COURSE OF DISEASE PROGRESSION AND TIME TO DEATH

AMONG DIFFERENT DEMENTIA TYPES IN A

POPULATION BASED STUDY

by

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ABSTRACT

Course of Disease Progression and Time to Death Among Different Dementia Types in a Population-Based Study

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Risk factors related to dementia onset have been well-described for Alzheimer's disease (AD), but less is known about disease course and mortality. This project aimed to describe factors associated with dementia type [AD, vascular dementia (VaD), or mixed presentations (AD-VaD, AD-Other, Non-AD dementia)], and identify modifiable and non-modifiable factors that influence disease course and mortality. Age, sex, family history, genetics (e.g. Apolipoprotein E (APOE)), and health conditions were examined. This project used extant data from the Cache County Study on Memory and Aging (CCSMA) and the ancillary study, the Cache County Dementia Progression Study (DPS).

Males were more likely to be diagnosed with AD-Other and Other dementia, compared to females. Cardiovascular conditions were more common in those with VaD, and poorer health was associated with an increased risk of VaD and mixed dementia types (AD-Other) or non-AD dementias. APOE ɛ4 was associated with lower cognitive scores while higher education and higher health ratings were associated with better cognitive and functional outcomes. Female sex and non-AD dementia were associated with lower MMSE scores. Males experienced a more rapid decline, five years post-diagnosis. Females with AD-Other dementia declined more rapidly on the MMSE for the first 7 years post-diagnosis. Those with AD-Other exhibited more rapid functional decline compared to those with AD. Males with AD-Other dementia or non-AD dementia alone experienced a more rapid rate of functional decline compared to females five or more years after diagnosis. Those with AD-Other, AD-VaD, and Other dementia, experienced poorer ADL functioning than those with AD alone. Later dementia onset age was associated with lower BMI on average. Shorter survival was found for AD-Other, VaD and Other dementia, compared to AD. Lower BMI was associated with higher risk of death.

This study identified distinguishing characteristics among dementia diagnoses, and risk factors related to disease course and mortality. The study is unique in the amount of follow-up time acquired among a population-based sample and included forms of dementia beyond AD and VaD. This study emphasizes early intervention and provides important information to healthcare providers and families, as they anticipate patients' unique disease course and beneficial interventions.

(188 pages)

PUBLIC ABSTRACT

Course of Disease Progression and Time to Death Among Different Dementia Types in a Population Based Study

Kaitlyn E. Kauzor

Alzheimer's disease (AD) is the most common type of dementia, Other types of dementia include vascular (VaD) and mixed forms, like AD with VaD, other mixed forms of dementia or dementia of unknown etiology (DUE). Research has focused on factors that may increase risk of developing dementia, with less understood about disease course. Factors associated with risk for developing dementia onset include age, female sex, family history of dementia, genetics (e.g. having the ɛ4 allele of the Apolipoprotein E or APOE gene), and various health conditions. This project used previously collected data from the Cache County Study on Memory and Aging (CCSMA) and the ancillary study, the Cache County Dementia Progression Study (DPS).

This study found that among different dementia types, there were modifiable and non-modifiable risk factors associated with type. General health status was among the most important factors associated dementia type and with severity of cognitive and functional impairment. Specific health conditions associated with increased risk for VaD included cardiovascular and cerebrovascular issues. Among males, diagnoses of AD with Other dementia or non-AD dementia alone, showed more rapid rate of functional decline, compared to females. Functionally, those diagnosed with VaD, AD with another form of dementia, or non-AD dementia alone, experienced worse functioning over time. Those with AD with another form of dementia, AD with VaD, and those with Other dementia, experienced poorer overall functioning in ADLs than those with AD alone. Lower BMI was associated with a higher risk for mortality, while those with higher BMI displayed longer survival. Poor health status and low BMI were strong predictors of mortality. Being diagnosed with AD with Other dementia, and non-AD dementia alone, followed by VaD alone had the greatest morality risk compared to having AD alone.

These results offer greater information on factors that influence dementia type and course, while providing possible avenues for intervention (e.g. managing specific health conditions). The identification of factors that influence mortality may inform healthcare providers' of duration of dementia and mortality.

DEDICATION

To my steadfast parents, John and Annetta, my witty brother, Brian, my loving grandparents, and dear Carter – For your unwavering support during this long journey, I will be forever grateful. I love and thank you all.

Kaitlyn E. Kauzor

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CHAPTER I:

GENERAL INTRODUCTION

Dementia prevalence is increasing as people live longer. The World Health Organization reported that 55 million people world-wide had dementia as of 2021, and that number is predicted to increase to 78 million by the year 2030 (World Health Organization, 2022). Alzheimer's disease (AD), the most common type of dementia, accounts for around 60-80% of all cases of dementia (Alzheimer's Association, 2019). Vascular and Diffuse Lewy Body (DLB) dementias are the next most common, accounting for 20% and 4-16% of dementia cases, respectively (Rizzi et al., 2014; Dementia Association, 2022). With each type of dementia comes a specific set of symptoms and characteristics, many of which present unique challenges as the disease progresses. This is not only relevant for the patient, but importantly, for their families, caregivers, and healthcare providers. While research has identified some patterns of decline among these dementias, several factors influence the course of disease progression. The more that is understood about what factors influence dementia progression, the more informed health care providers, caregivers, and families can be to provide the best care possible.

There are several factors typically examined when considering one's risk for developing dementia, but less studied are the factors that specifically impact the course of decline as the condition progresses. Factors that have been linked to a more rapid disease progression include age of onset, sex, and level of education. Specifically, studies have reported a more rapid decline associated with early onset (prior to age 65) of AD and in those with lower levels of education (Buccione, 2007; Contador et al., 2016; Holland et al., 2012). However, cognitive or neural reserve also suggests that higher levels of education may initially delay the identification of dementia, but ultimately result in more rapid decline after onset (Stern, 2012). Females have also been reported as experiencing a more rapid decline than males in those with dementia (Anstey et al., 2021), and specifically in AD (Henderson & Buckwalter, 1994; Ferretti et al., 2018).

In addition to these demographic factors, clinical features such as neuropsychiatric symptoms (NPS) have been associated with disease progression and severity of decline, though the literature is inconsistent. One study found a higher endorsement of NPS to be linked to more severe cognitive decline (Vik-Mo et al., 2018), though others have reported varying associations of NPS with cognitive decline, depending on dementia type (Cerejeira et al., 2012). Severity of dementia at baseline is thought to be an influencing factor in NPS expression, and overall, AD has been associated with lower levels of NPS compared to other dementia types (Brodaty et al., 2015). Other studies have even indicated shortened survival rates in individuals experiencing significant mood disturbances or elevated NPS (Choi et al., 2016; Huang et al., 2020).

Family history of dementia and genetic factors are often examined in risk of AD, but generally have not been examined in the context of other forms of dementia and their progression. One study that aimed to examine family history as a factor in AD progression found no association (Ferrari et al., 2018). With respect to the AD risk gene of Apolipoprotein $\varepsilon 4$ (APOE $\varepsilon 4$), there is mixed literature on its role in disease progression. Several studies have reported a more rapid decline in AD for those who are APOE $\varepsilon 4$ carriers (Albrecht et al., 2015; Rawle et al., 2018; Wilson et al., 2002), whereas other studies reported no such association (Ferrari et al., 2017; Foster et al., 2013; Tschanz, 2011).

While many factors discussed above are non-modifiable, there are also factors thought to influence decline that are considered modifiable. Health and medical conditions including diabetes, malnourishment, obesity, and vascular disease have not only been linked to risk for dementia onset (Kalaria, 2010; Kloppenborg et al., 2008; Knopman et al., 2018, Whitmer et al., 2005) but have been shown to influence disease progression (Sanders et al., 2018; Tschanz et al., 2013; Wengreen et al., 2009). Individuals with AD with poorer overall quality of health also tend to have poorer cognitive and functional outcomes (Leoutsakos et al., 2012).

Considering all of the above factors is challenging and often complicated by barriers in collecting accurate data. Determining onset of dementia is not always straight forward, though studies aim to identify onset ages as accurately as possible. Recognized cases of dementia at a given time (prevalent cases) while useful, present difficulties in the estimation of onset ages due to errors in recall and the lack of recognition of subtle changes in symptom onset. Additionally, the course of decline in dementia is not necessarily linear, and thus the rate of cognitive decline, for example, may vary depending on where along the course of dementia the various assessments have occurred. Finally, many studies have examined clinical populations, and while convenient, results from these studies may not generalize to the broader population of persons with dementia. Given the lack of information on the course of dementia (particularly with respect to non-AD forms), and the role of various factors on progression, this study considered a variety of dementia types and the role of various non-modifiable and modifiable factors on dementia course. In examining disease course, which has not been thoroughly examined across various forms of dementia, this study aimed to characterize duration of dementia (survival time) for various dementia types as well as factors that influence it. Importantly, this study also identified and classified by type, these characteristics in an older adult population with cases of dementia identified early in their disease course.

CHAPTER II

COURSE OF DISEASE PROGRESSION AND TIME TO DEATH AMONG DIFFERENT DEMENTIA TYPES IN A POPULATION BASED STUDY

Literature Review

Alzheimer's disease (AD) is the most common cause of dementia in late-life, accounting for 60-80% of all cases of dementia. In the United States alone, as of 2024, approximately 6.9 million people are living with AD (Alzheimer's Association, 2024. The next most common cause of dementia is Vascular Dementia (VaD), followed by Diffuse Lewy Body Dementia (DLDB), accounting for roughly 20% and 4-16% of cases of dementia, respectively (Rizzi et al., 2014; Lewy Body Dementia Association, 2022). Patterns of symptom onset, progression, and duration of dementia vary by the underlying cause of the condition. AD is most often characterized by initial difficulties related to short-term memory (Braak, H. & Braak E., 1988), followed by impairment in language (namely word-finding), executive functioning and long-term memory (Mucke et al., 2009; Perry et al., 1999), which coincide with impairments in independent activities of daily living (IADLs). VaD results from cerebrovascular disease, resulting in reduced circulation of oxygen to the brain (Khan et al., 2016). Executive function impairment is often the first prominent symptom of VaD, commonly exhibited by difficulty managing finances or occupational activities, as well as by increased confusion and forgetfulness (Smith et al., 2017). In contrast to AD and VaD, DLBD is characterized by early expression of neuropsychiatric symptoms (NPS) and motor symptoms, with progressive

deterioration of memory, and fluctuating cognitive status over time (Delenclos et al., 2017; Larsson et al., 2018; Matar et al., 2019). More recently, diagnostic criteria were modified to include rapid eye movement (REM) sleep behavior disorder (Delenclos et al., 2017; Matar et al., 2019) as a core feature. The occurrence of NPS is not a defining feature of DLBD as they are common in most forms of dementia (Vik Mo et al., 2018).

In addition to differences in the onset of symptoms by dementia type, the overall course differs as well. The course of AD from onset to death ranges from 3.4- 5.7 years, as reported by previous studies (Barclay et al., 1985; Mölsä et al., 1986; Tschanz et al., 2004). One study reported median survival times in AD by age groups as prior to 75 years of age, 75 to 84 years of age and older than 85 years of age, with survival being 6, 5 and 3.5 years, respectively (Brookmeyer et al., 2002). Updated statistics report by the Alzheimer's Association in 2024 report that at 70 years of age, those with AD are twice as likely to die prior to the age of 80, compared to those without AD (Alzheimer's Association, 2024). Some studies have indicated shorter survival times in DLBD than in AD, by a reported range of 2 to 3.5 years (Price et al., 2017; Magierski et al., 2010, Olichney et al., 1998), while others have reported no significant differences (Walker et al., 2000). VaD is often associated with shorter survival time (around 4 years postdiagnosis) than AD, possibly due to challenges in managing the effects of cerebrovascular disease (Eizaguirre et al., 2017; Fitzpatrick et al., 2005). However, because individuals are often diagnosed years after disease onset, it is difficult to ascertain accurate survival times, which may contribute to variable results. Other factors such as medical comorbidities and age of dementia onset may also affect survival duration (Tschanz et al., 2004).

While risk factors for the development of AD and related disorders (ADRD) have been the topic of numerous studies (de Oliveira, 2014; Lindsay, 2002; Locke, 2009; Tschanz, 2013), less studied are factors that affect the rate of dementia progression (e.g., cognitive and functional decline as a function of time). Additionally, much of the information regarding decline among dementia types varies or has not been extensively characterized. Even within a single form of dementia such as AD, the rate of dementia progression can vary widely (Tschanz et al, 2011). Younger onset age (prior to age 65) has been linked to a more rapid disease progression in dementia (Buccione, 2007; Holland et al., 2012; Karen et al., 2019; Mungas, 2001; et al., 2009; Wattmo & Wallin, 2017), as well as lower levels of education (Contador et al., 2016). Contador and colleagues (2016) found a more rapid rate of both cognitive and functional decline in individuals with lower levels of education. However, Stern (2012) suggests that under the cognitive reserve theory, those with higher education may experience later onset of AD, but a more severe/rapid decline. Sex differences have been described in numerous studies, with many reporting a more rapid cognitive decline in women than men (Anstey et al., 2021; Henderson & Buckwalter, 1994; Ferretti et al., 2018; Koran et al., 2017; Lin et al., 2015). Hippocampal and gray matter volume as well as general brain atrophy have also been shown to be more significant in females compared to males, in those with AD (Apostolova et al., 2006; Koran et al., 2017; Skup, 2011), though one study reported no difference between the sexes when measuring overall brain volume in individuals with AD (Edland et al., 2002). Ardekani et al. (2016) also reported more rapid progression of hippocampal atrophy in females with AD as compared to males with AD.

Family history of dementia and genetic factors have been linked to the development of AD (Donix et al., 2012; Kauwe et al., 2009; Locke et al., 2009; Paulson & Igo, 2011; Scarabino et al., 2016), but few studies have examined their role in dementia progression. Ferrari et al. (2017) published one of the few studies to examine family history of dementia as a factor of dementia progression. However, in a sample of 81 individuals with possible AD (as determined by presence of an AD biomarker), followed for a minimum of 2 years, they found no significant associations between a positive family history of dementia and rate of cognitive decline. Notably, family history of dementia and rate of cognitive or functional decline has been virtually unexamined in other forms of dementia.

With respect to genetic factors, the ɛ4 allele of the apolipoprotein (APOE ɛ4) gene is well established in the risk for AD (Albrecht et al., 2015; Launer et al., 1999; Lindsay et al., 2002; Liu et al., 2013; Rawle et al., 2018; Roses et al., 1993; Wilson et al., 2002). Many studies have also found a more rapid decline in AD among carriers of the ɛ4 allele (Albrecht et al., 2015; Rawle et al., 2018; Wilson et al., 2002), though several studies have reported no significant impact of this gene (Ferrari et al., 2017; Foster et al., 2013; Tschanz et al., 2011) on dementia progression.

Clinical features such as NPS are associated with progression rates. Vik-Mo and colleagues (2018) demonstrated an association between higher endorsement of NPS in those with more severe cognitive decline. However, these findings are not consistent across the literature or among various forms of dementia (see review by Cerejeira et al., 2012). Brodaty and colleagues (2015) showed that baseline dementia severity was related to severity of NPS, as well as dementia type, with frontotemporal dementia associated

with greater overall NPS, and AD associated with the lower levels of NPS compared to all other dementias. Other studies have supported similar findings, for example, Zahodne et al. (2015) found an association between higher levels of baseline psychosis and worse cognitive status, in those with mild AD. This study also reported more rapid decline in those with higher levels of psychosis and depression, at baseline. Mood symptoms (anxiety, apathy and dysphoria) have been associated with increased mortality in those with AD (Huang et al., 2020), and findings from Choi et al. (2016) suggest an increased risk for mortality among dementia patients with increased severity of NPS. Thus, greater severity of NPS may be an indicator of overall severity of dementia.

While health or nutritional factors as a risk for dementia onset have been well described (Kalaria, 2010; Kloppenborg et al., 2008; Gottesman, 2018; Tschanz et al., 2013; Wengreen 2009; Whitmer, 2005), it is less clear the degree to which these factors impact rate of decline after dementia onset. Poorer nutritional status has been associated with more rapid cognitive decline and functional impairment (Sanders et al., 2016) as well as shortened time to severe dementia and death in all-cause dementia (Sanders et al., 2018). Leoutsakos et al. (2012) reported that persons with AD in poor-to-fair health compared to good or excellent health exhibited poorer cognitive and functional outcomes. Studies have also indicated that weight loss in AD is associated with increased mortality (Soysal et al., 2021), and Albanese et al. (2013) reported an association between increased dementia severity and more significant weight loss. Garcia-Ptacek et al. (2014) reported that having a body mass index (BMI) that was considered typical or overweight, was associated with reduced mortality, in those with dementia. There is also evidence to suggest that acetylcholinesterase inhibitors, commonly prescribed to slow the progression of cognitive symptoms in dementia, are also associated with weight loss (Franx et al., 2019). While several studies have described the association between BMI and various outcomes in dementia overall and specifically AD, there is a dearth of information characterizing BMI across the disease course of other dementias. Examining BMI among different dementia types may help identify groups that are particularly vulnerable to frailty. Further clarifying relationship between BMI and mortality would also serve providers when predicting survival duration or imminent death.

A common challenge of examining disease course is to identify and diagnose individuals as close to their onset of dementia as possible. Many studies conducted to date have used samples of convenience, such as those available in clinical settings, the results from such sources may not generalize to the broader population. Additionally, reported rates of progression among different dementia types are inconsistent and not well characterized. While prevalent (extant) cases are informative, their use to model or describe the overall course of dementia poses a challenge. This is because it is often difficult to accurately identify onset age, so challenges exist in accurately modeling disease course in its entirety. As reviewed above, several factors such as sex and genetic factors have been examined, especially in AD, but few studies have examined other factors such as family history of dementia and health and nutritional factors on the course of dementia, particularly in non-AD forms of dementia.

The purpose of this project was to examine the characteristics and clinical course of various forms of dementia, in a population-based sample. Specific influences of family history on dementia progression (cognitive and functional decline), and survival duration were examined. Sex differences, medical comorbidities, and APOE ɛ4 genotype were also examined across the different forms of dementia. The following diagnoses were included in this study: AD, VaD, AD with another form of dementia, and Other dementia. The specific research questions of project are outlined below:

- Research question 1 examined factors that best differentiated dementia diagnoses from each other. Factors examined included: sex, age of onset, APOE ε4 status, vascular risk factors and other health conditions, NPS, BMI, first cognitive symptom affected, presence of motor symptoms, and maternal or paternal family history of dementia.
- Research question 2 described the clinical course of dementia in incident (new onset) cases, comparing rates of cognitive and functional decline among dementia groups.
- Research question 3 examined the physical aspects of dementia, examining the trajectory of BMI and overall survival duration in incident (new onset) cases from age of dementia diagnosis for BMI and age of onset to death for survival duration, by dementia type.

This project provided valuable and novel information to researchers and clinicians who wish to better understand and predict patterns of decline in people with dementia using factors that have yet to be considered collectively. The added factors of family history, APOE ɛ4 status and others, provided practical information regarding potential influences on the clinical course of dementia for patients, families, and their caregivers.

Methods

Participants

The current study used extant data from the Cache County Memory Study in Aging (reference Breitner et al., 1999) and the ancillary study, the Cache County Dementia Progression Study. Participant demographics and recruitment have been previously described (Breitner et al., 1999; Tschanz et al., 2013). In brief, 5,092 permanent residents of Cache County, Utah, aged 65 and older, were enrolled in this study, which consisted of four waves of dementia screening and ascertainment from approximately 1995 – 2007. Dementia screening progressed from administration of a brief cognitive screening test, the Modified Mini-Mental State Exam (3MS), and if screened positive (scores below the 25th percentile; Breitner et al., 1999; Miech et al., 2002), to a clinical interview with a knowledgeable informant of the subject's cognitive and functional abilities, as well as their engagement in daily living activities using the Dementia Questionnaire (DQ). Participants rated with dementia or significant cognitive impairment on the DQ or were members of a subsample randomly selected to complete all stages of dementia screening and assessment, were then invited to complete a clinical assessment (CA; Breitner et al., 1999; Miech et al., 2002). The CA consisted of neuropsychological assessment and neurological and physical exam with the participant as well as a clinical and medical health interview with an informant. The CA data were reviewed at an initial diagnostic case conference that included a study neuropsychologist, study physician and the CA team. Dementia diagnoses were assigned using Diagnostic and Statistical Manual, 3rd ed., revised (DSM-III-R) criteria. For those with a working diagnosis of dementia or prodromal AD, participants were invited to complete a

physician visit, laboratory studies, and neuroimaging as well as an 18-month follow-up CA. Final clinical diagnoses were determined by a consensus panel consisting of a boardcertified neurologist, geriatric psychiatrist, neuropsychologists, and a cognitive neuroscientist (Breitner et al., 1999). Specific dementia types were based on current research criteria at the time. For example, AD diagnosis was based on the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA, McKhann et al., 1984) criteria, and vascular dementia was based on the International Workshop of the National Institute of Neurological Disorders and Stroke (NINDS) and the Association Internationale pour la Recherche et l'Enseignement en Neurosciences (AIREN) (NINDS-AIREN; Erkinjunitti, 1994) criteria. Individuals whose dementia presentation was not consistent with a recognized form of dementia were designated as "Dementia, Undetermined Etiology." Age of dementia onset was assigned as the age at which the participant met DSM-III-R criteria for dementia, based on a chronological review of symptoms provided by the informant at the CA. Dementia severity at the diagnosis visit was also determined using the Clinical Dementia Rating (CDR) scale (Morris et al., 1997—see Breitner et al., 1999 for procedures). The participant's first cognitive symptom reported was also noted.

The subsequent waves followed a similar protocol, except that the cut scores on the 3MS differed in Wave 2 (Miech et al., 2002), and the DQ stage was eliminated in Waves 3 and 4. As a result of the four waves of dementia ascertainment, a total of 942 persons with dementia were identified. Based on the estimated onset ages, 359 were identified as prevalent cases in Wave 1 and 583 as incident cases at subsequent waves. The following diagnoses types were included in this study: Alzheimer's Disease (AD), Vascular dementia (VaD), other dementia excluding AD and VaD (Other), AD and VaD (AD-VaD), and AD with another non-vascular form of dementia (AD-Other). The "Other" dementia type category was comprised of several diagnoses such as diffuse Lewy Body dementia (DLBD), frontotemporal dementia (FTD), and Pick's disease, though highest category was dementia of undetermined etiology (DUE). Table 1 displays a summary of the diagnoses within the "Other" dementia group, for the full sample of participants as well as by prevalent or incident dementia in Wave 1. See Appendix A for full compilation of diagnoses included in the Other dementia group, displayed by research question.

Table 1

	All Participants	Prevalent Cases	Incident Cases
	n=173/931 (18.6%)	n = 57/349 (16.3%)	n = 116/582 (19.9%)
	(10.070)	(10.370)	(19.970)
Dementia of an undetermined etiology (DUE)	132 (14.2%)	43 (12.3%)	89 (15.3%)
Related to Parkinson's disease	18 (1.9%)	2 (0.6%)) 16 (2.7%)
Possible or probable dementia with Lewy Bodies	10 (1.1%)	4 (1.1%)) 6 (1%)
Related to Frontotemporal dementia (FTD)	7 (0.8%)	4 (1.1%)) 3 (0.5%)
Hypoperfusion	3 (0.3%)	2 (0.6%)) 1 (0.2%)
Amyotrophic sclerosis (ALS)	1 (0.1%)	1 (0.3%)) 0 (0%)
Normal Pressure Hydrocephalus (NPH)	1 (0.1%)	1 (0.3%)) 0 (0%)
Progressive Supranuclear Palsy (PSP)	1 (0.1%)	0 (0%)) 1 (0.2%)

Diagnoses Within the Other Dementia Type for Full Sample and Separated by Prevalence or Incident Dementia Status at Wave 1

Procedures

Surviving individuals identified as incident cases (new dementia onsets after enrollment visit into the CCSMA) were enrolled into the Dementia Progression Study (DPS) which began in 2002. The procedures of the DPS included semi-annual clinical assessments similar to that of the CCSMA CA, with a neuropsychological test battery, an abbreviated physical/neurological exam with the participant as well as a clinical health interview about the participant with a caregiver. Vital status was monitored from obituaries or information obtained from caregivers.

Measures of Dementia Progression and Course

Cognition: Mini-Mental State Exam (MMSE)

The Mini-Mental State Exam (MMSE) was used as a brief measure of global cognitive functioning (Folstein et al., 1975). The MMSE has been determined to be both valid and a reliable indication of cognitive status in older adults with cognitive impairments, with internal reliability being reported as ranging from α =.54-.81, according to several studies (Albert and Cohen, 1992; Jorm et al., 1988; Kay et al., 1985 & Tombaugh et al., 1996). For the purposes of this study, the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) version was used. This measure assesses orientation, recall, attention, language, and visuospatial skills, with a maximum score of 30. Higher scores are indicative of better cognitive performance. Sensory-motor adjusted scores were calculated by discarding items confounded by sensory or motor impairment and recalculating the score as the percentage correct of remaining items, multiplied by

30. If the participant was unable to comprehend instructions for the MMSE due to cognitive impairment, a score of "0" was applied. Scores ranged from 0 to a maximum of 30 points.

Function: Dementia Severity Rating Scale (DSRS)

The Dementia Severity Rating Scale [DSRS; (Clark, 1996)] was completed by the caregiver at the CA and each DPS visit. Aspects of functioning that are captured and maximum points with this measure include: memory (7 points), orientation (5 points), judgment (4 points), social interaction (4 points), home activities/responsibilities (4 points), personal care (4 points), speech/language (6 points), recognition of others (5 points), feeding (4 points), incontinence (4 points), and ambulation (6 points). Higher scores are indicative of more severe impairment (Clark, 1996). Total DSRS score was used as an indication of participants' overall functioning, and domain scores were used to indicate progression in specific cognitive and functional domains. These domain scores were created by collapsing individual item ratings as follows: ADLs (personal care, feeding, incontinence); cognition (memory, orientation, judgment, recognition of others and speech); social activity (social interactions, home activities/responsibilities). See Appendix B for DSRS and DSRS domain scores. The range of points possible for the DSRS total and domain scores were as follows: DSRS total = 0.54; ADL domain = 0.10, Cognition domain = 0-29; and Social Activity domain = 0-9.

Physical Change and Survival Duration

Height and weight were collected at the CCMS CA and each DPS visit, allowing for a calculation of body mass index (BMI; $BMI = \frac{weight (kg)}{height (m)2}$) (Nutall, 2015). BMI has historically been utilized as a common way to identify individuals as having body types of "normal" (18.5-24.9), "overweight" (25-29.9) and "obese" (30+) (World Health Organization, 1997; National Institutes of Health, 1998). Calculated BMI was used as an outcome over the course of dementia.

Participant deaths and ages at death were determined through published obituaries, reports from the Utah Department of Health, or through caregivers. Survival duration was calculated in years from the age of dementia onset to death.

Predictors: Demographic, Health, and Family History Information; Body Mass Index and APOE Genotype

Demographic information was collected from participants in the Wave 1 of the CCSMA. These factors, including age, education level, sex, and history of medical conditions, were obtained at the Wave 1 CCSMA enrollment interview with the participant and updated by the participant before dementia diagnosis at each subsequent CCSMA wave. After dementia diagnosis, health information was updated by the participant's caregiver at each DPS visit. The medical conditions obtained from the visits were coded and data entered. For this project, neurological, cardiovascular, and endocrine conditions were examined as predictors in each of the above categories. Due to the low frequency of endorsement of several medical conditions, individual conditions were grouped based on similar body systems and type of condition to create medical condition groups (e.g. conditions of the cardiovascular system). For predictors of sufficient sample size (for example, hypertension), presence or absence of the individual predictor was used instead of the category. Thus, the following groups of conditions were created for analyses: conditions of the brain, types of arrythmias and related conditions -rel-major cardiac conditions, minor cardiac conditions, congestive heart failure, hypertension, hyperlipidemia, cancers, depression, other psychiatric disorders, autoimmune conditions, chronic pain, gastric/digestive, pulmonary, urologic, thyroid, diabetes, and blood deficiencies. The decision to create the groups as described was based on a few considerations such as sample size (e.g. were there enough endorsements for this condition to be included in an analysis alone). For example, this was the case for the commonly occurring hypertension and hyperlipidemia, which are also known to be distinct risk factors for vascular-related dementia. Other factors which contributed to groupings included similarity/like-conditions (e.g. arrythmia NOS, arrhythmia, other atrial fibrillation grouped together) or level of severity of the medical event (e.g. arrythmia was not grouped with myocardial infarctions as the latter is a more severe and a distinct medical event). Additionally, sparsity of endorsements of specific conditions also contributed to specific conditions being grouped together, if only they were also previously linked by similar conditions (e.g. aortic aneurism, while severe, did not have the sample size to include in analyses alone, rather, it was grouped with minor cardiac conditions which also had very low N. See Appendix C for a list of all medical conditions assessed and Appendix D for specific medical conditions that were coded into categories.

Neuropsychiatric symptoms, including mood symptoms, were collected using the Neuropsychiatric Inventory (NPI; Cummings et al., 1994) (See Appendix E for full list of questions). The NPI is a 12-item inventory that assesses the occurrence and severity of NPS in dementia including: hallucinations, delusions, agitation/aggression, depression/dysphoria, anxiety, elation/euphoria, apathy/indifference, disinhibition, irritability/lability, motor disturbance, nighttime behaviors, and appetite and eating. The informant or caregiver is asked to evaluate whether the patient displays each of these symptoms (yes/no). For each "yes" item, a rating for the *severity* (mild, moderate, severe) and *frequency* of occurrence (occasionally= less than once per week, often= once per week, frequently= several times a week but not daily, very frequently= once or multiple times per day) is obtained. Overall severity scores were determined by multiplying the severity and frequency scores for each symptom. For the purposes of this study, the severity of each symptom was examined. Items of appetite and sleep disturbance were not collected at the first two waves of the CCMS, and due to the extremely low endorsements of euphoria across the sample, these items were excluded. As such, a 9item NPI was used for this study. Cluster scores were examined, as able, depending upon the distribution of symptoms by dementia group. Cluster scores include the affective cluster, comprised of a combination of depression, anxiety and irritability scores, while the psychotic cluster is a combination of hallucination and delusion scores (Steinberg et al., 2013).

