THE EFFECTIVENESS OF THE GERIATRIC DEPRESSION SCALE TO DISTINGUISH
APATHY FROM DEPRESSION IN ALZHEIMER’S DISEASE
AND RELATED DEMENTIAS

Tommy E. Davis, Jr., M.P.H.

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APPROVED:

James Hall, Major Professor
Claudia Coggin, Committee Member
Frank Collins, Committee Member
Charles A. Guarnaccia, Committee Member
Andrew W. Houtz, Committee Member
Linda Marshall, Chair of the Department of
Psychology
Sandra L. Terrell, Dean of the Robert B. Toulouse
School of Graduate Studies
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Early detection of Alzheimer’s disease (AD) and related dementias in the elderly is critical for improving treatment methods and is a necessary component for improving public health interventions. One of the earliest and most common behavioral syndromes of AD is apathy and is associated with executive dysfunction. Apathy in AD is often misdiagnosed as depression due to an overlap in symptoms. Studies that have found depression to be associated with executive dysfunction have not always controlled for the presence of apathy. The Geriatric Depression Scale (GDS) is a widely used instrument designed to assess depression in the elderly. This study utilized the GDS and a set of standard neuropsychological instruments to investigate the relationship between apathy, depression, and executive functions in individuals with AD and related dementias. The first objective of this study was to determine if apathy has a greater impact on executive functions compared to depression in AD and related dementias. The second objective was to determine the effectiveness of the GDS as a screen for apathy. The results of the analyses did not support the hypotheses. However, exploratory analyses suggested a possible non-linear relationship with apathy and various levels of dementia severity. Exploratory analysis also suggested mean levels of endorsement for apathy varied by diagnosis. Further research is warranted to investigate this relationship and the GDS endorsement patterns for caregivers regarding their impression of the demented individual.
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CHAPTER 1
INTRODUCTION

Alzheimer’s disease (AD) and related dementias represent a devastating burden to those afflicted and their caregivers. Furthermore, the economic and social burden will continue to grow as the demographic structure of the world’s population continues to change dramatically. The concept of global aging suggests the percentage of the world population over the age of 65 will continue to increase in proportion to other younger age groups dramatically through the year 2050. This shift in global population demographics will result in real number growth of those afflicted with AD and related dementias. Early detection of AD and related dementias is critical for improving treatment methods and is a necessary component for improving public health interventions (Cummings, 2003; Wimo & Winblad, 2004).

One early and common behavioral syndrome of AD is apathy (Kuzis, Sabe, Tiberti, Dorrego, & Starkstein, 1999; Landes, Sperry, Strauss, Geldmacher, 2001; McPherson, Fairbanks, Tiken, Cummings, & Back-Madruga, 2002; van Reekum, Stuss, & Ostrander, 2005; Ready, Ott, Grace, & Cahn-Weiner, 2003). The apathy found in AD is characterized behaviorally by a lack of interest in usual activities, hobbies, and pursuits; loss of interest in social engagements and interpersonal activities such as meeting friends or spending time with family members; and a loss of emotional engagement with reduced affect and diminished intimacy (Cummings, 2003). Apathy is a major psychological and physical burden for caregivers (Cummings et al., 1994; Thomas, Clement, Hazif-Thomas, & Leger, 2001). Apathy, or loss of motivation, may be described in behavioral terms as an absence of responsiveness to stimuli as demonstrated by a lack of self-initiated action (Stuss, van Reekum, & Murphy, 2000; van Reekum et al., 2005). While apathy has been associated with a number of adverse outcomes in AD, it is
underrecognized (Landes et al., 2001; van Reekum et al., 2005). The impact of apathy on AD is associated with the connection between apathy and impairment in executive functions (Boyle et al., 2003; Landes et al., 2001; McPherson et al., 2002; Ready et al., 2003). Executive functions involve the selection, initiation, direction, implementation, and regulation of cognitive skills and behaviors (Cummings, 2003; Landes et al., 2001). Consequently, executive dysfunction broadly impairs the successful performance of tasks by impacting the initiation, planning, and problem solving, that self-directed, goal-oriented behaviors require (Cummings, 2003; Landes et al., 2001).

Apathy in AD and related dementias is often misdiagnosed as depression due to an overlap in symptoms (Landes et al., 2001; Levy et al., 1998; Marin, 1990; Marin, Firinciogullari, & Biedrzycki, 1993). Instruments designed to assess depression often contain items related to apathy. Depression has often been found to be associated with executive dysfunction. However, many of these studies (Fitz & Teri, 1994; Lyketsos et al., 1997; Payne et al., 1998) have not always controlled for the presence of apathy (Kuzis et al., 1999; McPherson et al., 2002). The Geriatric Depression Scale (GDS; Brink, Yesavage, Lum, Heersema, Adey, & Rose, 1982) is a widely administered (Kieffer & Reese, 2002), single-dimension instrument designed to assess depression in the elderly. Several factor analyses of the GDS have determined certain items of the GDS to be associated with apathy and other items associated with dysphoria (Adams, 2001; Adams, Matto, & Sanders, 2004).

The present study investigated the relationship between apathy, depression, and executive functions in individuals with AD and related dementias. The first objective of this study was to determine if apathy had a greater impact on executive functions compared to depression. The second objective was to determine the effectiveness of the GDS as a screen for apathy. Two
hypotheses were established. First, AD and related dementia patients who endorsed a high level of apathy category items on the GDS will show a poorer performance on administered measures of executive functions than AD and related dementia patients who endorsed a lower level of apathy category items. Second, AD and related dementia patients who endorsed a high level of dysphoria category items on the GDS while endorsing a low level of apathy category items will not have significantly different findings on administered measures of executive functions than AD and related dementia patients who endorsed a low level of dysphoria category items.

Definition, Clinical Features, and Prevalence of Apathy

While the term apathy is generally defined as a lack of interest or emotion, numerous attempts have been made to define apathy more precisely, especially in clinical terms (Cummings, 2003; Marin, 1990; Stuss et al., 2000; van Reekum et al. 2005). Marin (1990) further suggests that the absence of a clear and precise definition impedes its systematic investigation. Marin (1990) also indicated that the clinical utility of a more precise definition of apathy may have not been considered in the past due to the difficulties of distinguishing it from other syndromes, such as depression, despair, and abulia. Marin (1990) suggests that if clinical observation and application is used to help define apathy, a lack of motivation is perhaps more precise than a lack of emotion. The example is given of a depressed patient who may be apathetic but is yet, at the same time, in great emotional pain. Stuss et al. (2000) further elaborates on these inconsistencies when trying to settle on a precise definition of apathy due to its secondary presence in a number of neurological and psychiatric disorders.

Marin (1990) clinically defines apathy as a state of primary motivational impairment that is not attributable to a diminished level of consciousness, an intellectual deficit, or emotional
distress. Marin (1990) indicates that when a lack of motivation is attributable to intellectual impairment, emotional distress, or diminished level of consciousness, then apathy is a symptom of some other syndrome such as abulia, akinesia and akinetic mutism, depression, dementia, delirium, despair, and demoralization (Stuss et al., 2000). While the definition by Marin is widely cited and clinically utilized (Landes et al., 2001; Levy, 1998; McPherson et al., 2002; Stuss et al. 2000; van Reekum et al., 2005), Stuss et al. (2000) argue that apathy cannot be clinically defined as simply a lack of motivation.

Stuss et al. (2000) suggest that amotivation describes a state of lacking an inner urge that moves or prompts one to action. Their argument is that the assessment of an inner urge, such as motivation, is problematic and could only be assessed through inferences based on affect or behavior. Therefore, Stuss et al. (2000) define apathy as, “an absence of responsiveness to stimuli as demonstrated by a lack of self-initiated action,” which allows for objective behavioral measurements. Initiation is central to this definition and the self-initiated response may be affective, behavioral or cognitive in nature (van Reekum et al., 2005). Stuss et al. (2000) conceptualize two types of apathy. The first being related to disturbed arousal perhaps associated with the reticular activating system, and the second associated with frontal-subcortical dysfunction, which they label executive apathy. Using this distinction, executive apathy would appear to be the type of apathy most associated with AD and related dementias.

A review (van Reekum et al., 2005) of various AD research with outpatients showed an overall point prevalence of apathy of 60.3% across the studies. Another review (Mega, Cummings, Fiorello, & Gornbein, 1996) showed a point prevalence of 70%. Apathy across dementia severity as determined by Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) showed a point prevalence of 47%, 80%, and 93%, in mild, moderate, and
severe conditions respectively (Cummings, 2003). Research by Cummings (2003) has shown point prevalence of apathy in up to 70% of patients with AD, 90% with frontotemporal dementia, dementia with Lewy bodies, and progressive supranuclear palsy; 40% of those with corticobasal degeneration, and 20% of those with Parkinson’s disease.

Causes of Apathy in Alzheimer’s Disease and Related Dementias

Causation of apathy in AD and related dementias has been explored through neuroimaging and autopsy studies, examination of apathy with general cognitive dysfunction and with executive dysfunction, and the relationship of apathy with depression.

Neuroimaging and Autopsy Studies

Neuroimaging and autopsy studies appear to implicate subcortical-frontal circuits, involving, in particular, the anterior cingulate and dorsolateral prefrontal cortex in the pathophysiology of apathy (Landes et al., 2001; Tekin & Cummings, 2002; van Reekum et al., 2005). Apathy in AD, as well, appears to be specifically related to the disruption of the anterior cingulate-subcortical circuit (Landes et al., 2001; McPherson et al., 2002; Stuss et al., 2000). The anterior cingulate mediates motivated behavior and dysfunction associated with lesions in this area is reflected behaviorally in decreased motivation (Tekin & Cummings, 2002). Functional imaging studies in AD patients show decreased activity in the anterior cingulate cortex correlating with apathy (Craig et al., 1996; Levy et al., 1998; Tekin & Cummings, 2002; van Reekum et al., 2005). A SPECT study of 40 AD patients reported a correlation between apathy and diminished blood flow to the right temporoparietal regions (Ott, Noto, & Fogel, 1996).
Apathy and Global Cognitive Dysfunction

Research that has focused on the impact of apathy on global cognitive impairment has had mixed results (Landes et al., 2001; McPherson et al., 2002; van Reekum et al., 2005). Most of this research has utilized the MMSE (Folstein et al., 1975), which does not provide a sensitive measure of executive function or frontal lobe impairment (Landes et al., 2001; van Reekum et al., 2005). Studies that have incorporated neuropsychological measures of executive function have shown apathy correlating with poorer performance on these measures and thus suggestive of executive dysfunction (Boyle et al., 2003; Kuzis et al., 1999; McPherson et al., 2002; Ready et al., 2003). Neuropsychological measures not dependent on executive functions were unrelated to apathy.

