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The effects of cognitive behavioral therapy designed to manage anxiety in people with Alzheimer's disease

Firas Abbas Fadhil
University of Technology- Iraq
100110@uotechnology.edu.iqEmail:
Telephone: 009647727481386
Orcid: 0000-0002-2447-2622

Abstract

Alzheimer's disease is frequently associated with anxiety, which increase cognitive deterioration. Using anxiety-management cognitive behavioral therapy may help to halt cognitive decline, but more research needed to prove it. The objective of this research is assess the impacts of a cognitive behavioral therapy targeting anxiety on the cognitive deterioration of persons with Alzheimer's disease. Participants randomly assigned to one of two groups: cognitive behavioral treatment (n = 7) or support group (n = 9). Both therapies administered once a week for a total of eight weeks. Cognitive measures (comprehensive cognition, verbal memory, semantic memory, fluency) and an anxiety measure were used to assess the intervention's effects before (T1) and after (T2). After 6 months (T3). Between T1 and T2, the results revealed that CBT had a larger impact on general cognition and anxiety among participants (g = 0.75) than the control group. Between T2 and T3, the support group had a larger improvement (g = 1.20) in participants' verbal fluency than the cognitive behavioral therapy group.

Keywords: clinical neuropsychological, psychosocial intervention, cognitive behavioral therapy, Alzheimer's type dementia, cognitive deterioration, anxiety,

Introduction

Alzheimer's disease is a major international societal issue. It results in a progressive and pervasive impact on people's daily lives. This impact, mainly known in its cognitive and behavioral aspect, also results in a shakeup of identity. Through the difficulty for people to integrate the disease as a new characteristic of their identity. This difficulty stems mainly from cognitive decline and psychological strategies to cope with the threat posed by the diagnosis.

The cognitive decline associated with Alzheimer's disease (AD) varies according to the stage of the disease, but at the early stage, cognitive difficulties are mainly located in episodic memory (events and their context), semantic memory (general knowledge) and verbal fluency (Budson & Kowall, 2011; Hodges, 2006; Noroozian, 2016). In addition to these cognitive symptoms, 39% of people with AD present with anxiety symptoms of varying intensity (Zhao et al., 2016). Several reviews of the literature have established that there is a

authors have shown that CBT could be used with the elderly who present with cognitive decline (Spector et al., 2015; Stanley et al., 2013).

1.1 Alzheimer's disease

1.1.1. Diagnosis and definition. The DSM-5 (American Psychiatric Association, 2013) categorizes Alzheimer's disease under the term "Major neurocognitive disorder due to Alzheimer's disease". This disorder is defined as the presence of a gradual and insidious cognitive decline (eg, memory, learning, or executive function) that interferes with activities of daily living (ADL), that may be accompanied by symptoms neuropsychiatric (eg, mood disturbance, listlessness, or irritability). To date, no biomarker has made it possible to make a 100% reliable diagnosis of the presence of this disease.

Thus, healthcare professionals can only make the probable or possible diagnosis. However, it is possible to assess the level of functional impact caused by the disease according to a categorization divided into 7 stages (Reisberg, 1984). The first stage being the absence of functional impact and cognitive decline and the seventh stage being a major functional and cognitive impact (ability to speak limited to 5 words, absent intelligible speech, difficult or absent psychomotor skills, etc.).

1.1.2. Cognitive impairment.

AD involves a multitude of cognitive impairments, including memory, attention, executive functions and language (Noroozian, 2016).

First, episodic memory is an explicit and conscious memory of the events of our life (Budson, 2011; Tulving, 1972). In AD, it is particularly affected when it is new information learned after the onset of the disease (i.e., episodic anterograde memory). This type of relationship between the presence of anxiety symptoms and development of Alzheimer's type dementia (Beaudreau & O'Hara, 2008; Becker et al., 2018)**.** All stress the significance of developing a treatment that targets both anxiety and cognitive problems in order to break the vicious circle between anxiety and cognitive decline. Moreover, to date, no treatment exists to cure this disease. Indeed, all of the existing treatments, mainly pharmacological (Campos et al., 2016; Mandell and Green, 2011), are only symptomatic (Hugo and Ganguli, 2014). It has been proposed to use psychosocial interventions more frequently to help this clientele (Zeisel, Reisberg, Whitehouse, Woods & Verheul, 2016).