Family history of dementia was determined in the interview by asking about family history of memory problems, including onset and progression of symptoms, effects on ability to live independently, and any formal diagnosis of the cause of dementia. Family history information was updated at each subsequent wave of the CCSMA (See Appendix F for Family History Section of interview). Family history of dementia was categorized as positive or negative individually for maternal or paternal relationships. Each family member had to survive to age 50 to be coded as positive or negative for a history of memory problems.

During the CA, neurological and motor symptom data were also collected. Neurological and motor symptoms were recorded as positive for each symptom and gait disturbances as evaluated by the study nurse (See Appendix G). Symptoms of interest for the current project included abnormal motor reflexes (left, right, both sides of the body in deep tendon reflexes), presence of primitive reflexes (snout, visual suck, tactile suck), gait abnormalities (walking, turning, stopping, presence of shuffling, accessory movements, tandem walk), bradykinesia, posture, motor impersistence (tongue protrusion, eye closure), weakness (shoulder elevation, pronator drift, strength differences), fluidity of speech (rapid repetition of words), upper extremity fluidity of motor movements (finger-to-nose, finger-thumb tapping, rapid alternating movements), muscle tone/rigidity (flexion/extension at elbow and wrist; cogwheel rigidity), and tremor. Presence or absence of symptom abnormalities were used as follows: left or right reflex hyperreflexia, left or right hyporeflexia, primitive reflexes, gait, bradykinesia, posture, left or right weakness, speech abnormalities, movement fluidity, muscle rigidity, and tremor. As sample size permitted, signs and symptoms were lateralized; for low sample sizes lateralization was not used, rather, symptoms were collapsed across similar features to reflect the presence or absence of impairment (e.g. left or right upper extremity weakness collapsed to upper extremity weakness). See Appendix H for a full description of the neurological symptom categories that were created from individual symptoms.

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With respect to additional predictors or covariates, a general medical health rating (GMHR) was determined at each CCSMA and DPS clinical assessment. The GMHR was assigned based on reports of the participant's health history, including any acute or unstable condition, number of medications, and if the person appeared frail (Lyketsos et al., 1999). Scores range from 1-4, with the following indications of health status: 4=excellent (maximum of 2 stable illnesses and 2 medications), 3=good (maximum 1 unstable but treated illness or maximum 4 stable illnesses and 4 medications), 2=fair (maximum 3 unstable illnesses) and 1=poor (patient is considered very ill/terminal). Fair and poor categories were collapsed when indicated by sparse numbers in the poor category. BMI as a covariate was examined according to the following categories: normal (18.5 to 24.9), overweight (25.0 to 29.9), and obese (30.0 and above) (Weir & Jan, 2019). APOE ɛ4 genotype was determined by polymerase chain reaction (Breitner et al., 1999) from DNA processed from buccal skin samples. Presence or absence of the ɛ4 allele was used in statistical analyses.

Statistical Analyses

Descriptive statistics were performed to determine baseline characteristics of the participants within each dementia type to better understand potential differences between these groups.

Research question 1 determined how participants with different types of dementia diagnoses differed cross-sectionally from each other at their diagnosis visit. The outcome variable was dementia type: AD, VaD, AD-VaD, AD-other and Other dementia. Predictor variables included sex, age of dementia onset, medical comorbidities, neurological symptoms, family history of memory problems, APOE ɛ4 status, BMI category at diagnosis, specific NPS, and first cognitive symptom. Variables included as covariates in the model were overall health status (GMHR), dementia duration, education and whether the participant was a prevalent or incident case. Due to the nominal nature of the dependent variable (5-category dementia type), multinomial logistic regression (MnLogR) was used to determine the role of the predictor in distinguishing likelihood of dementia type relative to the reference category of AD. Parameters estimated in MnLogR are interpreted similar to standard logistic regression and are exponentiated to create odds rations (OR). A sequence of models was run systematically by adding and removing each predictor (e.g., demographic factors, and neurologic and medical conditions) to determine which collection of variables best differentiated each diagnosis type from that of the reference category.

Research question 2 examined longitudinal trajectories of cognition and function for each dementia type, identifying meaningful differences in disease course. For this analysis, only incident cases of dementia were included. Outcome variables included MMSE (adjusted total score) and DSRS (domain and total scores). Predictor variables in these models included dementia type, maternal and paternal family history of memory problems, and sex. Variables included as covariates were overall health, age at dementia onset, dementia duration (years from onset age to diagnosis visit), education and APOE ɛ4 status. Due to repeated observations being gathered on participants, the analysis must account for the lack of independence amongst all observations. A Repeated measures Analysis of Variance (ANOVA) is not appropriate since the observations were not taken at the same time intervals for all participants and the number of observations per participant varies widely from 1-15. Linear mixed effects models (i.e. multilevel models, MLM) were used to explore trajectories of decline and test for effects of each predictor and covariate, while controlling for person-to-person differences (random intercepts and slopes, where applicable; Hox et al., 2017). The interaction between dementia type and sex was tested to examine sex differences in the rate of cognitive and functional progression.

Research question 3 was broken into two parts- (A) examined trajectory of BMI and (B) time to death among the different dementia diagnoses. For both analyses, only incident cases of dementia were included. To examine trajectory of BMI, calculated BMI was modeled over time, similar to the dependent variables in question 2. Predictor variables included dementia type, GMHR status, sex and age, and education as a covariate. MLM was used to explore trajectory of BMI and test for effects of each predictor and covariate, run separately by sex. For mortality, the dependent variable was time to death and/or right censoring (RC), in years along with an indicator to distinguish death from RC end points. Survival analyses with Cox proportional hazards regression was used to examine time to death. Because not all participants experienced death during the observation window or their death date was not able to be collected, time of RC was used as the last date of monitoring death data. An alternative, statistical modelling approach such as logistic regression was not used since most participants did have death dates, and the time span from baseline to death varied greatly. Additionally, restricting analysis of time-to-death amongst just those who died would also be less desirable as RC participants would need to be excluded. Thus, for the Cox regression, the predictor variables were dementia type, maternal and paternal family history of memory problems,

and sex. Other predictor variables included as covariates were GMHR at diagnosis, BMI category at diagnosis, dementia duration, education, and APOE ɛ4 status. Interactions included dementia type by sex and dementia type by maternal or paternal family history. Similar to logistic regression, parameter estimates for Cox regression are computed on the logistic scale and must be exponentiated to create hazard ratios (HR) for interpretation.

All statistical analyses were run using R statistical software (R Core Team, 2015).

Results

Sample sizes differed based on eligibility criteria for each research question and missing data; as such, descriptive data are presented by research question individually.

Research question 1: Participant Demographics

Out of 931 eligible participants with dementia, 10 participants were excluded due to missing data on education, APOE ε 4 genotype, and/or baseline health status, leaving a sample of 921. This sample was composed of 566 (61.5%) female participants, had a mean age of dementia onset at 81.99 years (SD = 7.06), and a mean education of 12.9 years (SD = 2.72) (see Table 2). Excluded participants had a longer mean duration of dementia pre-baseline (about 1.67 years longer; p = .054). Excluded and included participants did not significantly differ on age at diagnosis. Table 2 summarizes descriptive statistics for the final sample.

Participant Characteristics at Diagnosis, Prevalent and Incident Dementia Cases, n = 921

Variable	М	SD	n	%
Dementia onset age	81.99	7.06		
Dementia duration at first visit (years)	2.98	2.72		
Education (years)	12.91	2.72		
Sex				
Male			355	38.5
Female			566	61.5
APOE e4				
No ε4			482	52.3
1 or more ε4			439	47.7
Dementia type				
AD			530	57.5
AD-Other			27	2.9
AD-VaD			72	7.8
VaD			122	13.2
Other			170	18.5
General Medical Health Rating (GMHR)				
Total	1.97	1.14		
Poor			11	1.2
Fair			299	32.5
Good			516	56.0
Excellent			95	10.3
Living environment at first visit				
Home/Own residence			678	73.6
Residential/Assisted Living			78	8.5
Skilled Nursing Facility			165	17.9
MMSE total score	18.63	7.95		
NPI total score	6.42	8.88		

Notes. AD= Alzheimer's disease, AD-other = AD with another form for dementia, VaD = vascular dementia. MMSE = mini-mental state exam; NPI = neuropsychiatric inventory; APOE = apolipoprotein E; Higher score on GMHR rating total indicates more positive health rating. Prevalent cases refer to cases identified in Wave 1 of the study while incident cases refer to onset after Wave 1.

Research question 2: Participant demographics

Out of 576 eligible participants with dementia, 70 participants were excluded due to missing data on education, APOE ε 4 genotype, and/or baseline health status, leaving a sample of 506. This final sample was composed of 306 (60.5%) female participants, had a mean age of dementia onset at 83.09 (SD = 6.12), years, and a mean education of 13.4 (SD = 3.03) years. On average, excluded participants were older (M = 86.49 vs. 83.09), and had about one year less of education (M = 12.29 vs. 13.40). Excluded and included participants did not significantly differ on dementia duration. Table 3 provides a full summary of the incident dementia cases (ascertained after Wave 1), comparing the subset that was analyzed to the subset that was excluded from analysis due to missing data.

Included			Excluded						
		(n=50)6)			(n=7	70)		
Variable	Μ	SD	n	%	Μ	SD	n	%	р
Dementia onset age	83.09	6.12			86.49	6.79			<.001***
Dementia duration	1.72	1.24			1.68	1.48			.786
at first visit (years)									
Education (years)	13.40	3.03			12.29	2.93			.004**
Sex									.982
Male			200	39.5			27	38.6	
Female			306	60.5			43	61.4	
APOE ε4									.155
No ε4			283	55.9			46	65.7	
1 or more ε4			223	44.1			24	34.3	
Dementia Type			_						.235
AD			294	58.1			39	55.7	.200
AD-Other			17	3.4			0	0	
AD-VaD			40	7.9			3	4.3	
VaD			58	11.5			12	17.1	
Other			97	19.2			16	22.9	
General Medical Heal	lth		2,						
Rating (GMHR)									
Total	2.63	.64			2.49	.70			.075
Categories									.087
Poor	7	1.4					2	2.9	
Fair	209	41.3					38	54.3	
Good	253	50					24	34.3	
Excellent	37	7.3					6	8.6	
Living environment a	at first								.308
visit									
Home/Own Resi	idence		408	80.6			51	72.9	
Residential/Assi	sted		49	9.7			9	12.9	
Living									
Skilled Nursing	Facility		49	9.7			10	14.3	
MMSE total score	21.47	5.64			19.78	7.12			.204
DSRS total score	3.73	1.88			3.58	2.02			.598

Comparison of Participant Characteristics of Incident Dementia Participants by Inclusion Status for Question 2, n = 576

Note. AD= Alzheimer's disease, AD-other = AD with another form for dementia, VaD = vascular dementia, AD-VaD= AD with VaD. MMSE = mini-mental state exam; DSRS = dementia rating scale; APOE = apolipoprotein E; Higher score on GMHR rating total indicates more positive health rating. Incident cases refer to onset after Wave 1 of the study.

Research Question 3: Participant Demographics, Total Sample and by Sex

Parts A and B of this research question utilized the same sample of incident dementia cases and as such, demographic characteristics displayed in Tables 4 and 5 are representative of both studies. A full summary of participants is presented in Table 4 and Table 5 comparing males to females. Table 6 displays quartiles of survival times by dementia type. A total of 582 incident dementia case participants were included in these analyses. This overall sample was composed of 351 (60.3%) female participants, had a mean age of dementia onset at 83.49 (SD = 6.29) years, a mean education of 13.26 (SD = 3.04) years, and a mean survival of 3.95 years (SD = 3.07, median = 3.20). Compared to males, on average, females were older at dementia onset (est = 1.48, p = .006, d = 0.24) and had a longer dementia duration at the time of their first visit (est = .38, p < .001, d = 0.30). Females also had fewer years of education (est = 0.38, p < .001, d = 0.44), and on average were more likely to be either underweight or obese at diagnosis visit, than males ($\chi^2 = 33.15$, p < .001, V = 0.25). Males and females did not differ significantly on length of survival from diagnosis visit.

Variable	М	SD	n	%
Dementia onset age	83.49	6.29		
Dementia duration at first visit	1.74	1.29		
(years)				
Survival (years)	3.95	3.07		
Education (years)	13.26	3.04		
Sex				
Male			231	39.7
Female			351	60.3
APOE ε4				
No ε4			330	56.7
1 or more ε4			248	42.6
Dementia type				
AD			335	57.6
AD-other			17	2.9
AD-VaD			43	7.4
VaD			71	12.2
Other			116	19.9
BMI at Diagnosis visit				
Underweight			33	5.7
Normal			244	41.9
Overweight			172	29.6
Obese			76	13.1
General Medical Health Rating				
(GMHR)				
Total	2.61	0.65		
Poor			9	1.5
Fair			249	42.8
Good			280	48.1
Excellent			43	7.4

Participant Characteristics for Incident Dementia Cases for Question 3, n = 582

Notes. AD= Alzheimer's disease, AD-other = AD with another form of dementia, VaD = vascular dementia, AD-VaD = AD with VaD. Higher score on GMHR rating total indicates more positive health rating.

		Mal				Fema			
Variable	М	(n=2) SD	31) n	%	M	$\frac{(n=3)}{SD}$	/	%	
Dementia onset	82.60	6.40	п	70	84.08	6.16	n	70	<u>۲</u> **006.
	82.00	0.40			84.08	0.10			.000.
age Dementia	1.51	1.23			1.89	1.31			<.001***
duration at first	1.51	1.23			1.89	1.51			<.001
visit (years) Survival (years)	3.80	3.05			4.04	3.09			0.35
Education (years)	5.80 14.06	3.03 3.51			4.04	3.09 2.57			0.55
APOE ε4	14.00	5.51			12.74	2.37			.763
No ε4			133	57.6			197	56.1	.705
l or more $\varepsilon 4$			155 96	37.0 41.6			152	43.3	
			90	41.0			132	45.5	.003**
Dementia type AD			115	49.8			220	62.7	.005
AD-other			115	49.8			6	1.7	
AD-VaD			11	4.0 6.1			29	8.3	
VaD VaD			35	15.2			29 36	8.3 10.3	
Other			55 56	24.2			50 60	10.3	
			30	24.2			00	1/.1	<.001***
BMI at Diagnosis			7	3			26	7.4	<.001
Underweight Normal			88	38.1			156	7.4 44.4	
							72	44.4 20.5	
Overweight Obese			100 22	43.3 9.5			72 54	20.3 15.4	
	1.1		22	9.5			54	15.4	
General Medical He Rating (GMHR)	eann								
Total	2.65	0.61			2.59	0.67			.255
	2.05	0.01			2.39	0.07			.233
Categories Poor			9	1.5			6	1.7	.110
Fair			9 249	42.8			161	45.9	
Good			249 280	42.8 48.1			151	43.9 44.2	
Excellent			280 43	48.1 7.4			155 29	44.2 8.3	
Excenent			43	/.4			29	0.3	

Comparison of Participant Characteristics for Incident Dementia Cases by Sex for Research Question 3, n = 582

Notes. AD= Alzheimer's disease, AD-other = AD with another form of dementia, VaD = vascular dementia, AD-VaD = AD with VaD. BMI = Body Mass Index. APOE = apolipoprotein E. Higher score on GMHR rating total indicates more positive health rating. Incident cases refer to onset after Wave 1 of the study. * p < .05. ** p < .01. *** p < .001.

Dementia Type	n	М	(SD)	[Min.	Max]	Q1	Median	Q3
AD	335	4.52	(3.26)	[0.00,	16.96]	1.84	3.83	6.31
AD-other	17	3.45	(2.38)	[0.20,	9.21]	1.67	3.18	4.31
AD-VaD	43	3.69	(2.74)	[0.18,	11.48]	1.60	3.15	5.78
VaD	71	3.27	(2.54)	[-0.24,	13.72]	1.20	2.93	4.44
Other	116	2.86	(2.62)	[0.00,	15.75]	1.09	2.13	3.88

Summary of Survival Time for all Incident Dementia Cases by Dementia Type, n = 582

Note. Survival time is years spanned from first observation after dementia onset until death or right censoring (i.e. end of observation window). $Q1 = 25^{th}$ percentile, $Q3 = 75^{th}$ percentile. Incident cases refer to onset after Wave 1 of the study.

Research Question 1: Characterization of dementia diagnosis by risk factors

The results of multinomial logistic regression models estimated risk for specific dementia diagnoses, compared to the reference group of persons with AD (See Table 7). Among demographic predictors, higher onset age was associated with decreased risk of VaD (OR=0.93, 95% CI [0.89, 0.97]) or Other dementias (OR=0.97, 95% CI [0.93, 1.00]), as was being an APOE ε 4 carrier (OR=0.44, 95% CI [0.26, 0.73]; OR=0.56, 95% CI [0.36, 0.85], respectively). Dementia duration at first visit was also associated with decreased risk of Other dementia (OR=.90, 95% CI [0.82, 0.99]). Female sex was associated with decreased odds of AD-Other dementia (OR=0.25, 95% CI [0.09, 0.67]) and Other dementia (OR=0.63, 95% CI [0.41, .96]) diagnoses.

Overall health status was also a significant predictor of dementia type. Having a poor to fair health rating (reference category = good) was associated with increased risk of VaD (OR= 2.64, 95% CI [1.58, 4.42]) and Other dementia (OR= 1.83, 95% CI [1.19,

2.81]). Having a health rating of excellent was associated with a decreased risk for a diagnosis of Other dementia (*OR*=0.22, 95% CI [0.08, 0.64]).

Among neurological signs, chin or shoulder weakness was associated with increased risk for AD-VaD (OR = 2.11, 95% CI [1.03, 4.30]) and VaD (OR = 2.73, 95% CI [1.46, 5.11]). Similarly, the presence of lateralized signs was also associated with greater risk of AD-VaD (OR = 4.80, 95% CI [2.54, 9.06]) and VaD (OR = 11.62, 95% CI [6.53, 20.66]); worse quality of speech was associated with greater risk of VaD (OR = 2.32, 95% CI [1.35, 3.98]).

Specific health conditions were significantly associated with specific dementia types. Having a major cardiac condition was associated with increased risk for AD-VaD (OR= 3.27, 95% CI [1.84, 5.80]) and VaD (OR= 2.39, 95% CI [1.41, 4.09]). Hypertension, hyperlipidemia and diabetes were not significant predictors of dementia type (p > .05). A history of a neurological brain condition or Parkinson's disease (PD) was associated with increased risk for Other dementia (OR= 2.16, 95% CI [1.33, 3.51]; OR= 23.09, 95% CI [6.64, 80.31], respectively). PD was also associated with increased risk of having a diagnosis of AD-Other (OR= 95.40, 95% CI [18.78, 484.45]). Regarding psychiatric symptoms, the presence of hallucinations was associated with an increased risk of AD-Other dementia (OR = 3.34, CI [1.07, 10.40]) and Other dementia (OR = 2.10, CI [1.20, 4.09]).

Figures 1 and 2 display the probability of specific dementia diagnoses based on significant non-modifiable and modifiable health-related risk factors.

Adjusted Odds Ratios with 95% Confidence Intervals from the Multinomial Logistic Regression Model for Each Dementia Type Compared to Alzheimer's Disease

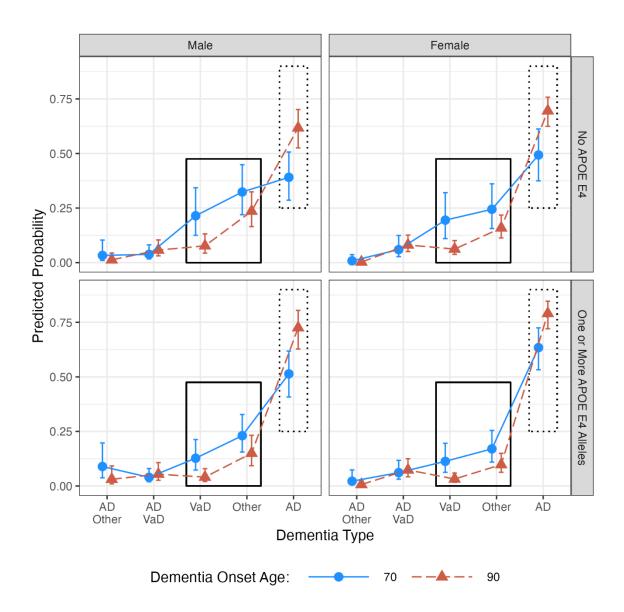
Variable	AD Other		VaD (without AD)	Other domentia
	AD-Other	AD-VaD	VaD (without AD)	Other dementia
(Intercept)	0.02 [0.00, 0.06]***	0.04 [0.02, 0.08]***	0.06 [0.03, 0.11]***	0.31 [0.18, 0.51]***
Dementia Onset Age	0.94 [0.87, 1.01]	1.00 [0.95, 1.04]	0.93 [0.89, 0.97]***	0.97 [0.93, 1.00]*
Dementia Duration at First Visit	0.97 [0.81, 1.17]	1.03 [0.92, 1.15]	0.96 [0.86, 1.07]	0.90 [0.82, 0.99]*
Sex, Female	0.26 [0.10, 0.69]**	1.22 [0.66, 2.23]	0.71 [0.42, 1.21]	0.62 [0.40, 0.96]*
APOE ε 4, at least one copy	2.11 [0.81, 5.50]	0.81 [0.46, 1.40]	0.44 [0.26, 0.73]**	0.56 [0.36, 0.85]**
General Medical Health Rating (GM	IHR, ref = Good			
Excellent	0.53 [0.13, 2.06]	0.78 [0.28, 2.18]	0.27 [0.07, 1.04]	0.22 [0.08, 0.64]**
Poor/Fair	0.51 [0.16, 1.64]	1.69 [0.95, 3.00]	2.64 [1.58, 4.42]***	1.83 [1.19, 2.81]**
Neurological Signs (ref = Normal/2	Absent, vs present/abnormal -or	NA = unavailable/unknown)	-	-
Chin/shoulder weakness	1.14 [0.28, 4.71]	2.11 [1.03, 4.30]*	2.73 [1.46, 5.11]**	1.66 [0.91, 3.03]
NA	4.38[0.30, 64.31]	0.39 [0.05, 2.86]	2.15 [0.34, 13.48]	1.32 [0.37, 4.70]
Abnormal ideational praxis	3.32 [1.16, 9.46]*	0.91 [0.51, 1.64]	0.85 [0.50, 1.45]	0.87 [0.56, 1.36]
NA	0.59 [0.04, 8.39]	1.45 [0.39, 5.44]	0.67 [0.16, 2.76]	1.47 [0.54, 4.01]
Lateralizing signs	2.27 [0.68, 7.56]	4.80 [2.54, 9.06]***	11.62 [6.53, 20.66]***	1.73 [0.99, 3.04]
NA	1.84 [0.36, 9.37]	2.14 [0.83, 5.51]	8.86 [4.15, 18.95]***	1.44 [0.61, 3.38]
Vertical gaze	0.25 [0.02, 3.60]	1.56 [0.64, 3.83]	0.60 [0.24, 1.53]	1.47 [0.67, 3.21]
U NA	0.30 [0.04, 2.05]	1.03 [0.40, 2.64]	0.41 [0.16, 1.06]	1.37 [0.70, 2.70]
Speech qualities	1.31 [0.48, 3.50]	1.15 [0.63, 2.13]	2.32 [1.35, 3.98]**	1.07 [0.69, 1.67]
NA	8.92 [0.79, 101.05]	3.68 [0.52, 25.54]	4.30 [0.72, 25.58]	0.37 [0.08, 1.73]
Psychiatric Symptoms	L / J		L / J	L / J
Hallucinations	3.34 [1.07, 10.40]*	0.71 [0.24, 2.16]	1.39 [0.57, 3.20]	2.21 [1.20, 4.09] *
NA	1.65 [0.15, 17.96]	1.62 [0.43, 5.98]	1.36 [0.39, 4.85]	3.30 [1.21, 8.99]*
Medical Conditions, endorsed vs.				· (·) · · · · j
Conditions of the brain	2.37 [0.85, 6.60]	1.20 [0.61, 2.39]	1.47 [0.82, 2.66]	2.16 [1.33, 3.51]**
Major cardiac conditions	0.80 [0.28, 2.32]	3.27 [1.84, 5.80]***	2.39 [1.41, 4.09]**	1.40 [0.87, 2.23]
Cardiac conditions, other	3.44 [0.60, 19.64]	3.52 [0.99, 12.53]	3.17 [0.93, 10.79]	1.13 [0.31, 4.16]
Parkinson's disease	95.40 [18.78, 484.45]***	1.95 [0.19, 20.46]	1.62 [0.28, 9.40]	23.09 [6.64, 80.31]***

Notes. AD= Alzheimer's disease, AD-other = AD with another form of dementia, VaD = vascular dementia, AD-VaD = AD with VaD. APOE =

Apolipoprotein E. Dementia onset age is centered at 82 years, the grand mean. CI = 95% confidence interval.

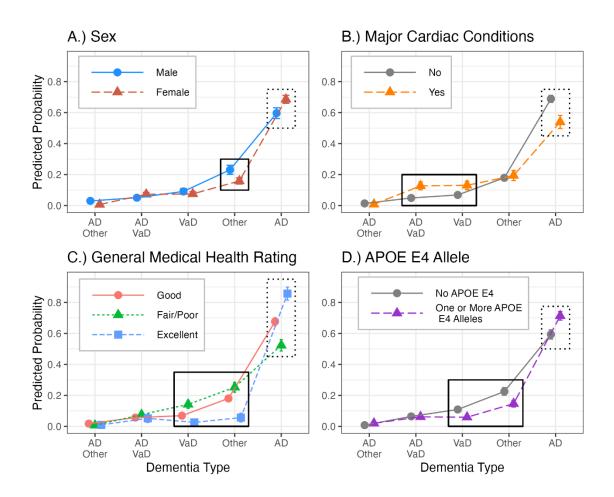
* p < .05. ** p < .01. *** p < .001.

Multinomial Logistic Regression Based Predicted Probabilities for Dementia Type Highlighting Discrepancies Due to Dementia Onset Age, Controlling for Sex and presence of APOE ε 4 allele



Note. AD = Alzheimer's Disease, VaD = Vascular Dementia, Other = Dementia other than Alzheimer's Disease and Vascular Dementia. APOE = Apolipoprotein E. Error bars display 95% confidence intervals for each predicted probability. The dementia onset age was modeled continuously, and two values (70 and 90) were selected for illustrative purposes only. Rectangles are displayed to highlight effects of interest.

Multinomial Logistic Regression Based Predicted Probabilities for Dementia Type Highlighting Discrepancies Due to Sex, Cardiac Condition, General Health and APOE ε4



Note. AD = Alzheimer's Disease, VaD = Vascular Dementia, Other = Dementia other than Alzheimer's Disease and Vascular Dementia. APOE = Apolipoprotein E. Error bars display 95% confidence intervals for each predicted probability. The dementia onset age was modeled continuously. Rectangles are displayed to highlight effects of interest.

Research Question 2: Cognitive and Functional Trajectories Over Time MMSE Total Score

In examining the effects of modifiable and non-modifiable risk factors on cognitive decline, several factors emerged with significant main effects on the MMSE, while others predicted rate of decline on the MMSE across all dementia types.

In fully adjusted models, dementia type did not differ in overall effects, but exhibited interactions with sex as well as sex and time course (p of model with interactions < .05). Specifically, females with a diagnosis of AD-Other dementia declined more rapidly on this measure up to the first 7 years post diagnosis compared to males. However, for those with Other dementia diagnosis, males experienced a more rapid decline after five years post diagnosis despite females having lower initial scores. This can be observed in Figure 3. No other interactions of diagnosis and sex were significant on trajectory of MMSE scores.

Older age of dementia onset (b = -0.11, SE = 0.04, p = .011), and duration of dementia at first visit (b = -1.30, SE = 0.19, p = <.001) were both associated with lower MMSE scores across all visits. Similarly, having at least one $\varepsilon 4$ allele of APOE (b = -1.35, SE = 0.45, p = .009) was associated with a lower MMSE score. Covariates associated with higher MMSE scores included level of education (reference = less than high school; high school: b = 3.12, SE = 0.66, p = <.001, greater than high school: b =3.51, SE = 0.61, p = <001) and overall health status (ref poor; fair: b = 11.05, SE = 1.94, p = <.001; good: b = 12.39, SE = 1.96, p = <.001; excellent: b = 11.43, SE = 2.11, p =<.001). Table 8 displays fully adjusted models for cognitive outcome. DSRS Total Score In examining factors that were significantly associated with this measure of functional ability, a diagnosis of Other dementia was associated with greater overall functional impairment (b = 0.83, SE = 0.30), p = .006). When examining change in trajectory of the DSRS over time, AD with other dementia exhibited substantially more rapid rate of functional decline compared to those with AD, particularly after 2.5 years post diagnosis (see Figure 4). Smaller differences were noted between AD and VaD where initially rate of decline diverged from that of AD after 5 years. There was a significant interaction between dementia diagnosis and sex such that in AD-Other dementia and Other dementia diagnoses, males exhibited more rapid increase in functional impairment than females. Figure 5 displays DSRS over time, by diagnosis and sex.

Among the covariates, older onset age of dementia was associated with slightly lower DSRS total score (b = -0.03, SE = 0.01, p = .016), indicating less overall functional impairment. Aspects of overall health status were also significant, such that being considered to have excellent overall health (as compared to poor) was also associated with less functional impairment (b = -1.37, SE = 0.67, p = .042). Higher education was indicative of higher degree of impairment in functioning (high school: b = 0.45, SE =0.19, p = .015, greater than high school: b = .543, SE = 0.17, p = .013), compared to those with less than high school education. Table 8 displays the fully adjusted models for functional outcomes.

