

1-1-2016

Fear Of Alzheimer's Disease And Its Role In Memory Monitoring And Control

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**FEAR OF ALZHEIMER'S DISEASE AND ITS ROLE IN
MEMORY MONITORING AND CONTROL**

by

ANNALISE RAHMAN

DISSERTATION

Submitted to the Graduate School

of Wayne State University,

Detroit, Michigan

in partial fulfillment of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

2016

MAJOR: PSYCHOLOGY (Clinical)

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ACKNOWLEDGEMENTS

Dr. John L. Woodard, Ph.D.

Dr. Peter Lichtenberg, Ph.D., ABPP

Dr. Jeske Damoiseaux, Ph.D.

Dr. Stephen J. Vangel, Ph.D.

Dr. Cathy Lysack, Ph.D.

Funding for this project was made available through the Wayne State University Dissertation Funding Award.

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CHAPTER 1: SPECIFIC AIMS

Although some age-related decline in memory functioning is normal, many middle-aged or older individuals interpret everyday memory lapses as indicators of the development of dementia, or Alzheimer's disease (AD) in particular. While a fleeting thought about the development of AD may not be harmful, repeated misperception of attention and memory failures can be much more detrimental. This phenomenon, known as fear of Alzheimer's disease (FAD) or anticipatory dementia, may lead to hyper-vigilance for AD symptoms, decreased self-efficacy, depression and anxiety. These symptoms reciprocally worsen attention and memory performance, potentially creating a self-fulfilling prophecy of memory decline.

Despite the negative impact of FAD in the middle-aged and older adult populations, few studies have been conducted to assess the construct. Little is known about the conceptual overlap between FAD and other important metacognitive processes, such as prospective appraisal of memory abilities and subjective memory complaints (SMCs). Furthermore, researchers have not yet established the relationship between FAD and objective memory ability. Although FAD has been tied to greater trait-level anxiety (French, Floyd, Wilkins & Osato, 2012), little is known about the practical implications that FAD has on compensatory cognitive and health behaviors.

A primary goal of this study was to establish the construct validity of FAD through assessing its conceptual overlap with Judgment of Learning (JOL) and SMCs. Although one scale currently exists to evaluate general fear, physical symptoms, and catastrophic attitude associated with FAD (French, Floyd, Wilkins & Osato, 2011), this scale has shortcomings. There are several potentially important aspects of the construct, including knowledge about AD development and prevalence, and locus of control/self-efficacy for preventing or treating

dementia, which are not included in the FAD construct. The present study has also developed and validated a new scale of FAD that assesses different dimensions of the construct more effectively than existing measures.

A second aim of the study was to investigate the relative ability of different types of metamemory tests to explain variance in objective memory functioning. The study assessed how SMCs, FAD, and metacognitive monitoring during a Judgment of Learning task account for variance on an objective memory test.

A third aim of the study was to investigate the relationships between FAD and metacognitive monitoring and control. The study examined how individuals with high and low FAD estimate their memory ability for a list-learning task. This judgment of learning task informs whether middle-aged and older adults with varying levels of FAD update their memory appraisals based on self-monitoring of performance on the task. Finally, the study investigated the extent to which individuals with different levels of FAD utilize metacognitive control, or behavioral strategies to compensate for their perceived memory problems.

Specific Aim 1

The study evaluated the construct validity of fear of Alzheimer's disease (FAD) using measures of general negative affect, subjective memory complaints (SMCs), judgment of learning (JOL), and a novel measure of FAD. It was hypothesized that (a) individuals with a family history of Alzheimer's disease would have greater FAD. Also, it was proposed that FAD is positively associated with (b) SMCs and (c) general negative affect. FAD was also expected to be (d) negatively associated with JOL appraisals and accuracy of those appraisals. It was also hypothesized that (e) negative affect, SMCs and JOL would account for a relatively small proportion of variance in FAD, as FAD is a unique construct. Last, it was hypothesized that (f)

scores on a newly developed scale of FAD are related to scores on existing measures of FAD, but do not overlap completely. It was proposed that the novel measure assesses new aspects of FAD not covered by these existing tests, including knowledge about normal versus abnormal forgetting in older adulthood, prevalence and causes of and treatments available for AD, health-specific and AD-specific locus of control, beliefs about personal susceptibility to AD, negative affect related to AD thoughts, and specific fears and living with AD.

Specific Aim 2

The study determined the relative ability of FAD, SMCs, JOL appraisal (metacognitive monitoring) and engagement in compensatory behaviors (metacognitive control) to account for variance in objective memory functioning in cognitively intact older adults. It was hypothesized that (a) SMCs, FAD and difference in study behavior (study time on self-paced list-learning trials as compared to matched computer-paced trials) are not significantly associated with objective memory functioning. However, (b) JOL estimates were expected to account for a significant proportion of variance in objective memory functioning. It was also hypothesized that (c) of all the FAD and metacognitive variables, metacognitive monitoring accounts for the most variance in objective memory functioning after controlling for age and education.

Specific Aim 3

The study determined whether FAD is associated with changes in metacognitive monitoring accuracy and implementation of control strategies in cognitively intact older adults. It was hypothesized that (a) metacognitive monitoring, in the form of JOL estimates, is significantly associated with metacognitive control, measured by study time on subsequent trials. It was also hypothesized that (b) individuals low in FAD produce smaller difference scores between the predicted and actual number of items remembered across all learning trials. They

were also expected to study a list of 20 words for a shorter amount of time than individuals high in FAD, and to vary study time based on perceived performance (i.e. utilize longer study time when objective memory performance was below expectation). Conversely, (c) individuals high in FAD were expected to underestimate their memory ability for all trials, creating larger difference scores between the predicted and actual number of items recalled. It was hypothesized that they would not increase their study time based on perceived task performance. Instead, they were expected to consistently utilize the maximum study time allotted, regardless of trial, perceived ability level or performance.

CHAPTER 2: GENERAL BACKGROUND AND SIGNIFICANCE

It is estimated that approximately 5.4 million Americans were diagnosed with Alzheimer's disease (AD) in 2012, over 95% of whom were over the age of 65 (Hebert, Scherr, Bienias, Bennett, & Evans, 2003). Furthermore, one in nine people over the age of 65 and one-third of people over the age of 85 will be diagnosed with the disease (Alzheimer's Association, 2014). Dementia, and AD in particular, is a problem of growing concern not only for the aging baby-boomer generation, but also for their caretakers, who make up over 15 million Americans (Alzheimer's Association, 2012). The costs of short- and long-term healthcare and hospice for individuals with AD are significant, totaling an estimated 150 billion dollars in the United States alone (Alzheimer's Association, 2014).

Public perception of AD has changed with increased scientific knowledge about the disorder. While the majority of people in the United States are aware of AD as a source of memory problems, very few individuals have specific knowledge about AD symptoms, preventative lifestyle changes, and treatments (Anderson, Day, Beard, Reed, & Wu, 2009). A lack of specific knowledge about AD or its etiology may contribute to widespread concern about development of the disorder. In the population of Americans 18 years of age and older, approximately 60% cite major concern about age-related memory loss (Anderson, Day, Beard, Reed & Wu, 2009).

For younger adults, fear of developing AD is poignant but abstract. For middle-aged and older adults, everyday 'normal' lapses in memory may provide concrete 'evidence' for greater worry. For example, while a failure to remember an appointment, the location of one's keys, or the name of a new acquaintance might not be interpreted in the context of AD risk in younger adults, middle-aged and older adults might perceive these instances as harbingers of a more

significant and frightening memory disorder. This fear of Alzheimer's disease (FAD), also known as anticipatory dementia, has significant impacts on health, regardless of whether the fear is warranted. FAD may contribute to the 6-10% prevalence rate of anxiety disorders among older adults (Schuurmanns & van Balkom, 2011). Anxiety disorders, in turn, have significant negative impacts on health, including seeking of more somatic health care visits but decreased use of mental health care services, poorer cardiac health, and increased mortality in men or frail older adults (Schuurmanns & van Balkom, 2011). Despite evidence to the contrary (Schuurmanns & van Balkom, 2011), there is a popular belief that anxiety and other affective problems may be an early indicator of dementia. In the case of FAD, this anxiety is circular; having greater fear of developing dementia causes anxiety, which is seen as confirmation of the impending cognitive decline.

Memory functioning is critical for the performance of day-to-day tasks and independent living, but awareness of one's memory and other cognitive abilities is equally important. Metacognition refers to the thoughts, beliefs, and mental processes used to assess and control cognition. In regards to memory specifically, metacognition refers to the *monitoring* of one's memory successes and failures, as well as the difficulty of encoding and retrieving information (Veenman, Van Hout-Wolters, & Afflerbach, 2006; Hertzog & Dunlosky, 2011). Meta-memory also refers to *control*, the motivation and direction of one's behaviors to compensate for these failures in order to increase successes (Veenman, Van Hout-Wolters, & Afflerbach, 2006; Hertzog & Dunlosky, 2011). Older adults fearful of developing dementia may monitor their memory failures more closely and may be more sensitive to retrieval struggles than older adults who feel less vulnerable to AD. Older adults high in FAD may also control their behavior in particular ways to accommodate their perceived memory impairments, such as utilizing

calendars and written mnemonic aids, relying on family members or caregivers for reminders, or seeking of medical treatment.

Significance of the Current Study

In the context of existing research that has examined these behaviors, it appears that a better understanding of the construct of FAD is needed. Furthermore, whether or to what extent it might relate to and impact objective memory functioning and meta-memory has received relatively little attention in the research literature. This study will develop and validate a novel measure of FAD, and it will use this measure to determine the extent to which FAD interferes with memory monitoring and compensation for poor perceived memory. This research also provides important benefits for clinical work. A novel measure will greatly improve assessment of dementia-specific worry, allowing for identification of older adults at greater risk for anxiety disorders and associated problems. Better understanding of the relationship between FAD, SMCs, metacognitive monitoring and actual memory performance may provide the basis for a predictive model of objective memory functioning. Furthermore, the study will allow researchers to identify individuals who might benefit from psychoeducation about normal memory aging and AD, balancing of expectations about cognitive decline, and training in compensatory memory strategies. This study may also identify individuals more likely to rely on memory aids, cognitively challenging activities and games to improve their own performance, versus individuals who may rely on caregivers to monitor and accommodate memory decline.

The following sections include a more detailed review of the current research on FAD, SMCs, and metacognitive monitoring and control of memory abilities. The function of this review is to provide a context for the current study and identify gaps in the literature that may be addressed by the current study.

Fear of Alzheimer's Disease

FAD, also known as anticipatory dementia, was originally conceptualized as the tendency of adult children of AD patients to be worried that their age-associated memory changes are a signal of impending cognitive decline due to AD (Cutler & Hodgson, 1996). This phenomenon is not restricted to children of AD patients but is a widespread concern for middle-aged and older adults (Cutler & Hodgson, 1996). Approximately 60% of American and European individuals (Anderson, Day, Beard, Reed & Wu, 2009; Cantegreil-Kallen & Pin, 2012) and 46% of Israeli older adults (Werner, 2002) cite memory impairment as a major source of concern. In France, perception of the seriousness of AD has increased in recent years (Leon et al., 2015). In parallel, belief in the efficacy of treatments and the normalcy of memory loss with age has decreased (Leon et al., 2015).

In attempting to explain why healthy people might perceive their memory problems as disordered, researchers have found that greater knowledge of the disorder not only fails to inoculate people against worry, but it may actually increase that worry (Cutler & Hodgson, 2001; Roberts and Connell, 2000). Despite having better knowledge of dementia symptoms, causes, and risk factors, many people overestimate the heritability of AD (Roberts & Connell, 2000), leading to greater fear in adult children of AD patients (Cutler & Hodgson, 1996; 2001; Roberts & Connell, 2000). Furthermore, number of family members with the disease is positively correlated with FAD (Cutler & Hodgson, 2001). Even without a genetic vulnerability to the disease, serving as a caregiver to someone with AD appears to increase one's personal concerns about memory impairments (Cantegreil-Kallen & Pin, 2012; Leon et al., 2015).

Perceived memory abilities also increase one's level of FAD, with greater frequency of SMCs (Cutler & Hodgson, 1996; 2001) or perception of a recent change in memory functioning

(Cutler & Hodgson, 2001) associated with more fear. Among demographic predictors of FAD, being married (Cutler & Hodgson, 2001), older (Cutler & Hodgson, 2001; Cantegreil-Kallen & Pin, 2012), female (Cantegreil-Kallen & Pin, 2012), having a higher educational or occupational level, or being unmarried or female *with a living relative diagnosed with AD* are associated with greater fear (Cutler & Hodgson, 1996; 2001). FAD has also been linked to overall health beliefs, with poorer perceived overall health being associated with greater fear of developing Alzheimer's disease (Cantegreil-Kallen & Pin, 2012).

FAD represents a barrier to early detection of Alzheimer's disease or other dementias (Corner & Bond, 2004). Individuals who are fearful of their memory problems may not be able to distinguish their everyday memory failures from true indications of decline. People high in personal concern about AD are more likely to seek medical attention for their memory complaints (Ramakers et al., 2009), which allows for better screening but greater cost for these individuals. Furthermore, these individuals are likely to use compensatory mnemonic strategies (ex. calendars, lists, schedules, memory training games) less often or less effectively than individuals with more accurate appraisal of their memory. If memory decline is seen as inevitable and uncontrollable, motivation for early identification, treatment, and preparation may be lessened. Ultimately, anxiety about loss of one's identity, dignity, or independence might cause individuals who are fearful about the etiology of their memory lapses to be less willing to ask for help (Corner & Bond, 2004).