Apathy and Executive Dysfunction

While research suggests that apathy found in AD and related dementias is related to executive dysfunction, questions of specifically how apathy impacts executive functions are being explored (Cummings, 2003; Damasio, 1994; Ott et al., 1996). Cummings (2003) defines executive functions as integrative, synthetic, and generative. Furthermore, Cummings (2003) suggests that these functions mediate the choice, planning, programming, implementation, and feedback refinement of volitional actions. Cummings (2003) also suggests that executive functions have multiple dimensions and are dependent on instrumental functions such as language, memory, praxis, and perception. This theoretical model for executive functions utilizes memory, emotional, and sensory information as inputs for the process of volitional activity. Clinical description of patients with frontal lobe damage, and thus probable executive dysfunction, are related by Luria (1980):
These patients were found to preserve all type of sensation, to have no sign of disturbance of movement, and to have no disturbances of gnosis, praxis, and speech; nevertheless, their complex psychological activity was grossly impaired. They were unable to produce stable plans and became inactive and aspontaneous. They could respond to ordinary questions or perform habitual actions, but they were quite unable to carry out complex, purposive, and goal-directed actions. They were unable to evaluate their attempts, they were not critical of their behavior, and could not control their actions; they continued to perform automatic actions which had long ceased to be meaningful, without any attempt at correction. They were no longer concerned about their failure, they were hesitant and indecisive, and, most frequently of all, they became indifferent or they exhibited features of euphoria, as a result of the loss of their critical awareness of their behavior (p. 247).

Luria (1980) noted, as other researchers (Tekin & Cummings, 2002), the rich connections of the frontal lobes with the underlying limbic structures and thus other areas involved in interoception to suggest that the frontal lobes are intimately involved in the regulation of body states.

Damasio (1994) goes further to specify the relationship between the goal-directed aspect of executive functions, what he labels as reasoning/decision making and emotion/feeling and the neuroanatomical substrates that are involved when these processes are impaired.

First, there is a region of the human brain, the ventromedial prefrontal cortices, whose damage consistently compromises, in a pure fashion as one is likely to find, both reasoning/decision making, and emotion/feeling, especially in the personal and social domain. One might say, metaphorically, that reason and emotion “intersect” in the ventromedial prefrontal cortices and that they also intersect in the amygdala. Second, there is a region of the human brain, the complex of somatosensory cortices in the right hemisphere, whose damage also compromises reasoning/decision making and emotion/feeling, and, in addition, disrupts the processes of basic body signaling. Third, there are regions located in prefrontal cortices beyond the ventromedial sector, whose damage also compromises reasoning and decision making, but in a different pattern: Either the defect is far more sweeping, compromising intellectual operations over all domains, or the defect is more selective, compromising operations on words, numbers, objects, or space, more so than operations in the personal and social domain…In short, there appears to be a collection of systems in the human brain consistently dedicated to the goal-oriented thinking process we call reasoning, and to the response selection we call decision making, with a special emphasis on the personal and social domain. This same collection of systems is also involved in emotion and feeling, and is partly dedicated to processing body signals (p. 70).

Anosognosia, or a lack of awareness of one’s cognitive deficits is often found in AD (Landes et al., 2001; Ott et al., 1996). Anosognosia in AD has been shown to be highly
correlated with apathy, which suggests that this unawareness of deficits is more closely related to emotional changes than to cognitive impairment (Landes et al., 2001). Damasio (1994) has investigated anosognosia in patients who have right somatosensory damage located in the right temporoparietal cortical region and suggests these individuals exhibit similar deficits as those with prefrontal damage. These patients with right temporoparietal damage are unable to make appropriate decisions on personal and social matters, and the patients with prefrontal damage, like patients with temporoparietal damage are usually indifferent to their health status or impairment. Marin (1990) also indicated right hemisphere lesions may produce apathy or indifference, along with anosognosia, with little or few elementary sensory or motor abnormalities.

As noted earlier, Ott et al. (1996) reported a correlation between apathy and diminished blood flow to the right temporoparietal regions and suggested that disruption of the temporolimbic structures in AD may result in a loss of the integration of emotional and sensory information necessary for goal-directed behavior and motivation. These findings would suggest that disruption of the anterior cingulate-subcortical circuit via limbic structures provides one explanation for the occurrence of apathy in AD (McPherson et al., 2002).

Research has defined five frontal-subcortical circuits. These circuits are named according to their function or site of origin in the cortex (Tekin & Cummings, 2002). The anterior cingulate circuit is suggested to mediate motivated behavior and lesions in this circuit are associated with decreased motivation. In other words, the anterior cingulate circuit facilitates the internal selection of environmental stimuli based on their internal relevance (Tekin & Cummings, 2002). This description of the function of the anterior cingulate circuit provides an elegant corollary to the Stuss et al. (2000) definition of apathy as an absence of responsiveness to
stimuli as demonstrated by a lack of self-initiated action. Thus, the relationship of apathy to executive functions is primarily seen in the dysfunction it creates in the inputs of emotion and somatosensory function. These are seen as ventromedial prefrontal region functions (Damasio, 1994; Ott et al., 1996) while memory and more general executive functions are viewed as dorsolateral prefrontal cortex functions (Tekin & Cummings, 2002).

*The Association of Apathy with Depression*

Landes et al. (2001) suggests the diagnosis of apathy may often be overlooked clinically because of the overlap of symptoms with depression. A greater awareness of the impact of apathy on AD and other related dementias and that it can be an early precursor to dementia has fostered efforts to clarify the criteria necessary to differentially diagnose apathy from depression (Kuzis et al., 1999; Landes et al., 2001; Levy et al., 1998; Marin, 1990; Marin et al., 1993; McPherson et al., 2002; Stuss et al., 2000; van Reekum et al., 2005). Furthermore, distinguishing apathy from depression has important treatment implications as these disorders respond to different interventions (Landes et al., 2001). Landes et al. (2001) note that an individual with dementia who has loss of interest, hypersomnia, fatigue, diminished ability to concentrate, and weight loss meets the criteria for a diagnosis of major depression without having a depressed mood or any other signs of dysphoria. Landes et al. (2001) further note that the high levels of apathy in AD may make AD patients more likely to meet criteria for the diagnosis of major depression in the absence of symptoms of dysphoria.

Studies investigating the symptoms of depression in the elderly have found that symptoms generally reflect two categories. The first category involves problems with mood such as dysphoria, guilt feelings, and suicidal ideation. The second category involves problems with
motivation such as lack of interest, low energy, and psychomotor slowing. The dysphoric symptoms such as sadness, guilt feelings, self-criticism, helplessness, and hopelessness distinguish depression from apathy (Adams, 2001; Adams et al., 2004; Landes et al., 2001; Marin, 1990; Marin, 1997; Marin et al., 1993). Marin (1990) suggests that often when a diagnosis of apathy resulting from depression is given, the patient is, paradoxically, not truly apathetic. Marin (1990) suggests, with some exceptions, depression is a dysphoric state, therefore depressed patients who claim to be disinterested or unmotivated will most likely also report symptoms along the line of low self-esteem, self-deprecatory thoughts, anxiety, depressed mood, and hopelessness.

Multiple studies suggest the perceived overlap between apathy and depression in AD may result from assessment instruments that contain questions about symptoms that are common to both conditions (Landes et al., 2001). This is normally seen in assessment instruments for depression, which contain questions related to apathy (Adams, 2001; Adams et al., 2004; Levy et al., 1998; Marin et al., 1993; van Reekum et al., 2005). Marin et al. (1993) studied the overlap of items between the Apathy Evaluation Scale (AES) and the Hamilton Rating Scale for Depression (HamD) and found the HamD items with the strongest correlations with the AES were diminished work/interest, psychomotor retardation, anergy, and lack of insight. Some studies suggest some of the overlap between apathy and depression may be due to both syndromes having prefrontal cortical involvement (Levy et al., 1998). Landes et al. (2001) indicate apathy can also be a side effect of treating depression with selective serotonin reuptake inhibitors (SSRIs) as this class of antidepressant can affect dopaminergic systems found in frontal-subcortical circuits.
Geriatric Depression Scale

The Geriatric Depression Scale (GDS; Brink et al., 1982) is an instrument that has been used extensively in clinical and research settings since it first appeared more than 20 years ago (Adams et al., 2004; Peach, Koob, & Kraus, 2001). This was the first depression scale primarily used as a screening instrument designed specifically for a geriatric population (Adams et al. 2004; Yesavage et al., 1983). The GDS has become prominent in the gerontological literature. It has been recommended as a standard measure for depression in the elderly to facilitate research comparison and replication in studies (Koder, Brodaty, & Anstey, 1996). The GDS consists of a 30-item self-rating scale, which was designed to remedy some of the purported problems in screening for depression in the elderly (Adams, 2001; Yesavage et al., 1983). Scales such as the Zung Self-Rating Depression Scale (SDS; Zung, 1965) and the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943) tended to overdiagnose depression in the elderly because of the many questions regarding somatic concerns, which are beneficial in diagnosing depression in younger persons (Adams, 2001; Yesavage et al., 1983). Furthermore, at the time of the development of the GDS, it was suggested the scale must be sensitive in addressing issues such as suicidality and sexuality as these types of questions could make the elderly cohort defensive (Adams, 2001; Yesavage et al., 1983). Another concern is that the instrument be in a simple, easy to understand format. Several of the depression self-rating scales have multiple answer, Likert-type scales, which have been found to be confusing to some of the elderly in that these involve a greater number of choices and discriminations (Yesavage et al., 1983). The GDS addresses this by containing items that are endorsed either yes or no. The GDS uses its 30 items as a single additive scale with one point being scored for each “depressed” response (Adams, 2001). Twenty items represent a depressed response with a “yes” answer and
ten items indicate a depressed response with a “no” answer. The direction of item endorsement is interspersed to inhibit perseverative yes or no response sets. Brink et al. (1982) suggest a cutoff score of 11 points or above to indicate a depressed individual. The cutoff score of 11 points resulted in a sensitivity of 84% and a specificity of 95% for their study (Yesavage et al., 1983). Kieffer and Reese (2002) published a reliability generalization study of 338 previously published research studies of the GDS and determined the reliability across these studies was .85 (SD = .09).

Factor Analyses of the Geriatric Depression Scale

While the GDS has been widely used in clinical and research settings, very few factor analytical studies have been performed to investigate the underlying structure of the scale (Adams et al., 2004). Adams et al. (2004) suggest only four principal components analyses (PCA) of the English-language, 30-item GDS and only one PCA of the shorter form of the GDS, the 15-item GDS-S (Sheikh & Yesavage, 1986) are found in the literature. At present, the literature indicates Adams et al. (2004) appear to have performed the only confirmatory factor analysis (CFA) on the GDS. The earliest published PCA study of the GDS (Parmelee, Lawton, & Katz, 1989) suggest a six factor structure, which included a 14-item factor labeled as Dysphoria, with additional factors of Worry (4-items), Withdrawal-Apathy (4-items), Vigor (3-items), Decreased Concentration (2-items), and Anxiety (3-items). The Parmelee et al. (1989) PCA of the GDS resulted in nine eigenvalues over one. The Cattell scree plot suggested a final six-component solution with a Varimax rotation that converged in nine iterations (Adams, 2001). Subsequent factor analyses all contained a factor that was designated as representing a construct of withdrawal or apathy (Adams et al., 2004). The recent CFA performed by Adam and
colleagues (2004) suggested a five-factor model of the GDS that achieved a high goodness of fit incorporating 26-items. These factors are labeled Dysphoric Mood (9-items), Withdrawal-Apathy-Vigor (6-items), Hopelessness (4-items), Worry (4-items), and Cognitive Impairment (3-items). Hall and Davis (2008, in press) investigated the factor structure of the GDS in a sample of cognitively impaired older adults and suggested a four-factor model incorporating all 30 items. The factors were labeled Dysphoria (11-items), Meaninglessness (7-items), Apathy (6-items), and Cognitive Impairment (6-items).