DSRS - ADL, Cognitive and Social Domains

The three domains created from the DSRS were also modeled to explore meaningful factors associated with performance on ADL, cognitive and social outcomes. While no predictors were significant in relation to rate of change in DSRS domains, several main effects were observed. Being diagnosed with AD-Other dementia (b = 0.42, SE = 0.14, p = .003, AD-VaD (b = 0.21, SE = 0.09, p = .026) or Other dementia (b = 0.21, SE = 0.09, p = .026) 0.25, SE = 0.07, p < .001) was associated with poorer performance on ADLs, as was dementia duration (b = 0.09, SE = 0.02, p < .001). Being a carrier of the $\varepsilon 4$ allele of APOE was associated with higher ADL performance (b = -0.10, SE = 0.05, p = .049), as was being in excellent (b = -0.87, SE = 0.28, p = .002) and good health (b = -0.70, SE = -0.26, p = .008, compared to poor health. Having a positive maternal family history of memory problems was associated with slightly worse functioning across diagnoses over time (b = 0.10, SE = 0.03, p = .004). Figure 6 displays the ADL domain over time, across all diagnoses, for those with and without a maternal family history of memory problems. Female sex was associated with poorer outcomes on all three domains (ADLs: b = 0.18, SE = 0.05, p < .001; Cognitive and Social: b = 0.04, SE = 0.02, p < .05). There were no other significant factors associated with performance cognitive and social domains.

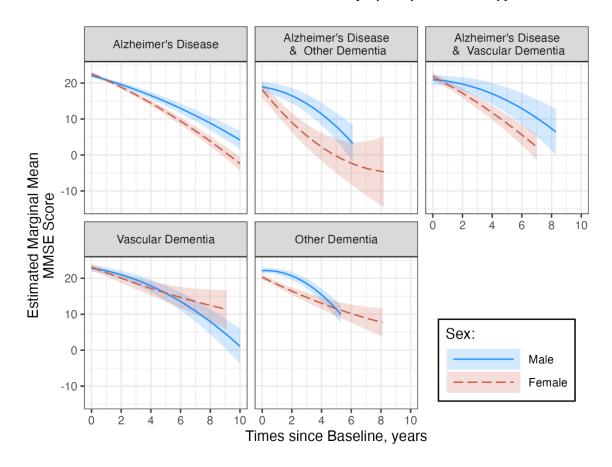
Adjusted Beta Coefficients with Standard Errors from the Multilevel Model for Cognitive and Functional Outcomes

Variable	MMSE Total Score	DSRS Total Score	ADL Domain of DSRS	Cognitive Domain of DSRS	Social Domain of DSRS
(Intercept)	10.21 (2.12) ***	3.50 (0.67) ***	1.33 (0.28) ***	2.07 (0.20) ***	1.88 (0.15) ***
Years	-1.11 (0.26) ***	0.31 (0.08) ***	0.19 (0.02) ***	0.04 (0.01) ***	0.07 (0.01) ***
Years ²	-0.07 (0.03) **	-0.03 (0.01) **	-0.01 (0.00) **	-0.000 (0.00) ***	-0.01 (0.00) ***
Dementia onset age	-0.11 (0.04) **	-0.03 (0.01) *	0.01 (0.00)	-0.000 (0.00)	-0.000 (0.00)
Dementia duration at first visit		0.07 (0.05)	0.09 (0.02) ***	0.000 (0.01)	0.000 (0.01)
Sex ($ref = male$)	0.59 (0.62)	0.33 (0.20)	0.18 (0.05) ***	0.04 (0.02) *	0.04 (0.02) *
APOE ε 4, at least one copy	-1.35 (0.45) **		-0.10 (0.05) *		
Education ($ref = less than high$			()		
Equivalent	3.12 (0.66) ***	0.45 (0.19) *	-0.05 (0.07)	0.02 (0.03)	0.05 (0.03)
More than	3.51 (0.61) ***	0.43 (0.17) *	-0.01 (0.07)	0.02 (0.03)	0.05 (0.03)
Family History of Memory Issu					
Maternal			-0.13 (0.07)		
Years x Maternal			0.10 (0.03) **		
Years ² x Maternal			-0.01 (0.00)		
General Medical Health Rating	(ref = Poor)				
Excellent	11.43 (2.11) ***	-1.37 (0.67) *	-0.87 (0.28) **	-0.16 (0.14)	-0.20 (0.15)
Good	12.39 (1.96) ***	-0.81 (0.63)	-0.70 (0.26) **	-0.11 (0.13)	-0.15 (0.15)
Fair	11.05 (1.94) ***	-0.37 (0.63)	-0.32 (0.26)	-0.08 (0.13)	-0.11 (0.15)
Dementia Type ($ref = AD$)					
AD-Other	-3.16 (1.66)	0.45 (0.55)	0.42 (0.14) **	-0.01 (0.05)	0.04 (0.06)
AD-VaD	-1.11 (1.52)	0.39 (0.47)	0.21 (0.09) *	-0.04 (0.03)	-0.02 (0.03)
VaD	0.74 (1.05)	0.19 (0.34)	0.10 (0.08)	0.01 (0.03)	0.03 (0.03)
Other	0.05 (0.90)	0.83 (0.30) **	0.25 (0.07) ***	0.03 (0.03)	0.04 (0.03)
Diagnosis by Sex Interactions					
AD-Other x Female	-1.29 (2.72)	-0.62 (0.90)			
AD-VaD x Female	0.33 (1.83)	-0.04 (0.59)			
VaD x Female	-0.26 (1.42)	0.17 (0.47)			
Other dementia x Female	-2.38 (1.19) *	-0.49 (0.40)			

Variable	MMSE Total Score	DSRS Total Score	ADL Domain of DSRS	Cognitive Domain of DSRS	Social Domain of DSRS
Time by Diagnosis and/or Sex 1	Interactions				
Years x AD-Other	0.56 (1.21)	-1.40 (0.53) **			
Years x AD-VaD	0.85 (0.97)	-0.08 (0.27)			
Years x VaD	0.50 (0.52)	-0.40 (0.16) *			
Years x Other dementia	1.32 (0.68)	-0.79 (0.24) **			
Years x Female	-0.64 (0.32) *	-0.28 (0.10) **			
Years ² x AD-Other	-0.26 (0.22)	0.31 (0.12) *			
Years ² x AD-VaD	-0.11 (0.11)	-0.02 (0.04)			
Years ² x VaD	-0.09 (0.06)	0.05 (0.02) *			
Years ² x Other dementia	-0.40 (0.13) **	0.11 (0.05) *			
Years ² x Female	-0.01 (0.03)	0.01 (0.01)			
Years x AD-Other x Female	-3.94 (1.78) *	1.98 (0.71) **			
Years x AD-VaD x Female	-1.25 (1.17)	-0.15 (0.37)			
Years x VaD x Female	-0.38 (0.82)	0.31 (0.26)			
Years x Other dementia x Female	-1.64 (0.85)	1.02 (0.32) **			
Years ² x AD-Other x Female	0.63 (0.30) *	-0.38 (0.14) **			
Years ² x AD-VaD x Female	0.09 (0.15)	0.05 (0.06)			
Years ² x VaD x Female	0.20 (0.10)	-0.03 (0.04)			
Years ² x Other dementia x Female	0.54 (0.15) ***	-0.17 (0.06) **			

Notes. The table displays parameter estimates from fully adjusted linear mixed effects models displayed by outcomes (column headings) and predictors (rows). AD= Alzheimer's disease, AD-other = AD with another form of dementia, VaD = vascular dementia, AD-VaD = AD with VaD. Dementia onset age is centered at 82 years, the grand mean. APOE = Apolipoprotein E. * p < .05. ** p < .01. *** p < .001.

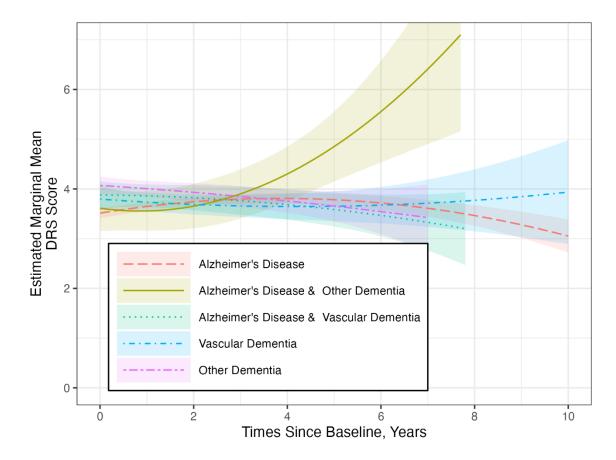
Multilevel Modeling of Estimated Marginal Means for Mini Mental State Exam (MMSE) Total Score Over Time, For Males and Females, Displayed by Dementia Type



Note. Error bands display one standard error of the mean (SEM) for each trajectory. Trajectories truncated at 99th percentile of follow-up time for each sex and dementia type. MMSE total score was modeled continuously.

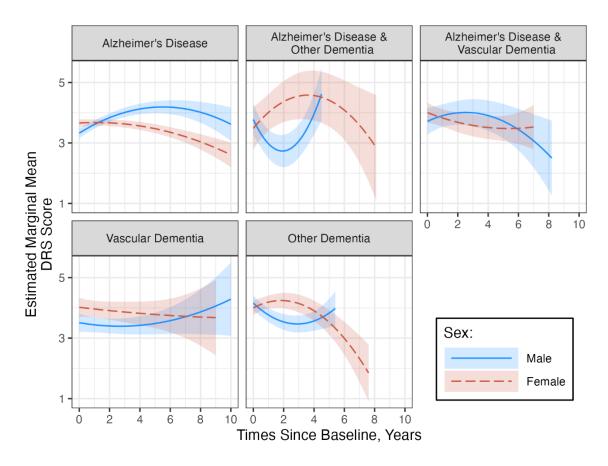
Multilevel Modeling of Estimated Marginal Means for Dementia Severity Rating Scale

(DSRS) Total Score Over Time, by Dementia Type



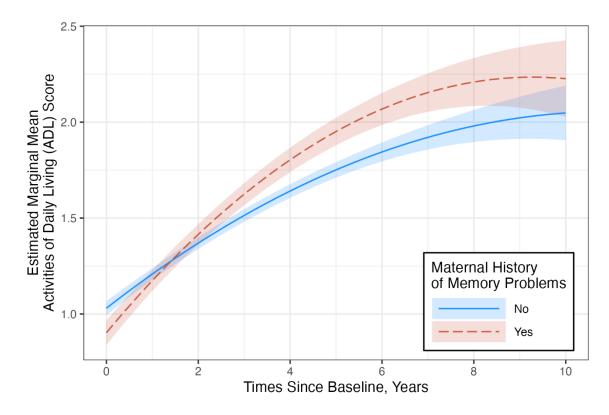
Note. Error bands display one standard error of the mean (SEM) for each trajectory. Trajectories truncated at 99th percentile of follow-up time for each dementia type. DSRS total score was modeled continuously.

Multilevel Modeling of Estimated Marginal Means for Dementia Severity Rating Scale (DSRS) Total Score Over Time, For Males and Females, Displayed by Dementia Type



Note. Error bands display one standard error of the mean (SEM) for each trajectory. Trajectories truncated at 99th percentile of follow-up time for each sex and dementia type. DSRS total score was modeled continuously.

Multilevel Modeling of Estimated Marginal Means for Activities of Daily Living (ADL) of Dementia Severity Rating Scale (DSRS) Over Time, Comparing Presence or Absence of Maternal Family History of Memory Problems



Note. Error bands display one standard error of the mean (SEM) for each trajectory. ADL score was modeled continuously.

BMI was modeled separately for each sex. Among males, few factors predicted the trajectory of BMI change across time. Overall, BMI showed a slight decline across all forms of dementia (b = -0.05, SE = 0.01, p < .001). Higher age of dementia onset was associated with lower BMI across visits (b = -0.21, SE = 0.04, p < .001) as was having greater than a high school education, compared to less than high school (b = -1.29, SE =0.60, p = .033).

Similarly, among females, BMI declined over time across all forms of dementia (b = -0.06, SE = 0.02, p = .010), and age of dementia onset was associated with lower BMI on average (b = -0.22, SE = 0.05, p < .001). BMI trajectory did not differ by dementia type.

See Table 9 for fully adjusted linear mixed effects models of BMI.

Figures 7 and 8 display BMI by dementia onset age for males and females, respectively.

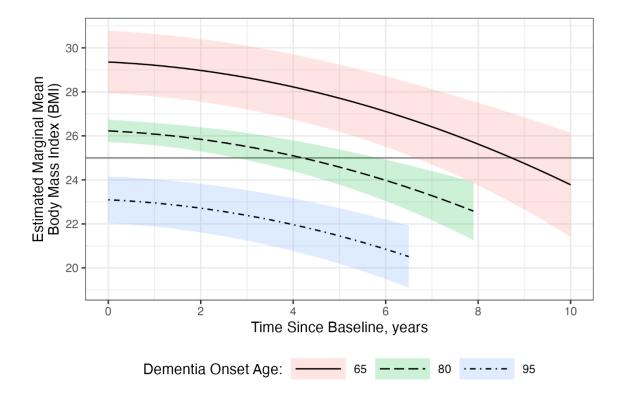
Variable	Males	Females
Intercept	27.49 (0.62) ***	23.40 (2.30) ***
Years	-0.10 (0.10)	0.09 (0.18)
Years ²	-0.05 (0.01) ***	-0.06 (0.02) **
Dementia onset age	-0.21 (0.04) ***	-0.22 (0.05) ***
Dementia duration at first visit	-0.21 (0.20)	-0.23 (0.21)
Education (ref = less than high school)	· · · ·	
Equivalent	-1.29 (0.72)	-0.23 (0.77)
More than	-1.29 (0.60) *	-1.00 (0.75)
General Medical Health Rating (GMHR, ref =	. ,	
Poor)		
Excellent		0.79 (2.30)
Good		2.86 (2.12)
Fair		3.61 (2.08)
Dementia Type ($ref = AD$)		
AD-Other	-1.50 (1.11)	-2.96 (1.97)
AD-VaD	0.07 (0.98)	-0.11 (0.97)
VaD	-0.46 (0.69)	0.34 (0.91)
Other	-0.82 (0.59)	-0.34 (0.78)
Years x Dementia Duration		-0.09 (0.08)
Years ² x Dementia Duration		0.02 (0.01)

Adjusted Beta Coefficients with Standard Errors from the Multilevel Model for Body Mass Index (BMI)

Notes. The table displays parameter estimates from fully adjusted linear mixed effects models displayed by sample (column headings) and predictors (rows). AD= Alzheimer's disease, AD-other = AD with another form of dementia, VaD = vascular dementia, AD-VaD = AD with VaD Dementia onset age is centered at 82 years, the grand mean. * p < .05. ** p < .01. *** p < .001.

Multilevel Modeling of Estimated Marginal Means for Body Mass Index (BMI) Over

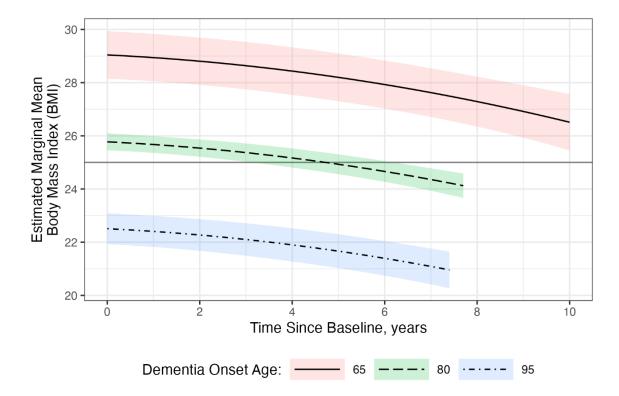
Time, Comparing Dementia Onset Age, for Male Participants



Note. Error bands display one standard error of the mean (SEM) for each trajectory. Trajectories truncated at 99th percentile of follow-up time for each dementia type. Dementia onset age values (65, 80, 95) were selected for illustrative purposes only.

Multilevel Modeling of Estimated Marginal Means for Body Mass Index (BMI) Over

Time, Comparing Dementia Onset Age, For Female Participants



Note. Error bands display one standard error of the mean (SEM) for each trajectory. Trajectories truncated at 99th percentile of follow-up time for each dementia type. Dementia onset age values (65, 80, 95) were selected for illustrative purposes only.

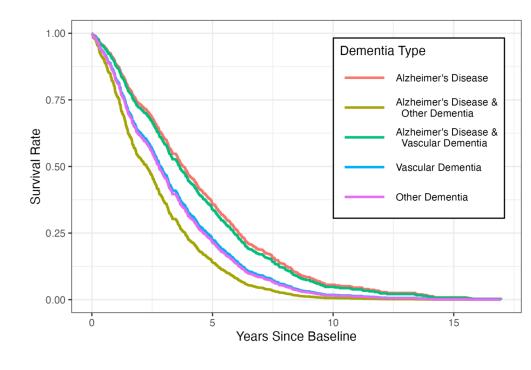
Several factors were significant predictors of survival from dementia diagnosis to death. Compared to persons with AD, higher hazard of death was found for Other dementia (HR = 1.63, p < .001; 95% CI [1.27-2.08]), AD-Other (HR = 2.23, p < .01; 95%CI [1.32-3.74]), and VaD (HR = 1.55, p < .01; 95% CI [1.16-2.01]). See Figure 9 for display of survival by dementia type. Age of dementia onset was associated with a 7% increased risk of death (HR = 1.07, p < .001; 95% CI [1.05-1.09]) and an 8% increased risk for dementia duration at first visit (HR = 1.08, p < .05; 95% CI [1.00-1.16]) for each increasing year of the predictor. As expected, health-related factors were also associated with a hazard of death. Higher BMI at initial visit was associated with a reduced risk for death (HR = 0.97, p < .01; 95% CI [0.95-0.99]) (See Figure 10). Similarly, overall health status at the diagnosis visit was also associated with a reduced risk for death, for those considered to be in fair (HR = 0.25, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17, p < .001; 95% CI [0.12-0.50]), good (HR = 0.17; 95% CI [0.12-0.50]), good (HR = 0.17; 95% CI [0.12-0 .001; 95% CI [0.08-0.34]), and excellent (*HR* = 0.14, *p* < .001; 95% CI [0.06-0.28]) health, compared to those considered to be in poor health (See Figure 11). Though not reaching statistical significance, being female was associated with a reduced risk of death (HR = 0.85, p = .08). Interactions tested between having a family history of memory problems and diagnosis, and between sex and diagnosis, were not significant. Table 10 displays the results of Cox Regression models.

Hazard Rations of Cox Regression for Time to Death

		<u>95% C.I.</u>		
Variable	HR	Lower	Upper	
Dementia onset age	1.07 ***	1.05	1.09	
Dementia duration at first visit, years	1.08 *	1.00	1.16	
Sex $(ref = male)$	0.84	0.70	1.03	
Dementia Type $(ref = AD))$				
AD-Other	2.22 **	1.32	3.74	
AD-VaD	1.06	0.75	1.50	
VaD	1.55 **	1.16	2.01	
Other	1.63 ***	1.27	2.08	
General Medical Health Rating (GMHR)	(ref = Poor)			
Excellent	0.13 ***	0.06	0.28	
Good	0.17 ***	0.08	0.34	
Fair	0.25 ***	0.12	0.50	
Body Mass Index (BMI)	0.97 **	0.95	0.99	

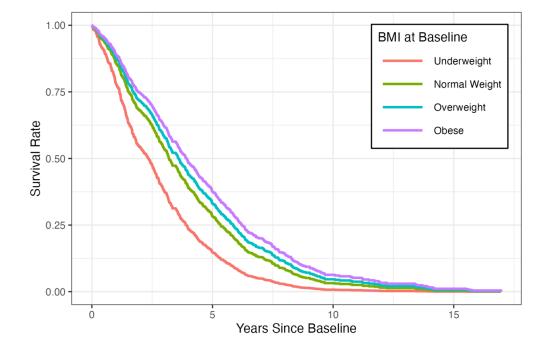
Notes. AD= Alzheimer's disease, AD-other = AD with another form of dementia, VaD = vascular dementia, AD-VaD = AD with VaD. Dementia onset age is

centered at 82 years, the grand mean. * p < .05. ** p < .01. *** p < .001.



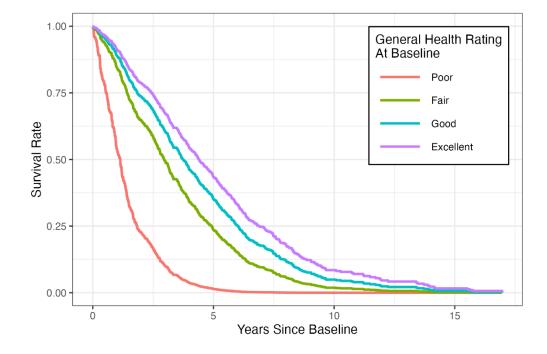
Survival Plot by Dementia Type

Note. Survival time to death was right censored at the date of last study visit when death date was not known.



Survival plot by Body Mass Index at Baseline Visit

Note. Survival time to death was right censored at the date of last study visit when death date was not known.



Survival plot by General Medical Health Rating (GMHR) at Baseline Visit

Note. Survival time to death was right censored at the date of last study visit when death date was not known.

Discussion

The aim of this project was to examine modifiable and non-modifiable risk factors associated with different causes of dementia, identify factors most impactful on cognitive and functional decline, and examine health-related outcomes of BMI and time to death, in a population-based cohort. This study identified several distinguishing factors characteristic of different dementia types, and highlighted the important role that type of dementia and overall medical health status have on the course of dementia and its duration.

Factors that distinguished different diagnoses included dementia onset age, where younger dementia onset age was associated with greater occurrence of VaD and Other dementia, compared to AD. Male sex was associated with higher occurrence of AD-Other or Other dementia. The latter is consistent with literature suggesting that AD (without another form of dementia) is more common in females than males (Henderson & Buckwalter, 1994; Ferretti et al., 2018). This study found that the ε 4 allele of APOE was associated with a reduced occurrence of VaD and Other dementias, consistent with literature describing the association between this gene and increased likelihood for AD diagnosis (Albrecht et al., 2015; Launer et al., 1999; Lindsay et al., 2002; Liu et al., 2013; Rawle et al., 2018; Roses et al., 1993; Wilson et al., 2002); however, other studies have reported an association between this gene and risk for VaD (Baum et al., 2006; Chuang et al., 2010; Ji et al., 1998). Further, despite these two diagnoses (AD and VaD) being considered separate disorders, Emrani et al. (2020) describes the relatively rare instance of having a "pure" form of either of these diagnoses, and argue for a classification that would allow AD and VaD to be considered a spectrum diagnosis. Other literature has

also highlighted this, suggesting that AD and VaD may be less distinct diagnoses given the overlapping cerebrovascular pathology and risk factors (e.g. high cholesterol, blood pressure) often identified in those with AD (Kalaria & Ballard, 1999; Kivipelto et al., 2001). Nonetheless, this study did find unique characteristics that differentiated VaD versus AD (younger age of onset, absence of the $\varepsilon 4$ allele of APOE, worse overall health, upper body weakness, lateralization of signs, and major cardiac conditions). The cooccurrence of dementia type, particularly for AD and VaD may be more common with increasing age. This was found to be the case in an autopsy-based study with the "oldest old" by Jellinger and Attems (2010) that described finding in their sample mixed dementia (AD with VaD) and AD with cerebrovascular lesions *increased* with age, while other diagnoses decreased. The distinctions between AD and VaD offer the opportunity for intervention better tailored to where on this "spectrum" an individual may fall. For example, prevention of major cardiac conditions [e.g., myocardial infarction, carotid artery disease and having a coronary artery bypass graft (CABG); refer to Appendix D for full list of conditions] may be a critical factor to reduce occurrence of VaD, singly or in combination with AD, whereas reduction of vascular risk factors of hypertension and hyperlipidemia may be a strategy applicable to all dementias. This study also found those with poorer health status have lower cognitive function overall; given this, knowing which diagnoses are more susceptible to poorer health status (e.g. VaD and AD-VaD) can point providers and patients towards possible interventions (e.g. early screening, preventative behaviors like diet and exercise) to bolster health (and cognition, function) among those with multiple comorbidities.

Neurological symptoms associated with specific diagnoses included upper body weakness, lateralization of signs and speech abnormalities, which were most associated with VaD singly or co-occurring with another form of dementia, likely reflecting the physical consequences of many cerebrovascular events. Further, consistent with what is known about medical comorbidities associated with cerebrovascular burden and poor health outcomes, cardiac conditions were also found to be associated with a diagnosis of VaD (Khan et al., 2016).

The Other dementia group is comprised of non-AD, mixed diagnoses, and most frequent, dementia of unknown etiology. Many of the primary and secondary diagnoses within this group reflect severe medical conditions and comorbidities [e.g. DUE with vascular changes, DUE with current alcohol abuse, amyotrophic lateral sclerosis (ALS), FTD, DLB- refer to Appendix A to view all diagnoses within this group). This mixed subset of diagnoses is not unique to this study; other literature has described difficulties in characterizing DUE based on the diversity of clinical presentations with several potential etiologies or a clinical presentation of major neurocognitive impairment while being inconsistent with diagnostic criteria for a specific form of dementia (Crystal et al., 2000). Crystal et al. (2000) presents a review of the literature, describing a wide range of estimated prevalence of DUE, from 2 to 65% (this included population and clinic based settings), though this clinical group does not appear to have been well described elsewhere. Given the mixed nature of clinical symptoms present among this group, this study's results suggesting that neurological conditions, including PD, and presence of hallucinations (early in disease course) places one at greater risk for a diagnosis of mixed dementia types, including AD-Other and Other dementia are understandable. While

hallucinations are not uncommon in AD, they tend to present towards the middle of the disease course rather than early in the disease course (Bassiony & Lyketsos, 2003). Further, aside from DUE alone or with a secondary diagnosis, Other dementia also included diagnoses for which more frequently occurring behavioral and psychiatric symptoms were quite common (FTD, DLBD). One may speculate that there may have been a wider array of unique behavioral and psychiatric features that distinguished dementia types, had these kinds of diagnoses been more prevalent in the Other dementia group. Nonetheless, with the exception of hallucinations, the results from this study suggest that other behavioral and psychological symptoms are similar across forms of dementia. Note, euphoria was rare across all forms of dementia and was therefore not examined amongst the neuropsychiatric symptoms.

Overall health status also reflected themes of poorer health being related to VaD and Other dementia, supported by known associations between health conditions, general health status and these specific diagnoses. Eizaguirre et al. (2017) and Hofman et al. (1997) describe that the higher occurrence of cerebrovascular and cardiovascular burden present in those with VaD may result in susceptibility to other co-occurring medical issues. Eizaguirre et al. (2017) also reported shorter survival in those with VaD compared to those with AD, consistent with this study's findings. Those with AD-Other and Other dementia actually experienced even higher hazard of death than VaD and AD alone. This underscores the shortened survival duration and course for non-AD forms of dementia, with or without medical comorbidities. While specific recommendations and conclusions regarding the Other group is difficult given the heterogeneity of its diagnoses, it appears that VaD is most associated with modifiable risk factors, offering possible intervention avenues for patients. Given the presence of the previously mentioned modifiable risk factors associated with VaD, a similar argument may be made for the treatment of comorbidities and enhancement of overall health.

Several studies have emphasized the utility of early intervention for modifiable risk factors. Kalaria (2010) describes that for those with a mixed presentation of AD and VaD, managing the cerebrovascular symptoms could help to slow progression of AD, which relates to the prior discussion surrounding the link between these two diagnoses. Borenstein et al. (2006) also describe modifiable factors as they arise across the lifespan, that are thought to impact AD development or expression, providing more pointed guidance on when specific interventions may be most beneficial. Though an association of cardiovascular risk factors and AD has been previously identified (Kalaria & Ballard, 1999; Kivipelto et al., 2001), this present study did not find hypertension or hyperlipidemia to be uniquely associated with dementia type. Other studies have found an association between hypertension and VaD, but not AD (Hayden et al., 2006). Although these health conditions alone were not uniquely significant in this study, vast literature highlights their overall significance as risk factors for AD (Eizaguirre et al., 2022; Hayden et al., 2006) or other forms of dementia (Wengreen et al., 2009; Rizzi et al., 2014) and warrant monitoring and intervention for primary prevention.

Those categorized as Other dementia were more impaired on functional outcomes and singly or with AD appeared to express more rapid cognitive and functional decline compared to AD. In examining individual domains, it appears that functional impairment in those with Other dementia may be driven by difficulties with basic ADLs (e.g. those related to self-care, grooming, feeding) over other aspects of functioning. Compared to AD, over time, those with VaD (minimally) and AD with another form of dementia, also experienced a greater decline in functioning overall compared to AD. This could be a consequence of the previously described physical difficulties that often arise as a result of related conditions affecting these groups such as stroke or mixed medical comorbidities directly influencing physical mobility.

Interestingly, while males with AD-Other dementia and non-AD dementia exhibited similar or better functional abilities than females, after approximately five years, they experienced greater impairment at an exponentially faster rate than females. Though the models controlled for overall health, it may be that a cumulative effect of comorbidities combined with increasing dementia severity result in the rapid increase in functional disability. This accelerated decline in performance was also observed in cognition among those with AD-Other dementia and non-AD diagnoses where females exhibited more rapid decline than males until years 5-7 post diagnosis where men appear to decline more rapidly.

With respect to specific functional domains (ADLs, cognitive and social), this study also found females to have worse performance than males. This may be related to females tending to decline more rapidly than males (at least earlier in the course of dementia across many dementia subtypes), perhaps also influencing functional outcomes (Wang et al., 2022; Zhang et al., 2018). It may be that females with dementia have a vulnerability towards frailty compared to males; while literature describes greater frailty in older females broadly (Bartley et al., 2016), evidence for greater frailty in females with dementia has not been well described. Additionally, older age of dementia onset, longer dementia duration, and being an APOE ɛ4 carrier, were associated with lower cognitive performance, on average, while higher education and better overall health were associated with better cognitive and functional outcomes. These findings are consistent with established literature describing risk for onset of cognitive and functional impairment being moderated by age, education and health status (Borenstein et al., 2006; Leoutsakos et al.,2012; Stern, 2012; Stern et al., 1999). Further, individuals in the upper age ranges are more likely to accumulate tau and amyloid beta (Katsumata et al., 2024), as well as having greater likelihood of age-related health conditions. One could hypothesize that those with an older onset age were also more likely to have developed these conditions, placing them at greater vulnerability for poorer cognitive outcomes. Older onset age was associated with higher functioning, likely reflective of the known association that being younger at onset age often is followed by a more rapid decline, compared to those who develop the disease in late life (Jacobs et al., 1994).