Until recently, FAD was usually measured using a single question, such as "I would like to ask how concerned you are about personally developing Alzheimer's disease. Would you say that you are *very* concerned, *somewhat* concerned, *not very* concerned, or *not at all* concerned?" (Cutler & Hodgson, 1996; Cutler & Hodgson, 2001, p. 338), or very few questions (Roberts &

Connell, 2000). In 2012, French, Floyd, Wilkins, & Osato developed the Fear of Alzheimer's Disease Scale (FADS) as a response to the limited assessment of FAD symptoms in research and clinical practice. The FADS contains three scales – General Fear, Physical Symptoms, and Catastrophic Attitude, and it is highly correlated with trait anxiety as measured by the STAI-T (French, Floyd, Wilkins & Osato, 2012). A Korean version of the scale has also been validated, with good psychometric properties for the assessment of anticipatory dementia (Moon, Kim, Hoi, Oh & Chan, 2014). This scale should be recognized as the first attempt to measure FAD comprehensively, using a psychometrically sound instrument; however, the FADS does not assess some important constructs associated with FAD. Alongside physical, emotional and cognitive reactions to thinking about AD, a more comprehensive measure of FAD would also assess knowledge of normal vs. AD-impaired memory functioning, knowledge about causes (including heritability) and treatments of AD, attitudes towards treatments for AD, beliefs about personal susceptibility to AD or quality of life with AD, and self-efficacy in handling an AD diagnosis.

In this study, FAD is conceptualized as developing from a combination of poor metacognitive appraisal of one's abilities, plus limited self-efficacy to change, control, or handle an AD diagnosis. As such, a comprehensive measure of AD must not only ask about one's perceived susceptibility to an AD diagnosis but also one's perceived ability to handle the diagnosis, if one were to be diagnosed. The Health Belief Model (HBM; Rosenstock et al., 1988) may provide a social learning theory model through which FAD may be better understood. The health belief model is used to understand and predict engagement in health and lifestyle behaviors and to assess attitudes about chronic illness. In its most basic form, the HBM asserts that *perceived threat* of a disorder or injury is combined with one's *outcome expectations* for

trying to address or change risk for the disorder to yield *self-efficacy*, or one's perceived ability to cope with or change the disorder or injury. In the case of FAD, the perceived threat of AD would arise from beliefs about one's personal susceptibility to the disorder and beliefs about the seriousness of the disorder. Outcome expectations for AD would arise from beliefs about availability and effectiveness of treatment options or preventative lifestyle changes to slow or stop the development of AD. High perceived susceptibility, strong negative beliefs about quality of life with AD, and few perceived ways to minimize AD course or severity would lead to lower self-efficacy and greater FAD.

Although it likely represents a distinct construct in its own right, FAD likely shares a conceptual overlap with tendency to notice and report everyday memory failures, or SMCs.

Subjective Memory Complaints

Subjective memory complaints (SMCs) are everyday complaints about perceived memory failures. The prevalence of memory complaints in healthy, non-demented older adults is at least 10% (Jungwirth et al., 2004). There is some evidence to suggest that SMCs may represent subtle functional changes in the brain before objective memory problems develop. Indeed, SMCs are among the diagnostic criteria for mild cognitive impairment, according to the most recent International Classification of Diseases (ICD-10, 2014). It is unclear if biological risk factors for AD are truly linked to greater subjective memory problems. Some studies conclude that the apolipoprotein E epsilon-4 allele, a major genetic susceptibility marker for AD, is associated with increased SMCs (Small et al., 1999), and others negate the association between AD biomarkers and SMCs (Buckley et al., 2013). Studies also question whether SMCs truly reflect objective memory deficits. There is evidence that older adults with poorer general intellectual ability or specific memory ability (Small et al., 1999; Balash et al., 2013) or older

men with poorer episodic memory functioning (Volz-Sidiropoulou & Gauggel, 2012) report more SMCs. Conversely, correlational and longitudinal studies of healthy older adults suggest that objective memory functioning does not predict frequency or severity of SMCs (Jungwirth et al., 2004; Ponds, Van Boxtel, & Jolles, 2000; Lenehan, Klekociuk, & Summers, 2012). England Amariglio et al. (2011) have presented evidence that different types of memory complaints may be differentially predictive of objective memory abilities, with certain SMCs (ex. difficulty following a group conversation, trouble finding one's way around familiar streets) accounting for a greater proportion of variance in objective memory abilities. Researchers have also questioned the ability of SMCs to predict conversion to Alzheimer's disease. A prospective study conducted with individuals above the age of 75 suggests that SMCs predict AD conversion only in individuals high in overall IQ, and that objective memory ability is a stronger, more universal predictor of AD conversion in individuals of any intellectual level (Jungwirth et al., 2008).

There is a large body of evidence suggesting that SMCs may be more influenced by affect or personality than actual memory functioning. Healthy older adults endorsing higher levels of anxiety and depression report more frequent or severe memory complaints (Balash et al., 2013; Buckley et al., 2013, Jungwirth et al., 2004, Ponds, Van Boxtel, & Jolles, 2000), and SMC severity is only associated with age (and not memory ability) in the mildly cognitively impaired (Buckley et al., 2013). Personality traits like neuroticism and negative affect may also account for variance in SMCs (Dux et al., 2008; Pearman, 2009; Merema, Speelman, Foster & Kaczmarek. 2013). More specifically, anxiety sensitivity may moderate the relationship between subjective and objective memory functioning such that individuals high in anxiety sensitivity will report more memory complaints, even in the absence of memory dysfunction (Dux et al., 2008). Independent of the influence of mood disorder, higher perceived stress also increases

memory complaints (Potter, Hartman, & Ward, 2009). This finding provides an explanation for the relationship between FAD and SMCs; greater FAD and associated worry about future healthcare and financial needs, relationships and losses may drive higher stress levels, which increases attention to memory functioning and, therefore, memory complaints.

While SMCs are unstructured private assessments of one's own personal memory functioning, it is unclear if they relate to structured assessments of memory ability or actual memory performance. Metacognitive tasks that require the participant to prospectively estimate his or her memory performance on a structured task may have greater objectivity and clinical utility.

Metamemory

Metacognition is defined as “thoughts, beliefs, and other cognitive processes devoted to assessing and controlling one's own cognitions” (Hertzog & Dunlosky, 2011, p. 61). Metamemory, an instance of metacognition, includes the cognitive processes related to appraisal and direction of one's memory. Although SMCs rely somewhat on metacognitive processes, the two constructs are not the same. While SMCs are subjective evaluations of everyday memory functioning, such as the failure to remember where one left one's keys, metamemory is assessed using formal and objective prospective and retrospective tasks. Two such paradigms, the *Judgment of Learning (JOL)* and *Feeling of Knowing (FOK)* tasks, ask the respondent to prospectively evaluate their performance on a memory task during the encoding or retrieval stage, respectively. In a JOL test, the respondent is presented with a stimulus – often word pairs or a word list – and must appraise the likelihood that the paired words or a certain number of items will be recalled at a later time (Souchay et al., 2013). In a FOK task, the respondent is shown a stimulus (again, either word pairs or a word list), is given an opportunity to recall the

associated pair or items, and he or she is then asked to appraise the likelihood of recognizing the missed targets at a later time (Souhay et al, 2013). Through these tasks, researchers are able to estimate the accuracy or ‘resolution’ of metacognitive appraisals as well as the confidence with which a respondent holds their appraisal.

Metacognition has also been conceptualized as involving a dual process of monitoring and control (Nelson and Narens, 1990 in Souhay et al., 2013). *Metamemory monitoring* describes the subjective evaluation of memory functioning in different situations. *Metamemory control* describes the behaviors that are enacted to maximize memory functioning (Souhay et al., 2013). In 1988, Nelson and Leonesio proposed a model of the relationship between these two concepts. Known as the ‘monitoring affects control hypothesis,’ this feedback model suggests that ongoing appraisal of one’s memory functioning in relation to the task demands of a given situation drives one to control one’s memory by guiding thoughts and behaviors and implementing compensatory or mnemonic strategies (in Souhay et al., 2013). Control strategies may range from small-scale changes in how a task is approached to significant lifestyle changes that might preserve memory functioning. There is some evidence to suggest that the perceived effectiveness of different control strategies changes with age, with younger adults rating task-specific control strategies (ex. using mnemonic devices) and older adults rating lifestyle changes (“use it or lose it” mental exercising) as most effective in improving memory (Hertzog, McGuire, Horhota, & Jopp, 2010).

Metacognitive Monitoring. While some research suggests that healthy older adults make accurate estimations of their memory performance, there is considerable individual variability in monitoring (Clare, Whitaker, & Nelis, 2010). Despite age-related changes in episodic memory and poorer source memory for encoded information, cognitively-intact older

adults appear to have similar FOK (Eakin & Hertzog, 2012) and JOL (Hertzog & Dunlosky, 2011) resolution to those of younger adults. Conversely, AD patients' monitoring of their memory is often inaccurate, with most individuals overestimating their abilities and some individuals underestimating their abilities (Clare, Whitaker, & Nelis, 2010).

Beyond Alzheimer's dementia and age, a range of factors may impact metacognitive resolution. Metacognitive monitoring accuracy is positively correlated with education in both healthy older adults and in older adults with diagnosed AD (Szajer & Murphy, 2013). Metacognitive monitoring appraisals are also shaped by social norms and cultural expectations. For instance, older adults from Sardinia, a cultural region known for longevity, are more likely to expect memory stability or even improvement than adults from a cultural region not known for longevity (e.g. Milanese; Bottiroli, Cavallini, Fastame, & Hertzog, 2013). The older adults from longer-living cultures may provide higher appraisals of their abilities based on their societal expectations. Furthermore, older adults from longer-living societies attribute memory stability or improvement throughout old age to a wider range of causes, such as fate, heredity, memory ability, memory training, nutrition, others' opinions, and personal task importance (Bottiroli, Cavallini, Fastame, & Hertzog, 2013).

Metacognitive Control. Even if monitoring resolution is high and one can identify when an item is or is not correctly encoded, control is essential for adapting behavior and cognition based on this knowledge. Without redirection of attention and implementation of strategies for poorly learned items, one cannot benefit from improved memory performance. It appears that, unlike metacognitive monitoring, metacognitive control may be impaired in healthy older adults. Although older adults can accurately estimate that items presented less frequently at encoding will be harder to recall, they cannot use this information to change the strategies by which they

learn the items or to identify the source of the items (Kuhlmann & Touron, 2011). Furthermore, while younger and older adults believe (incorrectly) that remembering read paired-associates would be easier than remembering generated pairs, younger adults allocate study time based on these ratings, while older adults do not (Froger et al., 2011).

Despite poor control abilities, researchers and clinicians can take advantage of spared monitoring and belief in the efficacy of memory training to instruct older adults in better compensatory strategies for declining memory. For instance, older adults can be taught to test their memory appraisals against actual performance immediately after learning, and to allocate further study time to items failed during self-test (Bailey, Dunlosky & Hertzog, 2010). Training on self-testing and study allocation is more effective than traditional strategies encouraged during learning, such as building of semantic or imagery mnemonics (Dunlosky, Kubat-Silman, & Hertzog, 2003; Bailey, Dunlosky & Hertzog, 2010). These training strategies may be critical for improving older adults metamemory, objective memory functioning and, ultimately, their ability to continue to live independently.

Study Summary

Despite the increased prevalence of AD and memory concerns more broadly, the relationship between metacognitive evaluations of memory functioning and objective memory performance has not been widely studied. It is also unclear how FAD might impact the relationship between perceived and actual memory performance. Furthermore, FAD has undergone limited validation as a construct related to but distinct from everyday SMCs or metacognitive monitoring and control. Last, research has not elucidated the impact of FAD on the monitoring-control process that appears to be important for adaptation to cognitive changes in late life. More specifically, it is not known whether having high FAD might interfere with

accurate monitoring of learning and retrieval or may interrupt one's ability and motivation to put into place effective compensatory control strategies. The current study aimed to address the above gaps in the literature by developing a novel measure of FAD that would tap into a more comprehensive set of domains than previous measures. This measure has the potential to be of considerable clinical utility. In addition, such a short questionnaire might be integrated into a larger psychological or cognitive battery or given by a primary care provider to assist with identifying older adults at risk for anxiety disorders. This study also aimed to understand the relationships between FAD, other metacognitive measures, and objective memory functioning. By understanding the relative amount of variance in objective memory abilities in healthy adults accounted for by SMCs, metacognitive appraisal of learning on a specific task, and FAD, researchers and clinicians may be able to characterize the nature of normal age-related memory changes more effectively. The current study also investigated the extent to which FAD impacts memory monitoring and the execution of compensatory control behaviors when memory is perceived as failing. This goal is critical from an intervention standpoint. Understanding these relationships will allow researchers to identify individuals who are more likely to use compensatory study or memory aids, to seek treatment for their memory complaints, and to successfully identify when their memory failures put them in dangerous situations. Conversely, the research will also allow for identification of older adults who may under- or over-estimate their memory abilities and may fail to implement strategies or seek treatments appropriately. This latter group of individuals may also prove to be more vulnerable to anxiety, depression, or a failure to pursue an engaged lifestyle. The study findings may allow researchers to identify those individuals who would be most at risk for these disorders, who are most likely to benefit from psycho-education about normal memory aging and training on metacognitive control strategies.

CHAPTER 3: METHOD

The following method was reviewed and approved by the Institutional Review Board of Wayne State University and the Institute of Gerontology Healthier Black Elders Review Board.

Participant Recruitment

A power analysis was used to calculate the number of participants required to achieve adequate power for the analyses used in the current study. For bivariate correlations, assuming a power level of .80, one-tailed $\alpha = .0042$ (Bonferroni-adjusted for multiple comparisons), and a predicted small to moderate r effect size of 0.35, the required sample size per group would be $n = 93$. For multiple regression, assuming a power level of .80, $\alpha = .00625$ (Bonferroni-adjusted for multiple comparisons), a predicted small to moderate R^2 effect size of 0.25 and a maximum of ten predictors, the required sample size per group would be $n = 85$. The study therefore recruited 94 participants to ensure adequate power to detect group differences.