Investigation of Apathy Related Subscales on the GDS

Virtually all of the factor analytic investigations of the GDS have resulted in a solution containing a factor, which generally expresses the construct of apathy (Adams, 2001; Adams et al., 2004). The investigation of an apathy dimension of the GDS has primarily focused on the relationship of that dimension with a normal, non-demented elderly population. Adams (2001) looked at the existence of a “Withdrawal-Apathy-(Lack of) Vigor” factor and its relationship to the theoretical geriatric developmental constructs of depletion syndrome (Gallo, Anthony, & Munthen, 1994; Newman, 1989; Newmann, Engel, & Jensen, 1991; Newmann, Klein, Jensen, & Essex, 1996) and gerotranscendence (Tornstam, 1989, 1997, 2000). “Depletion syndrome” was the term used by Newmann and colleagues (Newman et al., 1991; Neumann et al., 1996) to describe one half of two distinct symptom clusters found in the elderly, of which the other was “depression syndrome.” Each syndrome shared some constructs of the other; however the depression syndrome represented constructs associated with dysphoria, while the depletion syndrome represented constructs more associated with lack of interest and apathy (Adams, 2001). Adams (2001) discusses the concept of gerotranscendence, a normal developmental
process of aging in which, as one ages, one relinquishes broad, wide-ranging social interests for a more narrowly defined set of interests that reflect a natural increased desire for solitude and a less active lifestyle. The basic intent of Adams (2001) factor analytic research was to bring to light the possible tendency to over-diagnose the normal elderly with depression when, in fact, these individuals may simply be undergoing a normal developmental aging process. While, indeed, this diagnostic error may be occurring, concern is warranted as an over-reliance on these theoretical disengagement constructs as an explanation for withdrawal in the elderly could result in a tendency to overlook early precursors of possible dementia, in particular apathy.

Research by Newmann and her colleagues (1996) found that the depletion syndrome increased linearly with age, while the depression syndrome did not. Coincidentally, the prevalence of AD in the elderly increases substantially along with increases in age (Cummings, 2003; Wimo & Winblad, 2004). Additionally, executive functions are noted to be the cognitive process most susceptible to decline in aging (Cahn-Weiner, Malloy, Boyle, Marran, & Salloway, 2000). Furthermore, higher levels of apathy in AD have been shown to be associated with greater cognitive impairment (Craig et al., 1996; Mega et al., 1996; Paulsen, Stout, DeLaPena, & Romero, 1996) and older age (Cummings et al., 1994). To reiterate, while many studies (Fitz & Teri, 1994; Lyketsos et al., 1997; Payne et al., 1998) have found a link between depression and executive dysfunction, the majority of these studies have not controlled for the presence of apathy (Kuzis et al., 1999; McPherson et al., 2002). An initial exploratory factor analysis (Hall & Davis, 2008, in press) of the GDS in a sample of cognitively impaired older adults suggested a four-factor model incorporating all 30 GDS items. These four factors consisting of Dysphoria (11-items), Meaninglessness (7-items), Apathy (6-items), and Cognitive Impairment (6-items) will be utilized to assess the differential impact of apathy and depression on executive functions.
Hypotheses

Two hypotheses were proposed:

1. AD and related dementia patients who endorsed a high level of apathy category items on the GDS will show a poorer performance on administered measures of executive functions than AD and related dementia patients who endorsed a lower level of apathy category items.

2. AD and related dementia patients who endorsed a high level of dysphoria category items on the GDS while endorsement a low level of apathy category items will not have significantly different findings on administered measures of executive functions than AD and related dementia patients who endorsed a low level of dysphoria category items.
CHAPTER 2

METHOD

Participants

Participants for this study included 140 individuals (91 females, 49 males; mean age = 78.2 years old, $SD = 7.23$) who were obtained retrospectively from a database of community-dwelling patients, who presented during the period of 2003 to 2005 to a metropolitan outpatient clinic for a neuropsychological evaluation for dementia. Self-rating of depressive symptoms with the Geriatric Depression Scale (GDS) has been shown to be valid in patients with a Mini-Mental State Examination (MMSE, Folstein, Folstein, & McHugh, 1975) score of at least 15 (Katz, 1998) and thus participants with an MMSE below 15 on the date of evaluation were excluded. Participants from this database were largely composed of individuals in the ethnic/racial majority with some high school education or greater. Cognitive impairment diagnoses consisted primarily of Alzheimer’s disease (AD), vascular dementia (VaD), and mild cognitive impairment (MCI) (McKhann et al., 1984; Petersen, 2003; Román et al., 1993). Although AD, VaD, and MCI represent various stages and types of dementia, and may progress differently, similar dysfunction across diagnoses has been noted with respect to the executive aspects of independent functioning (Boone, Ponton, Gorsuch, Gonzalez, & Miller, 1998). Participants were at least 65 years of age on the date of evaluation.

Measures

Measures consisted of the Geriatric Depression Scale (GDS) and three standardized neuropsychological pencil and paper based testing instruments chosen to assess frontal systems and executive functions.
Geriatric Depression Scale (GDS)

The GDS (Yesavage et al., 1983) consists of a 30-item self-rating scale, which was designed to remedy some of the purported problems in screening for depression in the elderly (Adams, 2001; Peach et al., 2001; Yesavage et al., 1983). Scales such as the Zung Self-Rating Depression Scale (SDS; Zung, 1965) and the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943) tended to over-diagnose depression in the elderly because of the many questions regarding somatic concerns, which are beneficial in diagnosing depression in younger persons (Adams, 2001; Yesavage et al., 1983). Several depression self-rating scales have multiple answer, Likert-type scales, which have been found to be confusing to some of the elderly in that these involve a greater number of choices and discriminations (Yesavage et al., 1983). The GDS addresses this by containing items that are endorsed either yes or no. The GDS uses its 30 items as a single additive scale with one point being scored for each “depressed” response (Adams, 2001). Twenty items represent a depressed response with a “yes” answer and ten items indicate a depressed response with a “no” answer. The direction of item endorsement is interspersed to inhibit perseverative yes or no response sets. Brink et al. (1982) suggest a cutoff score of 11 points or above to indicate a depressed individual. The cutoff score of 11 points resulted in a sensitivity of 84% and a specificity of 95% for their study (Yesavage et al., 1983). Kieffer and Reese (2002) published a reliability generalization study of 338 previously published research studies of the GDS and determined the reliability across these studies was .8482 (SD = .0870).
The BDS (Grigsby, Kaye, & Robbins, 1992; Grigsby & Kaye, 1996) is based on Luria’s (1980) theory of frontal lobe functioning and was designed to predict the capacity for an individual to independently regulate their own behavior. It consists of simple cognitive and motor tasks that assess motor programming, inhibition, working memory, and insight (Suchy & Bolger, 1999). The BDS consists of nine items and has a possible total score of 19 points. The first seven of the items involve a motor response from the examinee, while the eighth item consists of a verbal response. The ninth item is not administered to the examinee, but rather represents the examiner’s assessment of the degree of insight demonstrated by the examinee into his or her performance (Grigsby et al., 1992; Grigsby & Kaye, 1996). The first eight items are scored on a 3-point scale. These scores represent examinee performance as adequate (2), mildly to moderately deficient (1), and very impaired (0). The rating of examinee insight utilizes a 4-point scale and assesses the individual’s awareness of the existence, nature, severity, and significance of errors in their performance (Grigsby et al., 1992). The specific criteria for administration and scoring are found in the BDS manual (Grigsby & Kaye, 1996). Test-retest reliability was high (.89 to .93), with good internal consistency (Cronbach’s alpha of .87), and high interrater reliability (total BDS score of .98) with trained raters (Grigsby et al., 1992; Kaye, Grigsby, Robbins, & Korzun, 1990). Kaye et al. (1990) suggest a BDS total score of 0 to 6 indicates severe impairment of behavioral control, scores from 7 to 10 suggest moderately severe impairment, and a score of 11 to 15 may indicate mild to moderate impairment. Elderly individuals scoring less than 11 are particularly susceptible to having difficulty independently regulating their behavior (Grigsby et al., 1992). Initial (Grigsby et al., 1992) and subsequent (Suchy, Blint, & Osmon, 1997) factor analytic examinations of the BDS revealed comparable
three-factor solutions. The three-factors by Grigsby et al. (1992) corresponded to (1) “the ability
to use intention and guide behavior”; (2) “the ability to use feedback”; and (3) “the capacity for
inhibition” (Suchy et al., 1997). A recent pilot factor analysis performed on sample of
cognitively impaired elderly outpatients to be used in this study (Hall & Harvey, 2008) revealed
a three-factor solution with BDS the following items: two go no-go tasks, the Head's test, a piano
exercise, and Luria's fist-edge-palm task loading on the Motoric Problem-Solving factor, two
items of alternate hand-tapping loading on the Simple Motoric Repetitive Behaviors factor, and
an examiner-rated item of the examinee's performance loading on the Insight factor. The BDS
has been shown to predict executive aspects of functional independence, particularly the impact
of apathy and disinhibition, while being unrelated to depression or apraxia (Kaye et al., 1990).
Performance on the BDS has been shown to be an independent predictor of both basic physical
and instrumental activities of daily living (pADLs and iADLs) performance as well as
discriminating between AD and MCI (Belanger et al., 2005).

Clock Drawing Task (CDT)

While numerous scoring systems (Freedman et al., 1994; Shulman, 2000) exist for the
CDT (Sunderland et al., 1989), a paper and pencil instrument, this study used a four-point CDT
scoring system (Nolan & Mohs, 1994), which consists of four criteria scored one-point each for
successful performance for an overall best possible score of four and a worst possible score of
zero. One point is given for successfully completing each of the following tasks: drawing a
closed circle, placing numbers in the correct positions, including all 12 correct numbers, and
placing the clock hands in the correct positions. The CDT administration performed with this
study consists of giving the examinee a blank 8.5 X 11 inch sheet of paper and reading the
instructions stated at the top of the paper, “Please draw the face of a clock and put the numbers in the correct positions. Now, draw in the hands at ten minutes after eleven.” No further instructions are given. The CDT has been suggested to be more sensitive in detecting early dementia than the Mini-Mental State Examination (MMSE; Folstein et al., 1975) (Fujii, 1992). The CDT performed in command administration with the instruction that the clock hands be placed at “ten after eleven” has been suggested to put greater demands on executive functions as mediated by the frontal lobes than other types of CDT administration (Freedman et al., 1994; Shulman, 2000). With respect to the four-point scoring criteria, results of a study by Davis and Houtz (2005) suggest that clock hand position places the greatest demands on executive functions followed by number position. Clock hand position requires the frontal system to interpret the meaning of “ten past eleven” and then to plan and implement a procedure to accomplish that part of the task. Correct number position placement also requires a strategy to effectively place each of the 12 numbers in the appropriate positions with each number evenly spaced around the clock face. It can be construed that more rote tasks such as listing numbers from 1 to 12 and drawing a circle are less susceptible to early cognitive dysfunction than the other two criteria.