BMI was not uniquely related to dementia diagnosis. In examining BMI by sex, both males and females displayed a significant decline in BMI over time, and older onset age of dementia resulted in lower BMI on average. For males, those with more education had a lower BMI on average. Borenstein et al. (2006) offers that higher levels of education may be associated with SES and in turn, healthful lifestyle circumstances that have downstream effects on health-related choices across the lifespan. Interestingly though, for older adults, research has described higher weight may be protective when considering risk for mortality (Winter et al., 2014), though it is also noted that higher BMI may negatively influence physical functioning in the older adult population (Woo et al., 2007), with the exception of frailty, which has been associated with lower BMI (Hubbard et al., 2010).

This study found several factors contributing to risk of mortality in dementia, including having a diagnosis of AD-Other, Other dementia, and VaD [median survival of 3.18, 2.13, 2.93 years, respectively; compared to AD alone (median survival of 3.83)]. As previously described, many of those with other dementias and VaD often have comorbid medical conditions, which likely contribute to shorter survival times than having AD alone. Consistent with findings by Winter et al. (2014), this study found that higher BMI (at initial visit) and a better overall health rating were both associated with a reduced risk for death, perhaps highlighting the important role that frailty and complex health comorbidities can have on one's disease course. While not statistically significant (p =.08), female sex was associated with reduced risk for death, similar to findings from Lee & Chodosh (2009). Another Cache County Study (Tschanz et al., 2004) examined mortality among the similar diagnostic groups as this present study, only among those with dementia at Wave 1. That study reported an increased risk for death among males (similar to this present study though we did not reach statistical significance), and among specific health conditions identified as cerebrovascular disease, coronary heart disease, diabetes, PD and pulmonary disorders to be significantly associated with mortality. Although the present study did not examine specific health conditions in relation to mortality, our results are generally consistent in that overall poorer health status was found to be related to higher risk for death. Further, the 2004 Cache County Study stratified mortality for dementia type by age group, reporting that among younger participants (65 - 74 years), those with AD had the highest mortality, while for those in

the older age group (\geq 85), those with VaD experienced higher mortality. While this present study did not stratify survival analyses by age group, this study also found those with VaD to be at an increased risk for death compared to those with AD for this sample.

Study Strengths and Limitations

The strengths of this study include the large, population-based cohort, as opposed to the commonly utilized clinical samples. This, and the longitudinal nature of the study (maximum follow up time up to 13.28 years post dementia diagnosis), may offer greater generalizability to persons with dementia overall than a study from restricted settings. This study also sought to describe disease course, to expand upon the body of literature that has well-described risk for onset of dementia, namely AD, and to discuss course of other dementias in comparison to that of AD. Further, much of the literature on aging, including that of risk factors and disease course, often described dementia broadly, or has limited the focus to AD. Though AD was the largest diagnosis group, our study examined other diagnoses to provide more nuanced characterization of clinical features and disease course.

Not without limitations, this study drew from a predominantly White sample, limiting generalizability in this regard. Also, the Other dementia group was comprised of a mix of diagnoses and undetermined etiology. Due to low numbers of these individual forms of dementia, specific distinctions by rare forms of dementia could not be examined. Another consideration is that the diagnosis groups were largely created based on clinical characteristics, with a smaller portion having neuropathological criteria; biomarkers both ante- or post-mortem may help better discriminate AD vs non-AD subtypes of dementia, bolstering diagnostic accuracy (Jack et al., 2024).

Clinical Utility and Future Directions

The clinical utility of this study lies in the identified non-modifiable and modifiable risk factors associated with dementia type, as well as in the described relationship of risk factors on cognitive, functional and health-related outcomes. This study was able to detect several risk factors that distinguish AD from non-AD types of dementia as follows: those specific to AD-Other (e.g. female sex, neurological signs, hallucinations and PD), AD-VaD (upper body weakness, lateralization of signs, and major cardiac conditions), VaD (including health status, APOE ɛ4 and neurological conditions) and Other dementia (age of onset, dementia duration, female sex, health status, hallucinations, conditions of the brain and PD). Notably, conditions of the brain and cardiac conditions that were found to be associated with increased risk for VaD, extends beyond the commonly studied effects of hypertension and hyperlipidemia on VaD, offering other important features characteristic of specific dementia types.

In considering the relevance of these findings in today's context (as this study utilized extant data spanning a few decades prior), consideration of current health protocols is warranted. Specific to this study's finding that cardiovascular conditions were more influential than other well-known predictors of health outcomes (hypertension, hyperlipidemia), one may hypothesize that in recent decades, Americans have become more health conscious (USDA, 2014; USDA 2024) which in turn, may lead to more healthful food and lifestyle choices. The finding that cardiovascular health conditions were so significant for vascular disease, may reflect poorer management of precursive conditions like hypertension and hyperlipidemia at the time of the current study's data collection. Since the data were collected for this study, the health field has continued to emphasize management of these factors as a way to prevent more serious, less modifiable conditions over time. Further, in recent years, medication adherence for the treatment of hypertension has improved with newer medications resulting in fewer side effects (Yarlagadda & Gupta, 2024). Despite these positive developments, Sidney et al. (2016) expressed concern for ongoing increases in cardiovascular disease (CVD) in the US, and even more recent reports raise concern for lack of decline in CVD between 2010-2022 (Woodruff et al., 2024). Based on these current reports of prevalence of CVD in the US, it appears that the medical concerns of our study's sample are not entirely unlike the current population.

Further, there continues to be no cure for AD or the other dementias examined within this study (e.g. FTD, DLBD). Although there have attempts to mitigate the progression and severity of AD with newer medications, the long-term effects and efficacy of new treatments remain unclear, and may also result in side effects that may impact patient adherence (Alzheimer's Associations, 2024). It also appears that those with APOE £4 may be more vulnerable to those side effects. With advancements in the medical field, people are living longer with dementia but continue to struggle with these challenges, and as such, our study's findings remain relevant and important for understanding factors that characterize dementia types and those that influence disease course.

It also appears that diagnoses of VaD alone, AD with VaD, and Other dementia may have the greatest opportunity for intervention related to modifiable risk factors and the significant role that overall health status and conditions of the heart, related to these diagnoses. In considering generations living today, trends of Americans choosing healthier foods has increased in recent decades (USDA, 2014), and further the USDA has continued to provide accessible data regarding dietary guidelines for consumers (USDA, 2024). However, despite these reports, other reports indicate that obesity in the US has continued to steadily rise since 1999 (Wang et al., 2020) and as such, overweight and obesity remain important health factors to study. The increase of accessible resources in recent decades provides support to providers too in that patients can more easily access health and nutrition related information outside of the medical setting and in conjunction with their care providers. This is also related to our study's finding that overall health status functions as a risk factor for negative outcomes (cognitive, functional, mortality) rather than a moderator of trajectory of decline. In recent years, guidelines for diagnosis of hypertension have been modified to include lowered cut points in an effort to support early identification and intervention (Armstrong, 2018). The utilization of updated health recommendations and information, along with early implementation of preventative measures will likely have the greatest positive outcome earlier on in life and prior to dementia onset.

Interestingly, higher education was associated with worse overall DSRS score, suggesting increased functional impairment. Given the known trend that cognitive reserve results in delays to identifying disease onset, it may be the case that this result is reflective of a more severe disease state that is being observed at baseline visit of this study, which in other cohorts has been associated with more rapid decline (Stern, 2012). Those in worse health and with a diagnosis of other dementia type also appear to be more vulnerable towards functional difficulties. This further highlights the need to attend to functional needs of patients, particularly knowing that females in this study struggled to perform ADLs more than males *and* integrating the finding that while males initially display stronger functional abilities overall, they do appear to go on to experience a more rapid decline than their female counterpart for those with non-AD forms of dementia.

Importantly, this study utilized incident cases when examining cognitive and functional changes over time and mortality. This is significant because incident cases tend to be more difficult to identify, with diagnoses often being made years after onset when it is more challenging to pinpoint when onset may have occurred. In our sample, incident cases on average, were identified within three years of dementia onset, and thus allowed for a more accurate observation of what trajectories look like among different dementia types (as opposed to capturing someone midway through their disease course). In recent years the medical field has made advancements in early detection of dementia [e.g. improvement in biomarkers (Forlenza, 2015), routine screenings at routine medical visits] and the growth in the development of medications to stave of disease progression upon onset. The advancement in identifying people with dementia earlier on in their disease course may result in an increase in incident cases of dementia; however, that coupled with some of the improvements to disease management, may also allow for earlier intervention and slower disease progression. This study's findings continue to offer value information for providers even in the context of these current day

advancements, and certainly remain relevant for those living in contexts that do not afford the same level of advanced care (e.g. those living in rural regions).

In examining mortality, those with a diagnosis of AD with another type of dementia were at the greatest risk for death, followed by Other dementia, and VaD (compared to having AD alone). Further, general health status was associated with reduced risk for death, as was greater BMI. This finding is notable in that high BMI is often considered a reflection of lower overall health, though for the older adult population, it appears that higher weight is actually protective, in the setting of other positive health factors; or, low BMI is an indication of an end stage of life. This offers insight into how interpretations of different indicators of health change across the lifespan and with dementia, and how special attention on maintaining an appropriate weight may be critical for some older adults. It appears that having dementia diagnoses that are associated with or are a consequence of cerebrovascular and cardiovascular issues, along with generally poorer health and those with low BMI later in life, suffer the greatest in regard to their cognitive and functional outcomes, as well as being vulnerable to lower survival rates.

Some studies have also offered key windows for intervention to effect change on either dementia onset or disease course (Borenstein et al., 2006; Livingston et al., 2020). Yaffe et al. (2021) described an association between early life cardiovascular risk factors and later in life cognitive decline, alluding to the importance of preventative action related to modifiable health factors. Managing overall health status and individual health conditions, with special attention to weight loss over time, may help promote longevity in older adults. As seen in this study, cerebrovascular and cardiovascular conditions were among the most important risk factors to influence risk for AD-VaD, VaD and Other dementia type, above several others included in the study. Those risk factors may also place someone at greater vulnerability for poorer overall health status, which was also closely related to risk for VaD and Other dementia, and to cognitive and functional outcomes.

This study offers many avenues for future research to expand upon, that would help bolster and better generalize these findings. While this study drew from a large population-based sample, a more representative sample of the general population would be one that is ethnically diverse. Within the "Other" dementia group, there are many comorbid diagnoses that have unique clinical characteristics, but due to low numbers of these individual diagnoses (e.g. FTD, LBD) this group could not be separated out further. In support of what this study sought to do, which included examining risk factors among a broader range of diagnoses within one sample, having the sample size in the future to further break down this kind of mixed group would be an important next step, as well as studying other health factors that might be associated with mortality among those diagnoses. Further, the integration of biomarkers would help clarify and strengthen diagnosis group accuracy. The complex nature of having dementia of an undetermined etiology or comorbid conditions (e.g. greater cognitive and functional difficulties, higher occurrence of health conditions and overall poor health status) was observed in this study, highlighting the important role that overall health can have on prognosis, regardless of dementia diagnosis. With the growing use of biomarkers as indicators specific dementia type, diagnostic specificity may be provided for those who have previously been identified as DUE here or in other cohorts (e.g. Crystal et al., 2000) to rule out other

types of dementia. One may hypothesize that as the field grows in its ability to identify more accurately the pathology associated with specific dementia types, there may be fewer individuals carrying diagnoses of DUE, as diagnostic specificity grows. As earlier described, Crystal et al. (2000) report wide ranging prevalence of DUE (2-65% depending on population sample), and this article also cautions that risk for having a DUE increases with age. It does not appear that prevalence of DUE since the publication of that article has been well described, and it remains unclear if the advancements in the medical field in the last few decades will influence rates of DUE in the future.

This present study examined overall health status and BMI on mortality, and it would be important to expand upon this, perhaps examining the influence of specific health factors most associated with dementia type in this study and examine how those may influence survival amongst all dementia diagnosis groups. It is the hope that this study has contributed to this growing body of literature, aiding healthcare providers in offering patient opportunities for early intervention by modifying risk factors as able, and providing patients and their families with expectations regarding disease course.

REFERENCES

Albanese, E., Taylor, C., Siervo, M., Stewart, R., Prince, M. J., & Acosta, D. (2013). Dementia severity and weight loss: a comparison across eight cohorts. The 10/66 study. *Alzheimer's & Dementia*, 9(6), 649-656.

Albrecht, M. A., Szoeke, C., Maruff, P., Savage, G., Lautenschlager, N. T., Ellis, K. A., Taddei, K., Martins, R., Masters, C. L., Ames, D., & Foster, J. K. (2015). Longitudinal cognitive decline in the AIBL cohort: The role of APOE ε4 status. *Neuropsychologia*, 75, 411–419. https://doi.org/10.1016/j.neuropsychologia.2015.06.008.

- Altmann, A., Tian, L., Henderson, V. W., Greicius, M. D., & Alzheimer's Disease Neuroimaging Initiative Investigators. (2014). Sex modifies the APOE-related risk of developing Alzheimer disease. *Annals of Neurology*, 75(4), 563-573.
- Alzheimer's Association. (2024). Alzheimer's disease facts and figures. *Alzheimer's & Dementia*, 20(5), 3708–3821. https://doi.org/10.1002/alz.13809.
- Anstey, K. J., Peters, R., Mortby, M. E., Kiely, K. M., Eramudugolla, R., Cherbuin, N., ...
 & Dixon, R. A. (2021). Association of sex differences in dementia risk factors with sex differences in memory decline in a population-based cohort spanning 20–76. years. *Scientific Reports*, 11(1), 1-10.
- APOE gene Genetics Home Reference NIH. (2020). National Institute on Health. Retrieved from https://ghr.nlm.nih.gov/gene/APOE#conditions.

Apostolova, L. G., Dinov, I. D., Dutton, R. A., Hayashi, K. M., Toga, A. W., Cummings,

J. L., & Thompson, P. M. (2006). 3D comparison of hippocampal atrophy in amnestic mild cognitive impairment and Alzheimer's disease. *Brain*, *129*(11), 2867-2873.

- Armstrong, R. A. (2013). What causes Alzheimer's disease? *Folia Neuropathologica*, *51*(3), 169-188.
- Armstrong, C. (2018). High blood pressure: ACC/AHA releases updated guideline. *American family physician*, 97(6), 413-415.
- Bartley, M. M., Geda, Y. E., Christianson, T. J. H., Shane Pankratz, V., Roberts, R. O., & Petersen, R. C. (2016). Frailty and Mortality Outcomes in Cognitively Normal Older People: Sex Differences in a Population-Based Study. *Journal of the American Geriatrics Society*, 64(1), 132–137. <u>https://doi.org/10.1111/jgs.13821</u>
- Bassiony, M. M., & Lyketsos, C. G. (2003). Delusions and Hallucinations in Alzheimer's Disease: Review of the Brain Decade. *Psychosomatics*, 44(5), 388–401.
 https://doi.org/10.1176/appi.psy.44.5.388.
- Baum, L., Lam, L. C., Kwok, T., Lee, J., Chiu, H. F., Mok, V. C., ... & Ng, H. K. (2006). Apolipoprotein E ɛ4 allele is associated with vascular dementia. *Dementia and geriatric cognitive disorders*, 22(4), 301-305.
- Bessi, V., Mazzeo, S., Padiglioni, S., Piccini, C., Nacmias, B., Sorbi, S., & Bracco, L.
 (2018). From subjective cognitive decline to Alzheimer's disease: the predictive role of neuropsychological assessment, personality traits, and cognitive reserve. A 7-year follow-up study. *Journal of Alzheimer's Disease*, 63(4), 1523-1535.

Borenstein, A. R., Copenhaver, C. I., & Mortimer, J. A. (2006). Early-Life Risk Factors

for Alzheimer Disease. *Alzheimer Disease & Associated Disorders*, 20(1), 63–72. https://doi.org/10.1097/01.wad.0000201854.62116.d7

Braak H, Braak E. Evolution of neuronal changes in the course of Alzheimer's disease. J. Neural Trans. Suppl. 53, 127–140 (1988).

Breitner, J. C., Wyse, B. W., Anthony, J. C., Welsh-Bohmer, K. A., Steffens, D. C., Norton, M. C., ... & Khachaturian, A. (1999). APOE-ε4 count predicts age when prevalence of AD increases, then declines: the Cache County Study. *Neurology*, 53(2), 321-321.

- Brookmeyer, R., Corrada, M. M., Curriero, F. C., & Kawas, C. (2002). Survival following a diagnosis of Alzheimer disease. *Archives of Neurology*, 59(11), 1764-1767.
- Buccione, I., Perri, R., Carlesimo, G. A., Fadda, L., Serra, L., Scalmana, S., & Caltagirone, C. (2007). Cognitive and behavioural predictors of progression rates in Alzheimer's disease. *European Journal of Neurology*, 14(4), 440-446.
- Burns, A., Jacoby, R., & Levy, R. (1991). Neurological signs in Alzheimer's disease. Age and Ageing, 20(1), 45-51.
- Canadian Study of Health and Aging. (1994). The Canadian Study of Health and Aging* Risk factors for Alzheimer's disease in Canada. *Neurology*, *44*(11), 2073-2073.
- Cannon-Albright, L. A., Foster, N. L., Schliep, K., Farnham, J. M., Teerlink, C. C., Kaddas, H., ... & Kauwe, J. S. (2019). Relative risk for Alzheimer disease based on complete family history. *Neurology*, 92(15), e1745-e1753.
- Cerejeira, J., Lagarto, L., & Mukaetova-Ladinska, E. (2012). Behavioral and psychological symptoms of dementia. *Frontiers in Neurology*, *3*(73).

- Choi, B., An, H., Son, S. J., & Kim, S. Y. (2016). PT585. Effect of Neuropsychiatric
 Symptoms on Mortality in Patients with Dementia: differences between
 Alzheimer's dementia, subcortical vascular dementia, and frontotemporal
 dementia. *International Journal of Neuropsychopharmacology*, 19(1), 15.
- Chuang, Y. F., Hayden, K. M., Norton, M. C., Tschanz, J., Breitner, J. C., Welsh-Bohmer, K. A., ... & Cache County Investigators. (2010). Association between APOE ε4 allele and vascular dementia: the cache county study. *Dementia and geriatric cognitive disorders*, 29(3), 248-253.
- Clark, C. M., & Ewbank, D. C. (1996). Performance of the dementia severity rating scale: a caregiver questionnaire for rating severity in Alzheimer disease. *Alzheimer Disease and Associated Disorders*, 10(1), 31-39.
- Costs of Alzheimer's to Medicare and Medicaid. (2019). Alzheimer's Association. Retrieved From <u>https://act.alz.org/site/DocServer/2012_Costs_Fact_Sheet_v</u> ersion 2.pdf?docID=7161
- Crystal, H. A., Dickson, D., Davies, P., Masur, D., Grober, E., & Lipton, R. B. (2000). The Relative Frequency of "Dementia of Unknown Etiology" Increases With Age and Is Nearly 50% in Nonagenarians. *Archives of Neurology*, 57(5), 713–719. https://doi.org/10.1001/archneur.57.5.713.
- Cummings, J. L., Mega, M., Gray, K., Rosenberg-Thompson, S., Carusi, D. A., & Gornbein, J. (1994). The Neuropsychiatric Inventory: comprehensive assessment of psychopathology in dementia. *Neurology*, 44(12), 2308-2308.
- Delenclos, M., Moussaud, S., & McLean, P. J. (2017). Lewy body dementia. In Disease-Modifying Targets in Neurodegenerative Disorders (pp. 175-198). Academic

- De Oliveira FF, Bertolucci PHF, Chen ES, et al. Risk factors for age at onset of dementia due to Alzheimer's disease in a sample of patients with low mean schooling from Sa o Paulo, Brazil. Int J Geriatr Psychiatr. 2014;29:1033–1039.
- Donix, M., Small, G. W., & Bookheimer, S. Y. (2012). Family History and APOE-4 Genetic Risk in Alzheimer's Disease. *Neuropsychology Review*, *22*(3), 298–309.
- Edland, S. D., Xu, Y., Plevak, M., O'Brien, P., Tangalos, E. G., Petersen, R. C., & Jack,
 C. R. (2002). Total intracranial volume: normative values and lack of association with Alzheimer's disease. *Neurology*, *59*(2), 272-274.
- Emrani, S., Lamar, M., Price, C. C., Wasserman, V., Matusz, E., Au, R., ... & Libon, D. J. (2020). Alzheimer's/vascular spectrum dementia: classification in addition to diagnosis. *Journal of Alzheimer's disease*, 73(1), 63-71.
- Kalaria, R. N., & Ballard, C. (1999). Overlap between pathology of Alzheimer disease and vascular dementia. *Alzheimer Disease & Associated Disorders*, 13, S115-S123.
- Erkinjuntti, T. (1994). Clinical criteria for vascular dementia: the NINDS-AIREN criteria. *Dementia and Geriatric Cognitive Disorders*, *5*(3-4), 189-192.
- Ferrari, C., Lombardi, G., Polito, C., Lucidi, G., Bagnoli, S., Piaceri, I., ... & Sorbi, S. (2018). Alzheimer's disease progression: factors influencing cognitive decline. *Journal of Alzheimer's Disease*, 61(2), 785-791.

Ferretti, M. T., Iulita, M. F., Cavedo, E., Chiesa, P. A., Schumacher Dimech, A.,

Santuccione Chadha, A., ... & Women's Brain Project and the Alzheimer Precision Medicine Initiative. (2018). Sex differences in Alzheimer disease—the gateway to precision medicine. Nature Reviews Neurology, 14(8), 457-469.

- Fiest KM, Roberts JI, Maxwell CJ, Hogan DB, Smith EE, Frolkis A, et al. (2016). The Prevalence and Incidence of Dementia Due to Alzheimer's Disease: a Systematic Review and Meta-Analysis. *Can J Neurol Sci.* 43 Suppl 1:S51–82
- Forlenza, O. V., Radanovic, M., Talib, L. L., Aprahamian, I., Diniz, B. S., Zetterberg, H., & Gattaz, W. F. (2015). Cerebrospinal fluid biomarkers in Alzheimer's disease:
 Diagnostic accuracy and prediction of dementia. *Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring, 1*(4), 455–463.
 <u>https://doi.org/10.1016/j.dadm.2015.09.003</u>
- Foster, J. K., Albrecht, M. A., Savage, G., Lautenschlager, N. T., Ellis, K. A., Maruff, P., Szoeke, C., Taddei, K., Martins, R., Masters, C. L., Ames, D., & the AIBL Research Group. (2013). Lack of reliable evidence for a distinctive ε4–related cognitive phenotype that is independent from clinical diagnostic status: Findings from the Australian Imaging, Biomarkers and Lifestyle Study. *Brain*, *136*(7), 2201–2216.
- Franx, B., Arnoldussen, I., Kiliaan, A. J., & Gustafson, D. R. (2017). Weight Loss in Patients with Dementia: Considering the Potential Impact of Pharmacotherapy. *Drugs & Aging*, 34(6), 425–436.

Fratiglioni, L., Ahlbom, A., Viitanen, M., & Winblad, B. (1993). Risk factors for late-

onset Alzheimer's disease: A population-based, case-control study. *Annals of Neurology*, *33*(3), 258-266.

- Fuller, S. J., Carrigan, N., Sohrabi, H. R., & Martins, R. N. (2019). Current and Developing Methods for Diagnosing Alzheimer's Disease. *Neurodegeneration* and Alzheimer's Disease: The Role of Diabetes, Genetics, Hormones, and Lifestyle, 43-87.
- García-Ptacek, S., Kåreholt, I., Farahmand, B., Cuadrado, M. L., Religa, D., &
 Eriksdotter, M. (2014). Body-mass index and mortality in incident dementia: a
 cohort study on 11,398 patients from SveDem, the Swedish Dementia
 Registry. *Journal of the American Medical Directors Association*, 15(6), 447-e1.
- Hayden KM, Zandi PP, Lyketsos CG, Khachaturian AS, Bastian LA, Charoonruk G,
 Welsh-Bohmer KA. Vascular risk factors for incident Alzheimer Disease and
 vascular dementia: The Cache County Study. *Alzheimer Disease & Associated Disorders*. 2006; 20(2):93–100.
- Hayden, K. M., Zandi, P. P., West, N. A., Tschanz, J. T., Norton, M. C., Corcoran, C., ...
 & Welsh-Bohmer, K. A. (2009). Effects of family history and apolipoprotein E ε4
 status on cognitive decline in the absence of Alzheimer dementia: The Cache
 County Study. *Archives of Neurology*, 66(11), 1378-1383.
- Hebert, LE, J Weuve, PA Scherr, DA Evans. Alzheimer disease in the United States (2010-2050) estimated using the 2010 Census. *Neurology*. 80: 2013; 1778–1783.
- Henderson, V. W., & Buckwalter, J. G. (1994). Cognitive deficits of men and women with Alzheimer's disease. *Neurology*, 44(1), 90–90. https://doi.org/10.1212/WNL.44.1.90.

- Hofman, A., Ott, A., Breteler, M. M., Bots, M. L., Slooter, A. J., van Harskamp, F., ... & Grobbee, D. E. (1997). Atherosclerosis, apolipoprotein E, and prevalence of dementia and Alzheimer's disease in the Rotterdam Study. *The Lancet*, 349(9046), 151-154.
- Honea, R. A., Vidoni, E. D., Swerdlow, R. H., Burns, J. M., & Alzheimer's Disease Neuroimaging Initiative. (2012). Maternal family history is associated with Alzheimer' disease biomarkers. *Journal of Alzheimer's Disease*, 31(3), 659-668.
- Hox, J., Moerbeek, M., & Van de Schoot, R. (2017). *Multilevel analysis: Techniques and applications*. Routledge.
- Huang, W., Qiu, C., von Strauss, E., Winblad, B., & Fratiglioni, L. (2004). APOE genotype, family history of dementia, and Alzheimer disease risk: a 6-year follow-up study. *Archives of Neurology*, 61(12), 1930-1934.
- Huang, M. F., Lee, W. J., Yeh, Y. C., Lin, Y. S., Lin, H. F., Wang, S. J., ... & Fuh, J. L.(2020). Neuropsychiatric Symptoms and Mortality Among Patients With MildCognitive Impairment and Dementia Due to Alzheimer's Disease
- Hubbard, R. E., Lang, I. A., Llewellyn, D. J., & Rockwood, K. (2010). Frailty, body mass index, and abdominal obesity in older people. Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences, 65(4), 377-381
- Jack Jr, C. R., Andrews, J. S., Beach, T. G., Buracchio, T., Dunn, B., Graf, A., ... & Carrillo, M. C. (2024). Revised criteria for diagnosis and staging of Alzheimer's disease: Alzheimer's Association Workgroup. *Alzheimer's & Dementia*.

Jacobs, D., Sano, M., Marder, K., Bell, K., Bylsma, F., Lafleche, G., ... & Stern, Y.

(1994). Age at onset of Alzheimer's disease: relation to pattern of cognitive dysfunction and rate of decline. Neurology, *44*(7), 1215-1215.

- Jagust, W., Gitcho, A., Sun, F., Kuczynski, B., Mungas, D., & Haan, M. (2006). Brain imaging evidence of preclinical Alzheimer's disease in normal aging. *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society*, 59(4), 673-681.
- Ji, Y., Urakami, K., Adachi, Y., Maeda, M., Isoe, K., & Nakashima, K. (1998). Apolipoprotein E polymorphism in patients with Alzheimer's disease, vascular dementia and ischemic cerebrovascular disease. *Dementia and geriatric cognitive disorders*, 9(5), 243-245.
- Kalaria, R. N. (2010). Vascular basis for brain degeneration: faltering controls and risk factors for dementia. *Nutrition Reviews*, 68(2), S74-S87.
- Kalaria, R. N. (2016). Neuropathological diagnosis of vascular cognitive impairment and vascular dementia with implications for Alzheimer's disease. *Acta Neuropathologica*, 131(5), 659-685.
- Kane, J., Surendranathan, A., Bentley, A., Barker, S., Taylor, J. P., Thomas, A. J., Allan,
 L. M., McNally, R. J., James, P. W., McKeith, I. G., Burn, D. J., & O'Brien, J. T.
 (2018). Clinical prevalence of Lewy body dementia. *Alzheimer's Research & Therapy*, *10*(1), 19.
- Katsumata, Y., Wu, X., Aung, K. Z., Gauthreaux, K., Mock, C., Forrest, S. L., Kovacs,G. G., & Nelson, P. T. (2024). Pathologic correlates of aging-related tauastrogliopathy: ARTAG is associated with LATE-NC and cerebrovascular

pathologies, but not with ADNC. *Neurobiology of Disease*, *191*, 106412. https://doi.org/10.1016/j.nbd.2024.106412

- Kauwe, J. S., Wang, J., Mayo, K., Morris, J. C., Fagan, A. M., Holtzman, D. M., & Goate, A. M. (2009). Alzheimer's disease risk variants show association with cerebrospinal fluid amyloid beta. *Neurogenetics*, 10(1), 13-17.
- Khan, A., Kalaria, R. N., Corbett, A., & Ballard, C. (2016). Update on vascular dementia. *Journal of Geriatric Psychiatry and Neurology*, 29(5), 281-301.
- Kivipelto, M., Helkala, E. L., Laakso, M. P., Hänninen, T., Hallikainen, M., Alhainen,
 K., ... & Nissinen, A. (2001). Midlife vascular risk factors and Alzheimer's
 disease in later life: longitudinal, population based study. *Bmj*, 322(7300), 1447-1451.
- Koran, M. E. I., Wagener, M., & Hohman, T. J. (2017). Sex differences in the association between AD biomarkers and cognitive decline. *Brain Imaging and Behavior*, 11(1), 205–213
- Lai, C. K. (2014). The merits and problems of Neuropsychiatric Inventory as an assessment tool in people with dementia and other neurological disorders. *Clinical interventions in aging*, 9, 1051.
- Larsson, V., Torisson, G., & Londos, E. (2018). Relative survival in patients with dementia with Lewy bodies and Parkinson's disease dementia. *PLOS ONE*, *13*(8).
- Launer LJ, Andersen K, Dewey ME, et al. (1999). Rates and risk factors for dementia and Alzheimer's disease—results from EURO- DEM pooled analyses. *Neurology*, *52*, 78–84.