Participants were recruited from multiple sources. A large proportion of participants were recruited through the participant research pool of the Healthier Black Elders (HBE) organization at the Wayne State University Institute of Gerontology (WSU IOG). The HBE is a group of professionals and volunteers from the Detroit area dedicated to advancing the health of older African-Americans through research, education, and healthcare initiatives. Part of the HBE's mission involves encouraging African-American older adults to participate in research. As such, the HBE maintains a list of Detroit-area residents interested in participating in approved research studies. The current study utilized a subset of this list based on inclusion/exclusion criteria to identify potential participants. The study was also advertised through short presentations at local community talks geared towards older adult health, flyers distributed in local public libraries, communities of faith, exercise groups, and senior centers, and through

word of mouth.

Inclusion and Exclusion Criteria

In an attempt to maximize the range of scores on the FADS and other FAD measures, researchers recruited an approximately equal number of individuals with and without a family history of Alzheimer's disease or other dementia. Family history positive participants were defined as having at least one blood relative with dementia diagnosed by a medical professional; cases who listed family members they *believed* to have dementia were not counted in the family history positive group.

Although participants were permitted to report SMCs, individuals who had been previously diagnosed with Mild Cognitive Impairment, dementia, or another memory disorder were excluded from the study. Given the focus of the study on metacognition, self-monitoring, and personal concern about susceptibility to AD, individuals who had previously ruled out dementia or AD through a clinical evaluation were also excluded from the study. All participants were living independently in the community.

Additional exclusion criteria included a history of significant neurological problems (i.e., stroke, head injury with loss of consciousness as an adult, seizures, or other neurological condition), current psychiatric disorder for which the individual is taking medication, or uncorrected visual or hearing impairments.

Sample Characteristics

Participants included 93 community-living individuals. Sample characteristics are summarized in Table 1. The majority of participants were female (75.30%) and the average age of the participants was 70.18 years. Most participants were retired, though 18.30% are currently working part-time and 9.70% are currently working full-time. Approximately 44% of the sample

was Caucasian and 49% was African-American. The average education of the participants was approximately 15 years. 53.80% of the participants denied a family history of dementia. 46.20% of the participants acknowledged a history of dementia or Alzheimer's disease in at least one blood relative. 24.70% also endorsed a history of caregiving, either professionally or personally, for someone with Alzheimer's disease or dementia.

The sample was also stratified by family history of dementia. Groups were compared via independent samples t-tests or chi-squared tests of significance to determine whether they differed in terms of demographic characteristics. Groups were not significantly different in terms of age ($t_{(90)} = 1.18, p = .241$, Cohen's $d = 0.244$) or education ($t_{(90)} = 0.024, p = .982$, Cohen's $d = -0.004$). Groups were also not significantly different in terms of gender ($\chi^2_{(1)} = 0.40, p = .627$, Cramer's $V = 0.065$), race ($\chi^2_{(3)} = 1.84, p = .796$), or employment status (Fisher's Exact $_{(2)} = 3.05, p = .211$, Cramer's $V = 0.186$).

Measures

Baseline Cognitive Functioning. In order to assess whether groups were equivalent in baseline intellectual functioning, all participants completed the Salthouse Synonym and Antonym Vocabulary tests and the Telephone Interview for Cognitive Status.

The Salthouse Synonym and Antonym Vocabulary Test (Salthouse, 1993). The Salthouse Synonym Vocabulary Test includes ten uncommon words. The respondent must choose the best synonym for each word from five provided options. The Salthouse Antonym Vocabulary Test (Salthouse, 1993) includes ten different words, to which respondents must match the best antonym from five options. The score for each test is the number of items answered correctly. The range in scores on the combination of the tests is 0 to 20, with higher scores denoting greater verbal abilities. The Synonym and Antonym vocabulary test has been

used as a proxy measure of general cognitive functioning, as verbal abilities are strongly correlated with overall IQ (Bowles & Salthouse, 2008).

Telephone Interview for Cognitive Status (TICS; Brandt & Folstein) – the TICS is a standardized measure of general cognitive ability designed to be given in situations in which face-to-face assessment would be impossible or inefficient. The measure includes eleven items that are summed to yield a total ability score. Test developers have suggested that the TICS correlates strongly with the MMSE and its scores have demonstrated high test-retest reliability for the detection of cognitive impairment in older adults age 60 to 98 years. Further studies of the psychometric properties of the TICS and the TICS-Modified have suggested that TICS scores validly assess cognitive functioning in Alzheimer’s disease and other dementias (Duff, Dennett, & Tometich, 2012) as well as amnesic mild cognitive impairment (Cook, Marsiske, & McCoy, 2009). Furthermore, TICS scores correlate highly with scores from several indices from a comprehensive neuropsychological assessment, including verbal memory (Rey Auditory Verbal Learning Test), orientation and mental tracking, fluency (category and animal naming), abstract reasoning (Raven Advanced Progressive Matrices), and attention and executive functioning (Stroop Color-Word Test, Trail-Making Test), and working memory (WAIS-III Digit Span subtest; van den Berg, Ruis, Biessels, Kappelle, & van Zandvoort, 2012).

Negative Affect. Negative affect, including general anxiety and depression, was assessed using multiple measures.

Geriatric Depression Scale - Short Form (GDS-SF; Sheikh & Yesavage, 1986). The short form of the GDS contains 15 phrases relating to symptoms of depression that older adults might have. The respondent is required to respond either “yes” or “no” based on whether the phrase describes how they have been feeling during the week prior to the assessment. Scores on

the GDS-SF range from 0 to 15, with a score equal to or greater than five suggesting the presence of depression. The short form of the GDS has been shown to have roughly equivalent specificity and sensitivity in the diagnosis of depression in older adults as compared to the long form of the GDS (Leshner & Berryhill, 1994). Older adults' scores on the long and short version of the GDS are also strongly correlated ($r = .66, p < .01$; Alden, Austin & Sturgeon, 1989).

State-Trait Anxiety Inventory for Adults (STAI-Y; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983). The STAI-Y is a measure of self-reported anxiety symptoms. The STAI-Y is divided into two subscales: State and Trait. The STAI-State subscale measures anxious feelings at the time of the assessment. The STAI-Trait subscale measures chronic anxious symptoms present across situations. The measure is often used to distinguish anxious symptoms from depressed symptoms or to differentiate situational anxiety from trait level anxiety.

The STAI-Y consists of two sets of 20 statements, with one set asking the respondent about current symptoms (STAI-Y1; ex. "I feel calm.") and a second set asking the respondent about general symptoms (STAI-Y2; ex. "I am "calm, cool, and collected"."). The respondent scores each item on a four-point Likert-style scale, with 1 = "Almost Always" and 4 = "Almost Never." The scores possible on each subscale range from 20 to 80, with higher scores denoting greater anxiety.

Scores from the STAI-Y have demonstrated reliability and validity for the assessment of trait and state anxiety in older adults. The test-retest reliability for STAI-Y scores may range from 0.31 to 0.86 using intervals of one to 104 days (Julian, 2011). As one might expect, the test-retest reliability for the state anxiety subscale of the STAI is much lower than for the trait anxiety subscale. Although little is known about the internal consistency of STAI scores when

used with older adults, previous research has shown that the STAI alpha coefficients may range from 0.86 for high school students to 0.95 for military recruits (Julian, 2011). STAI-Y scores have also been shown to share strong correlations with other measures of anxiety, indicating that it has construct validity (Julian, 2011). Recently, STAI-Y scores have demonstrated strong reliability and validity for the assessment of state (Potvin et al., 2011) and trait (Bergua et al., 2012) anxiety in adults over the age of 65 years.

Fear of Alzheimer's Disease. To assess each participant's level of anticipatory dementia and to validate the construct itself, several measures were used, including a novel measure aimed to tap into the broader construct of FAD.

Fear of Alzheimer's Disease Scale (FADS; French, Floyd, Wilkins & Osato, 2011). The FADS is a self-report measure that taps into three facets of FAD: general fear, physiological symptoms accompanying FAD, and catastrophic attitudes associated with FAD. Scores from the scale have been validated for the assessment of these facets of anticipatory dementia in adults age 65 to 91 (French et al., 2011). It includes 30 statements to which respondents indicate their level of agreement on a five-point Likert-style scale ranging from "never" to "always." Higher scores on the scale denote greater fear of developing Alzheimer's disease. Scores from the General Fear, Physical Symptoms, and Catastrophic Attitudes factors had Cronbach's alpha coefficients of .94, .87, and .80, respectively, in adults over age 65 years. The overall internal consistency coefficient of scores from the measure was .91, indicating acceptable reliability for research and clinical use in this context (French, 2011). FADS scores also demonstrated excellent construct validity and showed correlations with the total score on the STAI. However, stronger correlations were observed with the STAI trait subscale score (French, 2011).

Cutler & Hodgson (2001) Single Item Assessment. Previous studies have measured

FAD using a single question. In order to assess construct validity, the following item was also asked: “I would like to ask how concerned *you* are about personally developing Alzheimer’s disease. Would you say that you are *very* concerned, *somewhat* concerned, *not very* concerned, or *not at all* concerned?” Although the reliability of this single-item measure cannot be assessed, responses to the question have shown validity in the assessment of older adults’ personal concerns about AD. Cutler and Hodgson (2001) suggest that there is significant shared variance between worries about memory functioning, family history of AD, and FAD development.

Anticipatory Dementia Inventory (ADI). – The ADI is a novel measure of FAD designed for the purpose of this study. The ADI includes 50 statements regarding six different facets of FAD, each of which maps onto a part of the HBM (see Figure 1). Personal susceptibility is measured by (1) beliefs about normal vs. AD-related forgetting, (2) beliefs about the prevalence and etiology of AD, and (3) beliefs about personal likelihood of developing the disorder. Perceived seriousness of AD is assessed in terms of (4) immediate consequences (the physical and emotional symptoms experienced when thinking about AD), and (5) specific fears about AD sequelae (e.g., loss of independence, loss of relationships, physical pain). Outcome expectations about AD will be assessed in statements related to (6) beliefs about availability and efficacy of treatments/preventative lifestyle changes to slow or stop AD progression. Respondents rate each of the items for agreement on a five-point Likert-style scale. 0 = “strongly disagree”, 1 = “somewhat disagree”, 2 = “neutral – do not agree or disagree”, 3 = “somewhat agree”, and 4 = “strongly agree.” Higher scores on the ADI denote greater anticipatory dementia or fear of Alzheimer’s disease. The measure is included in Appendix A.

Subjective Memory Complaints. Self-reported memory difficulties were assessed using the Memory Functioning Questionnaire.

Memory Functioning Questionnaire (MFQ; Gilowski, Zelinski, & Schaie, 1990). The MFQ is a self-report measure used to assess how respondents perceive the frequency and severity of their memory problems, as well as compensatory strategies. The present study will utilize only the General Frequency of Forgetting scale (Frequency). The Frequency scale asks the respondent to rate how often they have difficulty remembering types of information (ex. names, faces, appointments). Each item is rated on a 7-point scale, with 1 = “Always” and 7 = “Never.” Scores on the MFQ Frequency scale range from 18 to 126, with lower scores suggesting greater frequency of perceived memory failures.

Gilowski, Zelinski and Schaie (1990) have found evidence that the four subscales of the MFQ, including the General Frequency of Forgetting scale, are a valid assessment of perceived memory functioning in adults age 16 to 89. The Cronbach’s alpha coefficient for scores from the Frequency subscale, in particular, is .94, suggesting high reliability (Gilowski, Zelinski, and Schaie, 1990).

Objective Memory Functioning and Metamemory. Objective memory functioning was assessed using a verbal list-learning task. The lists used in the current task were drawn from a prior study of verbal learning and recall (Woodard, 1991; see Appendix B). These 20-word lists were selected because items on the lists were matched for item difficulty, syllable length, and semantic and phonemic relatedness. The length of the word list was chosen to reduce the likelihood of ceiling effects and provide a task sufficiently challenging to encourage purposeful use of memory strategies by participants.

Words are presented visually using E-prime®, Version 2.0 (Psychology Software Tools, Inc.). All words were presented in large Arial font, in the center of the screen. Computer conditions were regulated to maximize reliability of word presentation across participants; a

dedicated laptop (2015 HP 15-f133wm with Intel Celeron N2840 Processor, 4GB Memory, 500GB Hard Drive, 15.6-inch screen diameter, Windows version 8.1), devoid of self-updating programs, the Internet, or anti-virus software that may interfere with task presentation or data recording, was used. Due to varying participant height, the angle of the computer was not standard; instead, efforts were made to have all participants seated comfortably, with the computer placed 18 to 24 inches away and the screen faced directly at them. Participants were seated away from windows to reduce screen glare. All instructions were provided visually and read to the participant, verbatim.

Condition 1: Computer-Paced Memory and JOL Task. In the computer-paced condition, 20 words are presented at a standard pace. The examiner begins by explaining that 20 words will be presented visually at a rate of one word every two seconds, and that the participant must try to remember as many words as possible so that he or she can recall them, in any order, after the list is presented. Prior to the list presentation, the examiner asks for an initial judgment of learning estimate to determine the participant's rating of their memory without behavioral feedback. The list is then presented at the center of the computer screen. Directly after the final word has been presented, the examiner asks for a judgment of learning estimate from the participant (*"Of the 20 words you just saw, how many do you think you can remember now?"*). As soon as the participant has given a JOL appraisal, the examiner prompts the participant to recall, out loud, as many of the words he or she can remember in any order. The participant is allowed up to 60 seconds to recall as many words as possible, with no corrections for repetition or feedback about accuracy. After completion of the recall trial, the participant completes two more repetitions of the above procedure for a total of three trials. The resulting data provides four JOL estimates and accuracy across trials, as well as information about learning and memory.