*Trail Making Test Part A and Part B (TMT, TMT-A, TMT-B)*

The TMT (Reitan & Wolfson, 1993), a paper and pencil instrument, consists of two parts. The TMT-A requires the examinee to draw a pencil line that sequentially connects 25 encircled numbers, beginning with 1 and ending with 25, distributed on an 8.5 X 11 inch sheet of paper. The TMT-B task is comparable except that the examinee must connect a distributed array of 25 encircled numbers 1 through 13 and letters A through L. The sequence on the TMT-B follows an alternating number-letter order (e.g., 1, A, 2, B, 3, C, etc.). The tasks are timed and examinees
are instructed to perform the task as quickly as possible. Examinees are redirected by the examiner if they make an error during administration. TMT-A and TMT-B are scored separately. The score for each part is the number of seconds required for completion of the task and the number of errors (Reitan & Wolfson, 1993). Both parts of the TMT require motor speed coordination, attention, visual scanning, and sequential processing (Lezak, Howieson, & Loring, 2004; Rasmusson, Zonderman, Kawas, & Resnick, 1998; Reitan & Wolfson, 1993; Spreen & Strauss, 1998). TMT-B most likely requires additional analytic logical cognitive processes known as set shifting, or attentional, mental, or cognitive flexibility (Lezak et al., 2004; Rasmusson et al., 1998; Reitan & Wolfson, 1993; Spreen & Strauss, 1998). According to the norms set by Reitan and Wolfson (1993), a time of 40 to 51 seconds and a time of 86 to 120 seconds are suggested to be indicative of mild to moderate neuropsychological impairment on the TMT-A and TMT-B respectively. A time of 52 seconds or greater and a time of 121 seconds or greater are suggested to be indicative of severe neuropsychological impairment on the TMT-A and TMT-B respectively. The presence of any errors on TMT-A or TMT-B is suggestive of neuropsychological impairment (Reitan & Wolfson, 1993). Studies reporting reliability coefficients vary greatly from .60s to .90s, however most are in the .80s (Lezak et al., 2004; Spreen & Strauss, 1998). Both TMT-A and TMT-B are very sensitive to the progressive cognitive decline associated with dementing processes (Lezak et al., 2004), while elderly individuals who perform poorly on TMT-B are likely to have difficulties with instrumental activities of daily living (iADLs) such as managing a home, medications, and finances even after statistically controlling for age, sex, and education (Bell-McGinty, Podell, Franzen, Baird, & Williams, 2002; Lezak et al., 2004). A study examining of error types in the TMT suggests an inhibitory deficit in AD (Amieva et al., 1998).
Design and Procedure

Institutional Review Board (IRB) approval was obtained from the University of North Texas and the University of North Texas Health Science Center. As outlined in the Participants section, participants for this study were obtained retrospectively from a database of community-dwelling patients, who presented during the period of 2003 to 2005 to a metropolitan outpatient clinic for a neuropsychological evaluation for dementia. The specific instruments investigated in this study were part of a larger battery of neuropsychological instruments administered to the participants. The database was derived from a review of the patient medical records. A data collection sheet with an assigned code number was utilized to transfer and aggregate the raw instrument data from the patient medical records for input into the computer database. After the participant data was transferred to the data collection sheet, the patient medical records are promptly returned to medical records. The assigned code number on the data collection sheet was cross-referenced to a separate list containing only the code number and medical record. This separate list is kept in a locked cabinet in which only the principal investigator has access. The code number was cross-referenced to an SPSS database where the participant’s age, race, gender, diagnosis, and neuropsychological evaluation data are recorded. Access to the database was limited to the principal investigator. De-linked and de-identified participant demographics, diagnoses, raw scores and individual item endorsements on the specific test instruments were analyzed in SPSS.
CHAPTER 3

RESULTS

Descriptive Statistics

Counts and percentages were calculated for the categorical demographic variables: gender, diagnosis, and diagnosis severity. Participants for this study included 140 individuals (91 females, 49 males) who were obtained retrospectively from a database of community-dwelling patients, who presented during the period of 2003 to 2005 to a metropolitan outpatient clinic for a neuropsychological evaluation for dementia. Cognitive impairment diagnoses consisted of 41 cases of Alzheimer’s disease (AD, 29.3%), 41 cases of vascular dementia (VaD, 29.3%), 24 cases of cognitive disorder NOS or mild cognitive impairment (MCI, 17.1%), 13 cases of mixed dementia (13.6%), nine cases of other (6.4%), and three cases of none (2.1%). Diagnosis severity ranged from a high of 68 cases of mild (48.6%) to a low of 1 case of severe (0.7%). These results are summarized in Table 1. Means and standard deviations were also calculated for the continuous demographic variables: age ($M = 78.2$, $SD = 7.23$) and Mini-Mental State Examination (MMSE; $M = 24.86$, $SD = 3.35$). These results are summarized in Table 2.

The pilot study (Hall & Davis, 2008, in press) exploratory factor analysis (EFA) of the Geriatric Depression Scale (GDS) was used to set the criteria for GDS subscales and the specific GDS items that comprise each of those subscales used in this research. The subscales are Apathy, Dysphoria, Meaninglessness, and Cognitive Impairment. The standardized neuropsychological pencil and paper based testing instruments chosen to assess frontal systems and executive functions consisted of the Behavioral Dyscontrol Scale (BDS), Trail Making Test – Part A and Part B (TMT-A, TMT-B), and the Clock Drawing Task (CDT). Means, standard deviations, skewness, and kurtosis were calculated for the GDS total score, subscale scores, and
the noted neuropsychological test instruments. These results are summarized in Table 3. Alpha internal consistencies were calculated for the GDS and noted subscales with higher internal consistencies observed in subscales with a greater number of items. These results are summarized in Table 4.

Inferential Statistics

In order to broaden readership and interest in this research, both categorical and continuous data statistical research methodologies were undertaken to investigate the hypotheses and the relationship between the GDS subscales and the standardized neuropsychological instruments representing executive functions, BDS, CDT, TMT-A, and TMT-B.

Initial bivariate correlations were calculated between the GDS subscales and the standardized neuropsychological instruments noted as executive measures. Statistically significant ($p < .01$, two-tailed) correlations were noted between the four GDS subscales. Similarly, statistically significant ($p < .01$, two-tailed) correlations were observed between the three executive measures. Modest statistically significant ($p < .05$, two-tailed) correlations were observed between the GDS subscales Meaninglessness, Cognitive Impairment and the executive measures TMT-A, TMT-B, and the BDS. No significant correlations were observed between the GDS subscales Apathy, Dysphoria, and the executive measures. These results are summarized in Table 5.

Clinical relevance was investigated through the use of categorical statistical processes, grouping the study participants by existing established cutoff scores (presence/absence) of executive dysfunction or categorical level (normal, mild to moderate, severe) of executive dysfunction across the standardized neuropsychological instruments. The BDS was examined
with both the established cutoff score and categorical levels. The CDT was examined with the established cutoff score. The TMT-A and TMT-B were examined with categorical levels.

The hypotheses were investigated by observing the categorical relationship between the standardized neuropsychological instruments and the level of endorsement (mean number of endorsed items in each factor) of the Apathy and Dysphoria factors of the GDS. One-way analyses of variance (ANOVA s) were calculated to compare the category groups for statistical differences. The Meaninglessness and Cognitive Impairment factors were comparably analyzed to investigate their relationship with the standardized neuropsychological instruments.

A one-way ANOVA was calculated on the BDS cutoff score and the GDS subscales Apathy and Dysphoria. The other subscales Meaninglessness and Cognitive Impairment were included in the analysis. The analysis for Apathy was not significant, $F(1, 138) = .518, p = .473$. The analysis for Dysphoria was not significant, $F(1, 138) = .842, p = .360$. The analysis for Meaninglessness was also not significant, $F(1, 138) = .268, p = .606$. The analysis for Cognitive Impairment was significant, $F(1, 138) = 7.768, p = .006$. These results with means and standard deviations for each GDS subscale in each BDS cutoff group are summarized in Table 6. The mean numbers of endorsed items for the GDS subscales across BDS cutoff levels of impairment are graphically represented in Figures 1 through 4.

A one-way analysis of variance (ANOVA) was calculated on the BDS categorical scores and the GDS subscales Apathy and Dysphoria. The other subscales Meaninglessness and Cognitive Impairment were included in the analysis. The analysis for Apathy was not significant, $F(3, 136) = .855, p = .466$. The analysis for Dysphoria was not significant, $F(3, 136) = .360, p = .782$. The analysis for Meaninglessness was not significant, $F(3, 136) = .257, p = .856$. The analysis for Cognitive Impairment was also not significant, $F(3, 136) = 2.600, p = .055$. These
results with means and standard deviations for each GDS subscale in each BDS categorical group are summarized in Table 7. The mean numbers of endorsed items for the GDS subscales across BDS categorical levels of impairment are graphically represented in Figures 5 through 8.

A one-way ANOVA was calculated on the CDT cutoff score and the GDS subscales Apathy and Dysphoria. The other subscales Meaninglessness and Cognitive Impairment were included in the analysis. The analysis for Apathy was not significant, $F(1, 138) = .996, p = .320$. The analysis for Dysphoria was not significant, $F(1, 138) = .749, p = .388$. The analysis for Meaninglessness was not significant, $F(1, 138) = 1.227, p = .270$. The analysis for Cognitive Impairment was also not significant, $F(1, 138) = .559, p = .456$. These results with means and standard deviations for each GDS subscale in each CDT cutoff group are summarized in Table 8. The mean numbers of endorsed items for the GDS subscales across CDT cutoff levels of impairment are graphically represented in Figures 9 through 12.

A one-way ANOVA was calculated on the TMT-A categorical scores and the GDS subscales Apathy and Dysphoria. The other subscales Meaninglessness and Cognitive Impairment were included in the analysis. The analysis for Apathy was not significant, $F(2, 137) = .532, p = .589$. The analysis for Dysphoria was not significant, $F(2, 137) = 1.370, p = .258$. The analysis for Meaninglessness was not significant, $F(2, 137) = .917, p = .402$. The analysis for Cognitive Impairment was also not significant, $F(2, 137) = .894, p = .412$. These results with means and standard deviations for each GDS subscale in each TMT-A categorical group are summarized in Table 9. The mean numbers of endorsed items for the GDS subscales across TMT-A categorical levels of impairment are graphically represented in Figures 13 through 16.

