocampal atrophy related to glucocorticoid drugs (Sapolsky, 1996) such as chronically elevated cortisol levels (Chapman, 2012). Increased cortisol has been associated with stress characteristics of negative affect (Buchanan, 1999) which may relate to neuroticism. In addition, emotional stress and glucocorticoids (associate characteristics of neuroticism) may also lead to structural changes in the hippocampus (atrophy and volume loss) further contributing to cognitive decline (Boyle, 2010; Gurvits, 1996; McEwen, 2000). Changes in these personality domains are significantly high in AD patients where premorbid personality traits have a moderate association with levels of cognitive functioning, and functioning in daily life (Chapman, 2012).

In females, midlife neuroticism seems to be associated with the early onset of dementia (Archer, 2009). In comparison to males, female midlife neuroticism may be accounted for by lower levels of the hormone estrogen at menopause (considering AD usually occurs after the mean age of 65 years). If not managed, stress can develop into distress, increasing risks of cognitive decline, impairment, and also AD (Wilson, 2007a).

Neuroticism, in comparison to other personality traits can be suggested to present with elevated activity of the limbic brain (Newbery, 2012), which includes the hippocampus, dentate gyrus, cornu ammonis, amygdala, basal ganglia, as well as other regions. The limbic system related to emotion and memory involves the amygdala and hippocampus. A correlation between activation levels in the amygdala and hippocampus (Poulin, 2011) suggests that atrophy in both may relate to less emotional reactivity in general and in association to memories. During mild AD, it is noted that amygdala activity is most associated with elevated emotions such as irritability and anxiety (Poulin, 2011), which highlights the function of brain alterations that occur in the hippocampus related to neuroticism and cognitive decline in AD (Boyle, 2010).

## **Mood and Emotional States Effect on Cognitive Decline:**

### **Loneliness**

Prolonged feelings of loneliness can harm individuals in many ways. Loneliness can lead to depression and higher risk of death (Holwerda, 2016). Feeling lonely can sometimes also lead to thoughts of being a burden or a liability on family and friends (Davis, 2014). Additionally, although patients with amnesia (and probably AD) show memory decline, they still feel emotions associated with memories ("University of Iowa - Health Science; Study: Patients with amnesia still feel emotions, despite memory loss," 2010). In attempt to improve patient rate of cognitive decline, it could be suggested to improve patients' psychological wellbeing, including stress levels. This can include providing comfort, care, and intimacy. Care is necessary for patients to feel understood and appreciated by others, especially by family, caregivers, nurses, and friends (Anderson, 2007). Observations indicate that loneliness doubles the risks of late-life AD-like dementia (Wilson, 2007b) however, there have been mixed findings. Autopsies suggest loneliness is not related to AD or cerebral infarction (Wilson, 2007b). However, we believe that although the impact of loneliness may not be directly observable, it can influence depression, stress and similar factors which do have direct impacts on cognition. As such, it is reported that depression associated with loneliness may be a contributing factor of AD development (Tanday, 2007).

### **Stress**

Stress and distress (including anxiety) can have many health implications. This includes increased risks and exacerbation of cognitive decline and functioning, MCI, and AD (Wilson, 2011, 2005, 2007a). This may be explained in part by stress-related release of the hormone cortisol, as it has been found that extreme stress leading to excessive release of cortisol affects the hippocampus (Buchanan, 1999; Gurvits, 1996; McEwen, 2000). Extreme elevated cortisol levels influence hippocampal function and result in impaired cognition, specifically memory (Archer, 2009). Reviewing research in the previous section on the

personality trait of neuroticism and now, stress, it is notable that a high level of cortisol is common in the two. The linkage concerns increased cortisol leading to hippocampal atrophy (Chapman, 2012) which, consequently contributes to memory declination (Archer, 2009). To reduce stress and cortisol levels and their impact on cognition, we suggest that individuals switch to a healthy diet, and participate in exercise regularly as part of a healthy lifestyle.

Stress is also a significant risk factor of amnesic MCI (aMCI). In a study conducted by Katz et al. (2016), participants were assessed three years after initial risk assessment of aMCI. It was found that the risk of developing aMCI increased by 30% per five-point score on the Perceived Stress Scale (PSS). Participants with scores in the 80<sup>th</sup> percentile were almost 2.5 times more likely to develop aMCI over this time.