Condition 2: Self-Paced Memory and JOL Task. In the self-paced list-learning condition, participants can vary the amount of time that they use to study the list of 20 words in order to maximize the number of words recalled. In the present study, metacognitive control is operationalized as amount of study time allocated to each item. The procedure for the self-paced condition mirrors the procedure in the computer-paced condition, with the exception of how word lists are paced. When the list is presented, the participant is given up to 10-seconds to study each word on the list, but can press a key to manually move on to the next word at any time. The participant can therefore shorten or lengthen their study time based on their assessment of task difficulty and memory ability. As in the computer-paced task, participants complete one JOL estimate prior to list presentation, then three presentation and recall trials, making a JOL estimate after each list presentation and before each 60-second recall trial.

Procedure

All interested individuals underwent a telephone screening to determine their eligibility for the study. Prior to this screen, participants provided verbal consent for recording of their responses. During the phone screen, participants were asked questions about their health and given the Telephone Interview for Cognitive Status to assess whether they were cognitively intact. Those individuals who met criteria for participation in the study were also asked to complete Cutler and Hodgson's single-item question about their level of FAD. Participants in the study were scheduled for an hour-and-a-half appointment to participate individually or in pairs. Testing was completed at the WSU research laboratory, the WSU IOG, or at a public library of their choice. All testing at public libraries was completed in private study rooms. Efforts were made to complete testing in only quiet, noise-controlled environments with adequate lighting to minimize computer glare. All individuals were asked to provide informed

consent before participation.

Questionnaires and Measures. Participants completed the Salthouse Synonym and Antonym Vocabulary tests, the State-Trait Anxiety Inventory, the MFQ Frequency Subscale, the FADS, and the ADI separately from the objective memory and metacognitive monitoring and control tasks.

It was thought that participants might be more anxious about their memory functioning after completing the questionnaires about memory concerns and fear of developing AD. This anxiety was expected to drive poorer performance on recall trials and underestimated or unusual JOL appraisals. Similarly, it was suggested that participants who had just undergone challenging memory tasks would over-estimate their concern about AD or memory-related worry. In order to control for the possibility of sensitization by task order, the order of the questionnaires and memory tasks was counter-balanced, with participants randomly assigned to receive either the questionnaires or the memory task first.

For participants with visual impairments, questionnaires with small print were read aloud by the examiner. The total time for participation was between 45 and 90 minutes. Participants were compensated with \$20 in cash.

CHAPTER 4: DATA ANALYSIS

Specific Aim 1

To validate FAD as a construct, the relationship between FAD and related constructs was assessed. First, independent samples t-tests were calculated to determine whether group differences exist on FAD measures based on family history. As data regarding caregiving was also available, FAD measure group means were also compared for individuals who had and had not reported a history of caregiving for someone with dementia. Pearson's r and Kendall's Tau-b correlations were computed to determine the relationship between FAD measures and the MFQ Frequency Subscale (subjective memory complaints; hypothesis b) and the STAI-State, STAI-Trait, and GDS scales (general negative affect; hypothesis c). To assess the relationship between FAD and metacognitive monitoring (hypothesis d), Pearson's r or Kendall's Tau-b correlations were calculated between FAD and JOL estimate score across all list-learning trials. A Bonferroni correction for familywise error was implemented.

Multiple regression was used to address hypothesis (e) of Specific Aim 1 – that negative affect, SMCs, and metacognitive monitoring account for a relatively small amount of variance in FAD. FAD was regressed onto STAI-State and Trait subscales, GDS, MFQ Frequency of Forgetting score, and JOL estimate summary scores simultaneously. The amount of variance unaccounted for ($1-R^2$) reflects the amount of residual variance in FAD not explained by other factors.

To address the final hypothesis (f) of Specific Aim 1, the Anticipatory Dementia Checklist (ADI) will be developed and analyzed. As previously mentioned, the ADI was developed to assess facets of FAD that map onto the Health Belief Model (HBM; Rosenstock, Stretcher and Becker, 1988) including beliefs about general and personal susceptibility to AD,

health and AD-specific locus of control, and knowledge about preventative and prescriptive treatments for AD. An initial pool of 50 questions was created by developing AD-specific items related to each component of the HBM (perceived susceptibility to AD, perceived seriousness of having AD, perceived barriers and benefits to taking action against AD development; see Figure 1). Items were created on a Likert-style bipolar scale, ranging from “Strongly Agree” to “Strongly Disagree.”

To assess construct validity, the correlations shared between different measures of FAD (Cutler & Hodgson single item question, FADS and ADI) were determined. Kendall’s Tau-b correlations were calculated for all analyses involving the Cutler and Hodgson single item. Pearson’s r correlations were computed for all other associations.

Although comprehensive scale validation is not possible with this limited sample size, scale validation procedures were used to make initial estimates of the ADI’s internal consistency and factor structure. Cronbach’s alpha was calculated to determine the internal consistency of the scale items. A principal components analysis was used to determine the independent facets of FAD assessed by the ADI. Item factor loadings were qualitatively and quantitatively reviewed to characterize each significant component.

Specific Aim 2

The second specific aim focuses on understanding which of the metacognitive variables utilized in this study might account for significant proportions of variance in objective memory functioning. To address hypotheses (a) and (b) of Specific Aim 2, Kendall’s Tau-b and Pearson’s r correlations were calculated between total number of items recalled across all self-paced list-learning trials and each of the metacognitive variables: MFQ-Frequency subscale scores (SMCs), FADS, ADI and Cutler & Hodgson’s single item scores (FAD), JOL estimate

summary score (metacognitive monitoring), and study time differences between computer-paced versus self-paced tasks (metacognitive control). A Bonferroni correction for family-wise error was implemented.

To address hypothesis (c) of Specific Aim 2, the relative proportion of variance in objective memory functioning accounted for by each of the metacognitive variables was assessed using multiple regression. Two regression equations were created to explain composite memory scores across all self-paced trials and computer-paced trials, separately. MFQ-Frequency subscale scores, FADS, ADI and Cutler & Hodgson's single item scores, JOL estimate summary score, and study time differences between computer- versus self-paced tasks were entered simultaneously. The squared semi-partial correlations of each predictor were evaluated to determine the relative unique contributions of each metacognitive variable to the overall model.

Specific Aim 3

The final objective of the study aimed to investigate the relationship between FAD and metacognitive monitoring and control. First, metacognitive monitoring was compared to metacognitive control through calculation of Pearson's r correlations between JOL estimates from self-paced tasks and study time on subsequent trials (Hypothesis a). Kendall's Tau-b and Pearson's r correlations were calculated to estimate the relationship between FAD measures (FADS, ADI, and the Cutler & Hodgson single item) and metacognitive monitoring accuracy (the absolute value of JOL resolution). Kendall's Tau-b and Pearson's r correlations were also calculated to estimate the relationship between FAD measures and metacognitive control (average study time difference for computer-paced versus self-paced trials). Again, a Bonferroni correction for multiple comparisons was implemented to correct for the proposed comparisons.

To address hypotheses (b) and (c) of Specific Aim 3, a continuous x continuous

moderated multiple regression analysis was planned. A moderated multiple regression was to be estimated for each of the measures of FAD. To assess whether the FADS scores moderates the relationship between JOL estimates and study time changes, JOL estimate summary scores across all trials and the interaction between JOL estimate and FADS were to be entered into a regression equation to predict average study time difference between computer- and self-paced trials. This analysis was to be repeated for each of the measures of anticipatory dementia.

CHAPTER 5: RESULTS

All analyses were performed using SPSS Version 22.0 (IBM Corporation, 2013).

Data Screening

Minimum and maximum values for each variable were assessed to ensure data entry accuracy. A missing value analysis was completed using SPSS. All variables had less than or equal to five percent missing data with the exception of the Memory Functioning Questionnaire, General Memory Estimate (Question 1: “How would you rate your memory in terms of the kinds of problems you have?”, 11.8% missing data), FADS Item 16 (“My hands become clammy when I think about getting Alzheimer’s disease”; 6.5% missing data), and the Cutler & Hodgson FAD Question (63.4% missing data). Due to the high proportion of missing data, which appeared to occur because of a misprint in test packets, the Cutler and Hodgson FAD question was removed from further analyses. Cases with and without missing data on these variables were dummy coded and compared to assess for patterns of missing data. No patterns emerged from these analyses; therefore, data were determined to be missing at random. Rather, observations of participants’ files suggest that items were skipped because of their placement on questionnaire pages or because of difficulty reading the item.

Frequency tables were created for each variable to assess the distribution of the data. To determine whether univariate outliers existed in the dataset, all continuous variables were converted into z-scores and compared against a cutoff of $z \pm 3.29$, which reflects an alpha level of $\alpha = .001$. Two univariate outliers were detected: a single outlier for computer-paced JOL resolution (Case #208; $z = 3.31$), and a single outlier for self-paced JOL resolution (Case #110; $z = -3.69$). No other univariate outliers were detected in the dataset. In order to assess for the presence of multivariate outliers, the 14 outcome variables of interest were entered into a

regression to calculate Mahalanobis' distance. Using a chi-squared cut-off of $\chi^2_{(13)} = 36.123$ consistent with an alpha level of $\alpha = .001$, only two multivariate outliers were detected: Case #233 (Mahalanobis' distance = 53.685), and Case 150 (Mahalanobis' distance = 39.262). Given the limited number of univariate and multivariate outliers, it was not considered necessary to perform a discriminant function analysis to assess differences in these cases. Cases were inspected individually to determine if they possessed unique characteristics compared to the rest of the data; they were demographically similar to other cases.

The outcome variables of interest were also assessed for normality. The distribution of data for each variable was evaluated visually using Q-Q plots. The skewness and kurtosis index of each variable was also calculated. Variables were classified as not skewed, moderately skewed, substantially skewed, and not kurtotic, moderately kurtotic, and substantially kurtotic using this information (see Table 2). Although some variables were significantly skewed or kurtotic, these measures represent phenomena that are not expected to be normally distributed in the population; therefore, no transformation was completed to adjust these variables.

Demographically Corrected Outcome Measures

Age was not significantly associated with measures of FAD. It was not significantly associated with scores on the measures of negative affect. Age was associated with objective memory performance, as evidenced by significant correlations between age and Trial 1 score ($r_{(90)} = -.414, p < .001$), Trial 2 score ($r_{(90)} = -.451, p < .001$), Trial 3 score ($r_{(90)} = -.389, p < .001$) and total score ($r_{(90)} = -.468, p < .001$) in the computer-paced condition and Trial 1 score ($r_{(90)} = -.221, p = .035$), Trial 2 score ($r_{(90)} = -.309, p = .003$), Trial 3 score ($r_{(90)} = -.316, p = .002$) and total score ($r_{(90)} = -.311, p = .003$) in the self-paced condition. Interestingly, age was associated with accuracy of the JOL estimate (resolution) on computer-paced Trial 1 ($r_{(90)} = -.212, p =$

.042), computer-paced Trial 2 ($r_{(90)} = -.245, p = .019$), and across computer-paced trials ($r_{(90)} = -.219, p = .036$), but was not associated with JOL resolution on any self-paced trials.

Education did not share a significant relationship with any measures of FAD or negative affect. Education was significantly associated with performance on self-paced Trial 1 ($r_{(90)} = .260, p = .012$), Trial 2 ($r_{(90)} = .270, p = .009$), and Trial 3 ($r_{(90)} = .311, p = .003$), and across self-paced trials ($r_{(90)} = .302, p = .003$), but was not associated with objective memory functioning in the computer-paced trial. Education was also significantly associated with the JOL estimate on self-paced Trial 2 ($r_{(90)} = .223, p = .032$) and Trial 3 ($r_{(90)} = .205, p = .049$). Education shared a significant relationship with JOL resolution on computer-paced Trial 1 ($r_{(90)} = .215, p = .039$) and overall ($r_{(90)} = .209, p = .045$). Finally, education was significantly associated with the difference in number of items recalled on self-paced Trial 2 compared to computer-paced Trial 2 ($r_{(90)} = .222, p = .033$). Similarly, education was significantly associated with the difference in self-paced Trial 3 score versus computer-paced Trial 3 score ($r_{(90)} = .312, p = .002$), and overall difference between self- and computer-paced task scores ($r_{(90)} = .298, p = .039$).

Given that age and education were correlated with the many of the outcome measures of interest in the study, demographically corrected scores were computed. This correction was accomplished by regressing the outcome measures onto age and education and saving the resulting standardized residuals. These demographically corrected scores were used in subsequent analyses.

Specific Aim (1)

Individuals with and without a family history of dementia do not differ on FAD (Hypothesis a). Independent samples *t*-tests revealed that there were no significant difference between individuals with or without family history of dementia on FADS score ($t_{(89)} = -1.29, p =$

.201, Cohen's $d = 0.28$) or ADI score ($t_{(89)} = -0.61, p = .546$, Cohen's $d = 0.13$). No significant differences existed between individuals with and without history of caregiving for someone with dementia on the FADS score ($t_{(89)} = -0.80, p = .425$, Cohen's $d = 0.17$) or ADI score ($t_{(89)} = -0.97, p = .337$, Cohen's $d = 0.21, ES = .10$). Results are summarized in Table 3.

Higher FAD is associated with more subjective memory complaints (Hypothesis b).

Pearson's r correlations were calculated to determine the relationship between measures of FAD and subjective memory complaints (MFQ total score). Given that two comparisons were calculated to address this hypothesis, a Bonferroni correction for family-wise error was implemented, resulting in a significance level of $\alpha = 2.50E-2$. Scores on the FADS were significantly negatively associated with self-reported quality of memory on the MFQ ($r_{(88)} = -.44, p < .001$). Scores on the ADI were also significantly negatively associated with MFQ total scores ($r_{(88)} = -.33, p = .002$). Results are summarized in Table 4.