The WHO published data on causes of death in WHO member states for the year 2019 in December 2020. According to the data, there were 2000 deaths in Iraq in 2019 as a result of neurological diseases, 1500 of which were caused by Alzheimer's and other dementias.

Alzheimer's Disease International (2017), as a militant association for people affected by the disease, highlights the impact of the disease whatever the level of development of the country with 10.5 million in Europe, 4 million in Africa, 22.9 million in Asia and 9.4 million cases in the United States.

The aim of this study therefore to evaluate the effect of cognitive behavioral therapy (CBT), designed to treat anxiety, on the cognitive decline of persons with AD. We believe CBT is the ideal intervention to achieve such objective, because it is a therapy which has been proven in the management of anxiety in the elderly (Gould, Coulson & Howard, 2012), which does not cause any major side effects unlike pharmacotherapy and which is particularly popular from people living with AD (Burgener et al., 2008; Muniz et al., 2015). Finally, several

The results of the other study indicated that people living with AD generated fewer words during a semantic fluency task (categories) than during a phonological fluency (letters) task (Adlam et al., 2006). Several cognitive functions are affected from the early stages of AD, including episodic memory, semantic memory and verbal fluency. The cognitive decline observed in people with AD has been associated with several mechanisms, including the presence of anxiety.

1.2. Anxiety and cognition

Anxiety is among the most common neuropsychiatric symptoms in people with cognitive decline (Forrester et al., 2016; Wadsworth et al., 2012; Zhao et al., 2016). The meta-analysis by Zhao and colleagues (2016) did show that 12% to 70% of people living with AD suffer from anxiety, with an average of around 39%. The authors explain the large variance between studies due to methodological differences, such as age of participants and method of assessment. Beaudreau and O'Hara (2008) indicated that elderly living with cognitive impairment experienced more anxiety, and that anxiety had a deleterious impact on cognitive performance, thus creating a vicious cycle. In addition, researchers followed 1998 participants annually (without baseline neurocognitive impairment) over a 12-year period to establish whether neuropsychiatric symptoms, such as anxiety, appeared before or after the onset of cognitive impairment (Wise, Rosenberg, Lyketsos and Leoutsakos, 2019). The results showed that 33% of their participants had anxiety before the onset of a neurocognitive disorder (NCD), compared to 9% who developed anxiety after the onset of NCD. The results of this study suggest that anxiety occurs mainly (but not exclusively) before the onset of dementia. impairment is particularly noticeable by difficulty remembering upcoming appointments, the inability to remember a recent meeting with a loved one or the meal the day before. retrograde memory, that is, autobiographical memories preceding the onset of cognitive decline (eg, childhood memories), are generally well preserved in the onset of illness (Piolino et al., 2003), but the forgetfulness becomes more and more marked as the disease progresses, following the model of Ribot's Law (Budson & Solomon, 2011). This law stipulates that the most recent memories forgotten first and the forgetfulness gradually progresses to the oldest memories (Ribot, 1882).

Like episodic memory, semantic memory is explicit and conscious memory. This gathers all the information and knowledge of factual types, such as words, concepts, definitions, categorization, historical facts or characteristics of an object (Budson, 2011; Tulving, 1972). Moreover, it is the deficits linked to this type of memory which can explain in particular the "lack of the word" or the difficulties in remembering the first name of a loved one, typical symptoms of AD (Tchakoute et al., 2017).

According to a recent review, people living with AD also have difficulty in terms of executive functions (verbal fluency, inhibition, perseverance and decision-making) and working memory, from the early stages of disease (Kirova, Bays & Lagalwar, 2015). Other researchers have found similar results in verbal fluency (Adlam et al., 2006; Stern et al., 2011). Indeed, the results from one of these studies showed that participants with AD performed significantly worse in verbal fluency than participants without cognitive impairment (Stern et al., 2011).

symptoms, only the presence of anxiety was associated with the severity of the neurocognitive symptoms.

