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ABSTRACT

THE RELATIONSHIP BETWEEN COGNITIVE FLEXIBILITY, DEPRESSION, AND ANXIETY IN OLDER ADULTS

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It has been hypothesized that the disruptive effects of negative emotional states, such as anxiety and depression, may contribute to poorer performance by the elderly (Deptula & Singh, 1995). Yet, while the relationship between emotional states and performance on cognitive tasks has been well studied in the young, there is a paucity of research of this type utilizing older persons (Rankin, Gilner, Geller, & Katz, 1994; Salzman & Lebowits, 1991). In this study, the relationship between cognitive flexibility (one aspect of executive functioning), anxiety, and depression in older adults was explored. A sample of 281 older adults (aged 55-86) was analyzed using structural equation modeling to assess whether age, anxiety, and/or depression will significantly predict cognitive flexibility as measured by the Stroop Color-Word Test, Trailmaking Part B, Wisconsin Card Sorting, and Word and Category Fluency. In addition, analysis of cognitive flexibility as a one- and two-factor model was completed. Results demonstrated that a two-factor model better fit the data; in addition, depression and age significantly predicted cognitive inflexibility in this sample. This result is striking given that average levels of depression for the sample were mild to moderate.

It is with great pride that I dedicate this thesis to my father, Edward Sheridan De Lano, who has continually served as a powerful influence in my life. His love, guidance, and undying belief in me has inspired me to strive to be my best and reach beyond my dreams.

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INTRODUCTION

During the past century, there has been an increase in average life expectancy that is unparalleled in any other period in history. According to current population estimates, over 51 million people in the United States (21% of the total population) are at least 55 years of age (U.S. Bureau of Census, 2000), a 10-fold increase over estimates in 1900. Recent projections indicate that the population of older adults aged 65 and older has been rising steadily from a 1900 level of 4.0% of the total U.S population, to 12.4% in 2000. Additionally, the U.S. Senate Special Committee on Aging (1991) has estimated that this age group will double by 2030 and will comprise 22.9% of the population by 2050. Furthermore, the "oldest old," or 85 and older age group, is expected to more than double from 4 million today to 8.5 million by 2030, making this age group the fastest growing segment of the population (Jeste, 1997).

The expected growth in the number of elderly persons—particularly those over age 85—has major implications for society. Anticipating health care and assistance needs will be important as these individuals tend to be in poorer health and require more services than people below age 85. Large numbers of older people find their health threatened by memory impairments, depression, chronic medical conditions, and disability which can substantially diminish quality of life. More specifically, older adults are increasingly seeking treatment for a range of psychiatric conditions including anxiety disorders, depression, chronic pain, psychotic disorders, sexual dysfunction, sleep disorders, somatoform disorders, and substance abuse disorders (American Psychological Association, 1997). According to the Federal Forum on Aging (2000), one-third or more

of men and women age 85 and older have moderate to severe memory impairment and about 30 percent of this group experience severe depressive and/or anxiety symptoms. In fact, complaints of anxiety and depression are encountered more frequently in this age group than in younger individuals (Zarit & Knight, 1996).

Along with higher rates of depressive and anxious symptoms in older populations, studies have shown that there is also a decrement in cognitive functioning with aging. In addition, researchers have linked this decline with the frontal lobes of the human brain. For example, in studies on the effect of increasing age on tasks sensitive to damage of a number of brain regions, older adults have been found to perform more poorly, relative to younger adults, on measures of frontal lobe function, while performing comparably on tasks of nonfrontal function (Ardila & Rosselli,1989; Whelihan & Lesher, 1985). Findings such as these have recently generated theories of aging that propose that most age-related cognitive decline in memory and attention are a result of deterioration of executive function, generally theorized to be associated with the frontal lobes (Lezak, 1995; Shimamura, 1995). In fact, neuropsychological data from a number of studies suggest that there is a decrement in executive ability with age (Albert & Kaplan, 1980; Daigneault, Braun, & Whitaker, 1992; Goldberh & Bilder, 1986; Hochanadel & Kaplan, 1984; Mittenberg Seidenberg, Oleary, & DiGiulio, 1989; Uchiyama, Mitrushina, & D'Elia, 1994; Veroff, 1980; Whelihan & Lescher, 1985). This deterioration of executive functioning has been linked to a degeneration of neurons and decreased neurotransmitter activity in the aging brain (Lezak, 1995).