FAD is positively associated with depressive symptoms and trait anxiety (Hypothesis

c). Pearson's r correlations were calculated to determine the relationship between measures of FAD and Anxiety (STAI-State and STAI-Trait, GDS total score). Given that eight comparisons were calculated to address this hypothesis, a Bonferroni correction for family-wise error was implemented, resulting in a significance level of $\alpha = 6.25E-3$. Both the FADS score and the ADI score were significantly positively associated with State Anxiety at the $\alpha = .05$ level, but not at the Bonferroni-corrected level of significance. FADS score was significantly associated with Trait Anxiety at the $\alpha = .05$ level, but not at the Bonferroni-corrected level of significance; however, scores on the ADI shared a significant positive relationship with Trait Anxiety at this corrected level ($r_{(88)} = .248, p = .002$). Finally, both the FADS ($r_{(88)} = .214, p = .003$) and the ADI ($r_{(88)} = .287, p < .001$) were positively associated with depressive symptoms. These

findings are also included in Table 4.

FAD is not associated with metacognitive monitoring or resolution (Hypothesis d).

Pearson's r correlations were calculated to determine the relationship between measures of FAD versus JOL estimates and resolution (see Tables 5 and 6). A Bonferroni correction was used to account for the 32 total comparisons made to address this hypothesis, resulting in a significance level of $p < 1.56E-3$. No measures of FAD were significantly related to any JOL estimate or the resolution of any JOL estimate on computer- or self-paced trials. These findings do not differ when examining family history positive and family history negative groups in isolation.

Subjective memory complaints, negative affect, and metacognitive monitoring account for a relatively small amount of variance in FAD (Hypothesis e). Because correlations indicated no significant relationship between measures of FAD and JOL appraisals or accuracy, metacognitive monitoring variables were excluded from analyses for this hypothesis. Separate multiple regression equations were calculated for the FADS and ADI score, entering MFQ total score, GDS score, STAI State and STAI Trait subscale scores simultaneously. $1-R^2$ was used to assess the amount of residual variance in the FAD measure unaccounted for by other measures of anticipatory dementia. Subjective memory complaints and negative affect accounted for a significant proportion of the variance in FADS scores ($R^2 = .269$, $F_{(4)} = 7.83$, $p < .001$), but 73.1% of the variance in FADS scores was not accounted for by these predictors. Similarly, subjective memory complaints and negative affect accounted for a significant proportion of the variance in ADI scores ($R^2 = .273$, $F_{(4)} = 7.97$, $p < .001$), but 72.7% of the variance in ADI scores was not accounted for by these predictors.

The ADI is related to other measures of FAD, but it also taps into other aspects of Alzheimer's disease fear not measured by existing instruments (Hypothesis f). First, a

Pearson's r correlation was calculated to assess the relationship between the ADI and the FADS. The ADI was significantly positively associated with the FADS score at the Bonferroni-corrected level of significance ($r_{(86)} = .697, p < .001$). The FADS score accounted for 48.6% of the variance in the ADI score, but approximately 51.4% of variance in the ADI score was unique.

Internal consistency of ADI scores was assessed using Cronbach's alpha. Cronbach's alpha of the scale was $\alpha = .896$, indicating a level of reliability adequate for both clinical and experimental use. An assessment of the Cronbach's alpha with item deletion indicated that reliability of the scale would not significantly differ with deletion of any single item.

For the purpose of exploring the potential components in the scale for future directions, a principal components analysis (PCA) using an orthogonal Varimax rotation was conducted. An initial assessment of the extractions showed that all components were significantly associated, with no extraction residuals $<.512$. Using an eigenvalue of greater or equal to 1 as cutoff for significance, 14 components were extracted. This solution would account for 75.35% of the variance in the measure. The 14-component solution resulted in 15% (194) residuals with absolute values greater than .05, indicating that this solution may not adequately capture the variance in the measure. Alternatively, a visual examination of the scree plot for the PCA (see Figure 2), indicate that a five-component solution may be more appropriate. This solution accounts for 47.87% of the variance in the measure. A parallel analysis was completed to determine the number of components to retain, based on the method suggested by O'Connor (2000). Parallel analysis uses the current study's sample size, number of variables in the PCA, and other parameters set by the researcher to calculate eigenvalues from randomly generated correlation matrices. These generated values were then compared to eigenvalues of the components extracted in the principal components analysis detailed above. The number of

components retained from the PCA are those components with eigenvalues greater than the corresponding random eigenvalues. Based on this method, a total of six factors should be retained for further analysis. This solution accounts for 52.48% of variance in the ADI. These data are summarized in Table 7.

A qualitative analysis was performed to determine the content of each potential component (see Table 8). The first component is related to overall negative affect at the thought of developing AD (e.g., “Thinking about getting AD makes me feel angry.”), as well as specific worries about AD (e.g., “I worry that I will be a burden on my family if I develop AD.”). The second component relates to physical symptoms associated with fear of AD (e.g., “When I think about AD, my stomach is in knots or I feel nauseated.”). The third component appears to assess the belief in efficacy of treatments for AD (e.g., “If I am diagnosed with AD, there is nothing doctors will be able to do to improve my outcomes.”). The fourth component appears to tap into belief in personal vulnerability to AD (e.g., “I believe that I am going to develop AD.”). The fifth component appears to assess beliefs in self-efficacy in reducing the likelihood of developing AD (e.g., “I can reduce my chances of developing AD by using my brain in new ways, like doing crossword puzzles, Sudoku, or reading.”). Items in the final component tap into resilience to worry about AD (“Thinking about AD does not make me worry.”). Other components were less defined.

Specific Aim (2)

Given the 48 comparisons completed for this aim, a Bonferroni-corrected significance level of 1.04E-3 was used.

Metacognitive control is positively associated with objective memory performance, but subjective memory complaints and FAD are not (Hypothesis a). Pearson’s r correlations

were calculated to determine the relationship between MFQ scores, FAD measures, study time differences in the self-paced condition, and objective memory functioning across computer- and self-paced trials. As indicated in Table 9, subjective memory complaints and anticipatory dementia measures were not significantly associated with objective memory functioning on any computer- or self-paced trial, or across trials. However, total study time difference between self-paced and computer-paced trials was significantly associated with performance on self-paced Trial 1 ($r_{(89)} = .379, p < .001$) and with the self-paced trials total score ($r_{(89)} = .345, p < .001$) at the Bonferroni-corrected level of significance.

Metacognitive monitoring is positively associated with objective memory performance (Hypothesis b). To address this hypothesis, Pearson's r correlations were calculated. Results are also summarized in Table 9. The total JOL estimate across all computer-paced trials was significantly positively associated with objective memory performance on computer-paced Trial 2 ($r_{(90)} = .407, p < .001$) and with performance across all computer-paced trials ($r_{(90)} = .396, p < .001$). In contrast, the total JOL estimate across all self-paced trials was significant positively associated with performance on computer-paced Trial 2 ($r_{(90)} = .492, p < .001$), computer-paced Trial 3 ($r_{(90)} = .472, p < .001$), computer-paced trials total ($r_{(90)} = .509, p < .001$), self-paced Trial 1 ($r_{(90)} = .436, p < .001$), self-paced Trial 2 ($r_{(90)} = .530, p < .001$), Trial 3 ($r_{(90)} = .504, p < .001$), and self-paced trials total ($r_{(90)} = .526, p < .001$).

Among metacognitive variables, metacognitive monitoring accounts for the greatest amount of variance in objective memory functioning (Hypothesis c). To address Hypothesis (c), MFQ total score, FADS total score, ADI total score, JOL estimates across all computer-paced trials, JOL estimates across all self-paced trials, and study time differences between self- and computer-paced trials were entered simultaneously into a regression equation to account for

variance in objective memory functioning. These measures were regressed onto computer-paced objective memory functioning and self-paced objective memory functioning separately. The results are summarized in Table 10. The squared semi-partial correlations were then assessed to determine the unique proportion of variance in the outcome accounted for by each of the metacognitive variables. Self-paced JOL estimates accounted for the highest proportion of unique variance in memory performance on computer-paced trials (12.53%, $t = 3.825$, $p < .001$) and self-paced trials (19.27%, $t = 5.00$, $p < .001$). Metacognitive control also accounted for a significant proportion of variance in computer-paced memory performance (4.37%, $t = -2.256$, $p = .027$), and self-paced memory performance (4.41%, $t = 2.389$, $p = .019$). None of the other metacognitive variables accounted for significant unique variance in objective memory functioning.

Specific Aim (3)

Metacognitive monitoring during self-paced trials is associated with metacognitive control (Hypothesis a). To assess this aim, Pearson's r correlations between JOL estimates for individual trials and across trials in the two conditions and study time differences were examined (see Table 11). Metacognitive monitoring was not significantly associated with metacognitive control on any of the computer-paced trials. In contrast, study time differences were significantly positively associated with JOL estimates on self-paced Trial 3 ($r_{(89)} = .248$, $p = .018$) and across all self-paced trials ($r_{(89)} = .226$, $p = .031$).

FAD is not associated with metacognitive monitoring, and it does not impact metacognitive control (Hypotheses b and c). Because none of the FAD measures were significantly associated with any of the JOL estimates, JOL resolutions, or study time differences, Hypothesis (b) was not analyzed further.

CHAPTER 6: DISCUSSION

The present study investigated a common problem among the ‘worried well’ population of middle- and older-aged adults: Fear of Alzheimer’s disease. The study provided further evidence of the shared variance between FAD and emotional constructs, including depression and trait-level negative affect. The study also examined the relationship between FAD and cognitive constructs, such as general subjective memory complaints, task-specific metacognitive monitoring and control, and actual memory performance. Results indicate that FAD may constitute perceptual and emotional experiences, as opposed to a cognitive phenomenon, as it is not linked to ability to accurately monitor one’s performance, ability to change behavior to improve performance, or objective recall ability. Finally, the study introduced a new, theory-driven approach to assessing FAD: the Anticipatory Dementia Inventory.

Results indicated that, contrary to prior research (Cutler & Hodgson, 1996; 2001; Roberts & Connell, 2000; Cantegreil-Kallen & Pin, 2012; Leon et al., 2015), individuals with a family history of dementia or of caregiving for individuals with dementia did not report a higher level of FAD. These findings may be explained by the relatively older sample assessed in the study, whose mean age was approximately 69 years. Although almost equal numbers of participants with and without a family history of AD were included, this family history was self-reported and largely based on participants’ perception, rather than on formal diagnosis of a parent with dementia. Many participants attributed their lack of knowledge of specific dementia diagnosis to the fact that knowledge of Alzheimer’s disease was more limited and formal diagnosis of dementia, let alone AD, was much less common when their parents were aging. Furthermore, anecdotal evidence from most family history positive participants indicated that parents with dementia often died early in their disease course, which may have lessened participants’ fear-

producing experiences with AD. The relatively older age of the sample may also have been associated with reduced FAD; older participants may have surpassed the perceived age of onset of AD, thereby lessening their level of fear. The finding that caregiving for AD is not associated with FAD, which also contradicts prior literature, may be accounted for by the fact that most caregiving positive participants reported doing so only professionally. Individuals whose occupation involved caregiving may have more accurate perceptions about AD prevalence and treatments, as well as greater self-efficacy with health behaviors, both of which would reduce FAD. Furthermore, a recent study proposed that proximity to AD through caregiving or family history may be a predictor of level of FAD only in younger to middle-aged adults (Cantegreil-Kallen & Pin, 2012). Nonetheless, the finding challenges the role of family history and caregiving in developing a 'worried well' presentation. While these experiences may contribute to perceived personal vulnerability to AD, neither family history nor personal exposure to AD independently produce concern that one will develop AD. Other characteristics, such as resilience to FAD, belief in treatments, or self-efficacy, may balance out the effect of family or caregiving history.

The current study corroborated prior findings (Cutler & Hodgson, 1996; 2001) that anticipatory dementia is associated with greater subjective memory complaints. However, the hypothesis that FAD would also be negatively associated with metacognitive monitoring was not supported. The number of items participants expected to recall across trials was the same, regardless of FAD level. This finding could be explained if all participants were providing similar, low estimates of performance, but only individuals high in FAD were accurately rating their poor performance. However, FAD also had no effect on metacognitive resolution, suggesting that individuals who are more fearful are no less accurate at rating their memory of a

list of words than those who are less fearful. Similarly, FAD level was not associated with scores on measures of overall cognitive functioning (TICS, Synonym or Antonym Vocabulary Tests), or with memory performance.

The above findings collectively differentiate subjective memory complaints from task-specific memory ratings. Indeed, investigation of the relationship between subjective memory complaints and metacognitive monitoring and resolution showed no significant association. Although a healthy, non-impaired individual may accurately monitor his ability to learn and remember information immediately and specifically to a memory task, an intervening process may cause him to misperceive the accumulation of these ratings over time to yield subjective memory complaints that drive FAD. Given the findings that FAD and subjective memory complaints are associated with trait-level negative affect, it is likely that this intervening process is affective in nature.

The association between subjective memory complaints and FAD, combined with the lack of relationships between metacognition and FAD and objective memory functioning and FAD provides support for the stereotype threat theory of FAD development. FAD may be interpreted as a failure to engage in task-specific metacognitive monitoring on a daily basis and, instead, an expectation of memory failure that increases anxiety, reduces attentional resources, and provides the individual with perceived 'evidence' that their memory *is* actually failing.

The study replicated prior findings that subjective memory complaints were not tied to objective memory performance (Jungwirth et al., 2004; Ponds, Van Boxtel, & Jolles, 2000; Lenehan, Klekociuk, & Summers, 2012). Given the hypothesized relationship between SMCs and FAD, it is not surprising that FAD is also not associated with memory functioning. However, this conclusion does highlight that FAD is based neither on actual memory failures,

nor an accurate appraisal of memory. Conversely, metacognitive monitoring was significantly associated with objective memory performance. This finding again underlines that, regardless of level of FAD, self-rating and performance should be related. An unexpected conclusion from the analyses is the relationship between metacognitive control and memory performance on the self-paced list-learning task. Participants who increased their study time were able to recall more words. Given the significant positive relationship between metacognitive monitoring and control, this association emphasizes that, in a healthy sample, accurate metacognitive monitoring can drive changes in behavior that can lead to improved performance.