More recently, other researchers have obtained similar results (Mah et al., 2015; Rosenberg et al., 2013). Mah's team (2015) investigated whether the presence of anxiety symptoms increased the risk of progression from MCD to AD. They also questioned whether there was an association between the presence of anxiety symptoms and neural mechanisms related to AD (e.g., hippocampus, tonsil, entorhinal cortex volume, cortical thickness). They followed 376 participants with MCD or MA over a period of 3 years. Their results showed that the level of severity of anxiety symptoms influenced the risk of progression from MCD to AD. Indeed, a mild, moderate or severe level of anxiety increased the chances of progression by 33%, 78% and 135%, respectively. In addition, their results suggested that the presence of anxiety was associated with greater annual atrophy of the entorhinal cortex (Mah et al., 2015). This structure median temporal lobes contributed a main role in the consolidation of declarative memory (Bear, Connors & Paradiso, 2007), thus supporting the association between anxiety symptoms and neural mechanisms related to memory difficulties in people with AD.

1.3. Psychosocial interventions

Psychosocial interventions include a multitude of so-called “non-pharmacological” treatments and are defined as “any intervention aimed at improving the quality of life and maximizing the functions of people, in the context of their current deficits” (Rabins and colleagues, 2007). Cognitive behavioral therapy (CBT) is one of the promising psychosocial interventions for individuals with AD, especially for its beneficial effect on anxiety symptoms.

Many have therefore wondered whether the presence of anxiety could influence cognitive decline, particularly when looking at the rate of progression from Mild Cognitive Disorder (MCD) to AD.

Researchers followed a MCD and dementia-free population aged 75 and over for approximately 4 years to evaluate the influence of neuropsychiatric symptoms on cognitive decline (Palmer, Berger, Monastero, Winblad, Bäckman & Fratiglioni, 2007). Their results suggested that the presence of anxiety symptoms, such as difficulty making decisions or the presence of lingering worries increased the risk of progression to AD, in people with or without MCD at baseline.

Ramakers and colleagues (2010) followed for 10 years 263 participants with TCL. They notably studied the presence of neuropsychiatric symptoms (anxiety-depressive symptoms, apathy and sleep difficulties). About half of the participants were living with one or more of these symptoms at the start of the study. By the end of the study, 90 participants had progressed to dementia, 88% of these were of AD. Among the neuropsychiatric symptoms studied, the researchers found that only anxiety was a predictor of progression from MCD to AD.

(Wadsworth et al., 2012) studied the impact of neuropsychiatric symptoms on disease progression, as well as their impact on symptom severity evaluated with Clinical Dementia Rating (CDR). Their data came from the Alzheimer's disease neuroimaging initiative, a 3-year longitudinal study including normal participants, MCD and living with AD. These researchers have shown that the more severe the anxiety symptoms at baseline, the greater the risk of progression from MCD to AD. In addition, among the neuropsychiatric

indicated that participants who received the multimodal intervention improved by 0.4 points on the *Mini Mental State Examination* (MMSE; Folstein et al., 1983) after the twenty-week follow-up compared to a deterioration of 0 , 5 points for people who did not receive the intervention.

In summary, only three studies have evaluated the effectiveness of CBT in seniors living with dementia. These studies have shown that it was feasible to perform CBT with this clientele and that this therapy could reduce the symptoms of anxiety (Spector et al., 2015; Stanley et al., 2013) or slow cognitive decline (Burgener et al., 2008). However, this last study has several limitations, which will be addressed in this research. In addition, the authors of an editorial recently published in the British Journal of Psychiatry stress the importance of having more studies that will better understand the effects of psychosocial interventions on cognitive decline associated with dementia (Savulich, et al., 2019).

Thus, in the light of the studies cited above, it is essential to better understand the effects of a CBT on the cognitive deterioration of persons with AD, all the more so with the accelerated aging of the population, the number of new cases of AD will increase over the next few years. Another reason to be interested in the beneficial effects of CBT is that the elderly clearly express a preference for this type of treatment, as it reduces the side effects and problems associated with polypharmacy (Rodakowski et al., 2015).