Lecanemab approved for treatment of early alzheimer's disease. Alzheimer's Disease

and Dementia. (n.d.). https://www.alz.org/alzheimers-

dementia/treatments/lecanemab-leqembi

- Leoutsakos, J.-M. S., Han, D., Mielke, M. M., Forrester, S. N., Tschanz, J. T., Corcoran, C. D., Green, R. C., Norton, M. C., Welsh-Bohmer, K. A., & Lyketsos, C. G. (2012). Effects of General Medical Health on Alzheimer Progression: The Cache County Dementia Progression Study. *International Psychogeriatrics / IPA*, 24(10), 1561–1570. https://doi.org/10.1017/S104161021200049X.
- Lin KA, Choudhury KR, Rathakrishnan BG, Marks DM, Petrella JR, Doraiswamy PM;
 Alzheimer's Disease Neuroimaging Initiative (2015) Marked gender differences
 in progression of mild cognitive impairment over 8 years. Alzheimers Dement (N
 Y) *I*(2), 103–110.
- Lindeboom, J., & Weinstein, H. (2004). Neuropsychology of cognitive ageing, minimal cognitive impairment, Alzheimer's disease, and vascular cognitive impairment. *European Journal of Pharmacology*, 490(1-3), 83-86.
- Lindsay, J., Laurin, D., Verreault, R., Hébert, R., Helliwell, B., Hill, G. B., & McDowell,
 I. (2002). Risk factors for Alzheimer's disease: a prospective analysis from the
 Canadian Study of Health and Aging. *American Journal of Epidemiology*, 156(5),
 445-453.
- Lewy Body Dementia Association. 2022. What is lewy body dementia? https://www.lbda.org/what-is-lbd/.
- Lindsay, J. (2002). Risk Factors for Alzheimer's Disease: A Prospective Analysis from the Canadian Study of Health and Aging. *American Journal of Epidemiology*, 156(5), 445–453.

- Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., ... & Mukadam, N. (2020). Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *The lancet*, 396(10248), 413-446.
- Liu CC, Kanekiyo T, Xu H, Bu G. (2013). Apolipoprotein E and Alzheimer disease: risk, mechanisms and therapy. *Nature Reviews Neurology*.
- Locke, D. E., Ivnik, R. J., Cha, R. H., Knopman, D. S., Tangalos, E. G., Boeve, B. F., ...
 & Smith, G. E. (2009). Age, family history, and memory and future risk for cognitive impairment. *Journal of Clinical and Experimental Neuropsychology*, *31*(1), 111-116.
- Magierski, R., Kłoszewska, I., & Sobów, T. M. (2010). The influence of vascular risk factors on the survival rate of patients with dementia with Lewy bodies and Alzheimer disease. *Neurologia i NneurochirurgiaPpolska*, *44*(2), 139-147.
- Matar, E., Martens, K. A. E., Halliday, G. M., & Lewis, S. J. (2020). Clinical features of Lewy body dementia: insights into diagnosis and pathophysiology. *Journal of Neurology*, 267(2), 380-389.
- McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, E. M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group* under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology*, *34*(7), 939-939.
- McKeith, I. G., Boeve, B. F., Dickson, D. W., Halliday, G., Taylor, J. P., Weintraub, D., .
 ... & Bayston, A. (2017). Diagnosis and management of dementia with Lewy
 bodies: Fourth consensus report of the DLB Consortium. *Neurology*, *89*(1), 88-100.

- Miech, R. A., Breitner, J. C. S., Zandi, P. P., Khachaturian, A. S., Anthony, J. C., & Mayer, L. (2002). Incidence of AD may decline in the early 90s for men, later for women: The Cache County study. *Neurology*, 58(2), 209-218.
- Mölsä, P. K., Marttila, R. J., & Rinne, U. K. (1986). Survival and cause of death in Alzheimer's disease and multi-infarct dementia. *Acta Neurologica Scandinavica*, *74*(2), 103-107.
- Morris, J. C. (1997). Clinical dementia rating: a reliable and valid diagnostic and staging measure for dementia of the Alzheimer type. *International psychogeriatrics*, 9(S1), 173-176.
- Mosconi L, Berti V, Swerdlow RH, Pupi A, Duara R, de Leon M. 2010. Maternal transmission of Alzheimer's disease: Prodromal metabolic phenotype and the search for genes. *Hum Genomics*, *4*:170–193.
- Mosconi, L., Brys, M., Switalski, R., Mistur, R., Glodzik, L., Pirraglia, E., ... & de Leon,
 M. J. (2007). Maternal family history of Alzheimer's disease predisposes to
 reduced brain glucose metabolism. *Proceedings of the National Academy of Sciences*, 104(48), 19067-19072.
- Mucke, L. (2009). Neuroscience: Alzheimer's disease. Nature, 461(7266), 895.
- Mungas D, Reed BR, Ellis WG, Jagust WJ (2001) The effects of age on rate of progression of Alzheimer disease and dementia with associated cerebrovascular disease. *Arch Neurol*, 58, 1243–1247.

Musicco, M., Palmer, K., Salamone, G., Lupo, F., Perri, R., Mosti, S., ... & Caltagirone,

C. (2009). Predictors of progression of cognitive decline in Alzheimer's disease: the role of vascular and sociodemographic factors. *Journal of Neurology*, *256*(8), 1288.

- National Institutes of Health (1998) Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults – the evidence report. *Obesity Research*, 6(2), 51s–209s.
- Nuttall F. Q. (2015). Body Mass Index: Obesity, BMI, and Health: A Critical Review. *Nutrition Today*, *50*(3), 117–128.
- Olichney, J. M., Galasko, D., Salmon, D. P., Hofstetter, C. R., Hansen, L. A., Katzman, R., & Thal, L. J. (1998). Cognitive decline is faster in Lewy body variant than in Alzheimer's disease. *Neurology*, 51(2), 351-35
- Oliveira, F. F., Chen, E. S., Smith, M. C., & Bertolucci, P. H. (2016). Predictors of cognitive and functional decline in patients with Alzheimer disease dementia from Brazil. *Alzheimer Disease & Associated Disorders*, 30(3), 243-250.
- Olney, N. T., Spina, S., & Miller, B. L. (2017). Frontotemporal dementia. *Neurologic Clinics*, *35*(2), 339-374.
- Ott A, Breteler MM, van Harskamp F, Stijnen T, Hofman A. (1998). Incidence and risk of dementia. The Rotterdam Study. *Am J Epidemiol*, 147:574–80.
- Paulson, H. L., & Igo, I. (2011). Genetics of Dementia. Seminars in Neurology, 31(5), 449–460.
- Perry, R. J., & Hodges, J. R. (1999). Attention and executive deficits in Alzheimer's disease: A critical review. *Brain*, 122(3), 383-404.
- Price, A., Farooq, R., Yuan, J.-M., Menon, V. B., Cardinal, R. N., & O'Brien, J. T.

(2017). Mortality in dementia with Lewy bodies compared with Alzheimer's dementia: A retrospective naturalistic cohort study. *BMJ Open*, 7(11), e017504.

- R Core Team. (2015). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing.
- Rabin, L. A., Wang, C., Katz, M. J., Derby, C. A., Buschke, H., & Lipton, R. B. (2012).
 Predicting Alzheimer's Disease: Neuropsychological Tests, Self-Reports, and
 Informant Reports of Cognitive Difficulties. *Journal of the American Geriatrics Society*, 60(6), 1128-1134.
- Rawle, M. J., Davis, D., Bendayan, R., Wong, A., Kuh, D., & Richards, M. (2018).
 Apolipoprotein-E (Apoe) ɛ4 and cognitive decline over the adult life course.
 Translational Psychiatry, 8(1), 1–8. https://doi.org/10.1038/s41398-017-0064-8.
- Rizzi L, Rosset I, Roriz-Cruz M. (2014). Global epidemiology of dementia: Alzheimer's and vascular types. Biomed Res Int.
- Roses, A. D., & Saunders, A. M. (1994). APOE is a major susceptibility gene for Alzheimer's disease. Current Opinion in Biotechnology, 5(6), 663–667. https://doi.org/10.1016/0958-1669(94)90091-4.
- Sanders, C., Behrens, S., Schwartz, S., Wengreen, H., Corcoran, C. D., Lyketsos, C. G., & Tschanz, J. T. (2016). Nutritional status is associated with faster cognitive decline and worse functional impairment in the progression of dementia: the cache county dementia progression study. *Journal of Alzheimer's disease*, 52(1), 33-42.
- Sanders, C. L., Wengreen, H. J., Schwartz, S., Behrens, S. J., Corcoran, C., Lyketsos, C.

G., ... & Cache County Investigators. (2018). Nutritional status is associated with severe dementia and mortality: The Cache County dementia progression study. *Alzheimer disease and associated disorders*, *32*(4), 298

- Scarabino, D., Gambina, G., Broggio, E., Pelliccia, F., & Corbo, R. M. (2016). Influence of family history of dementia in the development and progression of late-onset
 Alzheimer's disease. *American Journal of Medical Genetics Part B:* Neuropsychiatric Genetics, 171(2), 250-256.
- Sidney, S., Quesenberry, C. P., Jaffe, M. G., Sorel, M., Nguyen-Huynh, M. N., Kushi, L.
 H., ... & Rana, J. S. (2016). Recent trends in cardiovascular mortality in the
 United States and public health goals. *JAMA cardiology*, 1(5), 594-599
- Smith, E.; Clinical presentations and epidemiology of vascular dementia. (2017). *Clin Sci (Lond)*; *131*(11): 1059–1068.
- Skup, M, H Zhu, Y Wang, KS Giovanello, JA Lin, D Shen, et al. Sex differences in grey matter atrophy patterns among AD and aMCI patients: results from ADNI. *Neuroimage*. 56, 2011; 890–906
- Smits, L. L., van Harten, A. C., Pijnenburg, Y. A., Koedam, E. L., Bouwman, F. H., Sistermans, N., ... & van der Flier, W. M. (2015). Trajectories of cognitive decline in different types of dementia. *Psychological Medicine*, 45(5), 1051-1059.
- Sundermann E., Rubin, L., Lipton, R., Landau, S., & Maki, P. Does the female advantage in verbal memory contribute to underestimating Alzheimer's disease pathology in women versus men? J Alzheimers Dis. 56, 2017; 947–957
- Soysal, P., Tan, S. G., Rogowska, M., Jawad, S., Smith, L., Veronese, N., Tsiptsios, D.,

Tsamakis, K., Stewart, R., & Mueller, C. (2021). Weight loss in Alzheimer's disease, vascular dementia and dementia with Lewy bodies: Impact on mortality and hospitalization by dementia subtype. *International journal of geriatric psychiatry*, *37*(2), 10.1002/gps.5659. Advance online publication.

- Stern, Y. (2012). Cognitive reserve in ageing and Alzheimer's disease. *The Lancet Neurology*, 11(11), 1006-1012.
- Stern, Y., Albert, S., Tang, M. & Tsai, W. (1999). Rate of memory decline in AD is related to education and occupation. Neurology, 53(9), 1942-1947.
- 10 Early Signs and Symptoms of Alzheimer's. (2020). Alzheimer's Association. Retrieved from https://www.alz.org/alzheimers-dementia/10_signs.
- 2019 Alzheimer's disease facts and figures. (2019). Alzheimer's Association. Retrieved from https://www.alz.org/media/documents/alzheimers-facts-and-figures-2019-r.pdf.
- Tarry-Adkins, J. L., & Ozanne, S. E. (2017). Nutrition in early life and age-associated diseases. Ageing Research Reviews, 39, 96-105.
- Tjahyo, A. S., Gandy, J., Porter, J., & Henry, C. J. (2021). Is Weight Loss More Severe in Older People with Dementia? *Journal of Alzheimer's disease: JAD*, 81(1), 57–73.
- Tsai, M. S., Tangalos, E. G., Petersen, R. C., Smith, G. E., Schaid, D. J., Kokmen, E., Ivnik, R. J., & Thibodeau, S. N. (1994). Apolipoprotein E: risk factor for Alzheimer disease. *American journal of human genetics*, 54(4), 643–649.
- Tschanz, J. T., Corcoran, C. D., Schwartz, S., Treiber, K., Green, R. C., Norton, M. C., Mielke, M. M., Piercy, K., Steinberg, M., Rabins, P. V., Leoutsakos, J. M., Welsh-Bohmer, K. A., Breitner, J. C., & Lyketsos, C. G. (2011). Progression of

cognitive, functional, and neuropsychiatric symptom domains in a population cohort with Alzheimer dementia: the Cache County Dementia Progression study. *The American journal of geriatric psychiatry: official journal of the American Association for Geriatric Psychiatry*, 19(6), 532–542.

- Tschanz, J. T., Norton, M. C., Zandi, P. P., & Lyketsos, C. G. (2013). The Cache County Study on Memory in Aging: Factors affecting risk of Alzheimer's disease and its progression after onset. *International Review of Psychiatry*, 25(6), 673–685.
- Tyas, S. L., Manfreda, J., Strain, L. A., & Montgomery, P. R. (2001). Risk factors for Alzheimer's disease: a population-based, longitudinal study in Manitoba, Canada. *International journal of epidemiology*, 30(3), 590-597.
- Van Duijn, C. M., Clayton, D., Chandra, V., Fratiglioni, L., Graves, A. B., Heyman, A., ... & Rocca, W. A. (1991). Familial aggregation of Alzheimer's disease and related disorders: a collaborative re-analysis of case-control studies. *International Journal of Epidemiology*, 20(2), S13-S20.
- Walker, Z., Allen, R. L., Shergill, S., Mullan, E., & Katona, C. L. (2000). Three years s survival in patients with a clinical diagnosis of dementia with Lewy bodies. *International Journal of Geriatric Psychiatry*, 15(3), 267-27
- Wang, X., Hu, J., & Wu, D. (2022). Risk factors for frailty in older adults. *Medicine*, 101(34), e30169. <u>https://doi.org/10.1097/MD.000000000030169</u>.
- Wang, Y., Beydoun, M. A., Min, J., Xue, H., Kaminsky, L. A., & Cheskin, L. J. (2020).
 Has the prevalence of overweight, obesity and central obesity levelled off in the United States? Trends, patterns, disparities, and future projections for the obesity epidemic. *International journal of epidemiology*, 49(3), 810-823.

Weir, C. B., & Jan, A. (2019). BMI classification percentile and cut off points.

- Wengreen HJ, Neilson C, Munger R, Corcoran C. (2009). Diet quality is associated with better cognitive test performance among aging men and women. *The Journal of Nutrition*. 139(10):1944–1949.
- What Is Alzheimer's Disease? (2017). *National Institute on Aging*. Retrieved from https://www.nia.nih.gov/health/what-alzheimers-disease.
- What Is Dementia? (2020). Retrieved from https://www.alz.org/alzheimers dementia/what-is-dementia.
- Whitmer RA, Sidney S, Selby J, Johnston SC, Yaffe K. (2005). Midlife cardiovascular risk factors and risk of dementia in late life. *Neurology*, *64*(2):277–81.
- Wilson, R. S., Gilley, D. W., Bennett, D. A., Beckett, L. A., & Evans, D. A. (2000).Person specific paths of cognitive decline in Alzheimer's disease and their relation to age. *Psychology and Aging*, *15*(1), 18.
- Wimo, A., Guerchet, M., Ali, G. C., Wu, Y. T., Prina, A. M., Winblad, B., Jönsson, L., Liu, Z., & Prince, M. (2017). The worldwide costs of dementia 2015 and comparisons with 2010. *Alzheimer's & dementia: The journal of the Alzheimer's Association*, 13(1), 1–7.
- Winter, J. E., MacInnis, R. J., Wattanapenpaiboon, N., & Nowson, C. A. (2014). BMI and all-cause mortality in older adults: A meta-analysis123. *The American Journal of Clinical Nutrition*, 99(4), 875–890.

https://doi.org/10.3945/ajcn.113.068122.

Wolters, F. J., van der Lee, S. J., Koudstaal, P. J., van Duijn, C. M., Hofman, A., Ikram,

M. K., ... & Ikram, M. A. (2017). Parental family history of dementia in relation to subclinical brain disease and dementia risk. *Neurology*, *88*(17), 1642-1649.

- Woo, J., Leung, J., & Kwok, T. (2007). BMI, Body Composition, and Physical Functioning in Older Adults. *Obesity*, 15(7), 1886–1894.
 https://doi.org/10.1038/oby.2007.223.
- Woodruff, R. C., Tong, X., Khan, S. S., Shah, N. S., Jackson, S. L., Loustalot, F., &
 Vaughan, A. S. (2024). Trends in cardiovascular disease mortality rates and
 excess deaths, 2010–2022. *American Journal of Preventive Medicine*, 66(4), 582-589
- World Health Organization. (2019). *Dementia, 2019*. Retrieved from https://www.who.int/newsroom/factsheets/detail/dementia#:~:text=Although%20 dementia%20mainly%20affects%20older,60%E2%80%9370%25%20of%20cases

. Alzheimer's disease facts and figures. Alzheimer's Dementia, 15, 321-387.

- World Health Organization (1997) *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation on Obesity.* Geneva: WHO.
- World Health Organization (1997) *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation on Obesity.* Geneva: WHO.
- Yaffe, K., Vittinghoff, E., Hoang, T., Matthews, K., Golden, S. H., & Zeki Al Hazzouri,
 A. (2021). Cardiovascular Risk Factors Across the Life Course and Cognitive
 Decline: A Pooled Cohort Study. *Neurology*, *96*(17).
 https://doi.org/10.1212/WNL.00 000 000 000 1 1747.
- Zahodne, L. B., Ornstein, K., Cosentino, S., Devanand, D., & Stern, Y. (2015).

Longitudinal relationships between Alzheimer's disease progression and psychosis, depressed mood and agitation/aggression. *The American Journal of Geriatric Psychiatry : Official Journal of the American Association for Geriatric Psychiatry*, 23(2), 130–140.

Zhang, Q., Guo, H., Gu, H., & Zhao, X. (2018). Gender-associated factors for frailty and their impact on hospitalization and mortality among community-dwelling older adults: A cross-sectional population-based study. *PeerJ*, 6, e4326. <u>https://doi.org/10.7717/peerj.4326</u>.

Yarlagadda, S., & Gupta, K. (2024, March 18). New treatment available for high blood pressure. The University of Kansas Health System. <u>https://www.kansashealthsystem.com/news-room/news/2024/03/new-treatmentavailable-for-high-blood</u> pressure#:~:text=%E2%80%9COver%20the%20past%2050%20to,4%20people%

20who%20have%20it.

APPENDIX A: BREAKDOWN OF OTHER DEMENTIA BY RESEARCH

QUESTION

Table A

Diagnoses Comprised in "Other" Diagnosis Group, by Research Question

Primary Diagnosis	Secondary Diagnosis	Research Question 1	Research Question 2	Research Question 3A_Males	Research Question 3A_Females	Research Question 3B
DUE	-	56	34	8	20	34
DUE	Other medical diagnosis	11	11	4	6	11
DUE	Vascular changes	10	5	4	4	5
DUE	Other neurologic diagnosis	7	5	-	-	5
DUE	Stroke	5	5	-	4	5
DUE	Mild Ambiguous Breitner criteria	4	-	-	-	-
DUE	Major Depression	4	3	1	1	3
DUE	Neuropsychiatric disorder	3	2	1	1	2
DUE	Severe head trauma with residual	3	2	2	-	2
DUE	Parkinson's	2	2	1	-	2
DUE	Other psychiatric	2	2	1	1	2
DUE	Alcoholism current	1	1	-	1	1
DUE	Alcoholism past	2	2	2	-	2
DUE	Head injury	1	-	-	-	-
DUE	Mental Retardation/Developmental Disability	1	1	-	-	1
DUE	ALS without dementia	1	1	1	-	1
Parkinson's	Parkinson's	7	6	4	2	1
Parkinson's	Possible Lewy Body	6	3	2	1	3
Parkinson's	Mild Ambiguous Breitner criteria	1	1	-	1	1
Parkinson's	No AD contribution	1	1	1	-	1

Primary Diagnosis	Secondary Diagnosis	Research Question 1	Research Question 2	Research Question 3A_Males	Research Question 3A_Females	Research Question 3B
Parkinson's	Stroke	1	_	-	-	_
Parkinson's	Other neurologic diagnosis	1	1	1	-	1
Parkinson's	Other medical diagnosis	1	1	1	-	11
Parkinson's	Mild Impairment	1	1	1	-	1
Parkinson's	Major Depression	3	3	2	1	3
Parkinson's	Vascular changes	2	2	2	-	2
Frontal lobe dementia	-	5	3	3	-	3
Frontal lobe dementia	Mild Ambiguous Breitner criteria	1	-	-	-	-
FTD tangle only subtype	-	2	2	1	1	2
FTD tangle only subtype	Vascular changes	1	1	-	1	1
FTD tangle only subtype	Definite Vascular Changes on neuropathology	1	1	-	1	1
FTD tangle only subtype	Other neuropathology	1	1	-	1	1
FTD on neuropath, PPA subtype	-	1	1	-	1	1
Dem Lacking distinctive Histology	-	2	-	-	-	-
PSP	-	1	1	1	-	1
PSP per neuropath	-	1	1	-	1	1
Major Depression	DUE	1	1	1	-	1
Major Depression	Mild Impairment	1	1	1	-	1
Neuropsychiatri c disorder	DUE	1	-	-	-	-

Primary Diagnosis	Secondary Diagnosis	Research Question	Research Question	Research Question	Research Question	Research Question
2 100100		1	2	3A_Males	3A_Females	3B
Severe head trauma with residual	-	1	1	-	-	1
Severe head trauma with residual	Vascular changes	1	1	1	-	1
Severe head trauma with residual	Other psychiatric	1	1	1	-	1
Alcoholic Dementia	Mild Ambiguous Breitner criteria	1	-	-	-	-
Hypoperfusion dementia	-	1	1	1	-	1
Hypoperfusion dementia	Mild Ambiguous Breitner criteria	1	-	-	-	-
Hypoperfusion dementia	Definite Vascular Changes on neuropathology	1	-	-	-	-
NPH	Definite Vascular Changes on neuropathology	1	-	-	-	-
Definite Lewy Body	-	1	-	-	-	-
Possible Lewy Body	-	1	1	1	-	1
Possible Lewy Body	Other psychiatric	1	1	1	-	1
Dem Lacking distinctive Histology	Major Depression	1	-	-	-	-
Dem Lacking distinctive Histology	Other neurologic diagnosis	1	-	-	-	-
Hippocampal sclerosis	-	1	1	1	-	1
Possible Picks Disease	-	1	-	-	-	-
Other neuropathology	DUE	1	1	-	1	1

Primary Diagnosis	Secondary Diagnosis	Research Question 1	Research Question 2	Research Question 3A_Males	Research Question 3A_Females	Research Question 3B
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Notes. DUE = dementia of undetermined etiology. FTD = frontotemporal dementia. NPH = normal pressure hydrocephalus, Dem = dementia, PSP = progressive supranuclear palsy, ALS = amyotrophic lateral sclerosis, AD = Alzheimer's disease.

APPENDIX B: DEMENTIA SEVERITY RATING SCALE

DATE

DRS

START TIME: :

NO 2

1.	START TIME: Memory 1-8
M1-	Normal.
M2-	Occasional forgetfulness of no real significance.
M3-	Frequent noticeable forgetfulness, but still has partial recollection of events.
M4-	Moderate memory loss, more marked for recent events, and severe enough to interfere with everyday activities.
M5-	Severe memory loss; only well-learned material retained, with newly-learned material rapidly lost.
M6-	Usually unable to remember simple facts such as the day of the week, month and/or year, when the last meal was eaten or the name of the next meal.
M7-	Unable to evaluate memory because of speech or language difficulty and/or inability to follow instructions.
M8-	Questions about memory not meaningful; makes no attempt to communicate and is no longer aware of surroundings.
2.	Orientation 1-6
01-	Normal.
02-	Some difficulty keeping track of dates and times, but not severe enough to interfere with everyday activities.
03-	Frequently disoriented to time and sometimes disoriented to new places.
04-	Almost always disoriented to time and usually disoriented to place.
05-	Unable to understand or answer questions about time of day or name of present location
06-	Is unaware of questions and makes no attempt to respond.
3.	Judgement 1-5
J1-	Normal.
J2-	Only questionable impairment in problem solving ability.
J3-	Moderate difficulty in handling complex problems, but social judgement is usually maintained.
J4-	Severe impairment in handling problems, social judgement impaired.
J5-	Unable to exercise judgement in either problem solving or social situations.
4.	Social Interactions/Community Affairs 1-5
C1	

S1- No alteration in ability to participate in community affairs.

1 L7- 1 R1- 1 R2- 0 R3- 2 R3- 2 R4- 0 R5- 0 R6- 1 1 1 F1- 1 F2- 1 F3- 1 F4- 1 F5- 1 10. 1	Speech usually unintelligible or irrelevant. Unable to understand or answer questions or to follow verbal instructions. No response, no longer attempts to communicate. Recognition of Others 1-6 Normal. Occasionally fails to recognize distant acquaintances or casual friends. Always recognizes family and close friends but frequently fails to recognize others. Occasionally fails to recognize family members and/or close friends. Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Feeding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
8. 1 R1- 1 R2- 0 R3- 1 R4- 0 R5- 0 R6- 1 F1- 1 F2- 1 F4- 1 F5- 1 10. 1	Recognition of Others 1-6 Normal. Occasionally fails to recognize distant acquaintances or casual friends. Always recognizes family and close friends but frequently fails to recognize others. Occasionally fails to recognize family members and/or close friends. Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Feeding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
R1- 1 R2- 0 R3- 1 R4- 0 R5- 0 R6- 1 9. 1 17 1 F1- 1 F2- 1 F3- 1 17 1 F4- 1 10. 1	1-6 Normal. Occasionally fails to recognize distant acquaintances or casual friends. Always recognizes family and close friends but frequently fails to recognize others. Occasionally fails to recognize family members and/or close friends. Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Feeding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
R2- 0 R3- 1 R4- 0 R5- 0 R6- 1 9. 1 1 1 F1- 1 F2- 1 1 1 F4- 1 F5- 1 1 1 10. 1	Occasionally fails to recognize distant acquaintances or casual friends. Always recognizes family and close friends but frequently fails to recognize others. Occasionally fails to recognize family members and/or close friends. Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Eceding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
R3- 1 R4- 0 R5- 0 R6- 1 9. 1 1 1 F1- 1 F2- 1 1 1 F3- 1 1 1 1 1 1 1 1 1	Always recognizes family and close friends but frequently fails to recognize others. Occasionally fails to recognize family members and/or close friends. Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Feeding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
R4- (R5- (R6- 1 9. 1 1 1 F1- 1 F2- 1 F3- 1 1 1 F4- 1 F5- 1 1 1	Occasionally fails to recognize family members and/or close friends. Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Ecceding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
R5- (R6- 1 9. 1 1 1 F1- 1 F2- 1 F3- 1 F4- 1 F5- 1 10. 1	Only occasionally recognizes spouse or caregiver. No recognition or awareness of the presence of others. Feeding 1-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
R6- I 9. I F1- I F2- I F3- I F4- I F5- I I I	No recognition or awareness of the presence of others.
9.] F1- 1 F2- 1 F3- 1 F4- 1 F5- 1 10.]	Feeding I-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
F1- 1 F2- 1 F3- 1 1 F3- 1 1 F4- 1 F5- 7 1 10. 1	I-5 Normal. May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
F2- 1 F3- 1 F4- 1 F5- 1 10. 1	May require help cutting food but otherwise able to eat independently. Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
F3- 1 1 F4- 1 F5- 7 1 10. 1	Mostly able to eat independently but may require some assistance. Occasionally uses fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
1 F4- 1 F5- 7 1 10. 1	fingers or wrong utensils. Requires assistance. Uses fingers more than utensils. Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
F5- 1 10.]	Totally dependent on others for feeding. May have difficulty swallowing or require feeding tube.
10.]	feeding tube.
1	Incontinence
a	1-5
C1- 1	Normal.
C2- 1	Loses control of bladder rarely (generally less than one accident per month).
C3- 1	Loses control of bladder an average of two or more times a month.
C4- 1	Frequently loses control of bladder despite help to toilet (more than once a week).
C5- 1	Total loss of bladder control.
	Mobility/Walking 1-7
W1- 1	Normal
	May occasionally have some difficulty driving or taking public transportation but fully independent for walking without supervision.
	Able to walk outside without supervision for short distances but unable to drive or take public transportation.
W4-	Able to walk about the home without supervision but cannot go outside unaccompanied
	Requires supervision within the home but able to walk without assistance (may use can or walker).
W6-	Generally confined to a bed or chair. May be able to walk a few steps with help.
	4

12. By placing a mark on the line please rate the degree if any that the subject's present condition interferes with <u>your</u> ability to carry on a normal life style.

1	2	3	4	5	6	7	8	9	10
no problem			some stress but tolerable			highly stressful			can no longer cope

APPENDIX C: MEDICAL HISTORY

SECTION D: MEDICAL HISTORY

Now I'm going to ask you some specific questions about your medical history, your medications and some other related things.