In sum, the results of the present study point to a dissociation between more affectively-changed self-rating processes (subjective memory complaints, FAD) and more cognitive appraisals (metacognitive monitoring, resolution, and control). Although only the latter are grounded in actual memory functioning, both task-specific and general memory ratings appear to cause significant distress that may interfere with functioning in daily life.

A final goal of the study was to examine FAD through the Health Belief Model through the development of the Anticipatory Dementia Index (ADI). While prior studies have examined the symptoms of FAD using the Fear of Alzheimer's Disease Scale (FADS; French, Floyd, Wilkins & Osato, 2012; Moon, Kim, Hoi, Oh & Chan, 2014), these studies were limited in that they could not identify potential targets for intervention to reduce FAD. As FAD accounts for significant distress reported in large proportions of international samples (Anderson, Day, Beard, Reed & Wu, 2009; Cantegreil-Kallen & Pin, 2012; Werner, 2002; Leon et al., 2015) and drives change in health behaviors (Ramakers et al., 2009; Corner & Bond, 2004), a better understanding of causes of the phenomenon was sought. The study supports the notion that FAD can be examined through the lens of the Health Belief Model (Rosenstock et al., 1988).

The Health Belief Model (Rosenstock et al., 1988) proposes that health beliefs and behaviors are shaped not only by perceived threat of a disorder or problem, but also one's expectations for effectively reducing the threat. The primary component of FAD reflects overall negative affect and specific concerns about AD. This component maps cleanly onto the 'Perceived Seriousness of Consequences' aspect of the Health Belief Model (1988). The component is also most closely tied to other measures of FAD. Among specific concerns about AD, fear of not being able to contribute to society (Item #49), fear of being a burden on one's family (Item #43), concern about loss of decision-making abilities (Item #42), and concern over the financial burden of AD (Item #45) were most commonly endorsed by all participants, regardless of FAD level. These items demonstrate that FAD is not simply concern over memory loss or physical symptoms, but rather a concern over the larger impact of FAD on one's place within the family and society.

The second component of the ADI represents physical symptoms in response to these AD concerns. It likely also shares a large proportion of variance with the FADS measure, which also assesses physical reactions in FAD. Interestingly, very few participants endorsed the three major items comprising this component, suggesting that even those individuals who are most fearful do not experience sleep disturbances, shakiness or restlessness, or nausea in response to FAD. This component would also fall under the 'Perceived Seriousness of Consequences' facet of the Health Belief Model.

The fourth component of the ADI, which reflects personal vulnerability to AD, constitutes to 'Perceived Susceptibility' facet of the Health Belief Model. Items 28 ("I believe that I am going to develop AD") and 23 ("I believe that I am already showing signs of AD.") were the most commonly endorsed of Component 4, but were endorsed only by the most fearful

individuals. It was expected that other items related to perceptions of susceptibility to AD would load onto this component or form additional components. Although loadings are relatively lower, items from Components 9 through 12 represent beliefs about the normal versus abnormal memory loss with aging and perceptions of the heritability of AD. Given that recent studies have highlighted the importance of normative beliefs in FAD (Leon et al., 2015; Kim et al., 2015), future iterations of the scale will revise these items to more clearly tap into these constructs, as they constitute an important facet of FAD. In sum, Components 4, 9, 10, 11, and 12 likely measure Perceived Susceptibility, Components 1 and 2 measure Perceived Seriousness of Consequences, and all items in these components tap into some aspect of Perceived Threat of AD.

The third and fifth components of the ADI represent into the Outcome Expectations facet of the Health Belief Model. Items in Component 3 reflect belief in the efficacy of treatments for AD. Item #4 (“If I am diagnosed with AD, there is nothing doctors will be able to do to improve my outcomes.”) and #22 (“There are treatments that can slow or stop the progression of AD”; reverse-scored) were most strongly endorsed by individuals high in FAD. These items represent a mindset about the intractability of AD that would likely contribute to FAD. The fifth component assesses beliefs in one’s ability to effectively reduce risk for AD by changing behavior. Somewhat unexpectedly, the two self-efficacy items most commonly endorsed tapped into a belief that consuming a healthy diet (Item #12) and participating in social activities (Item #15) were the best strategies to reduce AD risk, as compared to engaging in physical or cognitively-stimulating activities. Nonetheless, these items reflect an important target for treatment of FAD. A recent review of a French public health intervention to decrease FAD determined that inaccurate knowledge about the availability and efficacy of medical and

behavioral interventions is a critical predictor of FAD (Leon et al., 2015).

The items of the final component tap into resilience against worry about AD (“Thinking about AD does not make me worry.”), and it may be a gross measure of overall level of FAD once Perceived Vulnerability, Perceived Seriousness of Consequences, and Outcome Expectations are processed.

These results corroborate the findings of a recent qualitative Australian study in which focus groups were interviewed to determine their knowledge of dementia risk factors and their motivation to engage in lifestyle changes that would reduce the likelihood of dementia (Kim et al., 2015). Although participants were able to list non-modifiable risk factors (age, genetics) and modifiable risk factors (diet, exercise, mental stimulation, social activity), there were considerable misperceptions about dementia as a normal part of aging and about relative benefit of risk factors. The study found evidence of three groups differing in level of perceived susceptibility: a fearful group, who were unable to approach actually estimating their likelihood of developing dementia because of their fear, a rational group, who reviewed their lifestyle and risk factors (with variable accuracy) to produce an estimate of dementia risk, and a cynical group, whose external locus of control drove them to estimate their chances of developing dementia as random, based on luck or chance. Avoidance of dementia and death due to dementia were the most commonly cited benefits of changing health behaviors, while limited education was the most commonly cited barrier. These findings echo the early conclusions from the ADI. The ADI may also be clinically useful as a measure of FAD that is more efficient than qualitative focus group interviewing to identify public perception of AD.

Limitations

Although the study provided important, novel information about the construct of FAD, it

was somewhat limited. The small sample size tested in this pilot study was not sufficient for true scale development. Furthermore, the sample was community dwelling and cognitively intact. Given these sample characteristics, relatively few individuals demonstrated high FAD. Future studies should investigate a clinical sample that includes individuals with true memory impairment and individuals whose concern is significant enough to warrant neuropsychological or other medical evaluation. This more clinical sample will allow researchers to investigate the convergent and discriminant validity between FAD and other constructs. A larger sample with a revised scale would permit confirmatory factor analytic studies to further conceptualize FAD as part of a health belief model.

Other characteristics of the sample may have also impacted FAD. The sample was highly educated and relatively older. As previously mentioned, older adults may believe that the older they are without a diagnosis of AD, the less likely they are to experience it (Kim et al., 2015). More educated adults may also have more accurate perceptions of AD prevalence and risk factors, lowering the overall level of FAD in the sample. Future studies should examine a greater proportion of middle-aged adults, with more varied education.

Finally, it may be helpful to examine the relationship between FAD and metacognition through a different metacognitive monitoring task. While the present study utilized a judgment of learning task that required assessment of learning prior to recall, a feeling of knowing (FOK) task, in which the participant rates how many items they recalled after the recall trial, may have been more appropriate. Anecdotally, some participants rushed through the JOL estimates, as they saw it as a distraction from working memory rehearsal of the final items on the list. If recall had already occurred as in the FOK task, more effort and concentration might have been allocated to the task of metacognitive monitoring, increasing the validity and reliability of these

ratings.

Implications

The study presents many clinical implications. The shared variance between FAD, negative affect and subjective memory complaints, when compared to the lack of association between FAD and metacognitive variables, suggests that treatment of FAD should focus on affective rather than cognitive strategies. Because FAD does not seem to be shaped by actual memory performance or self-awareness during a memory task, feedback strategies may not be effective in reducing FAD. Conversely, education about risk factors and normal versus abnormal memory decline may target perceived susceptibility to dementia, thereby reducing perceived threat. Similarly, education about availability of treatments and appropriate times to seek evaluation may decrease FAD. Finally, interventions to shift locus of control regarding AD development from external (genetics, chance, luck) to internal (lifestyle and health behavior) may increase self-efficacy and outcome expectations, also reducing FAD. As an example, a recent Australian public health intervention has shown promise in correcting misperceptions about dementia and increasing knowledge of and motivation for risk factor change via an informational website (Farrow, 2013).

The Anticipatory Dementia Index constitutes a promising new method for assessing not only intensity and implications of FAD, but also personal causes. It may be used to identify subtypes of FAD that may benefit differentially from interventional strategies. For instance, the ADI may allow clinicians to determine whether a ‘worried well’ patient simply has a misperception of his own vulnerability to the disorder, as opposed to a lack of understanding or motivation to reduce his vulnerability. The first patient may simply need normalizing education to reduce his fear, whereas the second patient may require more prescriptive lifestyle change to

address his FAD. Future studies should continue development of the scale with rewording and revisions of items to address specific facets of FAD.

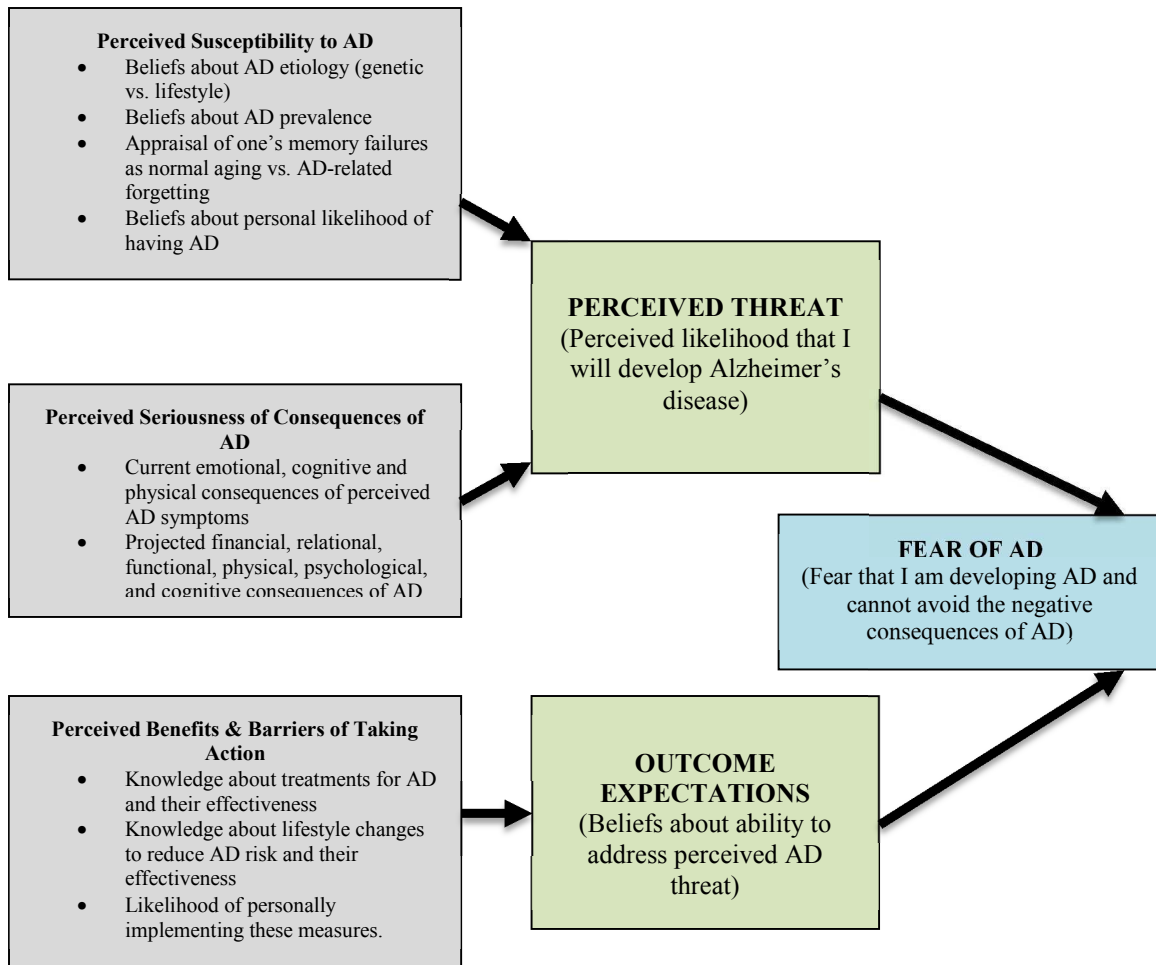


Figure 1. Conceptual model for anticipatory dementia

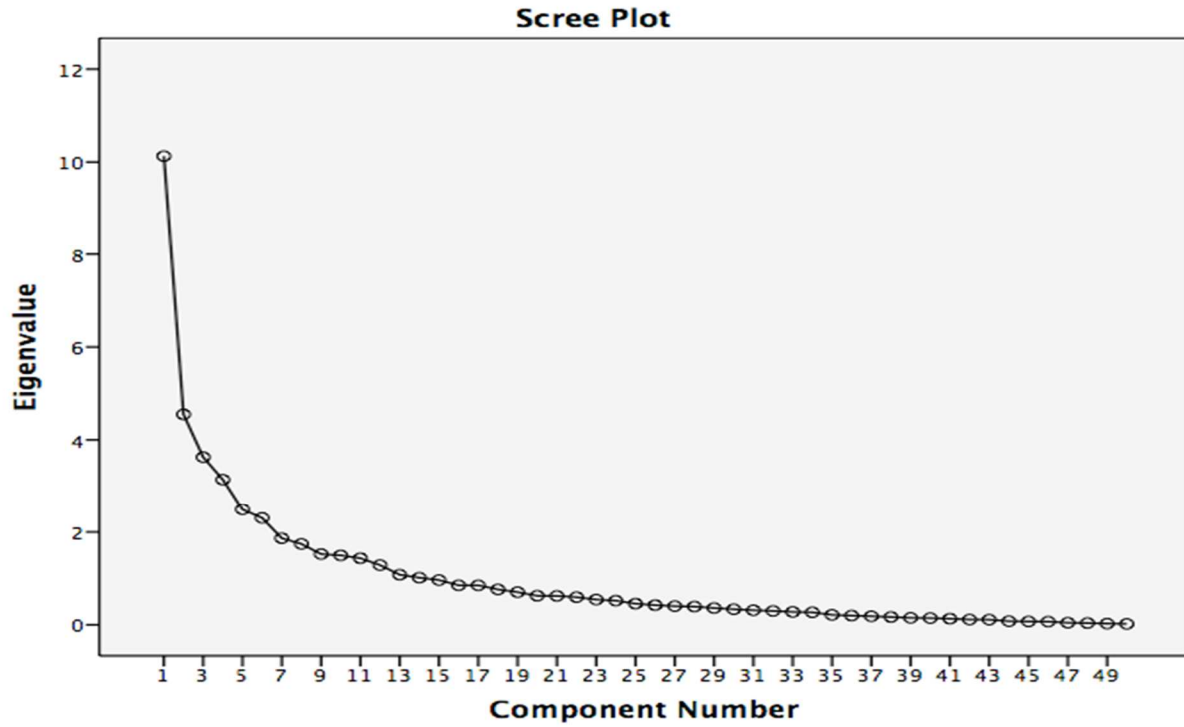


Figure 2. Scree Plot for Principal Components Analysis of the Anticipatory Dementia Checklist