2. Method

2.1. Participants

A total of 17 participants presented for the post-intervention assessment (T2). Between T2 and T3 (6 months after the intervention) only one

Indeed, the authors of a recent meta-analysis (Orgeta, Qazi, Spector & Orell, 2015) concluded that CBT was effective in reducing symptoms of anxiety in individuals with dementia. The studies presented in this meta-analysis evaluated the effectiveness of the Peaceful Mind program, an intervention based on cognitive and behavioral principles of CBT, in older people with dementia (Stanley et al., 2013). The experimental group was compared to a control group receiving usual care. The intervention has been shown to be influential in reducing anxiety and improving life quality. Other researchers have also evaluated the effect of a CBT on the anxiety level of persons with dementia (Spector et al., 2015). As in the previous study, the researchers compared the experimental group (N = 25) to a control group receiving usual care (N = 25). Participants who received CBT had fewer anxiety symptoms at the end of the procedure, and these treatment gains remained stable after 6 months. These results therefore suggest that CBT is a useful and effective therapeutic approach for the elderly suffering from mild to moderate dementia. However, these two studies did not verify the effects of CBT on the cognition of participants with dementia.

To our knowledge, only one research study has investigated the effectiveness of CBT in slowing cognitive decline in people living with dementia (Burgener, Yang, Gilbert & MarshYant, 2008). The objective of this study was to test the feasibility and effectiveness of a multimodal intervention (CBT, Taiji exercises and support group) on the cognitive, behavioral and physical functioning of people living with dementia (N = 43). More specifically, the effects of this intervention (n = 24) were compared with the effects of a control group receiving no treatment (n = 19). Their results

cognitive decline, used to assess participants' overall cognitive functioning (Costa et al., 2014; Lezak, 2012). This scale assesses 7 fields of cognition (i.e., visuospatial and executive functions, naming, memory, attention, language, abstraction and orientation). MoCA has excellent sensitivity to correctly identify the presence of dementia.

2.2.3.2. Memory and learning. Rey's 15-word task (Strauss, Sherman & Spreen, 2006) was used to assess participants' verbal memory. During this task, a list of 15 words read to the participant, who must then recall it immediately after hearing it, and then 30 minutes later. This task allows you to have a measurement of immediate memory (immediate recall) and long-term memory (delayed recall).

2.2.3.3. Verbal fluency. A verbal fluency test also used to assess the ability of participants to generate words quickly (Lezak, 2012). The test is divided into two stages: naming as many words as possible starting with a letter (phonological fluency), then belonging to a specific category (semantic fluency), for a period of 120 seconds. The choice of the letter (P or T) and the category (animals or clothing) was made by alternating between the measurement times, in order to attenuate the effect of practice.

2.3. Procedure

The evaluations were divided into two sessions: first, the participants underwent a medical and psychological evaluation of approximately 120 minutes carried out by a doctor from the medical city of Baghdad (medical evaluation) and doctoral students in psychology from the University of Montreal previously trained. Second, participants had to complete a neuropsychological assessment of approximately 90 minutes. Doctoral students in neuropsychology performed this assessment. participant (CBT group) dropped out of the study because the person who had been accompanying him from the start was no longer available. Finally, one participant was excluded from the statistical analyzes since his score at the first assessment (T1) was deemed too low to be representative of the sample. The final sample in T3 therefore included 15 participants (6 in the experimental group and 9 in the control group), or 2 less than in T2 and 4 less than in T1.

2.2. Material

2.2.2. Clinical interview and self-reported questionnaires.

2.2.2.1. Severity of dementia. Clinical Dementia Rating (CDR) used by the doctor to assess the level of severity of the dementia (eligibility criteria). This tool is divided into 6 areas: Memory, Orientation, Judgment and Problem Solving, Social Activities, Home and Leisure Activities, and Personal Care. The CDR has excellent inter-rater reliability (Burke et al., 1988) and is clinically valid for separating people with and without dementia as well as for differentiating between different levels of disease severity (Morris, 1997).

2.2.2.2. Anxiety. The intensity of anxiety symptoms was assessed by the Abbreviated Penn State Worry Questionnaire (PSWQ-A; Hopko et al., 2003), a self-reported questionnaire that included 8 items answered on a Likert scale ranging from 1 ("not at all corresponding") to 5 (" extremely corresponding"). The PSWQ-A can identify the presence of clinical anxiety in the elderly with a specificity of 92.5% and a sensitivity of 66.4% (Wuthrich, Johnco & Knight, 2014).