D1.	What is your current weight?	LBS
D2.	What is your current height?	
D3.	What is your current weight at 18?	LBS
D4.	What is your current height at 18?	
D5.	Has a doctor or nurse told you that you have Parkinson's Disease?	YES1 NO
D6.	Have you taken L-DOPA or Sinemet?	YES
D7.	Has a doctor or nurse told you that you had a stroke?	YES
D8.	How many strokes have you had in that interval?	NUMBER OF STROKES

Baseline Interview

D9. When did the (last) stroke take place?	MM YY
a. Did one side of your body, or one arm o leg, become weaker than the other?	r YES1 NO
b. Did you lose the ability to speak or understand what was said to you, for a da or more?	y YES
c. Did you see a doctor or go to a hospital?	SAW DOCTOR (RECORD)
RECORD NAME AND ADDRESS OF DOCTOR OR HOSPITAL.	
NAME:	
ADDRESS:	
#1 INTERVIEWER CHECKPOINT: IF D8=1 STRC TO D11)KE, GO
D10. When was the stroke before the last one?	

 a.
 Did one side of your body, or one arm or leg, become weaker than the other?
 YES
 1

 b.
 Did you lose the ability to speak or understand what was said to you, for a day or more?
 YES
 1

Baseline Interview

SECTION D: MEDICAL HISTORY

SECTION D: MEDICAL HISTORY

	c. Did you see a doctor or go to a hospital?	SAW DOCTOR (RECORD) 1 WENT TO HOSPITAL (RECORD) 2 NO DOCTOR OR HOSPITAL 3 RF 7 DK 8
	RECORD NAME AND ADDRESS OF DOCTOR OR HOSPITAL. NAME:	
	ADDRESS:	
D11.	Have you had a TIA, mini-stroke, or funny spell suggestive of a stroke?	YES 1 NO (GO TO D12) 2 RF (GO TO D12) 7 DK (GO TO D12) 8
	a. When did this first take place?	
D12.	Has a doctor or nurse told you that you have had a transient ischemic attack, TIA or mini-stroke?	YES 1 NO (GO TO D13) 2 RF (GO TO D13) 7 DK (GO TO D13) 8
	a. When did this first take place?	
	b. Did you see a doctor or go to a hospital?	SAW DOCTOR (RECORD) 1 WENT TO HOSPITAL (RECORD) 2 NO DOCTOR OR HOSPITAL 3 RF. 7 DK. 8
	RECORD NAME AND ADDRESS OF DOCTOR OR HOSPITAL.	
	NAME:	

Baseline Interview

	ADDRESS:	
D13.	Have you had a head injury so severe that you lost consciousness, lost your memory for a period of time, or had to see a doctor?	YES 1 NO (GO TO D17) 2 RF (GO TO D17) 7 DK (GO TO D17) 8
D14.	How many times did this happen?	# OF TIMES
D15.	Now I want you to think about your (last) head injury. How old were you at that time?	AGE
	a. Could you please describe the injury to me.	
	b. Did you see a doctor or go to a hospital?	SAW DOCTOR (RECORD) 1 WENT TO HOSPITAL (RECORD) 2 NO DOCTOR OR HOSPITAL 3 RF. 7 DK. 8
	RECORD NAME AND ADDRESS OF DOCTOR OR HOSPITAL.	WENT TO HOSPITAL (RECORD)
	RECORD NAME AND ADDRESS OF	WENT TO HOSPITAL (RECORD)
	RECORD NAME AND ADDRESS OF DOCTOR OR HOSPITAL. NAME:	WENT TO HOSPITAL (RECORD)

Baseline Interview

SECTION D: MEDICAL HISTORY

SECTION D: MEDICAL HISTORY

	30-59 MINS 3 1-24 HRS 4 >1 DAY 5 RF 7 DK 8
e. Sometimes, after a head injury, people experience amnesia or loss of memory. Did you have a period of amnesia after the injury?	YES
f. How long did you have this memory loss?	0-24 HRS
g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)?	YES 1 NO 2 RF 7 DK 8
INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN ONE HEAD INJURY IN D14?	YES (CONTINUE)
D16. Now I want you to think about your previous head injury. How old were you at that time?	AGE
a. Could you please describe the injury to me?	
b. Did you see a doctor or go to a hospital?	SAW DOCTOR 1 WENT TO HOSPITAL 2 NO DOCTOR OR HOSPITAL 3 RF 7 DK 8
RECORD NAME AND ADDRESS OF DOCTOR OR HOSPITAL.	
NAME:	
ADDRESS:	

Baseline Interview

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d. How long were you unconscious? <5 MINS d. How long were you unconscious? <5 MINS 30-59 MINS 30-59 MINS 1-24 HRS 1 DAY RF DK bit you have a period of amnesia after the injury? NO (GO TO g) f. How long did you have memory loss? VES f. How long did you have memory loss? 0-24 HRS g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YES INTER VIEWER CHECKPOINT: DID R. REPORT YES (GO TO HEAD INJ. SUPP.)NO (GO TO DIS) MORE THAN 2 HEAD INJURIES IN D14? YES (SPECIFY)NO (GO TO B) D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? YES (SPECIFY)			UTO
RF (GO TO e) DK (GO TO e) d. How long were you unconscious? <5 MINS	C	e. Dia you lose consciousness?	
d. How long were you unconscious? <5 MINS.			RF (GO TO e)
5-29 MINS 30-59 MINS 1-24 HRS >1 DAY RF DK (GO TO g) RF MOR (GO TO g) RF DK (GO TO g) RF DK RF DK RF G How long did you have memory loss? 0-24 HRS 2-6 DAYS >1 WEEK RF DK g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? MORE THAN 2 HEAD INJURIES IN D14? VES (GO TO HEAD INJ. SUPP.) NO (GO TO D18) NO (GO TO D18) SPECIFY: SPECIFY:			DK (GO TO e)
30-59 MINS 1-24 HRS >1 DAY RF DK e. Sometimes, after a head injury, people experience amnesia or loss of memory. Did you have a period of amnesia after the injury? f. How long did you have memory loss? f. How long did you have memory loss? g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? INTER VIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? SPECIFY:	ć	l. How long were you unconscious?	<5 MINS
1-24 HRS			5-29 MINS
>1 DAY RF. DK. e. Sometimes, after a head injury, people experience amnesia or loss of memory. Did you have a period of amnesia after the injury? f. How long did you have memory loss? f. How long did you have memory loss? 0-24 HRS. 2-6 DAYS >1 WEEK. RF. DK. g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? SPECIFY:			
RFDK. e. Sometimes, after a head injury, people experience amnesia or loss of memory. Did you have a period of amnesia after the injury? YES. f. How long did you have memory loss? 0-24 HRS. f. How long did you have memory loss? 0-24 HRS. g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YES. INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? YES (GO TO HEAD INJ. SUPP.)			
e. Sometimes, after a head injury, people experience amnesia or loss of memory. Did you have a period of amnesia after the injury? YES f. How long did you have memory loss? 0.24 HRS. f. How long did you have memory loss? 0.24 HRS. g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YES INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? YES (GO TO HEAD INJ. SUPP.). D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? YES (SPECIFY). SPECIFY:			RF
experience amnesia or loss of memory. Did you have a period of amnesia after the injury? f. How long did you have memory loss? f. How long did you have memory loss? g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? INTER VIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? SPECIFY: 			DK
Did you have a period of amnesia after the injury? RF (GO TO g)DK (GO TO D18)DK (GO TO D18)	e	. Sometimes, after a head injury, people	YES
injury? DK (GO TO g)		experience amnesia or loss of memory.	NO (GO TO g)
f. How long did you have memory loss? 0-24 HRS. g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YES. INTERVIEWER CHECKPOINT : DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? YES (GO TO HEAD INJ. SUPP.)			
2-6 DAYS >1 WEEK. RFDK. g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? SPECIFY: 		injury?	DK (GO TO g)
>1 WEEK. g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YES INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? YES (GO TO HEAD INJ. SUPP.)	f	How long did you have memory loss?	0-24 HRS
RFDK. DK g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YESNO			
g. At the time of this injury was there any penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? YES INTERVIEWER CHECKPOINT: DID R. REPORT MORE THAN 2 HEAD INJURIES IN D14? YES (GO TO HEAD INJ. SUPP.) D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? YES (SPECIFY) SPECIFY:			
penetration of the skull to the brain (e.g. such as from shrapnel, a bullet wound, or other object)? NO			DK.
such as from shrapnel, a bullet wound, or other object)? RFDK	Ę	. At the time of this injury was there any	YES
other object)? DK INTERVIEWER CHECKPOINT: DID R. REPORT YES (GO TO HEAD INJ. SUPP.) MORE THAN 2 HEAD INJURIES IN D14? NO (CONTINUE) D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? YES (SPECIFY) SPECIFY:			NO
INTERVIEWER CHECKPOINT: DID R. REPORT YES (GO TO HEAD INJ. SUPP.)			
MORE THAN 2 HEAD INJURIES IN D14? NO (CONTINUE) D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? NO (GO TO D18) SPECIFY: 		other object)?	
D17. Have you had brain surgery, a blast injury, or any other kind of brain injury such as a hematoma (blood clot on the brain)? SPECIFY:			YES (GO TO HEAD INJ. SUPP.)
other kind of brain injury such as a hematoma (blood clot on the brain)? SPECIFY:			
(blood clot on the brain)? RF (GO TO D18) SPECIFY:			
SPECIFY:			
SPECIFY:	(blood clot on the brain)?	
a. How old were you when this happened?	S	PECIFY:	
a. How old were you when this happened?			
	a	. How old were you when this happened?	AGE
		- •••	

Baseline Interview

SECTION D: MEDICAL HISTORY

	JI1 D.	MEDICAL HISTORY	
D18.	Hav fits?	e you had a new onset of epileptic seizures or	YES 1 NO (GO TO D19) 2 RF (GO TO D19) 7 DK (GO TO D19) 8
	a.	How old were you when you had your first seizure?	AGE
	b.	Did you take medication for this?	YES
	C.	How long were you on the seizure medication?	YEARS
D19.		you ever box regularly as an amateur or essional?	YES
	a.	How old were you when you began to box regularly?	AGE
	b.	How old were you when you stopped boxing regularly?	AGE
	C.	Were you ever knocked unconscious?	YES 1 NO (GO TO D20) 2 RF (GO TO D20) 7 DK (GO TO D20) 8
	d.	How many times were you knocked unconscious?	# TIMES

Baseline Interview

D20.	Now I'd like you to tell me how well you're doing with several routine daily activities. Do you need assistance with bathing or grooming (for example, running bath water, getting into or out of the tub, or washing body and hair)?	YES
D21.	Can you walk long distance (>1 BLOCK) without any sort of assistance?	YES (GO TO D22)
	a. Do you use a cane, a walker, or some other form of assistance?	YES
D22.	Do you need help traveling beyond walking distance (such as using the bus system or driving yourself)?	YES
D23.	Do you need help getting into or out of a bed or a chair?	YES
D24.	Do you need help using the restroom (including adjusting clothing or getting onto or off of the toilet)?	YES
D25.	Do you need reminders to use the restroom?	YES
D26.	Do you need help preparing meals for yourself (for example a hot meal, a sandwich, a TV dinner or microwaving food)?	YES
D27.	Do you need assistance with eating (for example, serving your food, using utensils, or drinking from a glass or cup)?	YES
D28.	Do you need assistance doing the laundry? This includes putting clothes into the washer and dryer, starting the machines, and unloading them when they're finished.	YES
D29.	Do you need help doing light housework such as dusting, washing dishes, or sweeping?	YES
D30.	Do you need help doing heavier chores such as vacuuming, yard work, moving furniture, or scrubbing?	YES
D31.	Do you need any kind of assistance using the	YES 1

Baseline Interview

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SECTION D: MEDICAL HISTORY

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	telephone, either answering the phone or placing calls? (This would include an amplifier, larger push button numbers, or preprogrammed phone numbers.)	NO (GO TO D32)
	a. What type of equipment?	
	RECORD:	
		EQUIP
D 32.	Do you need assistance with any kind of shopping (not including help with transportation)?	YES
	a. Do you need assistance with shopping for groceries and prescriptions?	YES
	b. Do you need assistance with shopping for things other than groceries and prescriptions?	YES
D33.	Do you need assistance or reminders to take your medications?	YES
D34.	Do you need help managing your finances, such as paying the bills or balancing your checkbook?	YES
D35.	Do you feel that you have enough contacts with other people or do you wish that you had more?	ENOUGH
D36.	This next group of questions is about cardiovascular or heart problems. Have you had a severe pain across the front of your chest lasting for a half an hour or more?	YES
D37.	Have you been told you had angina?	YES
D38.	Have you been troubled by shortness of breath when walking up a slight hill or when hurrying on level ground?	YES 1 NO 2 RF 7 DK 8
D39.	Have you ever had high blood pressure or been	YES

Baseline Interview

SECTION D: MEDICAL HISTORY

34

	told that you had hypertension?		NO(GO TO D40)
	a.	How old were you when you first learned you had high blood pressure?	AGE
	b.	Did a doctor evaluate your high blood pressure or prescribe treatment?	YES, EVALUATED
	C.	Who was the doctor who evaluated your high blood pressure?	
	NAI	ME:	
	ADI	DRESS:	
	d.	Do you have high blood pressure now or are you under current treatment for high blood pressure?	YES 1 NO 2 RF 7 DK 8
D40.		re you been told you had high blood lesterol triglycerides (lipids)?	YES
	e.	How old were you when you first learned you had high cholesterol or blood lipids?	AGE
	f.	Did a doctor evaluate your high cholesterol or blood lipids, or prescribe treatment?	YES, EVALUATED

Baseline Interview

2	ŀ	٢
-	۱.	2

	g.	Who was the doctor who evaluated your high cholesterol or blood lipids?	
	NAI	ME:	
	ADI	DRESS:	
	h.	Do you have high cholesterol or blood lipids now or are you currently under treatment for this condition?	YES
D41.	Hav	e you had coronary bypass surgery)?	YES 1 NO (GO TO D42) 2 RF (GO TO D42) 7 DK (GO TO D42) 8
	a.	How old were you when you first had a coronary bypass?	AGE
	Ъ.	How many of these operations have you had?	# OPERATIONS
	c. NAI ADI	Who performed the (last) bypass surgery? ME:	
D42.		e you ever had a heart attack, a myocardial rction, or a coronary thrombosis?	YES 1 NO (GO TO D43) 2 RF (GO TO D43) 7 DK (GO TO D43) 8
	a.	How old were you when you first had a heart attack (coronary)?	AGE

Baseline Interview

b. How many heart attacks have you had?	# HEART ATTACKS
c. Who was the (last) doctor who treated you for a heart attack?	
NAME:	
ADDRESS:	
D43 Have you ever had diabetes, high blood sugar or sugar in your urine?	YES1 NO (GO INT. CHECKPOINT)2
a. Did a doctor diagnose this condition?	YES
D44. What sort of treatment was prescribed for your diabetes?	DIET ONLY 1 ORAL TREATMENT (PILLS) 2 INSULIN 3 NONE 4 RF. 7 DK 8
INTERVIEWER CHECKPOINT: IS R. FEMALE?	YES (CONTINUE)
D45. Have you ever taken calcium supplements?	YES
a. For how many years?	YEARS (ROUND TO NEAREST INTEGER) RF
D46. Have you ever taken estrogen supplements?	YES 1 NO (GO TO E1) 2 RF (GO TO E1) 7 DK (GO TO E1) 8
a. For how many years?	YEARS (ROUND TO NEAREST INTEGER) RF

Baseline Interview

3/16/95



Cache County Study on Memory, Health & Aging Code Book

MEDICAL DIAGNOSIS FINAL 10/21/04

1.	Stroke, single, uncomplicated
2.	Stroke, single with aphasia or hemiparesis
З.	Strokes, multiple, uncomplicated
4.	Strokes, multiple with aphasia or hemiparesis
5.	Binswanger's disease
6.	Parkinson's Disease
7.	Mental retardation, any cause
8.	Hydrocophalus Normal prossure
9.	Cerebritis, any cause
10.	Cerebral palsy
11.	Hypoxic exposure acute
12.	Amyotrophic lateral sclerosis
13.	Myotonic dystrophy
14.	Multiple sclerosis
15.	B12 deficiency
16.	Seizure disorder
17.	Encephalopathy, any cause
18.	Tumor Brain, non-malignant
19.	Tumor Brain, primary malignant
20.	Tumor Brain, metastatic
20.	Hoad injury/trauma S/P
22.	Transient ischemic attack (single)
23.	Transient ischemic attack (multi)
23.	Neurotoxin Exposure
24.	Depression, major
25.	Bipolar disorder
20.	Schizophrenia
28.	Anxiety disorder
29.	Abuse/dependence, alcohol
30.	Abuse/dependence, alconor Abuse/dependence, substance
31.	Abusedependence, substance Psychosis or "breakdown", NOS
32.	Hypertension
33.	Peripheral vascular disease/claudication/ASVD
34.	Congestive heart failure
35.	Angina/CAD
36.	Myocardial infarction, single
37.	Myocardial infarction, single Myocardial infarction, multiple
38.	Cardiomyopathy
39.	Atrial fibrillation
40. 41.	Arrhythmia, othor atrial Arrhythmia, other ventricular
42.	Valvular heart disease
	CABG S/P
44.	Hyperlipidemia/ high cholesterol
45.	Carotid endarterectomy, S/P
46.	Chronic obstructive pulmonary disease, not oxygen-dependent
47.	Chronic obstructive pulmonary disease, oxygen-dependent
48.	Fibrosis, pulmonary
49.	Asthma
50.	Pulmonary Carcinoma Primary
51.	Rhinitis allergic /hay fever

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Cache County Study on Memory, Health & Aging Code Book

52.	Pneumonia	
53.	Tuberculosis	
54.	Bronchiectasis/chronic bronchitis	
55.	Irritable bowel syndrome/spastic colon/chronic colitis	
56.	Crohn's disease/Ulcerative colitis	
57.	Pancreatitis, acute	
58.	Pancreatitis, chronic	
59.	Hopatitis, acuto	
60.	Hepatitis, chronic	
61.	Gastric Carcinoma	
62.	Colon Carcinoma	
63.	Hepatic Carcinoma	
64.	Pancreatic Carcinoma	
65.	PUD	
66.	Renal insufficiency, chronic	
67.	Renal tailure, on hemodialysis	
68.	Renal failure, on peritoneal dialysis	
69.	Renal Carcinoma	
70.	Pyelonephritis, chronic	
71.	Nephrolithiasis	
72.	Kidney problem, other	
73.	Cystitis, occasional	
74.	Cystitis, chronic/recurrent	
75.	Fibroid, uterine	
76.	Hysterectomy	
77.	Opphorectomy	
78.	Prostatitis	
79.	Prostate Carcinoma	
80.	Breast Carcinoma	
81.	Diabetes Mellitus, diet controlled	
82.	Diabetes Mellitus, oral hypoglycemics	
83.	Diabetes Mellitus, insulin-dependent	
84.	Hypothyroidism	
85.	Hyperthyroidism	
86.	Diabetic Coma, Hx	
87.	Folate deficiency	
88.	Anemia, any cause	
89.	Gout	
90.	Arthritis rheumatoid	
91.	Tendonitis/Bursitis	
92.	Autoimmune disorder other severe (Sjogren's Scleroderma, etc.)	
93.	Systemic lupus erythematosus	
94.	Arthritis Osteoarthritis/Degenerative disc disease/Degenerative joint disease	
95.	Macular Degeneration	
96.	Neurosyphilis	
97.	HIV There have been with a track the second second second	
98.	Tuberculosis with extra-pulmonary involvement	
99.	Leukemia/blood dyscrasia/lymphoma	
100.	DVT	
101.	Embolism, pulmonary	
102	Sleep Apnea	

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104	Hudessenhalus Other
104.	Hydrocephalus Other
105.	Cirrhosis GERD
106.	Glaucoma
107.	
109.	CNS vasculitis Endometrial Carcinoma
1109.	UTI. chronic
111.	
112.	AD Rx by Subject's MD
112.	Dementia Senile /Hardening of the arteries Dementia Vascular
114.	Narcolopsy
114.	Subdural Hematoma
115.	Dementia Frontal Lobe
117.	Dementia Alcoholic
118.	Dementia Lewy Body
119.	Dementia
120.	Denentia Meniere's Disease
120.	Arteritis Temporal
121.	Carpal Tunnel
122.	Aneurism Cerebral
123.	Hemorrhage Intracerebral
125.	Delirium
126.	A.V. Malformation
127.	Pacemaker
128.	Cardiac Arrest
129.	Pericarditis
130.	Carotid Arterial Disease
131.	Neuropathy Peripheral
132.	Aneurism, abdominal aortic
133.	Andurism, adrtic
134.	Tachycardia
135.	Angioplasty/stent replacement
136.	Greenfield Filter. S/P
137.	Cholecystectomy
138.	Hepatitis A
139.	Hepatitis B
140.	Hepatitis C
141.	Hepatitis unspecified
142.	Gastritis
143.	GI Blood
144.	Uterine Carcinoma
145.	Benign Prostate Hypertrophy
146.	Bladder Carcinoma
147.	Ovarian Careinoma
148.	Psoriasis
149.	Lyme Disease
150.	Polio
151.	Gangrene
152.	Fever Rheumatic
153.	Decubitus
154.	Malignant Melanoma
155.	Head/Neck Carcinoma

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APPENDIX D: MEDICAL CONDITION GROUPINGS

Table D

Grouping	Individual Conditions			
Conditions of the Brain	A.V. Malformation			
	Aneurism Cerebral			
	Cerebritis, any cause			
	CNS vasculitis			
	Encephalopathy, any cause			
	Hydrocephalus, Other			
	Hypoxic exposure acute			
	Neurotoxin Exposure			
	Tumor Brain, non-malignant			
	Multiple sclerosis			
	Seizure disorder			
	Cerebral palsy			
	HIV			
	Neurosyphilis			
	Cerebral aneurism			
Arrhythmias and Related	Arrhythmia, NOS			
Procedures	Arrhythmia, other atrial			
	Arrhythmia, other ventricular			
	Pacemaker			
	Pacer/Defibrillator			
	Atrial fibrillation			
Major Cardiac	Angina/CAD			
	Angioplasty/stent replacement			
	CABG S/P			
	Myocardial infarction, multiple			
	Myocardial infarction, single			
	Carotid Arterial Disease			
	Carotid endarterectomy, S/P			
	DVT			
	Peripheral vascular disease/claudication/ASVD			
Cardiac, Minor	Cardiac Arrest			
	Cardiomyopathy			
	Pericarditis			

Breakdown of medical conditions comprised in the medical groups

	119
	Tachycardia
	Valvular heart disease
	Aneurism, aortic
Congestive Heart Failure	
Hyperlipidemia	
Hypertension	
Cancer	Basal Cell Carcinoma
	Bladder Carcinoma
	Breast Carcinoma
	Colon Carcinoma
	Head/Neck Carcinoma
	Leukemia/blood dyscrasia/lymphoma
	Malignant Melanoma
	Ovarian Carcinoma
	Prostate Carcinoma
	Renal Carcinoma
	Uterine Carcinoma
Depression	
Psychiatric	Abuse/dependence, alcohol
	Abuse/dependence, substance
	Anxiety disorder
	Bipolar disorder
	Psychosis or "breakdown", NOS
Autoimmune	Arthritis rheumatoid
	Autoimmune disorder other severe (Sjogren's Scleroderma,
	etc.)
	Fever Rheumatic
	Systemic lupus erythematosus
Chronic Pain	Arthritis Osteoarthritis/Degenerative disc
	disease/Degenerative joint disease
	Carpal Tunnel
	Fibromyalgia/Chronic Fatigue Syndrome
	Osteoporosis
	PMR
	Scoliosis
	Tendonitis/Bursitis
	Gout
Gastric/Digestive	Crohn's disease/Ulcerative colitis

	GERD		
	Irritable bowel syndrome/spastic colon/chronic colitis		
	PUD		
	Pancreatitis, acute		
	Hepatitis C		
	Hepatitis unspecified		
Pulmonary	Asthma		
	Bronchiectasis/chronic bronchitis		
	Chronic obstructive pulmonary disease, not oxygen- dependent		
	Chronic obstructive pulmonary disease, oxygen-dependent		
	Embolism, pulmonary		
	Fibrosis, pulmonary		
	Pneumonia		
	Tuberculosis		
Urologic	Benign Prostate Hypertrophy		
	Cystitis, chronic/recurrent		
	Cystitis, occasional		
	Kidney problem, other		
	Nephrolithiasis		
	Prostatitis		
	Pyelonephritis, chronic		
	Renal insufficiency, chronic		
	UTI, chronic		
Thyroid	Hyperthyroidism		
	Hypothyroidism		
Diabetes	Diabetes Mellitus, diet-controlled		
	Diabetes Mellitus, insulin-dependent		
	Diabetes Mellitus, oral hypoglycemics		
Blood Deficiencies	Anemia, any cause		
	B12 deficiency		

APPENDIX E: NEUROPSYCHIATRIC INVENTORY

Completed: YES...1 NO...2

NPI

A. <u>Delusions</u> In the last month, has (NAME) had beliefs that you know are not true? For example, insisting that people are trying to harm (HIM/HER) or steal from (HIM/HER). Has (s/he) said that family members are not who they say they are or that the house is not (HIS/HER) home? I'm not asking about mere suspiciousness; I am interested if (NAME) is convinced that these things are happening to (HIM/HER).

		V		<u> </u>				
YES		NO (SKIP TO B) 2	IV		8	NA (SKII	PTOB).	9
				YES	NO	DK	IV	NA
A1.		eve that (s/he) is in danger to hurt (HIM/HER)?	that	1	2	7	8	9
A2.	Does (NAME) belie (HIM/HER)?	eve that others are stealing i	îrom	1	2	7	8	9
A3.	Does (NAME) belie having an affair?	eve that (HIS/HER) spouse	is	1	2	7	8	9
A4.	Does (NAME) belie living in (HIS/HER)	eve that unwelcome guests :) house?	are	1	2	7	8	9
A5.	Does (NAME) belie are not who they cla	eve that (HIS/HER) spouse tim to be?	or others	1	2	7	8	9
A6.	Does (NAME) believe that (HIS/HER) house is not (HIS/HER) home?				2	7	8	9
A7.	Does (NAME) believe that family members plan to abandon (HIM/HER)?				2	7	8	9
A8.	Does (NAME) believe that television or magazine figures are actually present in the home? (Does (s/he) try to talk or interact with them?)				2	7	8	9
A9.	Does (s/he) believe haven't asked about	any other unusual things th ?	at I	1	2	7	8	9
	Specify		YES SPE	CIFY				IF
Ifthe	screening question i	s confirmed, determine the	frequency	and seve	rity of t	lhe delusion	ns.	
A10. Frequency: Occasionally - less than once per week. Often - about once per week. <u>Frequently</u> - several times per week but less than every day. <u>Very Frequently</u> - once or more per day.			OFT FRE	EN QUENI	ΊΑL ΓLΥ TLY			
A11.	Severity: <u>Mild</u> - delusior	as present but seem harmles	ss and	MIL	D			

Date

produce little distress in the subject. <u>Moderate</u> - delusions are distressing and disruptive. <u>Marked</u> - delusions are very disruptive and are a major source of behavioral disruption. (If PRN medications are prescribed, their use signals that the delusions are of marked severity).	MODERATE
 A12. Do these problems represent a change from the way (s/he) has always been? <u>Yes</u> - they represent a clear change. <u>Exaggeration</u> - They are an exaggeration of previous problems. No - they represent no change (life long characteristics). Don't know. 	YES

B. Hallucinations

In the last month, did (NAME) have hallucinations such as false visions or voices? Does (s/he) seem to see, hear or experience things that are not present? By this question we do not mean just mistaken beliefs such as stating that someone who has died is still alive; rather we are asking if (NAME) actually has abnormal experiences of sounds or visions.

YES.	1	NO (SKIP TO C)2	IV	8	NA	(SKIP	го с)		9
				YI	ES	NO	DK	IV	NA
B1.	Does (NAME) hears voices?	describe hearing voices	or act as if (s/he)	1		2	7	8	9
B2.	Does (NAME)	talk to people who are n	iot there?	1		2	7	8	9
B3.	or behave as if	describe seeing things n (s/he) is seeing things n ls, lights, etc.)?		1		2	7	8	9
B4.	Does (NAME) others?	report smelling odors no	ot smelled by	1		2	7	8	9
B5.		describe feeling things o ise appear to be feeling the HER)?		1		2	7	8	9
B6.	Does (NAME) cause?	describe tastes that are v	without known	1		2	7	8	9
B7.	Does (NAME) experiences?	describe any other unus	ual sensory	1		2	7	8	9
Speci	fy					I	F YES S	PECIFY	
If the	screening questic	m is confirmed, determir	ne the frequency ar	nd sev	erity	of the h	allucinat	ions.	
B8.	<u>Often</u> - about <u>Frequently</u> - s every day.	- less than once per week once per week. several times per week bu <u>tly</u> - once or more per da	ut less than	OFTI FRE(EN QUE	NTLY			2
B9.	cause little di: <u>Moderate</u> - ha disruptive to t	nations are present but h stress for the subject. Illucinations are distressi the subject. ucinations are very disru	ing and are	MOL	ER	ATE			2

major source of behavioral disruption. (PRN medications may be required to control them).	
Do these problems represent a change from the way s/he) has always been? <u>Y es</u> - they represent a clear change. <u>Exaggeration</u> - they are an exaggeration of previous problems. <u>No</u> - they represent no change (life long characteristics). <u>Don't know</u> .	YES

C. Agitation/Aggression

In the last month, has (NAME) had periods when (s/he) refuses to cooperate or won't let people help (HIM/HER)? Is (s/he) hard to handle?

YES	1	NO (SKIP TO D) 2	IV8			NA (SKIP T	0 D) 9
				YES	NO	D K	IV	NA
C1.		get upset with those tryi resist activities such as		1	2	7	8	9
C2.	Is (NAME) stu way?	ibbom, having to have th	ings (HIS/HER)	1	2	7	8	9
C3.	Is (NAME) un	cooperative, resistive to i	help from others?	1	2	7	8	9
C4.	Does (NAME) (HIM/HER) ha	have any other behavior ard to handle?	rs that make	1	2	7	8	9
C5.	Does (NAME)	shout or curse angrily?		1	2	7	8	9
C6.	Does (NAME)	slam doors, kick furnitu	re, throw things?	1	2	7	8	9
C7.	Does (s/he) att	empt to hurt or hit others	;?	1	2	7	8	9
C8.	Does (s/he) hav behaviors?	ve any other aggressive (or agitated	1	2	7	8	9

Specia	fy		IF
•	-	YES SPECIFY	
If the	screening question is confirmed, determine the freque	ncy and severity of the agitation.	
C9.	Frequency: <u>Occasionally</u> - less than once per week. Often - about once per week. <u>Frequently</u> - several times per week but less than daily. Very Frequently - once or more per day.	OCCASIONAL OFTEN. FREQUENTLY VERY FRQTLY	2
C10.	Severity: Mild - behavior is disruptive but can be managed with redirection or reassurance. <u>Moderate</u> - behaviors disruptive and difficult to redirect or control. <u>Marked</u> - agitation is very disruptive and a major source of difficulty; there may be a threat of personal harm. Medications are often required.	MILD MODERATE MARKED	2

 C11. Do these problems represent a change from the way (s/he) has always been? <u>Yes</u> - they represent a clear change. Exaggeration - they are an exaggeration of previous problems. <u>No</u> - they represent no change (life long characteristics). <u>Don't know</u>. 	YES
---	-----

D. Depression/Dysphoria

In the last month, did (NAME) seem sad or depressed? Does (NAME) say that (s/he) feels sad or depressed?