A one-way ANOVA was calculated on the TMT-B categorical scores and the GDS subscales Apathy and Dysphoria. The other subscales Meaninglessness and Cognitive Impairment were included in the analysis. The analysis for Apathy was not significant, $F(2, 179) = .532, p = .589$. The analysis for Dysphoria was not significant, $F(2, 179) = 1.370, p = .258$. The analysis for Meaninglessness was not significant, $F(2, 179) = .917, p = .402$. The analysis for Cognitive Impairment was also not significant, $F(2, 179) = .894, p = .412$. These results with means and standard deviations for each GDS subscale in each TMT-B categorical group are summarized in Table 10. The mean numbers of endorsed items for the GDS subscales across TMT-B categorical levels of impairment are graphically represented in Figures 17 through 20.
Impairment were included in the analysis. The analysis for Apathy was not significant, $F(2, 137) = 1.040, p = .356$. The analysis for Dysphoria was not significant, $F(2, 137) = .207, p = .813$. The analysis for Meaninglessness was not significant, $F(2, 137) = 2.539, p = .083$. The analysis for Cognitive Impairment was also not significant, $F(2, 137) = .136, p = .873$. These results with means and standard deviations for each GDS subscale in each TMT-B categorical group are summarized in Table 10. The mean numbers of endorsed items for the GDS subscales across TMT-B categorical levels of impairment are graphically represented in Figures 17 through 20.

Empirical relevance was investigated through the use of a continuous statistical process. A step-wise hierarchical multiple regression model was used to investigate the relative amount of variance in the standardized neuropsychological instruments that is explained by the GDS subscales of Apathy and Dysphoria respectively. The stated hypotheses suggested that Apathy would explain the majority of the variance, while Dysphoria should not statistically add value to the regression model. Additionally, the Meaninglessness and Cognitive Impairment subscales were also similarly analyzed to investigate their contribution to the variance across the standardized neuropsychological instruments.

Within the hierarchical multiple regression model, the step-wise method criteria for independent predictor variables was set with a probability of $F$ to enter the model at $p \leq .05$ and be removed from the model at $p \geq .10$. By model design, if the probability method criteria were not met the hierarchical multiple regression model could not be calculated.

A step-wise hierarchical multiple regression was attempted for the BDS with the GDS subscales Apathy and Dysphoria. Neither GDS subscale met the probability of $F$ at $p \leq .05$ entry criterion and thus the model was not calculated. Correlations with the BDS for GDS subscales Apathy and Dysphoria were .099 and -.004, respectively, as noted in Table 5. An identical
regression was attempted for the BDS with the GDS subscales Meaninglessness and Cognitive Impairment. Meaninglessness (correlation -.093 with BDS, as noted in Table 5) did not meet the model entry criterion, while the model was significant with Cognitive Impairment. Adjusted R square = .029; \( F(1, 138) = 5.146, p = .025 \). These results are summarized in Table 11.

A step-wise hierarchical multiple regression was attempted for the CDT with the GDS subscales Apathy and Dysphoria. Neither GDS subscales met the probability of \( F \) at \( p \leq .05 \) entry criterion and thus the model was not calculated. Correlations with the CDT for GDS subscales Apathy and Dysphoria were -.026 and .022, respectively, as noted in Table 5. An identical regression was attempted for the CDT with the GDS subscales Meaninglessness and Cognitive Impairment. Neither Meaninglessness or Cognitive Impairment (respective correlations -.078, .050 with CDT, as noted in Table 5) met the entry criterion and thus the model was not calculated.

A step-wise hierarchical multiple regression was attempted for TMT-A with the GDS subscales Apathy and Dysphoria. Neither GDS subscale met the probability of \( F \) at \( p \leq .05 \) entry criterion and thus the model was not calculated. Correlations with TMT-A for GDS subscales Apathy and Dysphoria were -.077 and .000, respectively, as noted in Table 5. An identical regression was attempted for TMT-A with the GDS subscales Meaninglessness and Cognitive Impairment. The model was significant with both Meaninglessness and Cognitive Impairment. Adjusted \( R \) square = .070; \( F(2, 137) = 6.197, p = .003 \). These results are summarized in Table 12.

A step-wise hierarchical multiple regression was attempted for TMT-B with the GDS subscales Apathy and Dysphoria. Neither GDS subscale met the probability of \( F \) at \( p \leq .05 \) entry criterion and thus the model was not calculated. Correlations with the BDS for GDS subscales Apathy and Dysphoria were .091 and -.001, respectively, as noted in Table 5. An identical
regression was attempted for TMT-B with the GDS subscales Meaninglessness and Cognitive Impairment. Cognitive Impairment (correlation -.093 with BDS, as noted in Table 5) did not meet the model entry criterion, while the model was significant with Meaninglessness. Adjusted R square = .035; $F(1, 138) = 6.017, p = .015$. These results are summarized in Table 13.

Hypotheses

Two hypotheses were proposed:

1. AD and related dementia patients who endorsed a high level of apathy category items on the GDS will show a poorer performance on administered measures of executive functions than AD and related dementia patients who endorsed a lower level of apathy category items.

2. AD and related dementia patients who endorsed a high level of dysphoria category items on the GDS while endorsing a low level of apathy category items will not have significantly different findings on administered measures of executive functions than AD and related dementia patients who endorsed a low level of dysphoria category items.

Hypothesis Testing

The ANOVAs and the step-wise hierarchical multiple regression analyses did not support the first hypothesis. With respect to the ANOVAs, the mean number of endorsed GDS Apathy items was not statistically different across the impaired and non-impaired groups of the administered measures of executive functions. Due to the lack of statistical differences, no additional analyses were undertaken to develop a cut score on the GDS Apathy subscale that
would suggest the clinical presence or absence of apathy and, thus, executive dysfunction. With respect to the hierarchical multiple regression analyses, the GDS Apathy predictor variable did not meet the minimum criterion to enter the model, and thus did not support the first hypothesis.

In order to verify support for the second hypothesis, the analyses would need to support the first hypothesis. With respect to the analyses, in the ANOVAs, the mean number of endorsed GDS Dysphoria items was not statistically different across the impaired and non-impaired groups. In the hierarchical multiple regression analyses GDS Dysphoria did not statistically add value to the regression model. In light of the fact that the first hypothesis was not supported, the second hypothesis could not be supported.
CHAPTER 4
DISCUSSION
Purpose of Study

This study investigated the relationship between apathy, depression, and executive functions in individuals with Alzheimer’s disease (AD) and related dementias. Two objectives were established. The first objective of this study was to determine if apathy had a greater impact on executive functions compared to depression. The second objective was to determine the effectiveness of the Geriatric Depression Scale (GDS) as a screen for apathy. Two hypotheses were established. First, AD and related dementia patients who endorsed a high level of apathy category items on the GDS will show a poorer performance on administered measures of executive functions than AD and related dementia patients who endorsed a lower level of apathy category items. Second, AD and related dementia patients who endorsed a high level of dysphoria category items on the GDS while endorsing a low level of apathy category items will not have significantly different findings on administered measures of executive functions than AD and related dementia patients who endorsed a low level of dysphoria category items.

Overall Summary of Results

This study yielded some interesting, yet albeit, non-supportive results. Significant mean differences were not observed for the GDS Apathy subscale across the levels of impairment on the examined executive measures. Furthermore, within the regression analyses, the GDS Apathy subscale did not significantly contribute to the model. These non-significant statistical analyses resulted in a lack of support for the aforementioned hypotheses and thus the two study objectives. The first study objective to determine if apathy had a greater impact on executive
functions compared to depression was not ascertained through the analyses. In light of these indeterminate results, the second objective to determine the effectiveness of the GDS as a screen for apathy was not empirically supported. The initial bivariate correlations that were calculated between the GDS subscales and the standardized neuropsychological instruments established as executive measures showed statistically significant \((p < .01, \text{two-tailed})\) correlations among the four GDS subscales as well as statistically significant \((p < .01, \text{two-tailed})\) correlations among the three executive measures. No significant correlations were observed between the GDS subscales Apathy, Dysphoria, and the executive measures. While not the focus of the hypotheses, modest statistically significant \((p < .05, \text{two-tailed})\) correlations between the GDS subscales Meaninglessness, Cognitive Impairment and the executive measures Trail Making Test – Part A and Part B (TMT-A, TMT-B) and the Behavioral Dyscontrol Scale (BDS) were of interest.

Possible Conceptual Explanations for Study Findings

While a multitude of possible explanations exist for these results, the following discussion will examine a few of perhaps the most reasonable explanations that are supported by the study findings, study limitations, and the relevant literature. This examination should set the framework and direction for future research. Four possible conceptual explanations for the study findings will be explored. First, the validity and reliability of the GDS subscales. Second, different diagnoses have different relationships with impairment and affective symptoms. Third, the relationship of symptoms, as noted by GDS subscales, to neuropsychological variables is not linear and is related to severity of impairment, not specific neuropsychological domains. Fourth, there is some particular interaction between diagnosis, symptoms, level of impairment, and neuropsychological domain that is obscuring significant relationships. It should be noted that the
four areas of explanation are theoretically and clinically overlapping between one another. Data limitations with respect to linearity and normality will be addressed throughout.

Validity and Reliability of the GDS Subscales

A discussion of the validity and reliability of the GDS subscales may best begin with a review of the relationship between constructs and measures. DeVellis (2003) noted items within a scale are usually a means to an end for the assessment of a construct. In a way, measures are substitutes for variables that we cannot observe. As the relationship between measures is assessed, the relationship between constructs is inferred. The underlying phenomenon or construct that a scale is intended to reflect is frequently called the latent variable (DeVellis, 2003). It is latent in that it is often not directly observable and variable in that its magnitude or strength changes. A scale or measure is designed to estimate the unobservable actual magnitude of a variable, which is known as its true score. DeVellis (2003) noted that this latent variable is regarded as the cause of the item or set of items to take on a certain score or value. It becomes evident that if an underlying latent variable or phenomenon is not clearly defined or well agreed upon, this will become problematic in creating items or scales of items that are generally accepted to represent the phenomenon. As DeVellis (2003) noted reliability is concerned with how much a variable influences a set of items while validity is concerned with whether the latent variable is the underlying cause of item covariation. As noted in the Introduction section, much work has been undertaken to precisely define the construct of apathy. These definitions often have philosophical underpinnings and a heterogeneous taxonomy. The abstract nature of general definitions for apathy, such as a lack of interest, emotion, or motivation, makes it difficult to empirically determine if an instrument actually corresponds to a latent variable it is purported to
measure. This difficulty or lack of precision resulted in Stuss et al. (2000) to define apathy as, “an absence of responsiveness to stimuli as demonstrated by a lack of self-initiated action,” which allows for objective behavioral measurements. As noted, initiation is central to this definition and the self-initiated response may be affective, behavioral or cognitive in nature (van Reekum et al., 2005). A recurring theme noted throughout the literature on apathy is the tendency for it to be defined based on its psychological cause and the associated neuroanatomical region involved (Marin, 1990; Marin, 1997; Stuss et al., 2000). While numerous theoretical conceptualizations exist, Stuss et al. (2000) conceptualized two basic types of apathy. The first being related to disturbed arousal perhaps associated with the reticular activating system, and the second associated with frontal-subcortical dysfunction, which they labeled executive apathy. Based on this distinction and general review of the literature, it appeared reasonable to interpret executive apathy as the type of apathy most associated with AD and related dementias.