2.2.3. Neuropsychological tests.

2.2.3.1. Global cognitive functioning. The Montreal Cognitive Assessment (MoCA), a recommended assessment for measuring

The average age of the final sample was 76.9 +/- 1.4 years, the majority of participants had a university education (56.3%) and half of the sample was women (56, 3%). The mean dementia severity level was 1.3 +/- 0.11 (mild-moderate dementia), but most had a mild severity level (62.5%). Table I shows the demographic and clinical characteristics of our sample before the start of the intervention, according to the group to which the participants belong (experimental or control). The groups were equivalent across all data demographic and clinical. Table 2 details all the results obtained during our comparisons (t-tests and effect sizes) for each of the groups. All reviewers were blind to the participant allocation group. Following these two meetings, eligible participants were randomly assigned to one of the following two groups by matching them according to their gender, age, and level of self-criticism: 1) experimental group receiving CBT designed to manage the disease or anxiety. 2) control group consisting of a support group. The two interventions are detailed in section 3.3. Participants were reassessed at the end of the intervention (T2) and six months later (T3). The Ethics Committee of university of Baghdad approved the research project.

3. Results

Table I Main characteristics of participants

Characteristics	Intervention group (n = 7)	Support group (n = 9)	Signification
	M (SD or%)	*M (SD or%)*	*p*
Demographic			
Age	*77,86 (6,64)*	*76,22 (5,22)*	*0,59*
Sex			
Man	*3 (43 %)*	*4 (44 %)*	*0,95*
Women	*4 (57 %)*	*5 (56 %)*	
Education (year)			*0,91*
Primary	*0 (0 %)*	*1 (11 %)*	
Secondary	*2 (29 %)*	*1 (11 %)*	
College	*1 (14 %)*	*2 (22 %)*	
University	*4 (57 %)*	*5 (56 %)*	

Cognitive measures			
Dementia severity (CDR)	*1,29 (0,49)*	*1,39 (0,49)*	*0,68*
MoCA	*17,14 (2,91)*	*19,56 (3,32)*	*0,15*
Verbal memory *	*0,44 (1,20)*	*-0.34 (0,42)*	*0,15*
Semantic memory *	*0,03 (0,66)*	*-0,27 (1,07)*	*0,90*
Verbal fluency	*18,29 (5,66)*	*17,39 (7,32)*	*0,79*
Psychoaffective measures			
Anxiety *	*0,36 (0,99)*	*-0,28 (0,82)*	*0,18*

* Composite variable in score ž

3.1. Overall cognitive decline

According to the results obtained with the t-tests, the differences in means obtained for each of the groups did not differ significantly between T1 and T2 (t (15) = 0.77, p = 0.45, g = 0.63) or T2 and T3 (t (14) = 2.24, p = 0.08, g = 1.71) at the level of cognition overall. However, obtaining a large effect size in favor of CBT between T1 and T2 suggests that our intervention had a greater effect on participants' global cognition than the control group. Indeed, participants in the CBT group improved slightly between T1 and T2 on their global cognition (M = 1.00; S-T = 6.08), an increase of up to 5 additional points on the MoCA. This improvement was however followed by a decline in global cognition between T2 and T3 (M = -2.33; S-T = 0.58). As for the participants in the support group, they deteriorated on their global cognition between T1 and T2 (M = -1.75; SD = 2.63), a decline that continued between T2 and T3 (M = -0.25; SD = 1.50).

3.2. Verbal memory

For verbal memory, our analysis indicated that the mean difference scores obtained for each of the groups did not differ significantly between T1 and T2 (t (15) = 0.34, p = 0.74, g = 0 .17) or T2 and T3 (t (14) = 1.03, p = 0.32, g = 0.55).

Among participants in the CBT group, verbal memory even deteriorated slightly between T1 and T2 (M = -0.09; SD = 1.11) and between T2 and T3 (M = -0.19; SD = 0.91). Conversely, our results suggested that the verbal memory of the participants in the support group improved slightly between T1 and T2 (M = 0.07; SD = 0.84) as well as between T2 and T3 (M = 0.17; SD = 0.46).