Table 1

Demographic characteristics of the total sample and family history positive versus family history negative participants

	Total Sample (n = 93)		Family Hx + (n = 43)		Family Hx - (n = 50)		p
	n (%)	M (SD)	n (%)	M (SD)	n (%)	M (SD)	
Age		70.18 (9.74)		71.31 (10.07)		68.91 (9.31)	.241
Gender							.627
Male	23 (24.80)		13 (26.00)		9 (20.90)		
Female	71 (75.20)		36 (72.00)		34 (79.10)		
Race							.796
Caucasian	41 (44.10)		21 (42.00)		20 (46.50)		
African-American	46 (49.50)		24 (48.00)		22 (51.20)		
Other	4 (4.40)		3 (6.00)		1 (2.30)		
Education		15.24 (2.67)		15.24 (2.83)		15.23 (2.51)	.982
< 9 years	0 (0.00)		0 (0.00)		0 (0.00)		
9 - 11 years	0 (0.00)		0 (0.00)		0 (0.00)		
12 years	21 (22.60)		12 (24.00)		9 (20.90)		
13-15 years	27 (29.00)		15 (30.00)		12 (27.90)		
16+ years	44 (47.40)		22 (44.00)		22 (51.10)		
Employment							.211
Retired/Disabled	66 (71.00)		35 (70.00)		31 (72.00)		
Part-Time	17 (18.30)		7 (14.00)		10 (23.30)		
Full-Time	9 (9.70)		7 (14.00)		2 (4.70)		

Table 2

Skewness and Kurtosis of Distributions of Outcome Variables

	<u>Skewness</u>			<u>Kurtosis</u>		
	<u>None</u>	<u>Moderate</u>	<u>Substantial</u>	<u>None</u>	<u>Moderate</u>	<u>Substantial</u>
STAI State		+		x		
STAI Trait		+		x		
GDS			+			+
MFQ	x			x		
FADS		+		x		
ADI	x			x		
CP Pre-estimate	x			x		
CP Trial 1 JOL Estimate	x			x		
CP Trial 2 JOL Estimate		+			+	
CP Trial 3 JOL Estimate			+			+
CP Trial 1 Resolution	x				+	
CP Trial 2 Resolution	x				+	
CP Trial 3 Resolution	x			x		
CP Total Resolution	x				+	
CP Trial 1 Raw Score	x				-	
CP Trial 2 Raw Score	x			x		
CP Trial 3 Raw Score	x			x		
CP Raw Total	x			x		
SP Pre-estimate		+				+
SP Trial 1 JOL Estimate			+			+
SP Trial 2 JOL Estimate		+				+
SP Trial 3 JOL Estimate		+			+	
SP Trial 1 Resolution		-				+
SP Trial 2 Resolution	x				+	
SP Trial 3 Resolution	x				+	
SP Total Resolution		-				+
SP Trial 1 Raw Score	x				-	
SP Trial 2 Raw Score	x				-	
SP Trial 3 Raw Score	x			x		
SP Raw Total	x			x		
Study Time Difference		+		x		

Note. STAI = State Trait Anxiety Inventory; GDS = Geriatric Depression Scale; MFQ = Memory Functioning Questionnaire; FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory; CP = Computer-Paced Condition; SP = Self-Paced Condition; JOL = Judgment of Learning. No skewness or kurtosis was characterized by skewness or

kurtosis statistics between $-.499$ and $.499$. Moderate skewness or kurtosis describes variable distributions with skewness and kurtosis statistics between $-.999$ to $-.500$ and $.500$ to $.999$. Substantial skewness or kurtosis describes variable distributions with skewness or kurtosis statistics less than -1.00 and greater than 1.00 .

Table 3

Group mean differences on FAD based on family history of dementia or caregiving history

	<u>Family History +</u> <u>M(SD)</u>	<u>Family History -</u> <u>M(SD)</u>	<u>t</u>	<u>p</u>	<u>Caregiving +</u> <u>M(SD)</u>	<u>Caregiving -</u> <u>M(SD)</u>	<u>t</u>	<u>p</u>
FADS	.142 (1.12)	-0.130 (0.837)	-1.291	.201	0.147 (1.232)	-0.048 (0.902)	-0.801	.425
ADI	.066 (1.02)	-0.061 (0.963)	-0.606	.546	0.172 (1.141)	-0.059 (0.933)	-0.966	.337

Note. FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory.

Table 4

Correlations between FAD, subjective memory complaints, and negative affect (n = 88)

		MFQ Total	STAI - State	STAI-Trait	GDS
FADS	<i>r</i>	-.444	.169	.154	.214
	<i>p</i>	1.20E-5**	.019*	.031*	2.82E-3**
ADI	<i>r</i>	-.326	.209	.248	.287
	<i>p</i>	1.69E-3**	3.61E-2*	5.31E-4**	6.2E-5**

Note. * = $p < .05$, ** = $p < 2.50 \text{ E-}2$ (subjective memory complaints Bonferroni-corrected significance level) or $p < 6.25 \text{ E-}3$ (negative affect Bonferroni corrected significance level); FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory; MFQ = Memory Functioning Questionnaire; STAI = State Trait Anxiety Inventory; GDS = Geriatric Depression Scale.

Table 5

Correlations between FAD versus JOL Estimates and JOL Resolution on Computer-Paced List-Learning Trials (n = 88)

		T1			T2		T3		Overall
		Pre-estimate	Estimate	Resolution	Estimate	Resolution	Estimate	Resolution	Resolution
FADS	<i>r</i>	-.093	-.002	.034	.085	-.068	.142	-.047	-.026
	<i>p</i>	.382	.985	.752	.423	.524	.183	.660	.811
ADI	<i>r</i>	-.062	-.008	.095	.070	.002	.120	.003	.042
	<i>p</i>	.564	.942	.374	.512	.982	.262	.980	.696

Note. * = $p < .05$, ** = $p < 1.56E-3$ (Bonferroni-corrected significance level); FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory; T1 = Trial 1; T2 = Trial 2; T3 = Trial 3.

Table 6

Correlations between FAD versus JOL Estimates and JOL Resolution on Computer-Paced List-Learning Trials (n = 88)

		<u>T1</u>			<u>T2</u>		<u>T3</u>		<u>Overall</u>
		Pre-estimate	Estimate	Resolution	Estimate	Resolution	Estimate	Resolution	Resolution
FADS	<i>r</i>	.128	.030	.057	.013	-.057	.055	-.175	-.065
	<i>p</i>	.228	.780	.597	.907	.595	.609	.100	.543
ADI	<i>r</i>	.110	.029	.090	.007	.068	.021	-.064	.038
	<i>p</i>	.304	.787	.398	.949	.527	.843	.549	.725

Note. * = $p < .05$, ** = $p < 1.56E-3$ (Bonferroni-corrected significance level); FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory; T1 = Trial 1; T2 = Trial 2; T3 = Trial 3.

Table 7

% Variance Accounted for by the Six Components of the ADI

Component	Content	Eigenvalue	% Variance	Cumulative % Variance
1	Negative Affect, AD-specific worry	10.128	20.255	20.255
2	Physical Symptoms of FAD	4.549	9.099	29.354
3	Belief in Efficacy of AD Treatments	3.628	7.256	36.610
4	Belief in Personal Vulnerability to AD	3.142	6.284	42.894
5	Self-Efficacy in Reducing Risk for AD	2.489	4.977	47.871
6	Resilience to FAD	2.304	4.607	52.478

Table 8

Component Loadings of ADI Items

Item	Loadings						
	C1	C2	C3	C4	C5	C6	C7
43. I worry that I will be a burden on my family if I develop AD.	.906						
45. I worry about the financial burden of developing AD.	.880						
44. I worry about feeling physically unwell if I develop AD.	.876						
42. I worry that I will not be able to make decisions independently if I develop AD.	.870						
48. I worry about having to move out of my home if I develop AD.	.848						
47. I worry about losing important relationships in my life if I develop AD.	.810						
41. I worry about losing my memory for loved ones if I develop AD.	.805						
49. I will not be able to contribute to society if I develop AD.	.689						
46. I worry about how others will judge me if I develop AD.	.672						
<i>17. If I have a family member with AD, I will likely develop it too.</i>	<i>.403</i>						
37. When I think about AD, my hands shake.		.929					
38. I lose sleep worrying about AD.		.904					
36. When I think about AD, my stomach is in knots or I feel nauseated.		.800					
32. Thinking about getting AD makes me feel hopeless/makes me want to give up.		.566					
18. There are no treatments to slow or stop the progression of AD.			.836				
22. There are treatments that can slow or stop the progression of AD.			.771				
9. If I am diagnosed with AD, there is nothing I can do to improve my outcomes.			.762				
4. If I am diagnosed with AD, there is nothing doctors will be able to do to improve my outcomes.			.721				
11. There is nothing I can do to slow the progression of AD.			.696				
23. I believe that I am already showing signs of AD.				.823			
26. I believe that I already have AD.				.807			
30. I do believe that I am showing early symptoms of AD.				.765			
28. I believe that I am going to develop AD.				.509			
<i>20. I do not believe that I will develop AD.</i>				<i>.497</i>		<i>.469</i>	
13. I can reduce my chances of developing AD by staying physically active.					.873		
14. I can reduce my chances of developing AD by using my brain in new ways, like doing crosswords, Sudoku, or reading.					.790		
12. I can reduce my chances of developing AD by eating a healthier diet.					.721		
15. I can reduce my chances of developing AD by spending time with my family and friends.					.552		
<i>7. I am in control of my health as I age.</i>					<i>.491</i>		

Table 8, Continued

Component Loadings of ADI Items

Item	Loadings						
	C1	C2	C3	C4	C5	C6	C7
21. Thinking about getting AD does not make me worry.						.736	
27. I feel calm when thinking about AD.						.733	
50. <i>AD would not really be that bad.</i>						-.476	
25. If I had AD and there was a treatment to SLOW it, I would take it.							.929
24. If I had AD and there was a treatment to STOP it, I would take it.							.900
34. <i>Thinking about getting AD makes me feel angry</i>	.419						
40. <i>Just because I have a family member with AD does not mean I will get it too.</i>							
35. <i>Thinking about getting AD makes me feel sad.</i>	.469						
31. <i>Thinking about getting AD makes me feel anxious</i>						.432	
29. AD is inherited from parents							
1. Some forgetfulness is normal for people my age and older.							
6. AD is a common problem among older adults.							
16. AD is relatively rare.							
2. It is normal to occasionally forget things like where I placed my keys and when I have scheduled appointments.							
8. I can make changes in my lifestyle that will help me live a longer, healthier life.							
19. Whether I will develop AD will depend on my lifestyle as well as whether a family member passed it down to me.							
3. Most older people with memory problems have AD.							
39. I find it difficult to concentrate because I am distracted by thoughts of developing AD.							
5. Forgetting dates and appointments or where I put things might mean I have AD.							
33. If I had AD and there was treatment to slow or stop it, I would NOT take it.							
10. There is nothing I can do to slow the aging process or improve my health as I age.							