With respect to the reliability and validity of the Apathy subscale as found in the study by Hall and Davis (2008, in press), the basic empirical questions that arise are how much the underlying latent variable or construct of executive apathy as defined by Stuss et al. (2000) influences the items in the Apathy subscale (reliability) and is the cause of the Apathy subscale item covariation (validity). When examining the reliability and validity of the subscales within the Hall and Davis (2008, in press) it is helpful to review the method used to establish the original unidimensional GDS. The GDS was designed by a team of clinicians and researchers involved in geriatric psychiatry who selected 100 questions that were believed to potentially distinguish the elderly depressed from normals (Yesavage et al., 1983). They looked for potential questions that were relevant to depression, such as somatic complaints, cognitive complaints, motivation, future/past orientation, self-image, losses, agitation, obsessive traits, and mood. The
initial data analysis was based on the rationale that the initial 100 items on the scale should have *prima facia* validity for depression. Ultimately they settled on 30 items that were most highly and significantly correlated. Based on the research, the remaining 30 items were viewed to be the core of geriatric depression and included lowered affect, inactivity, irritability, withdrawal, distressing thoughts, and negative judgments about the past, present, and future (Brink et al., 1982). With respect to validity, the GDS showed a comparable or superior ability to differentiate between depressed and normals as compared to other measures (Yesavage et al., 1983). With respect to reliability, the Cronbach’s alpha coefficient was used to measure overall internal consistency of the GDS and found to be .94 in the original study (Yesavage et al., 1983). GDS total score (30 items) Cronbach’s alpha coefficients for Hall and Davis (2008, in press) and the present study were .838 and .816, respectively.

The GDS subscales as derived in the Hall and Davis (2008, in press) research appeared to have reliability coefficients that are consistent with other literature. That study reported the GDS subscales with Cronbach’s alpha coefficients for Apathy (6 items), Dysphoria (11 items), Meaninglessness (7 items), and Cognitive Impairment (6 items) as .634, .793, .721, and .468, respectively with 173 participants. The present study obtained the respective Cronbach’s alpha coefficients of .505, .755, .635, and .596 with 140 participants. The present study had 140 participants while the Hall and Davis (2008, in press) had 173. Other GDS factor analyses performed on non-demented samples had comparable reliability coefficients (Adams, 2001; Adams et al., 2004). Parmelee et al. (1989) obtained a six subscale structure for the GDS with Cronbach’s alpha measures of internal consistency at .91, .60, .58, .46, .54, and .51 for Dysphoria (14 items), Worry (4 items), Withdrawal/Apathy (4 items), Vigor (3 items), Decreased Concentration (2 items), and Anxiety (3 items) respectively, with a sample size of
Based on the values of internal consistency found in their study, Parmelee et al. (1989) recommended the GDS continue to be used as a single total scale. Cronbach’s alpha represents the proportion of a subscale’s total variance that is attributable to a common source, which presumably would be the true score of the latent variable underlying the items (DeVellis, 2003). With respect to the Apathy subscale, the alpha values were .505 and .634, for the present study and the Hall and Davis (2008, in press) study, respectively. This suggests in the present study roughly half of the variation is common variance to total variance. This is less than desirable for statistical purposes.

Studies that have shown a statistically significant linear relationship between measures of apathy and executive dysfunction have often utilized apathy measures that were endorsed by the caregiver as opposed to the actual patient (Cummings et al., 1994; McPherson et al., 2002; Landes et al., 2001). The general rationale for using caregiver endorsement as opposed to the patient is that the psychological processes and neuroanatomical substrates involved in apathy may result in an impairment of awareness, and more particularly, self-awareness (Cummings et al., 1994; McPherson et al., 2002; Landes et al., 2001). The level of participant self-awareness may not be adequately assessed by the Mini-Mental State Examination (MMSE) (Folstein et al., 1975), which was used to determine inclusion in the present study. Tabachnick and Fidell (2001) noted that a good factor analysis will “make sense” while DeVellis (2003) reflected on a parsimonious account of the factors. Both of these concepts were considered with the Hall and Davis (2008, in press) factor analytic derived four subscale structure used in this study. While obviously designed as a unidimensional instrument, the widespread use of the GDS makes it a well-desired source to glean additional empirical and clinical information. Future studies utilizing the Hall and Davis (2008, in press) GDS subscales may benefit from concurrent
endorsement by caregivers. Cummings et al. (1994) developed the Neuropsychiatric Inventory (NPI), a 10-domain scale used for the assessment of psychopathology in dementia (McPherson et al., 2002). The information is gathered from an individual who has daily contact with the patient and is familiar with the patient’s behavior.

Relationships of Impairment and Affective Symptoms Vary across Diagnoses

A second explanation for the study findings may be the possibility that different diagnoses have different relationships with impairment and affective symptoms. The neuropsychiatric expression and neuroanatomical correlated pathology differ across Alzheimer’s disease and the related dementias (Cummings, 2003). The temporal pattern of symptomatology also varies across diagnoses. For example, the hallmark of frontotemporal dementia (FTD) is marked by the onset of behavioral changes, such as apathy and disinhibition with a later onset of cognitive impairment. Alzheimer’s disease (AD) follows a gradually progressive course. Vascular dementia (VaD) is frequently characterized by an abrupt onset with subsequent periods of sudden cognitive decline, often described as a stair-step progression (Cummings, 2003). The initial neuroanatomical substrates and the progressive spread of histopathology also vary by type of dementia (Cummings, 2003; Landes et al., 2001; Levy et al, 1998; Schultz, Del Tredici, & Braak, 2004; Tekin & Cummings, 2002). This variation in location of onset and progression may have implications on the severity and temporal onset of neuropsychiatric symptoms. A study performed by Levy et al. (1998) hypothesized that apathy was different from depression, speculated that the two symptoms are produced by different neuroanatomical or neurochemical substrates, and were manifested differently in various neurologic disorders. The results by Levy et al. (1998) noted a large number of AD patients and even more FTD and progressive
supranuclear palsy (PSP) patients had apathy without depression, whereas many Parkinson’s
disease (PD) and Huntington’s disease (HD) patients had depression without apathy. Levy et al.
(1998) determined that generally across diagnoses apathy was correlated with cognitive
impairment while depression was not. The relationship between apathy and depression appeared
to be disease-specific. AD, FTD, and PSP had more prevalent and severe apathy, while PD and
HD had more prevalent and severe depression. PSP patients exhibited the most frequent and
severe apathy and the least depression.

An exploratory one-way ANOVA with the current study noted a statistically significant
$F(5, 131) = 3.264, p = .008$ difference between the mean endorsements of the GDS Apathy
subscale for AD ($M = 1.15, SD = 1.15$) and VaD ($M = 2.00, SD = 1.34$). The higher mean
endorsement for VaD over AD is consistent with the literature noting the increased subcortical
involvement in VaD (Cummings, 2003; de la Torre, 2002). Cummings (2003) noted that some
VaD patients evidence a predominance of white matter ischemic injury with a clinical syndrome
that tends to be gradually progressive in contrast to one punctuated by periods of acute decline.
Cognitively and behaviorally, these patients manifest prominent frontal executive dysfunction
with diminished motivation, loss of insight, apathy, and abulia (Cummings, 2003). Studies with
the NPI (Cummings et al., 1994) revealed more severe agitation, depression, anxiety, and apathy
in patients with subcortical VaD compared with patients with AD (Cummings, 2003). Of the ten
domains of the NPI, the apathy domain had the highest mean endorsement in VaD compared to
the other domains (Cummings, 2003). Perhaps it is possible that the disruption of the anterior
cingulate-subcortical circuit so often implicated in apathy is more greatly impacted in VaD
versus AD. While a growing body of literature notes the lack of mutual exclusivity of VaD and
AD (de la Torre, 2002), differentiating levels of neuropsychiatric symptoms clearly exist between the two dementias (Cummings, 2003).

**Non-linear Relationship between Symptoms and Neuropsychological Instruments**

A third explanation for the study findings may be the possibility that the relationship of symptoms, as noted by GDS subscales, to neuropsychological variables is not linear and is related to severity of impairment, not specific neuropsychological domains. An exploratory one-way ANOVA with the current study did not show a statistically significant linear relationship with any of the GDS subscales and diagnosis severity. By study design and composition of patients in the database, moderate to severe and severely demented individuals were not included while mild, mild to moderate, and moderately demented individuals were included in the study. A review of the GDS subscale mean endorsement levels across diagnosis severity shows a non-linear relationship. This type of endorsement may be a result of a non-linear interaction between an increase in neuropsychiatric symptoms as described by the GDS subscales and an increasing lack of insight into these symptoms by the patient as the dementing processes become more severe. As noted in the introduction, anosognosia, or a lack of awareness of one’s cognitive deficits is often found in AD and related dementias (Landes et al., 2001; Ott et al., 1996). Anosognosia in AD has been shown to be highly correlated with apathy, which suggests that this unawareness of deficits is more closely related to emotional changes than to cognitive impairment (Landes et al., 2001). This begs the question as to the relationship of emotional changes to cognitive impairment. Is the relationship linear? How do severity of cognitive impairment and emotional changes impact the other? Is there an interaction? Also as noted in the introduction, Damasio (1994) investigated anosognosia in patients who had right somatosensory...
damage located in the right temporoparietal cortical region and suggested these individuals exhibited similar deficits as those with prefrontal damage. These patients with right temporoparietal damage were unable to make appropriate decisions on personal and social matters, and the patients with prefrontal damage, like patients with temporoparietal damage were usually indifferent to their health status or impairment. The relationship of the lack of insight into one’s emotional function longitudinally through the dementing process may not be a constant or in other words linear. The relationship between cognitive impairment and emotional function certainly bears further elucidation. A substantial body of literature has linked apathy and its behavioral correlates to executive dysfunction (Cummings, 2003; Kuzis et al., 1999; McPherson et al., 2002). However, executive dysfunction is not discrete from other cognitive domains. This also calls into question the relationship of a particular neuropsychological instrument to the domain it is purported to represent.