Y ES		NO (SKIP TO E)	2	IV		8		NA (SKIPT	°E)9
					YES	NO	DK	IV	NA
D1.	Is (NAME) currently b	eing treated for clinic	al depressio	n?	1	2	7	8	9
D2.	. Does (NAME) have periods of tearfulness or sobbing that seem to indicate sadness?			1	2	7	8	9	
D3.	Does (NAME) say or act as if (s'he) is sad or in low spirits?		1	2	7	8	9		
				IF NO, DI	ς IV, NA (G	O TO DS)			
D4.	Has that been for >2 weeks?		1	2	7	8	9		
D5.	5. Has (s/he) been irritable?		1	2	7	8	9		
				IF NO, DH	ς, IV, ΝΑ (G	O TO D7)	10	1	
D6.	Has that been for >2	weeks?			1	2	7	8	9
D7.	Does (NAME'S) moo	d change a lot from da	y to day?		1	2	7	8	9
D8.	Does (NAME) put (HIM/HER)self down or say that (s/he) feels like a failure?			1	2	7	8	9	
D9.	Does (NAME) say that (s/he) is a bad person or deserves to be punished?			1	2	7	8	9	
D10.	Does (NAME) seem to be discouraged or say that (s/he) has no future?		1	2	7	8	9		
D11.	Does(s/he) feel that li	fe is not worthwhile?			1	2	7	8	9
D12.	Does (NAME) say tha family would be better			r that the	1	2	7	8	9
D13.	Does (s/he) feel worth	less?			1	2	7	8	9
D14.	Does (NAME) expres: (HIM/HER)self?	s a wish for death or ta	lk about kil	ling	1	2	7	8	9
D15.	Has (s/he) tried to com	nmit suicide in the past	six months	:?	1	2	7	8	9
If the sc	reening question is confirm	ed, determine the frequer	ncy and seve	rity of the depress	sion				
D16.	Frequency: Occasionally - less th <u>Often</u> - about once p <u>Frequently</u> - several tin <u>Very Frequently</u> - es	er week. mes per week but less		day.	OF FR	CCASION TEN EQUENT RY FRQI	LY		2 3
D17.	reassurance. Moderate - depressio spontaneously voiced	Very Frequently - essentially continuously present. Severity: Mild - depression is distressing but usually responds to redirection or reassurance. Moderate - depression is distressing, depressive symptoms are spontaneously voiced by the subject and difficult to alleviate. Marked - depression is very distressing and a major source of suffering				ILD ODERA TE ARKED	5		

D18.	Do these problems represent a change from the way (s/he) has always	
	been?	
	<u>Yes</u> - they represent a clear change.	YES 1
	Exaggeration - They are an exaggeration of previous problems.	EXG. OF PROB 2
	No - they represent no change (life long characteristics).	NO
	Don't know.	DK

E. <u>A pathy/Indifference</u>

In the last month, has (NAME) lost interest in the world around (HIM/HER)? Has (s/he) lost interest in doing things or lacked motivation for starting new activities? Is (s/he) more difficult to engage in conversation or in doing chores? Is (NAME) apathetic or indifferent?

Y ES		8	NA (SKIP TO F)			9
		YES	NO	DK	IV	NA
E1.	Does (NAME) seem less spontaneous and less active than usual?	1	2	7	8	9
E2.	Is (NAME) less likely to initiate a conversation?	1	2	7	8	9
E3.	Is (NAME) less affectionate or lacking in emotions when compared to (HIS/HER) usual self?	1	2	7	8	9
E4.	Does (NAME) contribute less to household chores?	1	2	7	8	9
E5.	Does (NAME) seem less interested in the activities and plans of others?	1	2	7	8	9
Еб.	Has (NAME) lost interest in friends and family members?	1	2	7	8	9
E7.	Is (NAME) less enthusiastic about (HIS/HER) usual interests?	1	2	7	8	9
E8.	Has (s/he) lost interest in almost everything?	1	2	7	8	9
		IFN), DK, IV, 1	NA,(GO T	'O E10)	
E9.	Has that been for >2 weeks?	1	2	7	8	9
E10.	Has (s/he) lost pleasure in everything?	1	2	7	8	9
		IFN	IF NO, DK, IV, NA, (GO TO E12)			
E11.	Has that been for >2 weeks?			7	8	9
E12.	Does (NAME) show any other signs that (s/he) doesn't care about doing new things?	1	2	7	8	9
Specify		I	F YES SI	PECIFY		
If the sc	reening question is confirmed, determine the frequency and severity of the apathy/in	lifferer	ice.			
E13.	Often - about once per week. OFTEN Frequently - several times per week but less than every day. FREQU	i jentl	L Y .Y			2 3
E14.	routines, only mildly different from subject's usual behavior; subject MODE	MILD MODERATE MARKED			2	
E15.	Do these problems represent a change from the way (s/he) has always been? Yes - they represent a clear change. YES					1

Exaggeration-they are an exaggeration of previous problems.	EXG. OF PROB
<u>No</u> - they represent no change (life long characteristics).	NO
<u>Don't know</u> .	DK

F. Elation/Euphoria

In the last month, has (NAME) seemed too cheerful or too happy for no reason? I don't mean the normal happiness that comes from seeing friends, receiving presents, or spending time with family members. I am asking if (s/he) has a persistent and <u>abnormally</u> good mood or finds humor where others do not.

Y ES		NO (SKIP TO G)2	IV		8	NA (SKIP T	'O G)	
				YES	NO	DK.	IV	NA
F1.	Does (NAME) appear from (HIS/HER) usual	to feel too good or too happy, d . self?	ifferent	1	2	7	8	9
F 2.	Does (NAME) find humor and laugh at things that others do not find funny?			1	2	7	8	9
F3.	Does (NAME) seem to have a childish sense of humor with a tendency to giggle or laugh inappropriately (such as when something unfortunate happens to others)?		1	2	7	8	9	
F4.	Does (NAME) tell jok others but seem funny	es or make remarks that have littl to (HIM/HER)?	e humor for	1	2	7	8	9
F 5.	Does (s/he) play child: away" for the fun of it	sh pranks such as pinching or pla ?	ying"keep	1	2	7	8	9
Fб.	Does (s/he) "talk big" or claim to have more abilities or wealth than is true?		1	2	7	8	9	
F7.	Does (NAME) show any other signs of feeling too good or being too happy?			1	2	7	8	9
Specif	ÿ			IF YES S	SPECIFY			
If the s	screening question is conf	irmed, determine the frequency a	nd severity of	the elation	/euphoria.			
F8.			OF y day. FRI	TEN EQUENTL	Y			
F9.	disruptive. <u>Moderate</u> - elation is	ery pronounced; subject is eupho:	MC MA	MILD MODERATE MARKED				
F10.	always been? <u>Yes</u> - they represent : Exaggeration -they a problems.	resent a change from the way (sh a clear change. re an exaggeration of previous o change (life long characteristic	YE EX NO	G. OF PRO)B			

G. Anxiety

In the last month, has (NAME) been very nervous, worried, or frightened for no apparent reason? Does (s/he) seem very tense or fridgety? Is (NAME) afraid to be apart from you?

Y ES				8 NA (SKI	Р ТО Н)	
		Y ES	NO	DK	IV	NA
G1.	Does (NAME) say that (s/he) is worried about planned events?	1	2	7	8	9
G 2.	Does (NAME) have periods of feeling shaky, unable to relax, or feeling excessively tense?	1	2	7	8	9
G 3.	Does (NAME) have periods of [or complain of] shortness o breath, gasping, or sighing for no apparent reason other that nervousness?		2	7	8	9
G4.	Does (s/he) complain of butterflies in (HIS/HER) stomach, or of racing or pounding of the heart in association with nervousness? (Symptoms not explained by ill health).	1	2	7	8	9
G5.	Does (s/he) avoid certain places or situations that make (HIM/HER) more nervous such as riding in the car, meeting with friends, or being in crowds?	g 1	2	7	8	9
G6.	Does (NAME) become nervous and upset when separated from you [or (HIS/HER) care giver]? [Does (s/he) cling to you to keep from being separated?]	1	2	7	8	9
G7.	Does (s/he) show any other signs of anxiety?	1	2	7	8	9
Specif	fy	IFYESS	PECIFY			
If the s	screening question is confirmed, determine the frequency and s	everity of the a	anxiety.			
G8.	Frequency: <u>Occasionally</u> - less than once per week. Often - about once per week. <u>Frequently</u> - several times per week but less than every day. <u>Very Frequently</u> - once or more per day.	OFTEN FREQUE	NTLY			2
G9.	Severity: <u>Mild</u> - anxiety is distressing but usually responds to redirection or reassurance. <u>Moderate</u> - anxiety is distressing, anxiety symptoms are spontaneously voiced by the subject and difficult to alleviate. <u>Marked</u> - anxiety is very distressing and a major source of suffering for the subject.	MODERA MARKEI	ATE			
G10.	Do these problems represent a change from the way (s/he) has always been? <u>Yes</u> - they represent a clear change. Exaggeration - they are an exaggeration of previous problems. No - they represent no change (life long characteristics). <u>Don't know</u> .	EXG. OF NO	PROB			

· ·	o or say unings that are not	t usually done or said in public? D NO (S KIP TO D	oes (s/he) do IV			assing to you or A (SKIP TO I)	
1.00			YES	NO	DK	IV	NA
H1.	Does (NAME) act imp consider the conseque	oulsively without appearing to nces?	1	2	7	8	9
H2.	Does (NAME) talk to them?	total strangers as if (s/he) knew	1	2	7	8	9
H3.	Does (NAME) say this or hurt their feelings?	ngs to people that are insensitive	1	2	7	8	9
H4.	Does (s/he) say crude that (s/he) would not v	things or make sexual remarks isually have said?	1	2	7	8	9
H5.	Does (s/he) talk openly matters not usually dis	y about very personal or private scussed in public?	1	2	7	8	9
H6.	Does (s/he) take libert: that is out of character	ies or touch or hug others in a way for (HIM/HER)?	1	2	7	8	9
H7.	Does (s/he) show any (HIS/HER) impulses?	other signs of loss of control of	1	2	7	8	9
Specify	у		IF YES SP	ECIFY			
If the s	creening question is conf	irmed, determine the frequency and	severity of th	ne disinhibitio	on.		
H8.	Frequency: Occasionally - less ti <u>Often</u> - about once p Frequently - several <u>Very Frequently</u> - es	er week.	OFTEN FREQUEN	ITLY		1 2 3 4	
H9.	redirection and guida <u>Moderate</u> - disinhibit overcome by the carr <u>Marked</u> - disinhibitic	tion is very evident and difficult to e giver. on usually fails to respond to any are giver, and is a source of	MODERA	ΤΕ		1 2 3	
H10.	(s/he) has always been <u>Y es</u> - they represent:	a clear change. are an exaggeration of previous	EXG. OF F NO	PROB		1 2 3 7	

I. Irritability/Lability

In the last month, did (NAME) get irritated and easily disturbed? Are (HIS/HER) moods very changeable? Is (s/he) abnormally impatient? We do not mean frustration over memory loss or inability to perform usual tasks; we are interested to know if (s/he) has <u>abnormal</u> irritability, impatience, or rapid emotional changes different from (HIS/HER) normal self.

YES	1	NO (SKIP TO J)	2 I	W			NA (SKIP T	о <u>л</u>	
			7	TES	NO	DK	г	7	NA
I1.	Does (NAME) have a handle" easily over li	a bad temper, flying "off the ttle things?		1	2	7	8	:	9
I2.		y change moods from one eing fine one minute and		1	2	7	5	1	9
I3.	Does (NAME) have s	sudden flashes of anger?		1	2	7	8		9
I4.	Is (NAME) impatient delays or waiting for	, having trouble coping with planned activities?		1	2	7	8	:	9
I5.	Is (NAME) cranky an	nd irritable?		1	2	7	5	;	9
I6.	Is (s/he) argumentativ with?	re and difficult to get along		1	2	7	8	;	9
I7.	Does (s/he) show any	other signs of irritability?		1	2	7	8	:	9
Specify				I	F YES SF	ECIFY			
If the scr	reening question is confi	irmed, dete m ine the frequenc	y and :	severity	of the irritabil	ity/lability	7.		
I8.	every day.		t.	OFTEI FREQU	SIONAL 9. JENTLY FRQTLY				
I9.	Severity: <u>Mild</u> - irritability or responds to redirect: <u>Moderate</u> - irritabilit and difficult to over <u>Marked</u> - irritability usually fail to respo giver, and they are a	t they	MODE	ED					
I10.	(s/he) has always bee <u>Y es</u> - they represent	a clear change. are an exaggeration of previ		EXG.0 NO	OF PROB				

J. Aberrant Motor Behavior

In the last month, did (NAME) pace, do things over and over such as opening closets or drawers, or repeatedly pick at things or wind string or threads?

YES	1	NO (S KIP TO K) 2	IV	10	8 NA (S	KIP TO K.)	10
			YES	NO	DK	IV	NA
J1.	Does (NAME) pace a apparent purpose?	around the house without	1	2	7	8	9
J2.	Does (NAME) rumm unpacking drawers o	age around opening and r closets?	1	2	7	8	9
J3.	Does (NAME) repea clothing?	tedly put on and take off	1	2	7	8	9
J4.	Does (s/he) have rep (s/he) performs over	etitive activities or "habits" that and over?	1	2	7	8	9
J5.		ge in repetitive activities such as king, wrapping string, etc.?	1	2	7	8	9
J6.		cessively, seem unable to sit still,) feet or tap (HIS/HER) fingers a	1	2	7	8	9
J7.	Does (s/he) do any of	ther activities over and over?	1	2	7	8	9
Specify	,	IF YES	SPECIFY				
J8.	Frequency: Occasionally - less Often - about once Frequently - several	irmed, determine the frequency an than once per week. per week. I times per week but less than ever <u>ently</u> - essentially continuously	OCCAS OFTEN y FREQU	SIONAL ENTLY FRQTLY			
J9.	little interference w <u>Moderate</u> - abnorm can be overcome by <u>Marked</u> - abnormal	al motor activity is very evident; 7 the care giver. motor activity is very evident; it ond to any intervention by the care	MODEI MARKI	RATE ED			
J10.	(s/he) has always bee Yes - they represen <u>Exaggeration</u> - they problems.		EXG. 0 s). NO	F PROB			

K. Other Behaviors Affected by Aging

		YES	NO	DK	NA
<u>Appetite</u> K1.	Has (HIS/HER) appetite been reduced?	L	2	7	9
K2.	Has (s/hc) lost weight?	L	2	7	9
K3.	Has (HIS/HER) appetite increased a lot?	l	2	7	9
К4.	Has (s/hc) gained weight?	1	2	7	9
<u>Sleep</u> K5.	Does (NAML) have trouble falling asleep?	L	2	7	9
K6.	Does (s/he) wake a lot?	L	2	7	9
К7.	Does (s/he) wake too early?	I	2	7	9
K8.	Does (s/he) sleep too much?	1	2	7	9
Energy K9.	Does (NAME) feel overly tired?	L	2	7	9
K 10.	Does (s/he) have enough energy?	L	2	7	9
K∐,	Has (s/hc) lost get up and go?	1	2	7	9
Cognition K12,	Does (NAME) describe trouble thinking clearly?	l	2	7	9
K 13.	Does (s/he) describe trouble concentrating?	l	2	7	9
К14.	Does (s/he) describe trouble making a decision?	1	2	7	9
<u>Psychome</u> K15.	t <u>tor</u> Are (NAML'S) movements slower than usual?	L	2	7	9
<u>Other Sor</u> K16.	natic Has (NAME/S) interest in sex decreased recently?	1	2	7	9
		IF YES SK	IP ТО К18		
K 17.	Has (HIS/HER) interest in sex increased recently?	l	2	7	9
К18.	Does (S/HE) have trouble with constipation?	1	2	7	9

L. Reliability of Informant Report

I. Ilow reliable is the informant's report of the subject	VERY RELIABLE
---	---------------

END TIME: :

APPENDIX F: FAMILY HISTORY

H1. This section is a brief family history. Please tell me the names of your brothers and sisters starting with oldest and continuing to the youngest. Please include yourself in the list. Do not include siblings that are adopted, step, half brothers or sisters.

RECORD FIRST AND LAST NAME OF EACH SIBLING THEN ASK QUESTIONS ACROSS COLUMNS. FOR R. ONLY RECORD NAME IN BIRTH ORDER.

\square												Birth oro	ler of R
	FIRST NAME	LAST NAME	S	EX	l (NA livi	is ME) ng?	What is (NAME"S) approximate age or age at time of	have stroke,	NAME) a heart a or other 1 ar prob	ttack, cardio	Did (N mem	AME) ev ory probl	er have em s?
			м	F	Y	N	tum e or death?	Y	И	DK	Y	N	DK
1.			1	2	1	2		1	2	8	1	2	8
2.			1	2	1	2		1	2	8	1	2	8
3.			1	2	1	2		1	2	8	1	2	8
4.			1	2	1	2		1	2	8	1	2	8
5.			1	2	1	2		1	2	8	1	2	8

Baseline Interview

3/16/95

Ν.		1	2	I	2	1	2	8	1	2	8
7.		l	2	I	2	1	2	8	1	3	8
Χ.		1	2	I	2	1	2	8	1	2	8
ч.		1	2	I	2	1	2	8	1	2	8
		1	2	1	2	1	2	8	1	2	8
н.		1	2	1	2	1	2	ж	1	2	×

SECTION II: FAMILY HISTORY

	50. II. 1. I. II. III. 11151 C										
12.		1	2	1	2	1	2	×	1	2	×
1 3.		1	2	1	2	1	2	8	1	2	8
1 4.		1	2	1	2	7	2	8	1.	2	8
1 5.		1	2	1	2	1	2	8	1	2	8
1 6.		1	2	1	2	1	2	8	1	2	8

SECTION II: FAMILY HISTORY

Baseline Interview

3/16/95

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ADOPTED NO INFO (GO TO INTERVIEWER CHECKPOINT)......1

	FIRST NAME	LAST NAME			(NAME) living? a		What is (NAME ^{**} S) approximate age or age at	have stroke.	(NAME) a heart a or other utar prob	ttack, cardio	ha	NAME) ve memo problems	ту
			мГ	г	Ŷ		time of death?	Ŷ	N	DΚ	Y	~	DK
30.			1	2	1	j		1	2	8	1	i	8
31.			1	2	1	2		1	2	8	1	2	8

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INT	ERVIEWER CHECKPOINT: DID ANY SIBLINGS OR PARENTS HAVE MEMORY PROBLEMS?	YES (CONTINUE)
HB.	Now I'd like to ask you a few questions about those relatives who experienced memory problems.	
	RECORD NAME FROM II OR II2 WITH MEMORY PROBLEMS.	
	A, NAME:	RELATIONSHIP
	 How old was (NAMI!) when (he/she) started having memory problems? 	AG!!

Baseline Interview

3/16/95

SECTION H: FAMILY HISTORY

ECTION H: FAMILY HISTORY	
Did the memory problems begin suddenly or slowly?	SUDDENLY
Did the memory problems get worse over time?	YES
 (Did/do) the memory problems cause limitations with daily activities? SPECIFY TYPES OF LIMITATION RECORD: 	YES (RECORD)1 NO (GO TO 5)2 DK (GO TO 5)
 Did a doctor ever give a diagnosis for the cause of the memory trouble? SPECIFY THE DIAGNOSIS RECORD: 	YES (RECORD)
	MEM
INTERVIEWER CHECKPOINT: IF DECEASED CONTINUE IF LIVING GO TO NEXT CHECKPOINT	
6. IF DECEA SED: What was the cause of death?	
RECORD:	
7. Was an autopsy performed?	YES
INTERVIEWER CHECKPOINT: ARE THERE OTHER RELATIVES LISTED WITH NEW MEMORY PROBLEMS?	YES1 NO (GO TO H4)
RECORD NEXT NAME FROM H10R H2 WITH MEMORY PROBLEMS.	
B. NAME:	RELATIONSHIP
 How old was (NAME) when (he/she) started having memory problems? 	AGE

Baseline Interview

3/16/95

SECTION H:	FAMILY	HISTORY

SECTION H: FAMILY HISTORY	
Did the memory problems begin suddenly or slowly?	SUDDENLYSLOWLYDK
 Did the memory problems get worse over time? 	YES NO DK
 (Did/do) the memory problems cause limitations with daily activities? SPECIFY TYPES OF LIMITATION 	YES (RECORD) NO (GO TO 5) RF (GO TO 5) DK (GO TO 5)
RECORD:	
5. Did a doctor ever give a diagnosis for the cause of the memory trouble? SPECIFY THE DIAGNOSIS	YES (RECORD) NO (GO TO INT. CHECKPOINT) RF (GO TO INT. CHECKPOINT) DK (GO TO5 INT. CHECKPOINT)
RECORD:	
	MEM
INTERVIEWER CHECKPOINT: IF DECEASED CONTINUE IF LIVING GO TO NEXT CHECKPOINT	
6. IF DECEASED: What was the cause of death?	
RECORD:	
7. Was an autopsy performed?	YES NO DK
INTERVIEWER CHECKPOINT: ARE THERE OTHER RELATIVES LISTED WITH NEW MEMORY PROBLEMS?	YES NO (GO TO H4)

Baseline Interview

SECTION H: FAMILY HISTORY

RECORD NAME FROM H1 OR H2 WITH MEMORY PROBLEMS.	
C. NAME:	RELATIONSHIP
 How old was (NAME) when (he/she) started having memory problems? 	AGE
Did the memory problems begin suddenly or slowly?	SUDDENLY
3. Did the memory problems get worse over time?	YES
4. (Did/do) the memory problems cause limitations with daily activities?	YES (RECORD)
SPECIFY TYPES OF LIMITATION	RF (GO TO 5)
RECORD:	
5. Did a doctor ever give a diagnosis for the cause of the memory trouble?	YES (RECORD)
SPECIFY THE DIAGNOSIS	DK (GO TO INT. CHECKPOINT)
RECORD:	
	1
	MEM

Baseline Interview

3/16/95

SECTION H:	EAMIL V	HISTORY
SECTION II.	LUMMER	INDIONI

INTERVIEWER CHECKPOINT: IF DECEASED CONTINUE IF LIVING GO TO NEXT. CHECKPOINT.				
6. IF DECEASED: What was the cause of death?				
RECORD:				
7. Was an autopsy performed?	NO			
NTERVIEWER CHECKPOINT: ARE THERE OTHER RELATIVES LISTED WITH MEMORY PROBLEMS?	YES (GOTO NO (CONTI	0 FAMILY H INUE)	IS. SUPP.)	
H4. Now I am going to read you a list of problems relatives we've been talking about, your full br parents, please tell me if any of them have been	others and siste	ers and your bio	ological	
	YES	NO	DK	
A. Alzheimer's disease?	1	2	8	
B. Parkinson's disease?	1	2	8	
C. Down's Syndrome?	1	2	8	
D. Senile dementia?	1	2	8	
E. "Hardening of the arteries?"	1	2	8	
F. Mini-strokes of TIA's?	1	2	8	
G. "Arteriosclerosis of the brain"?	1	2	8	
H. Any other neurological conditions?	1	2	8	
SPECIFY:				
		MEM		
SPEC IF Y:		MEM		
SPECIFY:		MEM		

Baseline Interview

3/16/95

SECTION G: INTERVAL FAMILY HISTORY

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- 1. PREPARE 4 LABELS
- 2. PUT ON GLOVES.

from the inside of your cheek.

- 3. OPEN SWAB.
- 4. INSERT SWAB IN R'S RIGHT CHEEK FOR 30 SECONDS.
- 5. ATTACH RIGHT LABEL TO BRUSH.
- 6. PLACE SECOND LABEL ON PACKET.
- 7. TAKE LEFT SAMPLE AND REPEAT.
- 8. TAKE OFF GLOVES
- 9. PLACE SAMPLE IN COOLER.

Baseline Interview

APPENDIX G: NEUROLOGICAL EXAMINATION

COMPLETE	YES
DATE	

NEUROLOGICAL EXAMINATION

Check "Normal, Abnormal, Can't execute or Missing for each question". If you check "Abnormal", also check the appropriate reason why or explain in comment field.

For each question, "Can't execute and Missing" will mean the following: -Can't execute: subject will not/cannot attempt task secondary to dementia. -Missing: examiner omits task, subject refuses (not secondary to dementia), or subject unable to do task secondary to physical reason.

1.	Visual fields by confrontation.	NORMAL No field cut in any quadrant
2.	Range/extent of lateral gaze.	NORMAL Complete gaze to left/right
3.	Range/extent of vertical gaze.	NORMAL Complete up & down gaze 1 ABNORMAL Incomplete up & down gaze 2 Complete absence of up & down gaze 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT 4
4.	Pupillary reflex	NORMAL PERRLA

5.	Close eyes, resist opening by examiner.	NORMAL No weakness of upper eyelid 1 ABNORMAL Unilateral or bilateral weakness 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT
б.	Opening and closing jaw.	NORMAL No deviation of mandible
7.	Pharyngeal movements(subject opens mouth, says"ah").	NORMAL No deviation of uvula or tongue .1 ABNORMAL Deviation to left or right .2 OTHER (SPECIFY) .3 .3 CAN'T EXECUTE .8 .9 SPECIFY/COMMENT .9
8.	Blow out cheeks with mouth closed.	NORMAL Can perform evenly bilateral .1 AB NORMAL Cannot perform evenly .2 Cannot perform evenly .2 Cannot perform with mouth closed .3 OTHER (SPECIFY) .4 CAN'T EXECUTE .8 MISSING .9 SPECIFY/COMMENTS
9.	Wide smile-show teeth.	NORMAL NO weakness noted 1 ABNORMAL Flattened nasolabial fold 2 Inability to raise corner of mouth on left or right 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9

		SPECIFY/COMMENT
10.	Frown with knit brows	NORMAL No weakness noted
11.	Wink with other eye open	NORMAL Can perform with either eye 1 Can perform with one eye only 2 AB NORMAL 2 Deviation to left or right 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPEC IF Y/C OMMENT 9
12.	Rapid tongue movement.	NORMAL 4 touches/second
13.	Motor impersistence. A. Eye closure X 20 seconds	NORMAL Maintains act for 20 seconds
	B. Tongue protrusion x 20 seconds (Do not allow subject to hold tongue in place with his/her teeth).	NORMAL Maintains act for 20 seconds. 1 AB NORMAL Pulls tongue in before 20 seconds. 2 OTHER (SPECIFY). 3 3 CAN'T EXECUTE. 8 8 MISSING 9 9

	SPECIFY/COMMENT
 A coustic nerve A. Rubbing of fingers. 1. Right 	NORMAL Able to hear 1 ABNORMAL Unable to hear 2 OTHER (SPECIFY) 3 2 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT 9
2. Left	NORMAL Able to hear 1 ABNORMAL 1 1 Unable to hear 2 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT 1 1
B. Whispering. 1. Right	NORMAL A ble to hear 1 ABNORMAL Unable to hear 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT 8 9
2. Left	NORMAL Able to hear 1 ABNORMAL 1 1 Unable to hear 2 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT 3 3
15. Chin resistance.	NORMAL No weakness noted on either side ABNORMAL Unilateral weakness 2 Bilateral weakness 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT

1		
16.	Shoulder elevation (shrug).	NORMAL 1 ABNORMAL 1 Unilateral weakness 2 Bilateral weakness 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT
17.	Is subject mute?	YES (GO TO 21)1 NO
18.	Repeat A. "La, La, La, La, La".	NORMAL Regular rate, rhythm and > 4 syllables/second, each syllable clear
	B. "Go, Go, Go, Go, Go".	NORMAL Regular rate, rhythm and > 4 syllables/second, each syllable clear 1 ABNORMAL 2 Arrhythmic 2 <4 syllables/second
	C. "Kitty, Kitty, Kitty, Kitty, Kitty".	NORMAL Regular rate, rhythm and > 4 syllables/second, each syllable clear AB NORMAL Arrhythmic 2 <4 syllables/second

		CDECLEV/COMMENTS
		SPECIFY/COMMENTS
19.	Rate of conversation.	NORMAL Normal Speed
		ABNORMAL Too fast2
		Too slow
		SPECIFY/COMMENTS
20.	Clarity of conversation.	NORMAL
		Normally understandable1 ABNORMAL Examiner must listen carefully
		Subject must repeat to be understood
		OTHER (SPECIFY)
		MISSING
		SPECIFY/COMMENTS
21.	Finger-to-nose touching.	NORMAL Ouickly, smooth and accurate
		ABNORMAL Slow but accurate
		Dysmetria noted
		OTHER (SPECIFY)4 CAN'T EXECUTE
		MISSING9
		SPECIFY/COMMENTS
22.	Finger-thumb tapping. A. Right	NORMAL 4 taps/second1
		ABNORMAL 3 taps/second or faster but arrhythmic2
		<3 taps/ second
		OTHER (SPECIFY)
		MISSING
	B. Left	NORMAL 4 taps/second1
		ABNORMAL 3 taps/second or faster but arrhythmic2
L		

	<pre><3 taps/second</pre>
23. Diadochokinesis. A. Right	NORMAL At least 3 pats/second and smooth 1 ABNORMAL 2 pats/second or faster but anthythmic 2 < 2 pats/second
B. Left	NORMAL At least 3 pats/second and smooth 1 ABNORMAL 2 pats/second or faster but arrhythmic 2 < 2 pats/second
24. Hand praxis tasks. A. Inter-locking fingers (No verbal cues)	NORMAL Performs correctly. 1 AB NORMAL Performs incorrectly. 2 OTHER (SPECIFY). 3 3 CAN'T EXECUTE 8 MISSING 9 SPEC IF Y/COMMENT 9 9
B. Mirrored fingers (No verbal cues)	NORMAL Performs correctly
25. Grasp. A. Right	NORMAL Absent

	Subject grasps examiners hand on stimulation 2 Grasps after verbal request not to do so
B. Left	NORMAL Absent 1 ABNORMAL 1 1 Subject grasps examiners hand on stimulation 2 2 Grasps after verbal request not to do so 3 3 OTHER (SPECIFY) 4 4 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT
 Muscle tonus (passive flexion/extension at elbow and wrist). A. Right 	NORMAL Normal muscle tone, no rigidity 1 AB NORMAL Rigidity or stiffness present 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT
B. Left	NORMAL Normal muscle tone, no rigidity 1 ABNORMAL Rigidity or stiffness present 2 OTHER (SPECIF Y) 3 3 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIF Y/COMMENT 1 1
27. Cogwheel phenomenon. A. Right	NONE NOTED 1 PALPABLE AT BICEPS TENDON 2 GROSSLY VISIBLE 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT
B. Left	NONE NOTED

		GROSSLY VISIBLE
28.	Tremor at rest.	NORMAL None
29.	Tremor with arms outstretched (while seated).	NORMAL None
30.	Pronator drift (arms out-stretched, palms up, eyes closed while seated).	NORMAL Absence of drift
31.	Strength difference with downward pressure and then sudden release by examiner (arms out-stretched, resists examiner's pressure. Conducted while subject is seated.) If Subject has arthritis or injury to hands, replace with biceps pull. Indicate task attempted.	NORMAL No strength difference, equal rebound
32.	Upper extremity motor strength. Grip strength, or biceps pull. Strength difference between L and R finger grasp or biceps pull. Subject grasps	Circle one Grip strength=1 Biceps pull=2 NORMAL No strength difference between R and L trials

examiner's extended fingers (2 digits) with R and L trials.	Specify weakness (L or R)
Primitive reflexes. A. Snout (tap closed lips with reflex hammer)	NORMAL Absent 1 ABNORMAL Present 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 MISSING SPECIFY/COMMENT 9 SPECIFY/COMMENT
B. Visual suck	NORMAL Absent 1 ABNORMAL Present 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT
C. Tactile suck	NORMAL Absent 1 ABNORMAL 2 OTHER (SPECIFY) 3 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT 9

34.Deep	tendon	reflexes.