3.3. Semantic memory

The results of our analyzes indicated that there was no significant difference between the differences in means obtained in the two groups on semantic memory (T2 vs T1: t (15) = 1.15, p = 0, 27, g=0.58; T3 vs T2: t(14)=1.06, p=0.31, g=0.50).

Moreover, just as the results obtained in verbal memory, the performance of the CBT group deteriorated slightly between T1 and T2 (M = -0.18; SD = 0.55) as well as between T2 and T2. T3 (M = -0.11; SD = 0.29). Conversely, our analysis suggested that the semantic memory of the participants in the support group improved slightly between these same measurement times (M = 0.14; SD = 0.55 and M = 0.11; SD = 0.51 respectively).

3.4. Verbal fluency

Our analysis demonstrated that participants in the support group had significantly improved on verbal fluency between T2 and T3 compared to those allocated to the CBT group (t (14) = 2.27, p = 0.04, g = 1.20). This change significance supported by a very large effect size. Thus, between T2 and T3, the mean difference obtained for verbal fluency differed significantly between the support group (M = 1.78; SD = 3.44) and the CBT group (M = -2.25; SD = 3.24). However, no significant difference was found between T1 and T2 (t (15) = 0.77, p = 0.45, g = 0.39).

3.5. Anxiety

Regarding anxiety, our analysis indicated that the differences in means obtained by the two groups did not differ significantly between T1 and T2 (t (15) = 1.49, p = 1.16, g = 0.75) and between T2 and T3 (t (14) = -1.05, p = 0.31, g = 0.55). However, obtaining a large effect size in favor of CBT between T1 and T2 suggests that our, intervention had a greater effect on anxiety compared to the support group. Indeed, between T1 and T2, the anxiety of the participants in the CBT group decreased (M = -0.15; SD = 0.32) while that of the support group increased (M = 0.12; SD = 0.38). Our results, however, suggested that the trend reversed between Q2 and Q3; the anxiety of participants in the CBT group increased (M = 0.12; S-T = 0.32) while that of the support group decreased (M = -0.13; S-T = 0.52).

Table 2 Comparison Analysis Results

Cognitive and psychoaffective measures	Measurement time	T-test	Control	Experimental	Signification	Effect size
		t	*M (SD)*	*M (SD)*	*p*	*g of Hedges*
MoCA	Δ T2-T1	*+1,00* (6,08)	*-1,75* (*2,63*)	0,77	0,45	0,63
	Δ T3-T2	*-2,33* (*0,58*)	*-0,25* (*1,50*)	2,24	0,08	1,71
MEM_VER	Δ T2-T1	*-0,09* (*1,11*)	*+0,07* (*0,84*)	0,34	0,74	0,17
	Δ T3-T2	-0,19 (*0,91*)	*0,17* (*0,46*)	1,03	0,32	0,55

	Δ T2-T1	*-0,18* (0,55)	*+0,14* (*0,55*)	1,15	0,27	0,58
MEM_SEM						
	Δ T3-T2	*-0,11* (0,29)	*+0,11* (0,51)	1,06	0,31	0,50
	Δ T2-T1	*-4,00* (5,09)	*-2,11* (*4,67*)	0,77	0,45	0,39
FLUENCE						
	Δ T3-T2	*-2,25* (*3,24*)	*+1,78* (*3,44*)	2,27	0,04*	1,20
	Δ T2-T1	*-0,15 (0,32)*	*+0,12 (0,38)*	1,49	1,16	0,75
ANXIETY						
	Δ T3-T2	*+0,12 (0,32)*	*-0,13 (0,52)*	-1,05	0,31	0,55

* = significant at $p \leq 0.05$

MEM_VER: variable measuring verbal memory, MEM_SEM: variable measuring the semantic memory, Δ T2-T1: difference between after the intervention and before the intervention, Δ T3-T1: difference between six months after the project and immediately after the intervention, g of Hedges (Cohen, 1988): > 0.20 = small effect size; <0.50 = average effect size; <0.80 = large effect size.

people with a major neurocognitive disorder. Our results also suggest that the improvement in overall cognition observed in our CBT group occurred at the same time as a decrease in anxiety, which confirms our third hypothesis (H3), and suggests that anxiety can play a role in cognitive decline. These results corroborate the studies which argued that anxiety played an important role in cognitive decline and the rate of progression. Indeed, a 5-point improvement in MoCA, as we observed in one of our participants between the start and the end of CBT, could make the difference between a mild cognitive impairment and a major neurocognitive disorder.