Moreover, females often express higher rates of distress, which is associated with a decline in cognitive functioning over an average of five years (Wilson, 2005). Increased experience of stress in females may explain why worse cognitive functioning is more common in females than males (Wilson, 2005).

As well as increasing the risk of cognitive decline, negative mood states and experiences involving stress, anxiety and so on may also contribute to an acceleration of cognitive decline in people with diagnoses of various forms of dementia (Wilson, 2005). Chronic distress and emotional states (including misery and agony) are common in AD patients. Irritability, agitation and similar emotions also contribute to worse AD patient health and may also affect cognition (Hurt, 2009). High levels of continual distress are associated with decline in multiple cognitive functions, primarily episodic memory (Wilson, 2007a). Moreover, chronic distress is also linked to higher rates of MCI. In addition to an association with global cognitive impairment, chronic distress is also related to limbic system functioning (Wilson, 2007a). However, chronic distress is suspected to specifically influence certain aspects of cognition more than others, predominantly episodic memory (Wilson, 2007a). This association between chronic distress and memory may be tied to the relationship between the hippocampus and limbic system. The anatomic structure of the limbic cortex is connected with the hippocampus therefore, if the limbic system is altered by stress, the hippocampus may also be affected, likely to disrupt memory (Tortora, 2006). This reflects a chain-like link or hierarchical structure where all factors associated with cognitive decline lead or contribute to one another.

Studies of post-traumatic stress disorder (PTSD) patients may additionally help shed light on the connection between stress, distress and cognitive decline. There are increased risks of developing dementia in male veterans under the age of 75 with PTSD (Yaffe, 2010), which suggests that an individual's stressful life experiences increase the likelihood of developing neurodegenerative diseases. An increased risk of disease development has been attributed to damage to hippocampal structures. Suffering from head injury, witnessing traumatic events, and experiencing chronic stress can damage or shrink the hippocampus, causing disruption to learning and memory (Yaffe, 2010). Stressful experiences tend to overlap with ongoing anxiety which is very common in elderly individuals with cognitive decline and dementia (Bierman, 2007). As anxiety increases so does global cognitive decline, making individuals more prone to stress (Wilson, 2011). Moreover, cognitive decline is

related to the amount of stress an individual experiences, increasing chances of developing AD (Wilson, 2011).

### **Depressive symptoms**

Individuals experiencing depression are suggested to also score high on neuroticism (Farmer, 2002), highlighting the importance of considering both factors in AD. It is proposed that depression is associated with hippocampal volume loss (Sheline, 2003) and contributes to cognitive decline and dementia (Yaffe, 1999). However, much of the literature consider depression and stress and their associated emotions separately when assessing and evaluating individual's mental wellbeing, cognition, and risk of AD, despite the fact that such emotional psychological states are closely related (Nima, 2013). We attempt to bridge this gap by discussing the mentioned states and emotions, considering their pathological symptoms and suggesting strategies for minimising the effect of psychological distress on individuals in mid- and late-life.

Depression has been found to be a contributing factor to dementia and AD (Cherbuin, 2015). In elderly individuals' assessments of depression, each point increase on the CES-D/HAM (Center for Epidemiological Studies Depression)/Hamilton Depression Scale) contributes to a 5% increase risk of dementia over a period of 5.2 years (Cherbuin, 2015). Furthermore, scores above 20/21 on the CES-D and similar scales used to measure depression are linked to an 83% increase in the risk of dementia, after an 8.2 year follow up. Similarly, a one-point increase on the CES-D/HAM was found to increase AD development by 6% over a 5.9 year period (Cherbuin, 2015). In more detail, Cherbuin et al. (2015) also note that scores above 20/21 on the CES-D is linked to a 97% risk increase of AD over 6.3 years. These findings are also where dementia patients tend to score high on the CES-D (Fuhner, 2003), implying that depression is related to cognitive decline and dementia development.