Cognitive flexibility is an important aspect of executive functioning that is central to successful strategic decision making. It has been described by Spreen & Strauss

(1991) as the "ease with which a person can shift his or her perceptual set to conform to changing demands and suppress a habitual response in favor of an unusual one" (p. 46). Although the bulk of research has evaluated cognitive flexibility in terms of brain damage (Lezak, 1995; Luria, 1981; Stuss & Benson, 1986; Eslinger & Grattan, 1993) and mental illness (Galdi, 1993), there has been a recent shift to looking at cognitive flexibility in terms of general aging (Eslinger & Grattan, 1993; Parkin & Lawrence, 1994; Schultz, Kaye, & Hoyer, 1980). In addition, diminished cognitive flexibility is known to be one of the earliest deficits associated with dementing disorders of later life (Lezak, 1995; La Rue, 1992), thus it is hypothesized that a milder, age-related decline in the executive abilities may be responsible for some of the cognitive and behavioral changes observed in normal aging (Kaye & Grigsby, 1991). An individual may be affected by an inability to attend to more than one source of stimuli at a time, difficulty with problem solving, and trouble remembering information due to inability to actively choose and employ optimal strategies. Thus, in extreme cases, an individual may appear to be severely impaired in tests of cognition (i.e. memory) though the actual deficit may involve executive functioning (cognitive flexibility).

It has been hypothesized that the disruptive effects of negative emotional states, such as anxiety and depression, may contribute to poorer performance by the elderly. In fact, studies are emerging that are beginning to demonstrate that even subclinical levels of depression can interfere with neuropsychological testing in older adults. Yet, while the relationship between emotional states and performance on cognitive tasks has been well studied in the young, there is research of this type utilizing older persons is scarce (Rankin, Gilner, Geller, & Katz, 1994; Salzman & Lebowits, 1991). Both anxiety and

depression have been extensively studied in relation to memory; however the impact of mood on executive functioning—specifically, cognitive flexibility--has received little attention. Understanding the relationship between aging and mood disorders in later life--even of a subclinical nature--will help researchers further elucidate the cognitive changes that occur in later life as well as help guide researchers and clinicians in the treatment and prevention of executive cognitive decline in association with mood disturbances.

Cognitive Flexibility

The functions of the frontal lobes in human cognition is complex, but it is now well established that the prefrontal lobes are thought to control the executive cognitive functions (Lezak, 1995, La Rue, 1992; Parkin, 1992, 1997). Executive functions involve regulation, organizing, self-monitoring, planning, control and cognitive flexibility. More specifically, executive function is described as the cognitive capacity to control goal-directed behavior and more basic cognitive processes and to use environmental feedback to modify responses (Welsh, 1991; Welsh, Cicerello, Cuneo, & Brennan, 1995; Welsh & Pennington, 1988). Recognition that the frontal lobes appear to be the brain area affected most by cortical atrophy in aging (Haug & Eggers, 1991), and that older adults characteristically perform more poorly than younger adults on neuropsychological tests of executive function (Daigneault & Braun, 1993; Daigneault, Braun, & Whitaker, 1992; Mittenberg, Seidenberg, O'Leary, & DiGiulio, 1989; Parkin & Walter, 1992) has lead to the executive decline hypothesis of memory and aging. Indeed, there appears to be a striking similarity between the types of memory deficits evidenced by older adults and by those with frontal lobe lesions (Moscovitch & Winocur, 1992). Given the importance of

executive functioning to human adaptive behavior, researchers are beginning to closely examine these functions as they relate to aging.

One important aspect of executive functioning is cognitive flexibility which was initially described as a concept occurring on a "rigidity-flexibility" dimension by Shaie (1970, 1958, 1975) and Schulz (1980). Central to successful strategic decision making, cognitive flexibility has been more recently described by Spreen & Strauss (1991) as the "ease with which a person can shift his or her perceptual set to conform to changing demands and suppress a habitual response in favor of an unusual one." Specifically, flexibility is defined as the capability of perceiving not only interrelationships between concepts but also important distinctions between them. Broadly considered, cognitive flexibility includes actively choosing cognitive strategies that fit individuals' goals at that time (Showers & Cantor, 1985), intelligently adapting to ones' environment (Berg & Sternberg, 1985; Simon, 1990), and creative thinking, or finding unusual yet relevant solutions to problems. Furthermore, cognitive flexibility gives rise to the number and types of strategies used for decision-making and memory. Thus, an individual with impairment in cognitive flexibility may be mistaken as having serious memory decline when, in fact, the impairment may be due to a deficiency in cognitive flexibility.