An Interaction Obscuring Significant Relationships

A fourth explanation for the study findings may be the possibility that there is some particular interaction between diagnosis, symptoms, level of impairment, and neuropsychological domain that is obscuring significant relationships. A substantial portion of the discussion has focused on specific possible interactions between study variables. It is possible that there is an interaction that is either obscuring or cancelling out various relationships based on the statistical methods employed in this study. One possibility that could be obscuring significant relationships between GDS subscale endorsement and executive measures may be an interaction between a patient’s normative sense of decline associated with aging versus an autobiographical awareness of their own dysfunction or impairment. This is consistent with Damasio’s (1994) description of
anosognosia in which, in the absence of self-awareness, a patient will apply what they perceive as a normative, hypothetical endorsement or response pattern to their own situation based on generalized stereotypical conceptualizations. While maybe not fully analogous with the concept of confabulation, the substitution of perceived normative responses for responses based on actual self-awareness would appear comparable. Concurrent endorsement of the GDS by caregiver perception of the patient’s level of function could help differentiate this type of hypothetical normative endorsement by a patient from an endorsement that actually reflects self-awareness.

As noted by Landes et al. (2001), apathy was found to be the main correlate of anosognosia which, like apathy is associated with decreased cerebrovascular perfusion of the frontal lobe. This line of thought sheds light on the heterogeneity and overlap between diagnosis, symptoms, level of impairment, and neuropsychological domain. This heterogeneity is also observed in research into the etiology and neuropathology of late-life depression.

A substantial body of literature investigating the course of cognitive deficits associated with depression in the elderly often implicates specific impairments associated with apathy under an inclusive conceptualization of depression (Alexopoulos et al., 2000; Keller, Buckley, & Schatzberg, 2004; Krishnan, 2004; Schultz, Del Tredici, & Braak, 2004; Small, 2004). While apathetic behaviors exist within the clinical definition of depression, this lack of precision may lead to a lack of clarity in the literature. The importance of this precision is critical as much of the literature often notes depression as a precursor to dementia (Keller, Buckley, & Schatzberg, 2004; Krishnan, 2004; Rapp et al., 2006; Steffens et al., 2000; Steffens et al., 2002).

Krishnan (2004) noted the links between depression and cognition being complex and multifactorial. Krishnan (2004) further noted that the neuropathology associated with AD and VaD is overlapping with both neurodegenerative and vascular processes occurring in both
dementias. The neurodegenerative process involves hippocampal volume and APOE genotype while the vascular process specifically related to depression involves frontal and striatal areas, particularly the prefrontal cortex, caudate (Krishnan, 2004) and hypometabolism of the rostral anterior cingulate (Alexopoulos, 2000). Memory impairment is the hallmark of dementia. While memory function or dysfunction is often related to hippocampal integrity and volume (Rapp et al., 2006; Steffens et al., 2000; Steffens et al., 2002), successful memory encoding and retrieval strategies are frontally mediated (Luria, 1980; Lezak et al., 2004; Stuss & Levine, 2002) providing further evidence of the multifactorial nature of successful memory function.

The literature further describes the multifactorial relationship between hippocampal volume (Rapp et al., 2006; Steffens et al., 2000; Steffens et al., 2002), amygdala volume, hypothalamic-pituitary-adrenal axis dysregulation (Keller et al., 2004), executive dysfunction (Alexopoulos, 2000), apathy (Landes et al., 2001; van Reekum, 2005), depression, and memory with respect to AD and the related dementias (de la Torre, 2002; Krishnan, 2004; Schultz et al., 2004; Small, 2004). While these relationships are complex and overlapping, it becomes clear for the necessity of precise definitions and operationalizations in the ongoing research.

Another possibility that may be obscuring the results is the relative insensitivity of standard neuropsychological executive function measures to executive dysfunction in the ventral prefrontal cortex (VPFC). Stuss et al. (2001) indicated that patients with VPFC damage were not impaired on the TMT-B. Stuss and Levine (2002) noted that much of what is known about frontal or executive functions is based on neuropsychological studies on patients with dysfunction in the dorsolateral prefrontal cortex (DLPFC) and that this is the frontal area most of the standardized neuropsychological instruments are suggested to assess. They indicated patients with VPFC damage can appear normal on frontal tests of PLPFC functions. Stuss and Levine
(2002) further noted that the primary distinction between the function of the DLPFC and the VPFC is cognitive and affective, respectively. They indicated that within the frontal lobes further functional/anatomical distinctions can be made. Specifically pertinent to this discussion is the fact that superior medial lesions, particularly in the anterior cingulate, can cause an apathetic syndrome in which the functional basis of this impairment is a lack of initiation (Stuss et al., 2000). The frontal poles, which are anatomically close to the anterior cingulate, are involved in more recently evolved human processes—particularly autonoetic (or “self-knowing”) consciousness and self-awareness (Stuss & Levine, 2002). This speaks directly to the decreasing self-awareness that is seen in AD and the related dementias. Damage in the VPFC region has a tremendous impact on decision making and strategic self-regulation (Damasio, 1994; Stuss & Levine, 2002). Stuss and Levine (2002) use the term “self-regulatory disorder” (SRD) to describe patients who have an inability to regulate behavior according to internal goals and constraints. They noted that it arises from the inability to hold a mental representation of the self on-line and to use this self-related information to inhibit inappropriate responses. Stuss and Levine (2002) further indicated that SRD becomes most apparent in unstructured situations as opposed to structured situations where an individual can rely upon over-learned routines or pick up on environmental cues such as in a structured neuropsychological examination. The evidence of frontal lobe involvement in self-awareness has been obtained through memory research (Stuss & Levine, 2002). Right frontal lobe activation is associated with episodic memories. These memories are described as temporally tagged memories that are personally relevant, emotionally salient to the individual, and involved in self-awareness (Stuss & Levine, 2002). Stuss & Levine (2002) further noted that research supports a preeminent role of the right frontal lobe for personal memory, self-reflective memory, and when retrieving emotional memories from the past. Stuss
Levine (2002) relate frontal lobe impairment and deficits in self-awareness that are difficult to detect with standard neurological or neuropsychological assessment:

Performance on executive tests was normal/superior in one, and only mildly impaired in the other. Both patients could clearly identify their failings, exhibited concern about their problems, and could identify appropriate corrections to the problems. Neither, however, could return to their high-level executive work. The deficits in these patients were not at the level of executive control. Rather, they had a lack of real understanding of the implications of the problems and an inability to act in their own self-interest, despite knowing what to do and at least verbalizing an intent to change. These patients lack a mental model, not of the world, but of their own capacities and role in the world. The discrepancy between their mental model and their experience leaves them without a purpose or ability to organize perceptions and actions for future goals. (p. 421-422)

While these patients were not suffering from the behavioral precursors of AD or any related dementias, the functional impact of their impairment is analogous to the loss of self-reflection and loss of a personal sense of relationship to the world. This loss of self-awareness, while devastating to the individual, is perhaps even more catastrophic to family members and caregivers. The role of affect in the cognitive process appears critical to successfully initiate one’s cognitive capacity adaptively in service to one’s self.

General Conclusions

This study investigated the relationship between apathy, depression, and executive functions in individuals with AD and related dementias. The first objective of this study was to determine if apathy had a greater impact on executive functions compared to depression. The second objective was to determine the effectiveness of the GDS as a screen for apathy. The two hypotheses established to meet these objectives were not supported with the analyses. However, exploratory analyses suggested a possible non-linear relationship with apathy and various levels of dementia severity. Exploratory analysis also suggested mean levels of endorsement for apathy
varied by diagnosis. This study, while not confirming the established hypotheses, did suggest there some type of relationship with apathy and dementia.

Study Limitations

This study had several limitations that may have impacted the results. First, most clinical databases will have limitations such as missing data as well as skewed or kurtotic variables that impact the conclusions that can be reached under the general linear model. This database was no exception. Furthermore, the study design itself limited the range of dementia severity and thus may have decreased the opportunity to observe a statistically significant relationship between the study variables.

Future Research

The next step for future research would be to perform a confirmatory factor analysis (CFA) on the existing principal components analysis (PCA) to further validate the GDS factor structure on the cognitively impaired elderly. Additional executive measures that tap various aspects of executive function, particularly functions found to be associated with the VPFC, should be incorporated to further elucidate the relationship between apathy as measured by the GDS subscale and executive dysfunction. Non-parametric statistical procedures could be applied to the existing data to further understand the possible non-linear relationship between the variables. For future patients, it would be interesting to have a caregiver concurrently fill out the GDS to see if the caregiver’s endorsement pattern differs from that of the patient. Other studies have used caregiver endorsement measures to minimize the effect of the patient’s diminished
insight due to the dementing process. The existing widespread use of the GDS makes it a desirable measure to increase its utility on the growing population of demented elderly.

Table 1

Counts and Percentages of Categorical Demographic Data (N = 140)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>49</td>
<td>35</td>
</tr>
<tr>
<td>Female</td>
<td>91</td>
<td>65</td>
</tr>
<tr>
<td>Diagnosis</td>
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<td></td>
</tr>
<tr>
<td>Alzheimer’s Dementia</td>
<td>41</td>
<td>29.3</td>
</tr>
<tr>
<td>Vascular Dementia</td>
<td>41</td>
<td>29.3</td>
</tr>
<tr>
<td>Cognitive Disorder NOS</td>
<td>24</td>
<td>17.1</td>
</tr>
<tr>
<td>Mixed</td>
<td>13</td>
<td>13.6</td>
</tr>
<tr>
<td>Other</td>
<td>9</td>
<td>6.4</td>
</tr>
<tr>
<td>None</td>
<td>3</td>
<td>2.1</td>
</tr>
<tr>
<td>Diagnosis Severity</td>
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<tr>
<td>Mild</td>
<td>68</td>
<td>48.6</td>
</tr>
<tr>
<td>Mild to Moderate</td>
<td>16</td>
<td>11.4</td>
</tr>
<tr>
<td>Moderate</td>
<td>39</td>
<td>27.9</td>
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<tr>
<td>Moderate to Severe</td>
<td>2</td>
<td>1.4</td>
</tr>
<tr>
<td>Severe</td>
<td>1</td>
<td>0.7</td>
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Table 2

*Descriptive Statistics for Continuous Demographic Variables (N = 140)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age</td>
<td>78.20</td>
<td>7.23</td>
<td>28.0</td>
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<tr>
<td>Mini-Mental State Exam</td>
<td>24.86</td>
<td>3.35</td>
<td>15.0</td>
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</table>

Table 3

*Descriptive Statistics for Test Variables (N = 140)*

<table>
<thead>
<tr>
<th>Test Instruments</th>
<th>M</th>
<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
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<tr>
<td>Geriatric Depression Scale</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apathy</td>
<td>1.66</td>
<td>1.39</td>
<td>3.12</td>
<td>-0.49</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>2.71</td>
<td>2.52</td>
<td>3.71</td>
<td>-0.13</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>1.26</td>
<td>1.45</td>
<td>6.80</td>
<td>4.81</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>2.11</td>
<td>1.52</td>
<td>2.29</td>
<td>-0.78</td>
</tr>
<tr>
<td>Total Score</td>
<td>7.74</td>
<td>4.97</td>
<td>2.75</td>
<td>-0.30</td>
</tr>
<tr>
<td>Behavioral Dyscontrol Scale</td>
<td>12.00</td>
<td>3.68</td>
<td>-1.77</td>
<td>-1.32</td>
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<tr>
<td>Trail Making Test – Part A</td>
<td>77.96</td>
<td>55.02</td>
<td>11.76</td>
<td>16.27</td>
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<tr>
<td>Trail Making Test – Part B</td>
<td>228.59</td>
<td>79.75</td>
<td>-2.61</td>
<td>-3.10</td>
</tr>
<tr>
<td>Clock Drawing Task</td>
<td>2.93</td>
<td>1.05</td>
<td>-3.54</td>
<td>-0.85</td>
</tr>
</tbody>
</table>
Table 4