Depression is suggested to also relate to pathological issues such as neuroinflammation, oxidative stress, white matter lesions, and cerebral atrophy (Cherbuin, 2015). Such pathological issues are evident even in young individuals with depression. Therefore, for practitioners to minimise chances of developing dementia and AD at a young age, it would be best to consider the cause, treatment and management of pathological issues linked with the development of depression. In fact, it has been argued that a 25% reduction in depression prevalence could result in 827,000 fewer cases of AD worldwide (Barnes, 2011). While managing depression, it should be noted that verbal types of therapy may not be across the board effective because individuals respond differently to forms of therapy. For example, elderly and young males are less likely to engage in emotion talk therapy or even seek support from such therapies (Anderson, 2011). Therefore, it may be more beneficial for individuals to seek support from a wider range of therapies. For instance, in mild and moderate dementia, music therapy not only assists individuals to cope with and reduce depressive symptoms but also improves cognitive functioning and processes (mainly recall) in a positive and effective manner (Chu, 2014).

Apathy, an affective state involving a lack of interest, also affects cognition. Although apathy and depression are different, they can co-occur but are reliant on different neural mechanisms. As mentioned previously, it is important to account for factors such as

depression and apathy as occurring if not combined, then hierarchically in dementia development. Depression and apathy may relate to each other on the basis of similar emotional affects (Benoit, 2012). This is supported by the finding that depressed individuals tend to exhibit apathy, and individuals with apathy tend also to show depressive symptoms (Benoit, 2012). Therefore, we could suggest a unique symptom representative of the overlap between depression and apathy (Benoit, 2012) in cognitive decline. Regarding neuropathological effects of apathy, highly apathetic patients develop cortical emaciation in the caudal anterior cingulate cortex, lateral orbitofrontal cortex, and pars triangularis where neurofibrillary tangles occur (Tunnard, 2011). Changes in these brain regions are suggested to contribute to cortical thinning of the left anterior cingulate, affecting memory among other cognitive processes (Tunnard, 2011). Therefore, it is hypothesised that the underlying psychopathological mechanism connecting apathy and depression with cognition is the role of the anterior cingulate cortex (ACC). It could be suggested that damage to the ACC affects emotional experience and expression, and cognitive functions like learning.

Awareness of one's disease (AD), also known as insight, has been associated with depression, anxiety, and apathy (Horning, 2014). Insight is defined as one's self-understanding, knowledge and awareness of cognitive decline, impairment, and disease diagnosis. On the other hand, anosognosia is the lack of knowing and understanding one's own cognitive and functional difficulties. In elderly individuals and AD patients, anosognosia can limit some abilities, leading to risky and harmful behaviour (e.g., unknowingly overdosing on medications or accidentally keeping the stove on high heat, causing a fire) (Starkstein, 2007). Risky behaviour may be related to the idea that AD patients experience problems with verbal comprehension, fluency, and constructional praxis which may be linked to completing certain processes and functions in a proper and safe manner (Starkstein, 2007). Impairment in aspects of cognition is influenced by patients' awareness and knowledge of their physical and mental health. Lack of self-knowledge continues to degrade with cognitive functioning as supported by Mini-Mental State Examination (MMSE) scores (Hurt, 2009). With continued lack of insight and cognition, anxiety levels start to decrease because anxiety invoking experiences start to have no significant emotional effect (Bierman, 2007).

### **Distinguishing Cognitive Decline from Typical Mood State Symptoms:**

Many individuals in mid and late life exhibit neuroticism, and experience loneliness or stress. However, it is imperative to distinguish between symptoms inherent to aging and those indicative of reversible or solvable loneliness, stress, and depression. In doing so, it is vital to consider both observable behaviour and neurological changes. For example, clinicians often ask if patients are feeling depressed and if they complain of memory problems. If depressed, it is likely that they do not have dementia, as depression has a major impact on episodic memory functioning.

When investigating the differences between physical symptoms of mood changes and age-related cognitive decline, it is important to monitor amygdala activity and hippocampal changes. This includes observing expressions of irritability and anxiety (Poulin, 2011) as indicative of possible signs of the cognitive aging process. It may be possible that if all factors of amygdala activity, hippocampal changes and expressions of irritability and anxiety

occur together or are subsequent to each other, individuals may be in the early stages of behavioural and cognitive decline. If this is found not to be the case, it is essential to consider other factors such as environment. For example, individuals' living spaces are another important factor in cognition as a lonely environment may contribute to global cognitive impairment (O'Lunaigh, 2012). It is also important to not only note individuals' emotional experiences of loneliness but also test memory loss ("University of Iowa - Health Science; Study: Patients with amnesia still feel emotions, despite memory loss," 2010), and assess individuals for stress and depression, and compare scale scores to that of MMSE scores (Katz, 2016; Wilson, 2005).