	ABSENT	REDUCED	NORMAL	BRISK, NO CLONUS	BRISK WITH CLONUS	OTHER	CAN'T EXECUTE	MISSING	SPECIFY/ COMMENT
Biceps, R	0	1	2	3	4	5	8	9	
Biceps, L	0	1	2	3	4	5	8	9	
BRACH IAL RADIA	0	1	2	3	4	5	8	9	

LIS. R	ABSENT	REDUCED	NORMAL	BRISK, NO CLONUS	BRISK WITH CLONUS	OTHER	CAN'T EXECUTE	MISSING	SPECIFY/ COMMENT
BRACH IAL RADIA LIS., L	0	1	2	3	4	5	8	9	
Knees, R	0	1	2	3	4	5	8	9	
Knees, L	0	1	2	3	4	5	8	9	
ANKLE JERKS, R	0	1	2	3	4	5	8	9	
ANKLE JERKS, L	0	1	2	3	4	5	8	9	
35.	Ankle Clor A. Right B. Left	OTHE CAN' MISS SPEC NORM AB NO OTHE CAN' MISS	Absent DRMAL (SP R (SPECIF' T EXECUTI NG IFY/COMM MAL Absent DRMAL (SP R (SPECIF' T EXECUTI NG	ECIFY) Y) ENT ECIFY) Y) E					
36.	Kinesthesis A. Rig		AB SE OTHE CAN' MISS	PRESENT 1 AB SENT 2 OTHER (SPECIFY) 3 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT 9					2
B. Left			AB SE OTHE CAN'	NT R (SPECIF' T EXECUTI	Y) E				2

		SPECIFY/COMMENT
37.	Vibrating Sensation. A. Right	PRESENT 1 AB SENT 2 OTHER (SPECIFY) 3 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT 9
	B. Left	PRESENT
38.	Plantar response. A. Right	NORMAL Plantar flexion of great toe ABNORMAL Extension of great toe No reflex present 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT
	B. Left	NORMAL Plantar flexion of great toe ABNORMAL Extension of great toe No reflex present 3 OTHER (SPECIFY) 4 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT
39.	Heel-to-shin test. A. Right	NORMAL Moving foot is dorsi flexed and motion down shin is smooth, slow and accurate 1 ABNORMAL 2 Path of heel is shaky, jerky, wavering 2 Knee is overshot 3 Slide down skin accompanied by action tremor 4 OTHER (SPECIFY) 5 CAN'T EXECUTE 8

		MISSING
	B. Left	NORMAL Moving foot is dorsi flexed and motion down shin is smooth, slow and accurate 1 ABNORMAL 1 Path of heel is shaky, jerky, wavering 2 Knee is overshot 3 Slide down skin accompanied by action tremor 4 OTHER (SPECIFY) 5 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENT 1
40.	Romberg's sign: stand with feet together and eyes closed for 10-15 seconds.	NORMAL Normally still or slight weaving 1 AB NORMAL Falls to one side with eyes closed 2 Falls to one side with eyes open 3 3 Needs widened base to stay in one place 4 OTHER (SPECIFY) 5 5 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENT
41.	Gait activities. A. Walking down a hall at least 10 paces.	NORMAL Normal gait, narrow base. 1 AB NORMAL Deviation from straight line. 2 OTHER (SPECIFY) 3 3 CAN'T EXECUTE 8 8 MISSING 9 9 SPECIFY/COMMENTS 1 1
	B. Pivot while turning.	NORMAL Pivots on narrow base 1 ABNORMAL 1 1 ABNORMAL 2 2 Widens base or moves feet 3 3 Turns slowly or awkwardly 4 OTHER (SPECIFY) 5 CAN'T EXECUTE 8 MISSING 9 SPECIFY/COMMENTS 9

	C. Stopping on unexpected command.	NORMAL Stops on command or takes one small step	2 3 4 8
	D. Festination/ Shuffling.	NORMAL None ABNORMAL Slow start Shuffling gait OTHER (SPECIFY) CAN'T EXECUTE MISSING SPECIFY/COMMENTS	2 3 4 8
	E. Accessory movements	NORMAL Normal ABNORMAL Decrease of arm swings Trunk/neck rigid and flexed Width of gait gets smaller. OTHER (SPECIFY) CAN'T EXECUTE MISSING SPECIFY/COMMENTS	2 3 4 5 8
42.	Heel- to- toe pacing for 10 paces.	NORMAL No deviations or 1 deviation ABNORMAL >1 deviation Excessive truncal weaving Can't complete 10 paces OTHER (SPECIFY) CAN'T EXECUTE MISSING SPECIFY/COMMENTS	2 3 4 5 8
43.	Bradykinesia.	NORMAL Voluntary movements are normal ABNORMAL Mild or marked slowness. OTHER (SPECIFY) CAN'T EXECUTE	2 3

		MISSING
		SPECIFY/COMMENTS
44. Myo	clonus.	NORMAL.
		Absent
		ABNORMAL
		Mild myoclonus
		Occasional myoclonus
		Frequent myoclonus 4
		Severe myoclonus
		OTHER (SPECIFY)
		CAN'T EXECUTE
		MISSING
		SPECIFY/COMMENTS
45. Postu	лс.	NORMAL.
		Normal, erect
		ABNORMAL
		Slightly stooped2
		Very stooped
		Leans to one side
		OTHER (SPECIFY)
		CAN'T EXECUTE
		MISSING
		SPECIFY/COMMENTS

46.Praxis tasks (prefend to).

	NORMAL PERFORMS CORRECTLY	ABNORMAL (SPECIFY)	OTHER (SPECIFY)	CAN'T EXECUTE	MISSING	SPECIFY/COMMENTS
COMB YOUR	1	2	3	20	9	
HAMMER A NAIL	1	ī	3	8	9	
BRUSH YOUR TEETH	I	2	3	8	9	

COMMENT

₿NØ TIME: :

APPENDIX H: NEUROLOGICAL GROUPINGS

Table D

Breakdown of medical conditions comprised in the medical groups

Breakdown of medical conditions comprised in t	
Neurologic Grouping	Conditions/Signs/Symptoms
Facial Movements	Jaw movement
	Wide smile
Upper Body	Chin Strength
	Shoulder strength
Muscle Tone, Pronator Drift Upper Extremity	Upper extremity strength
Strength, Lateralized	Muscle tone
	Pronator drift
Speech Features	Repetition (kitty, go, la)
	Rate
	Clarity
Pharyngeal movement	
Visual Fields	
Pupillary Reflex	
Lateral Gaze	
Vertical Gaze	
Primitive Reflexes	Grasp
	Snout
	Visual suck
	Tactile suck
Myoclonus	
Tremor	Tremor
	Postural tremor
Pressure and Release	
Cogwheeling	Tendonitis/Bursitis
	Gout
Deep Tendon Reflexes, lateralized	Biceps
	Knees
	Brachial
Plantar Reflex	
Romberg	
Festination/shuffle	
Gait width	
Bradykinesia	
Posture	
Ideational Praxis	Comb, hammer, brush

Ankle clonus Finger tapping, lateralized

Left and right finger tapping

CURRICULUM VITAE

KAITLYN E. KAUZOR, M.A.

4150 Clement St., San Francisco, CA 94121 • (661) 904-5387 • kaitlyn.kauzor@gmail.com

EDUCATION

2024 (Expected)	 Doctoral Candidate, Utah State University Major: Clinical-Counseling Psychology (APA-Accredited Program) Dissertation: Course of Disease Progression and Time to Death Among Different Dementia Types in a Population Based Study – Anticipated defense: Fall 2024 Advisor: JoAnn Tschanz, PhD
08/15 – 05/17	Master of Arts, California State University, Northridge Major: Clinical Psychology Thesis: Cognitive and Functional Differences of Cognitively Impaired Individuals With and Without Caregivers Advisor: Jill Razani, PhD
08/12-05/15	Bachelor of Arts , California State University, Northridge Major: Psychology, Honors Program
2013	Certified Phlebotomy Technician 1 , UCLA Center for Prehospital Care License #00045637

AWARDS

2024	Society of Clinical Geropsychology Student Travel Award
2024	APA Assessment Community Student Poster Award
2018	Gloria Foster-George Scholarship, Utah State University
2015	Delmar Nicks Research Award, California State University, Northridge

SUPERVISED CLINICAL EXPERIENCE – DOCTORAL

07/23 - 06/2024	Neuropsychology Intern
	San Francisco Veterans Healthcare System – SFVA Hospital
	Supervisors: Johannes Rothlind, PhD, Brian Yochim, PhD, ABPP
	& Nicole Torrence, PhD
	Experiences:
	 Conduct comprehensive neuropsychological evaluations in person
	and via tele-health in the Neuropsychology Clinic and the interdisciplinary Memory Clinic
	 Conduct clinical interviews and feedback sessions
	 Scoring, interpretation, data integration and writing neuropsychological
	reports with tailored recommendations
	• Provide differential diagnoses among adults and older adults (65+) with
	various neurodegenerative processes and/or psychiatric diagnoses
	• Provide mental health services (e.g., weekly therapy, evidence-based intervention and
	assessment, behavioral and emotional support) and interdisciplinary
	consultation in inpatient setting (Community Living Center) to veterans
/ /	admitted for short-stay rehab and long-stay continuing care
06/22 - 05/23	• Attend weekly didactic seminars, including SFVA Internship and Research Seminar, Geropsychology Didactic and Grand Rounds, Neuropsychology Didactic; UCSF
	Psychopharmacology Seminar; other weekly group case conference/presentations with clinical research staff and post- doctoral
	fellows
	Population:
	• Adult/Geriatric; military veterans ages 18+; diverse socioeconomic
	status (SES)
	• Neurologic (e.g., dementia, stroke, TBI) and psychiatric inpatient and
	outpatient

Neuropsychology Extern Neuropsychology Center of Utah *Supervisor*: Adam Schwebach, PhD, Clinical Psychologist Experiences:

- Conduct clinical interviews and feedback sessions with patients and families
- Conduct comprehensive neuropsychological evaluations
- Score, interpret data, and write integrated reports
- Provide differential diagnoses to patients ranging from adolescence to older-adulthood with various neurodegenerative (dementia, TBI, stroke), developmental (ADHD, LD, intellectual impairment) and/or psychiatric (e.g. bipolar, OCD, MDD, borderline) conditions
- Evaluate cases related to capacity, guardianship and legallymandated evaluations

Population:

- Lifespan; diverse SES
- Community mental health, outpatient

08/21 - 06/22

Neuropsychology Extern

Lifespan Neuropsychology Clinic, Department of Psychiatry, University

of Utah School of Medicine

Supervisor: Kelly Konopacki, PhD, Clinical Assistant Professor Experiences:

- Conducted clinical interviews and feedback sessions with patients and
- families, communicating findings to referring provider
- Conducted comprehensive neuropsychological evaluations using a flexible battery approach
- Scored, interpretated data and wrote integrated reports
- Provided differential diagnoses and diagnostic clarifications to patients

• ranging from young adult to older-adulthood with various neurodegenerative (dementia, TBI, stroke), developmental (ADHD, LD, intellectual impairment) and/or psychiatric (e.g. bipolar, OCD, MDD,

borderline) conditions

- Participate in neuropsychological fact-finding didactics
- Participate in didactic seminars, including Neurology Grand Rounds,
- Psychiatry Grand Rounds, and other weekly group case conferences/presentations with clinical research staff

Population:

- Lifespan; diverse SES
- Medically complex neurologic (e.g. stroke, dementia, TBI) and

psychiatric (bipolar, schizophrenia, MDD) outpatient

06/20 - 05/22	Clinical Extern Student Health and Wellness Center at Utah State University <i>Supervisor</i> : Scott DeBerard, PhD, Executive Director of Student Health and Wellness Center Experiences:	
	 Conducted intake assessments and treatment planning Provided weekly psychotherapy in person and via telehealth Utilized evidence-based interventions of psychotherapy in the context of a brief, primary care model of treatment (ACT, CBT, BA, CPT) 	
	 Attended staff meetings and consulted with attending physicians and psychiatrists regarding treatment planning Attended weekly individual and group supervision to discuss clients' 	
	therapy goals and strategize about optimizing interventions Population:	
	• College students, age 18+	
	• Outpatient treatment of various conditions including eating disorders, depressive and anxiety disorders, OCD and religious trauma	
08/19 – 07/20	Clinical and Assessment Extern Behavioral Health Clinic at Utah State University <i>Supervisors</i> : Sara Boghosian, PhD, Director of Psychology Services; Susan Crowley, PhD, Professor; Marietta Veeder PhD, Professor Experiences:	
	 Conducted clinical interviews and feedback sessions with clients and families 	
	 Conducted comprehensive psychological evaluations and psychotherapy interventions (individual therapy, parent- management training) 	
	 Scored, interpreted data and wrote integrated reports Provided differential diagnoses to patients ranging from childhood to 	
	adulthood with various developmental and/or psychiatric conditions	
	Population:	
	• Children, adolescents, adults; wide range of SES/sliding scale	

• Community mental health, outpatient

PRE-DOCTORAL CLINICAL EXPERIENCE

08/15 – 07/17 **Psychological Assessment Intern**

Assessment Clinic at California State University, Northridge *Supervisor*: Gary Katz, PhD, Associate Professor Experiences:

- Conducted psychoeducational evaluations
- Scored, interpreted data and wrote integrated reports Population:
 - Children, adolescents, adults
 - Outpatient clinical specializing in ADHD and LD evaluations

RESEARCH EXPERIENCE AND SCHOLARLY PROJECTS

08/23 – Present Doctoral Intern Research Project

Description: Data were recently obtained from Uniform Data Set (UDS) and item response theory will be utilized to examine differential item functioning of the Verbal Naming Test. Aims of this project include identification of problematic items in this measure and suggested adaptations, including removal of the item(s) as necessary.

Research Assistant

08/18 - 07/20

Alzheimer's Disease and Cognitive Disorders Lab at Utah State University

Project: The Cache County Family Study (National Institute on Aging R01AG11380, R01AG031272, R01AG21136) *Principal Investigator*: JoAnn Tschanz, PhD, Professor Experiences:

- Administered and scored neuropsychological assessments, collected demographic and medical history
- Participated in case consensus meetings to render neurocognitive diagnoses
- Trained undergraduate RAs on neuropsychological testing and other study protocols
- Assisted with data analyses and poster presentations at national conferences

Population:

• Community-dwelling older adults living in Cache County, Utah with cognitive impairments and/or a family history of cognitive impairments

PRE-DOCTORAL RESEARCH EXPERIENCE

- 2015 2018 Research Coordinator
- 2013 2015 Undergraduate Research Assistant

California State University Northridge, Neuropsychology, Dementia and Multicultural Research Lab *Principal Investigator:* Jill Razani, PhD, Department of Psychology

Chair Experiences:

- Managed lab administration for NIH funded study examining cognitive and functional changes among individuals with MCI or early AD
- Created administration/scoring manuals, study database, and study codebooks
- Trained and supervised incoming RAs on study-related tasks (e.g. neuropsychological testing and scoring, lab administration tasks)

Project: "MCI, Dementia and Caregiver Longitudinal Study "

• Administered neuropsychological batteries to older adults with MCI or dementia, to examine longitudinal patterns of cognitive and functional decline, as well as caregiver burden

Population:

• Older adults with MCI or mild AD

Project: "The Multicultural and Acculturation Project"

• Conducted sociocultural/demographic interviews and administered neuropsychological batteries to individuals of diverse ethnic backgrounds to examine effects of acculturation on neurocognitive test performance

Population: Cognitively healthy Iranian and Hispanic adults

06/17 – 06/18 Research Assistant

Mary S. Easton Center for Alzheimer's Disease Research, David Geffen School of Medicine at UCLA

Principal Investigator: Ellen Woo, PhD, Associate Professor Experiences:

- Administered neuropsychological batteries to older adults with dementia or mild cognitive impairment
- Scored and interpreted data
- Wrote neuropsychological assessment feedback letters under the supervision of board- certified clinical neuropsychologist and physicians within multidisciplinary care team

Population:

• Outpatient, older-adults

08/17 – 11/17 **Research Assistant**

Department of Psychiatry at Harbor UCLA Medical Center Principal Investigator: Matthew Wright, PhD, Assistant Professor Experiences:

- Administered neuropsychological batteries to adults with traumatic brain injuries
- Scored and interpreted data

Population:

- Adults; diverse SES
- Outpatient, TBI

6/16 – 5/17 **Research Assistant**

Department of Psychiatry, Cedars Sinai Medical Center Supervisor: Waguih W. IsHak, MD, Vice Chair Experiences:

- Contributed to the writing of two original research articles
- Attended weekly case meetings with psychiatric team and participated in case presentations
- Attended inpatient rounds with a psychiatrist and observed pre-surgical evaluations
- Involved in the designing of new studies

Population:

• Inpatient psychiatric hospital

PUBLICATIONS AND PRESENTATIONS

Peer Reviewed Journal Articles

- Kauzor K., Drewel, M., Gonzalez, H., Rattinger, G., Hammond, A., Wengreen, H., Lyketsos, C., Tschanz, J. (2023). Malnutrition and neuropsychiatric symptoms in dementia: The Cache County Dementia Progression Study. *International Psychogeriatrics*. 1-11.
- Lara-Ruiz, J., Kauzor, K., Gonzalez, K., Nakhla, M., Banuelos, D., Woo, E., Apostolova, L.G., & Razani, J. (2019). Functional Ability of MCI and Alzheimer's Patients Predicts Caregiver Burden. *Journal of Gerontopsychology and Geriatric Psychiatry*. 32(1), 31-39.
- IsHak, W. W., Steiner, A., Klimowicz, Kauzor, K., Dang, J., Vanle, B., Elzhaby, C., Reid, M., Sumner, L., & Danovitch, I. Major Depression Comorbid with Medical Conditions: Analysis of Quality of Life, Functioning and Depressive Symptom Severity. *Psychopharmacology Bulletin*. 2018; 48(1): 1-18.
- Avila, J., Verney, S., Kauzor, K., Flowers, A., Mehradfar, M., & Razani, J. (2018). Normative Data for Farsi-Speaking Iranians in the U.S. on Measures of Executive Functioning. *Journal of Applied*

Neuropsychology: Adult. 26(3), 229-235

 Steiner, A. J., Recacho, J., Vanle, B., Dang, J., Wright, S. M., Miller, J. S., Kauzor, K., Reid, M., Mirocha, J., Danovitch, I., & IsHak, W. W. (2017). Quality of life, functioning and depressive symptom severity in older adults with major depressive disorder. *Journal* of *Clinical Psychiatry*. 78(7), 16420.

Invited Presentations

Kauzor, K. & Bergeson, J. (2018, 2019). *Alzheimer's Disease: Presentations, Course and Current State of Research*. Talk presented to the USU Department of Nursing at Utah State University.

Symposium Presentations

- Lara-Ruiz, J., Kauzor, K., Flowers, A., & Razani, J. (2016, April). *The* association between cognitive functioning. Functional ability and depression as Alzheimer's disease progresses. Cognitive and functional impairment in older adults with MCI and dementia. Symposium presented at the annual meeting of the Western Psychological Association, Long Beach, CA.
- Lara-Ruiz, J. & Kauzor, K. (2016, May). Mild cognitive impairment and mild Alzheimer's disease patients' cognitive and functional status predict caregiver burden. Symposium presented at the annual CSU, Social Science Research and Instructional Center Social Science Student Symposium, San Diego, CA.

Poster Presentations

- 1. Kauzor, K., Pennington, D., & Yochim, B. (2024, August). *Diagnostic Accuracy of the Verbal Naming Test Among White and Black Participants in the NACC*. Poster to be presented at the annual meeting of the American Psychological Association, Seattle, WA.
- Drewel, M., Gonzalez, H., Rattinger, G., Matyi, J., Hammond, A., Kauzor, K., Buhusi, M., & Tschanz, J. (2021, July). BDNF SNP C270T Modifies the Association Between History of Head Injury and Cognitive Status in Older Adults. Poster presented at the annual meeting of the Alzheimer's Association International Conference, Amsterdam, Netherlands.
- Hammond, A., Vernon, E., Kauzor, K., Rattinger, G., & Tschanz, J. (2020, February). Baseline Cognitive Status of Two Classifications of MCI and Conversion to Alzheimer's Disease: The Cache County Memory Study. Poster presented at the annual meeting of the International Neuropsychological Society, Denver, CO.
- Kauzor, K., Schwartz, S., Hammond, A., Tubbs, Z., Rattinger, G., & Tschanz, J. (2019, July). Patterns of neuropsychiatric symptoms and survival among older adults with various subtypes of dementia: The Cache County Dementia Progression Study. Poster presented at the annual meeting of the Alzheimer's Association International Conference, Los Angeles, CA.
- Kauzor, K., Flowers, A., Banuelos, D., Gonzalez, K., Apostolova, L., Woo, E., & Razani, J. (2018, February). *Test performance of MCI patients with and without caregivers as compared to AD and NC*. Poster presented at the annual meeting of the International Neuropsychological Society, Washington, DC.
- Kauzor, K., Flowers, A., Castillo, G., Nakhla, M., Herrera, J., Banuelos, D., & Razani, J. (2017, February). *Hispanic Performance on Verbal and Non-verbal Neuropsychological Tests*. Poster presented at the annual meeting of the International Neuropsychological Association, New Orleans, LA.
- Lara-Ruiz, J., Kauzor, K., Castillo, G., Banuelos, D., Nakhla, M., & Razani, J. (2017, February). *The impact of PTSD symptoms and cognitive performance on student veterans' academic achievement*. Poster presented at the annual meeting of the International Neuropsychological Society, New Orleans, LA.
- 8. **Kauzor, K.**, Lara-Ruiz, J., Castillo, G., Banuelos, D., Flowers, A., Alostaz, J., Nakhla, M., & Razani, J. (2016, May). *Effects of acculturation on attention test in ethnically diverse populations*. Poster presented at the annual convention of the Association for Psychological Science, Chicago, IL.

- Kauzor, K., Flowers, A., Lara-Ruiz, J., & Razani, J. (2016, April). Cognitive and functional performance of MCI with and without caregivers. Cognitive and functional impairment in older adults with MCI and dementia.
 Symposium presented at the annual meeting of the Western Psychological Association, Long Beach, CA.
- Castillo, G., Kauzor, K., Flowers, A., Alostaz, J., & Razani, J. (2016, April). *Performance differences between normal control, MCI, FTD and vascular dementia.* Poster presented at the annual meeting of the Western Psychological Association, Long Beach, CA.
- 11. Kauzor, K., Flowers, A., Lara-Ruiz, J., Apostolova, L., Woo, E., Ringman, J., & Razani, J. (2016, February). *Daily functioning in MCI patients with and without caregivers*. Poster presented at the annual meeting of the International Neuropsychological Society, Boston, MA.
- 12. **Kauzor, K**., Castillo, G., Alostaz, J., & Razani, J. (2015, November). *Financial capacity of cognitively impaired individuals using ADL tasks*. Poster presented at the annual meeting of the National Academy of Neuropsychology, Austin, TX.
- 13. Kauzor, K., Flowers, A., Razani, J., Apostolova, L., Woo, E., & Ringman, J. (2015, May). Cognitive function of Alzheimer's and MCI individuals on categories of the direct assessment of functional status. Poster presented at the annual convention of the Association for Psychological Science, New York, NY.
- 14. Kauzor, K., Castellanos, C., Castillo, G., Flowers, A., Avila, J., Razani, J., Woo, E., Ringman, J., Lu, P., & Apostolova, L. (2015, February). *Memory performance in MCI using memory and ADL tasks*. Poster presented at the annual meeting of the International Neuropsychological Society, Denver, CO.
- 15. **Kauzor, K**., Castillo, G., Rathje, G., Sarkissians, S., & Razani, J. (2014, May). *Acculturation and measures of executive functioning in Caucasian and Hispanic individuals*. Poster presented at the annual psychology undergraduate research conference of UCLA, Los Angeles, CA.
- 16. Castillo, G., Kauzor, K., Rathje, G., & Razani, J. (2014, April). Cultural differences and performance on measures of executive functioning in Caucasian and Hispanic individuals. Poster presented at the annual convention of the Western Psychological Association, Portland, OR.

TEACHING EXPERIENCE

08/18 - 07/21	Teaching Assistant
	Department of Psychology, Utah State University
	Course: Integrated Practicum with Adults, Adolescents and
	Children
	Supervisors: Sara Boghosian, PhD & Susan Crowley, PhD

	Course: Health Psychology Supervisor: Christopher Johnson, PhD
08/14 - 07/17	Teaching Assistant California State University, Northridge <i>Course:</i> Statistical Methods in Psychology <i>Supervisor:</i> Jill Razani, PhD
	<i>Course:</i> Psychology Research Methods <i>Supervisor:</i> Sheila Grant, PhD

LEADERSHIP AND PROFESSIONAL SERVICE

2023- Present	Intern Representative
	Externship and Internship Training Committee at SFVA
2022	Content Contributor – ACT and Exposure Therapy for
	Anxiety Dravis Continuing Education and Tasining Educational
	Praxis Continuing Education and Training – Educational training in
08/21-05/22	therapeutic interventions
00/21-03/22	incrapeutie interventions
	Graduate Student Representative
02/21	Combined Clinical-Counseling Psychology Program
	Founding Member
	Meedk – A brain health network for Armenians around the
	world,
	via social media. Information is shared in English and
	Armenian.
02/19 – Present	Member
	Multicultural Neuropsychology SIG, International
	Neuropsychological
	Society- A group dedicated to evaluating and describing the
	most pressing
	needs of clinical and research neuropsychologists as a call to
	action.
01/16 - 06/17	Community and Special Events Volunteer
01/10 = 00/17	Los Angeles LBGT Center

PROFESSIONAL AFFILIATIONS

2018 – Present	Hispanic Neuropsychological Society (HNS)
2018 – Present	American Psychology Association, Society for Clinical Neuropsychology (Division 40)
2015 – Present	International Neuropsychological Society (INS)

PROFESSIONAL TRAINING

2016	R Grant Writing Workshop, California State University,
	Northridge
	Clinical and Transitional Science Institute (CTSI), UCLA,
	Los Angeles, CA

TESTING EXPERIENCE

*Indicates (typically) weekly administration; otherwise proficient, with less frequent administration

(BDI and BAI)(IBenton Judgment of Line Orientation*(JoLO)SBoston Diagnostic Aphasia Examination**Boston Naming TestSBrief Visuospatial Memory Test-Revised*(BVMT-R)T*California Verbal Learning Test-IIT(CVLT-II)*Cognitive Assessment System (CAS)T*Clock Drawing TestVConnor's Continuous Performance TestV(CPT)In*Controlled Oral Word Association Test*(COWAT)ex*Delis-Kaplan Executive FunctioningVSystem (DKEFS)(C*Dementia Rating Scale (DRS)**Geriatric Depression Scale (GDS)(CGrooved Pegboard Test*Hand Dynamometer/Grip StrengthVHooper Visual Organization Test (HVLT)**Hopkins Verbal Learning Test (HVLT)**Aufman Test of Educational Achievementan	 *Rey-Osterrieth Complex Figure Test (RCFT) *Stroop Color-Word Test Structured Clinical Interview for DSM-5 (SCID) Symbol Digit Modalities Test (SDMT) *Test of Memory Malingering (TOMM) Test of Premorbid Functioning (TOPF) Test of Memory and Learning (TOMAL) *Trail Making Test (A & B) Trail Making Test (A & B) - Oral Verbal Naming Test Wechsler Abbreviated Scale of Intelligence (WASI) *Wechsler Adult Intelligence Scale - 4th edition (WAIS-IV) Wechsler Individual Achievement Test (WIAT) Wechsler Memory Scale (WMS) Wechsler Test of Adult Reading (WTAR) *Wide Range Achievement Test - 3rd and 4th edition (WRAT-III and WRAT-IV)
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*Mini-Mental State Examination/3MS Minnesota Multiphasic Personality Inventory (MMPI) *Montreal Cognitive Assessment (MoCA) *Multilingual Naming Test (MiNT) *Neuropsychological Assessment Battery (NAB) *Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) Wide Range Assessment of Memory and Learning (WRAML) Wisconsin Card Sorting Test (WCST)