*Reliability Coefficients of the Geriatric Depression Scale and Subscales (N = 140)*

<table>
<thead>
<tr>
<th>GDS Variable</th>
<th>No. of Items</th>
<th>Cronbach’s α</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphoria</td>
<td>11</td>
<td>.755</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>7</td>
<td>.635</td>
</tr>
<tr>
<td>Apathy</td>
<td>6</td>
<td>.505</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>6</td>
<td>.596</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>.816</td>
</tr>
</tbody>
</table>
Table 5

*Correlations Between Geriatric Depression Subscales and Executive Measures (N = 140)*

<table>
<thead>
<tr>
<th>GDS Subscale</th>
<th>TMT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Apathy</td>
</tr>
<tr>
<td>Apathy</td>
<td>—</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>—</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>—</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>—</td>
</tr>
<tr>
<td>Part A</td>
<td>—</td>
</tr>
<tr>
<td>Part B</td>
<td>—</td>
</tr>
<tr>
<td>CDT</td>
<td>—</td>
</tr>
<tr>
<td>BDS</td>
<td>—</td>
</tr>
</tbody>
</table>

*p < .05 (2-tailed). **p < .01 (2-tailed).
### Table 6

**One-way ANOVA Mean Differences of GDS Subscale Endorsements: BDS Cutoff**

<table>
<thead>
<tr>
<th>GDS Subscale</th>
<th>Impaired (n = 46)</th>
<th>Non-Impaired (n = 94)</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apathy</td>
<td>1.54 (1.24)</td>
<td>1.72 (1.45)</td>
<td>138</td>
<td>.518</td>
<td>.473</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>2.43 (2.30)</td>
<td>2.85 (2.62)</td>
<td>138</td>
<td>.842</td>
<td>.360</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>1.35 (1.51)</td>
<td>1.21 (1.42)</td>
<td>138</td>
<td>.268</td>
<td>.606</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>1.61 (1.31)</td>
<td>2.35 (1.56)</td>
<td>138</td>
<td>7.768**</td>
<td>.006</td>
</tr>
</tbody>
</table>

**p < .01.

### Table 8

**One-way ANOVA Mean Differences of GDS Subscale Endorsements: CDT (N = 140)**

<table>
<thead>
<tr>
<th>GDS Subscale</th>
<th>Impaired (n = 89)</th>
<th>Non-Impaired (n = 51)</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apathy</td>
<td>1.75 (1.35)</td>
<td>1.51 (1.45)</td>
<td>138</td>
<td>.996</td>
<td>.320</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>2.85 (2.67)</td>
<td>2.47 (2.23)</td>
<td>138</td>
<td>.749</td>
<td>.388</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>1.36 (1.52)</td>
<td>1.08 (1.29)</td>
<td>138</td>
<td>1.227</td>
<td>.270</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>2.18 (1.56)</td>
<td>1.98 (1.44)</td>
<td>138</td>
<td>.559</td>
<td>.456</td>
</tr>
</tbody>
</table>
Table 7

One-way ANOVA Mean Differences of GDS Subscale Endorsements: BDS Categorical ($N = 140$)

<table>
<thead>
<tr>
<th>GDS Subscale</th>
<th>Severe (n = 15)</th>
<th>Moderate (n = 31)</th>
<th>Mild (n = 69)</th>
<th>Non-Impaired (n = 25)</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apathy</td>
<td>1.13</td>
<td>0.99</td>
<td>1.74</td>
<td>1.32</td>
<td>1.70</td>
<td>1.45</td>
<td>1.80</td>
<td>1.50</td>
<td>136</td>
<td>.855</td>
<td>.466</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysphoria</td>
<td>2.60</td>
<td>2.38</td>
<td>2.35</td>
<td>2.29</td>
<td>2.91</td>
<td>2.75</td>
<td>2.68</td>
<td>2.27</td>
<td>136</td>
<td>.360</td>
<td>.782</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>1.40</td>
<td>1.45</td>
<td>1.32</td>
<td>1.56</td>
<td>1.27</td>
<td>1.51</td>
<td>1.04</td>
<td>1.36</td>
<td>136</td>
<td>.257</td>
<td>.856</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>1.53</td>
<td>1.36</td>
<td>1.64</td>
<td>1.30</td>
<td>2.38</td>
<td>1.61</td>
<td>2.28</td>
<td>1.43</td>
<td>136</td>
<td>2.600</td>
<td>.055</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 9

One-way ANOVA Mean Differences of GDS Subscale Endorsements: TMT-A (N = 140)

<table>
<thead>
<tr>
<th>GDS Subscale</th>
<th>Severe (n = 89)</th>
<th>Mild to Moderate (n = 25)</th>
<th>Non-Impaired (n = 26)</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apathy</td>
<td>1.74 1.43</td>
<td>1.64 1.29</td>
<td>1.42 1.36</td>
<td>137</td>
<td>.532</td>
<td>.589</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>2.79 2.42</td>
<td>3.16 3.20</td>
<td>2.04 2.05</td>
<td>137</td>
<td>1.370</td>
<td>.258</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>1.36 1.53</td>
<td>1.24 1.20</td>
<td>.923 1.35</td>
<td>137</td>
<td>.917</td>
<td>.402</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>1.98 1.38</td>
<td>2.32 1.75</td>
<td>2.35 1.72</td>
<td>137</td>
<td>.894</td>
<td>.412</td>
</tr>
</tbody>
</table>
Table 10

*One-way ANOVA Mean Differences of GDS Subscale Endorsements: TMT-B (N = 140)*

<table>
<thead>
<tr>
<th>GDS Subscale</th>
<th>Severe (n = 121)</th>
<th>Mild to Moderate (n = 14)</th>
<th>Non-Impaired (n = 5)</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apathy</td>
<td>1.69 1.37</td>
<td>1.79 1.58</td>
<td>.80 1.30</td>
<td>137</td>
<td>1.040</td>
<td>.356</td>
</tr>
<tr>
<td>Dysphoria</td>
<td>2.74 2.57</td>
<td>2.71 2.46</td>
<td>2.00 1.41</td>
<td>137</td>
<td>.207</td>
<td>.813</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>1.35 1.50</td>
<td>.93  .83</td>
<td>.00  .00</td>
<td>137</td>
<td>2.539</td>
<td>.083</td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>2.11 1.50</td>
<td>2.21 1.80</td>
<td>1.80 1.30</td>
<td>137</td>
<td>.136</td>
<td>.873</td>
</tr>
</tbody>
</table>
### Table 11

*Summary of Step-wise Hierarchical Multiple Regression Analysis for GDS Subscales Predicting Variance in BDS (N = 140)*

<table>
<thead>
<tr>
<th>Geriatric Depression Subscale</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>.460</td>
<td>.203</td>
<td>.190*</td>
</tr>
</tbody>
</table>

*Note. Adjusted R² = .029 for Step 1; F(1, 138) = 5.146, *p < .05.*

### Table 12

*Summary of Step-wise Hierarchical Multiple Regression Analysis for GDS Subscales Predicting Variance in TMT-A (N = 140)*

<table>
<thead>
<tr>
<th>Geriatric Depression Subscale</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>-7.371</td>
<td>3.026</td>
<td>-.203*</td>
</tr>
<tr>
<td>Step 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive Impairment</td>
<td>-8.710</td>
<td>3.018</td>
<td>-.240*</td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>7.897</td>
<td>3.163</td>
<td>.208*</td>
</tr>
</tbody>
</table>

*Note. Adjusted R² = .034 for Step 1; Adjusted R² = .070 for Step 2; F(2, 137) = 6.197, *p < .05.*

### Table 13

*Summary of Step-wise Hierarchical Multiple Regression Analysis for GDS Subscales Predicting Variance in TMT-B (N = 140)*

<table>
<thead>
<tr>
<th>Geriatric Depression Subscale</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meaninglessness</td>
<td>11.271</td>
<td>4.595</td>
<td>.204*</td>
</tr>
</tbody>
</table>

*Note. Adjusted R² = .035 for Step 1; F(1, 138) = 6.017, *p < .05.*
**Figure 1.** Mean number of endorsed GDS Apathy subscale items across BDS Cutoff levels of impairment.

**Figure 2.** Mean number of endorsed GDS Dysphoria subscale items across BDS Cutoff levels of impairment.
Figure 3. Mean number of endorsed GDS Meaninglessness subscale items across BDS Cutoff levels of impairment.

Figure 4. Mean number of endorsed GDS Cognitive Impairment subscale items across BDS Cutoff levels of impairment.
Figure 5. Mean number of endorsed GDS Apathy subscale items across BDS Categorical levels of impairment.

Figure 6. Mean number of endorsed GDS Dysphoria subscale items across BDS Categorical levels of impairment.
Figure 7. Mean number of endorsed GDS Meaninglessness subscale items across BDS Categorical levels of impairment.

Figure 8. Mean number of endorsed GDS Cognitive Impairment subscale items across BDS Categorical levels of impairment.
Figure 9. Mean number of endorsed GDS Apathy subscale items across CDT Cutoff levels of impairment.

Figure 10. Mean number of endorsed GDS Dysphoria subscale items across CDT Cutoff levels of impairment.
Figure 11. Mean number of endorsed GDS Meaninglessness subscale items across CDT Cutoff levels of impairment.

Figure 12. Mean number of endorsed GDS Cognitive Impairment subscale items across CDT Cutoff levels of impairment.
**Figure 13.** Mean number of endorsed GDS Apathy subscale items across TMT-A Categorical levels of impairment.

**Figure 14.** Mean number of endorsed GDS Dysphoria subscale items across TMT-A Categorical levels of impairment.
Figure 15. Mean number of endorsed GDS Meaninglessness subscale items across TMT-A Categorical levels of impairment.

Figure 16. Mean number of endorsed GDS Cognitive Impairment subscale items across TMT-A Categorical levels of impairment.
Figure 17. Mean number of endorsed GDS Apathy subscale items across TMT-B Categorical levels of impairment.

Figure 18. Mean number of endorsed GDS Dysphoria subscale items across TMT-B Categorical levels of impairment.
Figure 19. Mean number of endorsed GDS Meaninglessness subscale items across TMT-B Categorical levels of impairment.

Figure 20. Mean number of endorsed GDS COGNITIVE IMPAIRMENT subscale items across TMT-B Categorical levels of impairment.
REFERENCES