However, the improvement we observed between onset and end of CBT not sustained over the long term, invalidating one of our hypotheses (H1.2). It is important to note that the observed long-term cognitive decline also accompanied by an increase in anxiety symptoms, reinforcing the idea that anxiety is associated cognitive functioning.

4. Discussion

Our results partially confirm our first research hypothesis which predicted that participants in the CBT group would have a lower overall cognitive decline than those distributed in the support group between T1 and T2 (H1.1 = confirmed) and between T2 and T3 (H2.2 = invalidated). In fact, participants who followed CBT had a smaller decline between T1 and T2 compared to those distributed support group (H1.1). Our results even suggest that overall cognition improved between the onset and end of CBT. More specifically, we observed an average increase of 1 point in MoCa (maximum of 5 points) between the start and the end of the intervention, which is notable in the context of neurodegenerative disease where cognitive decline is generally expected. These results are in the same direction as those found by Burgener's team (2008) who observed an improvement of 0.4 in MMSE following a non-pharmacological intervention followed by

heterogeneity between the participants was noted, in particular with regard to the cognitive profile of the symptoms (memory vs executive) and the severity of the disease (CDR). This heterogeneity has possibly had impacts on several levels: on the group dynamics during the intervention, on the ability to understand the instructions, integrate the concepts and perform the exercises well at home. Therefore, it is possible that CBT had different inter-subject effects, which our statistical analyzes could not capture. Thirdly, the absence of an inactive control group (e.g. waiting list) did not allow us to control for the potentially beneficial effects of meeting with people living the same situation, of feeling taken care of by professionals, breaking the isolation, etc. It is therefore possible that the absence of a significant difference that we observed in several respects between the groups explained by this effect of support felt by the participants regardless of their group to which they belong. Then, although the content of the intervention was adapted for the AD population, it was of short duration (8 weeks). We believe that a longer-term intervention could be beneficial in order to better consolidate learning and better integrate concepts into daily life, especially in the presence of memory difficulties. Finally, studies have suggested that anxiety and AD are frequently associated with depression (Savulich et al., 2019; Steinberg et al., 2008; Zhao et al., 2016) and that several other factors may influence cognition (social network, feeling of loneliness, basic cognitive level) (Pitkala et al., 2011; McHugh et al., 2019).

Our second research hypothesis (H2) which predicted that participants in the CBT group would have less cognitive decline in several cognitive functions compared to the control group is however invalidated.

We even found, contrary to what was expected, that the support group had a higher impact on long-term verbal fluency, that is, between the end of the group and the 6-month follow-up, than the CBT group. This surprising result could be explained by the functioning of groups. Indeed, the participants of the support group, unlike those of the CBT, could freely discuss among themselves non-imposed topics, thus encouraging the generation of ideas and words spontaneously. This interpretation supported by a study, which has shown that it is possible to improve the verbal fluency of elderly people (without dementia) by having conversations with an interlocutor, even for short periods (about ten minutes). (Mochizuki-Kawai et al., 2008). Thus, it is possible that CBT, more structured in content, limited participants to spontaneously generating discussion topics, which would have had less effect on their fluency than a non-directive support group. Furthermore, just like what was observed for overall cognition, our results suggest that verbal fluency performance fluctuates according to the level of anxiety; when anxiety decreases, verbal fluency improves and vice versa.

These results interpreted in light of the limitations of the study. As a first step, as mentioned previously, the small size of our sample reduced the statistical power of our analyses. Interpreting effect sizes, however, allowed us to work around this limitation. Secondly, although the inclusion criteria were limited to diagnoses of Alzheimer's type dementia in a mild to moderate stage, a

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