An additional approach to distinguishing temporary reversible emotional states and moods from those that are age induced is to consider the crucial aspects of cortisol levels and brain alterations, such as increased risk of dementia, atrophy and lesions to regions of the limbic system such as the hippocampus, amygdala, and ACC (Chapman, 2012; Tunnard, 2011). Such assessments will assist researchers and therapists to identify whether an individual is experiencing cognitive impairment that is induced by reversible, temporary or environmental factors (pseudo-dementia) rather than confusing it with other types of dementia (AD).

## **Lifestyle Factors in Cognitive Decline:**

### **Nutrition**

Diet has been found to be related to cognitive decline (Nurk, 2007). There is evidence that tea, fish, fruits, nuts, and turmeric (curcumin) may protect against dementia (Cardoso, 2016; Chen, 2011; Mishra, 2008; Nurk, 2007; Pe'neau, 2011; Poulouse, 2014; Qin, 2014). It is suggested that vitamins, proteins and amino acids may explain their neuroprotective effects (Joseph, 2009; Ng, 2008; Rest, 2013; Xu, 2009). Below, we discuss the benefits of some foods associated with improved cognition and physical health.

Tea is understood to have overall health benefits, including that of reduced cognitive decline and improved mild cognitive impairment (Ng, 2008; Park, 2011). Green tea is found to be associated with lower rates of cognitive impairment (Kuriyama, 2006; Ng, 2008) and, and black fermented and semi fermented tea (oolong) have both been shown to significantly reduce cognitive decline in old age (Ng, 2008). This is possibly due to naturally occurring chemical compounds such as theanine in both black and green tea (Ng, 2008; Park, 2011). Natural compounds found in various types of tea reduce acetylcholinesterase activity and protect  $\beta$ -amyloid driven effects on cognition (Ng, 2008).

Fish may not only improve cognition but may also act as a neuro-protective agent against AD. Elderly individuals who consume fish or seafood weekly show a delay from four to seven years of developing dementia and AD (Nurk, 2007). Consuming more than one serving (e.g., 100g) of fresh water fish is associated with 2.5 times slower cognitive decline in verbal, immediate, and delayed memory (Qin, 2014). Potential explanations of these findings may be Omega-3 fatty acids (w-3 PUFA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) concentrations in fish which have been shown to reduce inflammation and oxidative stress in the central nervous system (Qin, 2014). However, it is



suggested that reduction of inflammation and stress prevent cognitive decline but do not prevent or treat dementia (Qin, 2014). Nurk et al. (2007) also identified that both unprocessed fatty and lean fish lead to significant improvements in overall cognitive performance. However, it is suggested that consumption of fatty fish has more protective effects against dementia and AD than lean fish (Huang, 2005), which may be related to the nutritional factors found in fatty fish such as higher DHA and PUFA (Zhang, 2016). Moreover, processed fish is noted to be the second most common contributor to improving episodic and short term memory, visospatial and motor skills, naming and recall among other cognitive functions (Nurk, 2007). Enhanced cognition has also been associated with the consumption of fish oil (O'Connor, 2012) which is apparent in semantic memory, verbal fluency, and psychomotor speed (Nurk, 2007).

Various nutrients have been found to affect cognition differently. Vitamins C and E contained in fruits such as strawberries and blueberries have shown to improve verbal memory in middle to old aged individuals (Pe´neau, 2011). Moderate fruit consumption is positively correlated with cognition in individuals with MCI (Chen, 2011). Antioxidants and polyphenolics present in some fruits (e.g., concord and blueberry juice ) are thought to mediate improvements in cognitive functions such as memory by contributing to the reversal of aging neurons and forgetful behaviours typically observed in elderly individuals (Joseph, 2009).