Table 8, Continued

Component Loadings of ADI Items

Item	Loadings						
	C8	C9	C10	C11	C12	C13	C14
43. I worry that I will be a burden on my family if I develop AD.							
45. I worry about the financial burden of developing AD.							
44. I worry about feeling physically unwell if I develop AD.							
42. I worry that I will not be able to make decisions independently if I develop AD.							
48. I worry about having to move out of my home if I develop AD.							
47. I worry about losing important relationships in my life if I develop AD.							
41. I worry about losing my memory for loved ones if I develop AD.							
49. I will not be able to contribute to society if I develop AD.							
46. I worry about how others will judge me if I develop AD.							
<i>17. If I have a family member with AD, I will likely develop it too.</i>							
37. When I think about AD, my hands shake.							
38. I lose sleep worrying about AD.							
36. When I think about AD, my stomach is in knots or I feel nauseated.							
32. Thinking about getting AD makes me feel hopeless/makes me want to give up.							
18. There are no treatments to slow or stop the progression of AD.							
22. There are treatments that can slow or stop the progression of AD.							
9. If I am diagnosed with AD, there is nothing I can do to improve my outcomes.							
4. If I am diagnosed with AD, there is nothing doctors will be able to do to improve my outcomes.							
11. There is nothing I can do to slow the progression of AD.							
23. I believe that I am already showing signs of AD.							
26. I believe that I already have AD.							
30. I do believe that I am showing early symptoms of AD.							
28. I believe that I am going to develop AD.							
20. I do not believe that I will develop AD.							
13. I can reduce my chances of developing AD by staying physically active.							
14. I can reduce my chances of developing AD by using my brain in new ways, like doing crosswords, Sudoku, or reading.							
12. I can reduce my chances of developing AD by eating a healthier diet.							
<i>15. I can reduce my chances of developing AD by spending time with my family and friends.</i>							
<i>7. I am in control of my health as I age.</i>							
					.405		
							.424

Table 8, Continued

Component Loadings of ADI Items

Item	Loadings						
	C8	C9	C10	C11	C12	C13	C14
21. Thinking about getting AD does not make me worry.							
27. I feel calm when thinking about AD.							
50. AD would not really be that bad.							-.439
25. If I had AD and there was a treatment to SLOW it, I would take it.							
24. If I had AD and there was a treatment to STOP it, I would take it.							
34. Thinking about getting AD makes me feel angry	.669						
40. Just because I have a family member with AD does not mean I will get it too.	.644						
35. Thinking about getting AD makes me feel sad.	.535						
31. Thinking about getting AD makes me feel anxious	.467						
29. AD is inherited from parents		.760					
1. Some forgetfulness is normal for people my age and older.		-.747					
6. AD is a common problem among older adults.			.715				
16. AD is relatively rare.			.705				
2. It is normal to occasionally forget things like where I placed my keys and when I have scheduled appointments.				.725			
8. I can make changes in my lifestyle that will help me live a longer, healthier life.				.607			
19. Whether I will develop AD will depend on my lifestyle as well as whether a family member passed it down to me.				.533			
3. Most older people with memory problems have AD.					.833		
39. I find it difficult to concentrate because I am distracted by thoughts of developing AD.					.526		
5. Forgetting dates and appointments or where I put things might mean I have AD.					.447		
33. If I had AD and there was treatment to slow or stop it, I would NOT take it.						.711	
10. There is nothing I can do to slow the aging process or improve my health as I age.							.739

Table 9

Correlations between metacognitive variables and objective memory functioning (n = 88)

		Computer-Paced Memory Task				Self-Paced Memory Task			
		T1	T2	T3	Total	T1	T2	T3	Total
MFQ	<i>r</i>	.097	.058	.001	.052	-.081	-.064	.018	-.039
	<i>p</i>	.356	.584	.994	.623	.445	.544	.863	.712
FADS	<i>r</i>	.049	.019	.074	.057	.055	-.019	-.052	-.016
	<i>p</i>	.649	.862	.489	.595	.608	.857	.628	.878
ADI	<i>r</i>	.134	.081	.052	.101	.126	.084	-.010	.064
	<i>p</i>	.209	.447	.630	.342	.238	.431	.922	.547
Study Time Difference	<i>r</i>	-.091	-.088	-.084	-.096	.379	.283	.276	.345
	<i>p</i>	.391	.409	.429	.365	2.08E-4**	6.51E-3*	8.14E-3*	8.23E-4**
CP JOL Total	<i>r</i>	.273	.407	.320	.396	.182	.286	.270	.261
	<i>p</i>	8.44E-3*	5.80E-5**	1.85E-3*	9.20E-5**	.083	5.70E-3*	9.37E-3*	.012*
SP JOL Total	<i>r</i>	.324	.492	.472	.509	.436	.530	.504	.526
	<i>p</i>	1.65E-3*	6.22E-7**	2.00E-6**	2.26E-7**	1.40E-5**	5.51E-8**	2.99E-7**	7.41E-8**

Note. * = significant at $p < .05$; ** = significant at $p < 1.04 \text{ E-}3$ (Bonferroni-corrected significance level); MFQ = Memory Functioning Questionnaire; FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory; CP = Computer-Paced Condition; SP = Self-Paced Condition; JOL = Judgment of Learning; T1 = Trial 1; T2 = Trial 2; T3 = Trial 3.

Table 10

Unique variance in objective memory functioning accounted for by metacognitive variables

Predictor	Computer-Paced Objective Memory Functioning				Self-Paced Objective Memory Functioning			
	<i>t</i>	<i>p</i>	<i>r_{sp}</i>	<i>r_{sp}²</i>	<i>t</i>	<i>p</i>	<i>r_{sp}</i>	<i>r_{sp}²</i>
				<i>R² = .315</i>				<i>R² = .383</i>
MFQ	0.510	.611	.047	.002209	-0.382	.703	-.034	.001156
FADS	-0.291	.772	-.027	-.000729	-1.112	.269	-.098	.009604
ADI	1.170	.245	.108	.011664	0.795	.429	.070	.0049
CP Total JOL Estimate	-0.251	.802	-.023	-.000529	-1.953	.054	-.171	.029241
SP Total JOL Estimate	3.825	2.58E-4*	.354	.125316	5.000	3.00E-6*	.439	.192721
Study Time Difference	-2.256	2.68E-2*	-.209	.043681	2.389	1.93E-2*	.210	.0441

Note. * = significant at $p < .05$; ; MFQ = Memory Functioning Questionnaire; FADS = Fear of Alzheimer's Disease Scale; ADI = Anticipatory Dementia Inventory; CP = Computer-Paced Condition; SP = Self-Paced Condition; JOL = Judgment of Learning

Table 11

Correlations between Measures of Metacognitive Monitoring and Metacognitive Control (n = 88)

		Computer-Paced Task				Overall	Self-Paced Task				
		Pre-Task JOL	JOL 1	JOL 2	JOL 3		Pre-Task JOL	JOL 1	JOL 2	JOL 3	Overall JOL
Study Time Difference (Computer- vs. Self-Paced Task)	<i>r</i>	0.039	0.12	.165	-.061	.074	.168	.198	.205	.248	.226
	<i>p</i>	0.712	0.257	.118	.563	.483	.110	.059	.051	.018*	.031*

Note. * = significant at $p < .05$; ** = significant at $p < .005$ (Bonferroni-corrected significance value); JOL = judgment of learning.

APPENDIX A

Anticipatory Dementia Inventory (ADI) - Page 1

Below is a list of statements that middle- and older-aged adults might say about Alzheimer's disease and how it might affect one's life. Please read each statement carefully, and then put an 'X' in the box that best represents how much you agree or disagreement with the statement right now, today.

0 = Strongly Disagree 1 = Somewhat Disagree 2 = Neutral /Neither Agree nor Disagree 3 = Somewhat Agree 4 = Strongly Agree

		0	1	2	3	4
1	Some forgetfulness is normal for people my age and older.					
2	It is normal to occasionally forget things like where I placed my keys or when I have scheduled appointments.					
3	Most older people with memory problems has Alzheimer's disease.					
4	If I am diagnosed with Alzheimer's disease, there is nothing doctors will be able to do to improve my outcomes.					
5	Forgetting dates and appointments or where I put things might mean I have Alzheimer's disease.					
6	Alzheimer's disease is a common problem among older adults.					
7	I am in control of my health as I age.					
8	I can make changes in my lifestyle that will help me live a longer, healthier life.					
9	If I am diagnosed with Alzheimer's disease, there is nothing I can do to improve my outcomes.					
10	There is nothing I can do to slow the aging process or improve my health as I age.					
11	There is nothing I can do to slow the progression of Alzheimer's disease.					
12	I can reduce my chances of developing Alzheimer's disease by eating a healthier diet.					
13	I can reduce my chances of developing Alzheimer's disease by staying physically active.					
14	I can reduce my chances of developing Alzheimer's disease by using my brain in new ways, like doing crosswords, Sudoku, or reading.					

APPENDIX A CONTINUED

Anticipatory Dementia Inventory (ADI) – Page 2

0 = Strongly Disagree 1 = Somewhat Disagree 2 = Neutral /Neither Agree nor Disagree 3 = Somewhat Agree 4 = Strongly Agree

15	I can reduce my chances of developing Alzheimer's disease by spending time with my family and friends.					
16	Alzheimer's disease is relatively rare.					
17	If I have a family member with Alzheimer's disease, I will likely develop it too.					
18	There are no treatments to slow or stop the progression of Alzheimer's disease.					
19	Whether I will develop Alzheimer's disease will depend on my lifestyle as well as whether a family member passed it down to me.					
20	I do not believe that I will develop Alzheimer's disease.					
21	Thinking about getting Alzheimer's disease does not make me worry.					
22	There are treatments that can slow or stop the progression of Alzheimer's disease.					
23	I believe that I am already showing signs of Alzheimer's disease.					
24	If I had Alzheimer's disease and there was a treatment to STOP it, I would take it.					
25	If I had Alzheimer's disease and there was a treatment to SLOW it, I would take it.					
26	I believe that I already have Alzheimer's disease.					
27	I feel calm when thinking about Alzheimer's disease.					
28	I believe that I am going to develop Alzheimer's disease.					
29	Alzheimer's disease is inherited from parents.					
30	I do believe that I am showing early symptoms of Alzheimer's disease.					
31	Thinking about getting Alzheimer's disease makes me feel anxious.					
32	Thinking about getting Alzheimer's disease makes me feel hopeless/makes me want to give up.					

APPENDIX A CONTINUED

Anticipatory Dementia Inventory (ADI) - Page 3.

0 = Strongly Disagree 1 = Somewhat Disagree 2 = Neutral /Neither Agree nor Disagree 3 = Somewhat Agree 4 = Strongly Agree

		0	1	2	3	4
33	If I had Alzheimer's disease and there was a treatment to slow or stop it, I would NOT take it.					
34	Thinking about getting Alzheimer's disease makes me feel angry.					
35	Thinking about getting Alzheimer's disease makes me feel sad.					
36	When I think about Alzheimer's disease, my stomach is in knots/I feel nauseated.					
37	When I think about Alzheimer's disease, my hands shake.					
38	I lose sleep worrying about Alzheimer's disease.					
39	I find it difficult to concentrate because I am distracted by thoughts of developing Alzheimer's disease.					
40	Just because I have a family member with Alzheimer's disease does not mean I will get it too.					
41	I worry about losing my memory for loved ones if I develop Alzheimer's disease.					
42	I worry that I will not be able to make decisions independently if I develop Alzheimer's disease.					
43	I worry that I will be a burden on my family if I develop Alzheimer's disease.					
44	I worry about feeling physically unwell if I develop Alzheimer's disease.					
45	I worry about the financial burden of developing Alzheimer's disease.					
46	I worry about how others will judge me if I develop Alzheimer's disease.					
47	I worry about losing important relationships in my life if I develop Alzheimer's disease.					
48	I worry about having to move out of my home if I develop Alzheimer's disease.					
49	I will not be able to contribute to society if I develop Alzheimer's disease.					
50	Alzheimer's disease would not really be that bad.					

APPENDIX B**Word Lists for List-Learning Task (Woodard, 1991)****Computer-Paced Word List**

reflex

flag

salad

month

idea

plain

winter

ocean

tank

flower

boulder

insect

dust

honor

garden

death

air

heaven

jail

inn

Self-Paced Word List

hint

impulse

link

toast

mood

queen

pepper

silence

odor

limb

iron

market

woods

hall

clock

rock

anger

bar

dirt

lord

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ABSTRACT**FEAR OF ALZHEIMER'S DISEASE AND ITS ROLE IN METACOGNITIVE MONITORING AND CONTROL**

by

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Introduction: Fear of Alzheimer's disease (FAD), or Anticipatory Dementia, is a healthy adult's misinterpretation of everyday memory failures as indicators of developing dementia. The current study investigated the construct of FAD and aimed to contextualize FAD within the Health Belief Model through development of a new scale, the Anticipatory Dementia Index (ADI). The study also assessed the relationship between FAD and metacognitive monitoring and metacognitive control.

Methods: 94 cognitively-intact community-dwelling older adults with and without a history of family history of AD completed questionnaires regarding their subjective memory complaints, state and trait anxiety, depression, and multiple measures of FAD, including the ADI. Participants also completed a list-learning task in which they were required to provide Judgment of Learning estimates of their recall after each trial (metacognitive monitoring) and to adjust their study time based on their perceived performance (metacognitive control).

Results: There were no differences in FAD based on family history of AD or caregiving. FAD was significantly associated with subjective memory complaints, trait-level negative affect, and depression. FAD was not associated with metacognitive monitoring or accuracy of

monitoring, metacognitive control, or objective memory functioning. Metacognitive monitoring and increased study time were associated with memory performance. Finally, the ADI is comprised of six components that map FAD onto the Health Belief Model.

Conclusions: FAD appears to be more affective than cognitive. Interventions for FAD should increase public knowledge of prevalence and risk for AD, and increase self-efficacy and motivation for health and lifestyle changes to reduce AD risk.

AUTOBIOGRAPHICAL STATEMENT

Annalise Rahman is originally from Surrey, England, but completed her undergraduate degrees in Biology, Psychology, and Neuroscience at the College of Charleston in Charleston, SC. During her undergraduate career, she studied social psychological models of health behaviors as well as neuropsychological assessment and treatment of traumatic brain injury. She moved to Detroit, MI in 2009 to pursue a Ph.D. in Clinical Psychology at Wayne State University.

Annalise's program of research as a graduate student has focused on physical health and lifestyle factors that predict differences in cognitive trajectories from mid- to late-life. Since 2009, she has worked with the consortium of researchers from the Georgia Centenarian Study to examine psychological and cognitive functioning in the oldest old population – centenarians. She has also been involved in grant-funded research on functional neuroimaging of episodic and semantic memory in aging, mild cognitive impairment, and Alzheimer's disease in Dr. John Woodard's research laboratory. She was also involved in data collection and analysis for a DOD-funded project to investigate the clinical utility of neuroimaging and neuropsychological assessment of mild traumatic brain injury.

Annalise's clinical experience includes practica at the Detroit Medical Center, Wayne State University Psychology Clinic, and the DMC Rehabilitation Institute of Michigan. She is currently completing her pre-doctoral internship in Clinical Psychology at the Ann Arbor Veterans Affairs Health Systems/University of Michigan consortium. Annalise will be completing her Neuropsychology Postdoctoral Fellowship in traumatic brain injury and geriatric neuropsychology at the Ann Arbor Veterans Affairs Health Systems/University of Michigan consortium from 2016-2018.