Nuts have many benefits including that of improving cognitive function (e.g., reducing memory decline)(Nooyens, 2011). Specifically in relation to walnuts they are suggested to reduce chances of AD by managing A $\beta$  peptides, and protecting cells from toxicity of free damaging radicals with its antioxidant properties (Muthaiyah, 2011). Walnuts, rich in polyphenol and melatonin when taken in moderate amounts improve cognition (memory) and motor function (Willis, 2009), possibly because polyphenols reduce oxidative stress and inflammation (Poulose, 2014). Walnuts also have a high content of PUFAs (EPA and DHA) found to improve and maintain cell and nerve functioning (Yehuda, 2002). PUFAs enhance synaptic plasticity and stability of the neural membrane which contributes to neural transmission, and also play a role in gene expression and neurogenesis (Yehuda, 2002). Also found in walnuts is the amino acid tryptophan which may improve cognitive functioning of elderly individuals. Tryptophan produces serotonin, improving aspects of verbal and short-term memory by influencing effective transmission of neural signals and messages to and from the brain (Rest, 2013). In Brazilian nuts, the mineral Selenium is also suggested to be beneficial in the prevention of dementia. With its antioxidant properties, selenium protects the body from free radicals, oxidative stress and other neuropathological complexities which negatively influence neurotransmission (Cardoso, 2016). Brazilian nuts are found to reduce and restore selenium in elderly individuals, improving semantic verbal fluency and constructional praxis among other less focused cognitive functions (Cardoso, 2016).

Turmeric (curcumin) is a natural anti-inflammatory plant that is used as a spice and natural herbal medicine. Curcumin has been shown to reduce inflammation commonly found in neurodegenerative diseases such as AD (Mishra, 2008). Additionally, curcumin can reduce infarction, deficit and oxidative stress, and prevent excessive formation of amyloid plaques (Mishra, 2008). Curcumin has effects on proteins and cells such as cytokines and

macrophages which have a crucial role in internal bodily responses against amyloid plaques that occur in AD (Xu, 2009). Curcumin also has beneficial effects similar to that of omega-3 fats in that it reduces inflammation and oxidative stress affecting the brain (Xu, 2009).

### **Fitness and exercise**

It is suggested that physical activity reduces cognitive decline associated with AD (Larson, 2008). Fitness influences mental and physical wellbeing and is associated with improved cognition. Regular exercise contributes to protecting individuals from experiencing hippocampal volume loss, brain atrophy, and declined memory which are characteristics of AD (Erickson, 2011). Young individuals with high fitness levels and who regularly participate in cardio-respiratory fitness are twice less likely to develop dementia, including AD (Liu, 2011). Similar findings were found in healthy individuals without dementia, in that individuals with increased rates of daily physical activity had significantly reduced risks of AD over 4 years (Buchman, 2012). Data analyses also showed a negative correlation between physical activity and decline in episodic, working memory and visuospatial cognitive functions (Buchman, 2012).

Furthermore, moderate participation in fitness in general, specifically aerobics, protects from and reduces old-aged hippocampal volume loss (Erickson, 2011). Aerobic exercise is found to reverse volume loss, and increase volume of the anterior hippocampus (Erickson, 2011). In addition, an increase in amount of grey and white matter of the prefrontal cortex is apparent in exercise participation (Erickson, 2011). Exercise is also assumed to be associated with increases in brain-derived neurotrophic factor (BDNF) serum levels and enhancements to anterior hippocampal volume, which in turn improves memory (Erickson, 2011). Thus, it can be suggested that exercise improves neuronal growth and cerebral blood flow (CBF), increases brain volume, and improves learning and memory (Erickson, 2011; Sullivan, 2008). High levels of fitness in AD patients are also related to a four time reduction in brain atrophy than individuals with low levels of fitness. Exercise also contributes to increased white matter volume in the hippocampal, inferior temporal gyrus, and precentralgyrus brain regions (Sullivan, 2008).

In sum, involvement in physical activity such as aerobics may improve executive control, and train regions of the brain such as the hippocampus. This may relate to exercise increasing dentate gyrus cerebral blood volume which improves cognition (learning) (Pereira, 2007). The benefits of fitness are important to advocate in order to reduce young individuals' risks of cognitive and functional impairment in later life.

### **Sleep**

Low hours of sleep at night are also associated with lack of exercise (Ohayon, 2005), which may result in having fewer protections against cognitive decline. Also related is the idea that neurogenesis in individuals who exercise is the correlation of oxygen volume used by individuals (Pereira, 2007), suggested to take part in neurogenesis which sleep dysfunction may interrupt, leading to cognitive decline. In support of this idea is sleep disturbance and fewer hours of sleep also been shown to be risk factors for cognitive decline, dementia, and AD development (Osorio, 2011; Spira, 2014). Ohayon and Vecchierini (2005) reported

obstructive sleep apnea (OSA), rapid eye movement (REM), and other sleep factors as contributing to cognitive decline.

Presenting a potential relationship between OSA and cognitive processing is Stranks and Crowe (2016) suggesting impaired patients presenting worse executive function (Alchanatis, 2004; Ancoli-Israel, 2008), specifically non-verbal memory, concept formation, and other functionality, and psychomotor speed (Stranks, 2016). Individuals who received intervention for OSA using nasal continuous positive airway pressure (CAP) have shown improved cognition in aspects of memory and executive function which is suggested to be affected by interruption of breathing during sleep (Ancoli-Israel, 2008). The way in which OSA may impact on cognition includes lack of oxygen reaching brain tissue (hypoxia) leading to brain cell injury, associated with individuals bodily functioning experiencing a state of oxygen deficiency (anaerobic glycolysis) (Alchanatis, 2004). Although CAP apnea machines can support breathing (oxygen intake) to lessen chances of cognitive impairment, they cannot completely repair damage caused by OSA or make up for the benefits of normal, healthy sleep function (Alchanatis, 2004; Ancoli-Israel, 2008) which may involve the role of neurogenesis.

Another sleep factor shown to affect cognition is REM sleep (Ohayon, 2005). Some studies show how REM sleep may be related to cognitive impairment in elderly individuals and in relation to emotional and mood states. In middle to elderly aged men, decreased time in the REM stage of sleep was associated with elevated cortisol during evening time of the day contributing to memory decline (Van Cauter, 2000). It has also been reported that individuals with MCI are likely to develop rapid eye movement sleep behavioural disorder (RBD)(Gagnon, 2012). The signs of cognitive impairment upon developing RBD involve memory, attention, and concentration dysfunction among many other symptoms which commonly occur in neurodegenerative diseases such as AD, Parkinson's disease, and Lewy Bodies dementia (Gagnon, 2012).

## **Conclusion**

Factors associated with cognitive decline in elderly individuals include personality, mood, and lifestyle. Personality and mood states either negatively or positively influence memory performance, most likely by affecting hippocampus function. The extent factors contribute to risks of cognitive decline usually determines the delay or onset of neurodegenerative diseases such as dementia and AD. Making appropriate and necessary lifestyle changes may minimize the risks and severity of cognitive decline and impairment.

In response to factors contributing to cognitive decline, further considerations need to be made and studied in the future. Findings of Katz et al. (2016) emphasise the importance of considering different aspects of individuals' living environment. Creating a positive environment, especially for the elderly can lessen the chances of experiencing sadness, stress, anxiety and similar emotions associated with cognitive decline, aMCI, AD, and other age-related diseases. Some necessary lifestyle changes include nutrition, fitness, and the care and company of family, friends and caregivers which can contribute to creating a positive and socially rich environment for elderly individuals. The significance of such implications

involves lifestyle and environmental changes not only influencing mood states, personalities, symptoms and disease progression, but are also associated with brain changes.

Other factors contributing to cognitive decline that can be investigated in the future regard individual differences in terms of education and language quality. One interesting aspect is the association of individuals' level of education and age-related neurodegenerative diseases (Katz, 2016). Individuals who tend to be highly educated and use sophisticated vocabulary, present good language expression of thoughts, ideas, and have complex sentence structure tend to develop dementia at a later age than usual (Marx, 2005). On the other hand, those with poor language qualities suggesting low education levels indicate that education may be beneficial and exercise cognition, reducing risks of AD, thus, making education and language of future interest to explore.

An additional recommendation for future work involves study of the complex properties of languages. As a main form of communication, languages with different characteristics may influence cognitive reserve, decline and impairment, dementia, and AD. It is also important to note that how and when language abilities are developed influence levels of language comprehension, processing and use (Bialystok, 2012), which, in turn might affect levels of cognition, cognitive reserve and control.

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