The capacity for flexibility in behavior extends through perceptual, cognitive, and response dimensions. Conceptual inflexibility appears in concrete or rigid approaches to understanding and problem solving, and also as stimulus-bound behavior in which these patients cannot dissociate their responses or pull their attention away from whatever is in their perceptual field or current thoughts (Lhermite, 1983). Such inflexibility may

evidence itself as an inability to shift perceptual organization, train of thought, or ongoing behavior to meet the varying needs of the moment.

Inflexibility of response results in perseverative, stereotyped, nonadaptive behavior and difficulties in regulating and modulating motor acts. In each of these problems there is an inability to shift behavior readily, or to conform behavior to rapidly changing demands on the person. This disturbance in the programming of behavior appears in many different forms and is closely associated with lesions of the frontal lobes (Luria, 1966; Le Gall et al., 1995). These combined deficits may underlie the inability of frontal lobe damaged patients to return to productive work or to have appropriate social interactions. Even when frontal damaged individuals know what and how to do something, they are impaired by an inflexibility of behavior characterized by perseveration of inappropriate behaviors and/or inability to maintain productive efforts.

The ability to vary one's mode of approach to solving complex problems is an important factor in achieving a successful outcome in nearly all facets of life. Having the capacity to view a problem from a flexible stance, in which alternate options are freely considered, is an essential feature of adaptive behavior and is particularly important in higher-order cognitive functioning (e.g., Buetcher, 1972; Torrance, 1972; Campbell, 1960; Gavurin, 1975). Cognitive flexibility is thought to be important because it allows for the consideration of a variety of behavioral options or solutions prior to executing an overt response.

Reactive and Spontaneous Flexibility

Review of clinical and experimental studies provides support for at least two forms of cognitive flexibility which have been identified as spontaneous and reactive

flexibility (Eslinger & Grattan, 1993; Grattan, 1990; Hanes, Andrewes, & Pantelis, 1995). Spontaneous flexibility is described by Eslinger & Grattan (1993) as the "ready flow of ideas and answers, often in response to a single question." Encompassing the notion of "fluency," spontaneous flexibility requires the intrinsic generation of responses or alternatives specific to the task at hand. More specifically, spontaneous flexibility concerns the production of alternative solutions utilizing fluency resources. In these tasks, the flexibility demands are found in those processes that underlie the generation of a diversity of responses. Some avoidance of automatic and habitual responses and strategies is necessary in order to attend to other features and aspects of knowledge. Thus spontaneous flexibility tasks typically require subjects to generate diverse and creative solutions by utilizing effective search strategies to successfully move among classes and categories of knowledge. Spontaneous flexibility is seemingly mediated by the prefrontal cortex (Eslinger & Grattan, 1993) and is typically assessed by verbal (Borkowski et al., 1967) and design (Jones-Gotman & Milner, 1977) fluency measures. Spontaneous flexibility tasks are likely to be more sensitive to frontal lobe integrity because they typically require subjects to access various classes and categories of knowledge in novel ways, bypassing more automatic or conventional strategies.

In contrast, reactive flexibility refers to the ease in freely shifting cognition and behavior in accordance with the distinctive demands and context of a situation. Hanes, Andrewes, & Pantelis (1995) describe this concept as the "ability to realign a behavioral predisposition according to altered contingencies." The concept was first described by Goldstein (1943) after he assessed a patient who could initiate activity but could not shift responses when required by the situation. A further dichotomy may be made between

set shift and set maintenance aspects of reactive flexibility; the former necessitating a change from current predispositions and the latter the ability to avoid distraction (Flowers & Robertson, 1985; Hanes, Andrewes, & Pantelis, 1995). The Wisconsin Card Sorting Test (WCST) has been widely employed for assessing reactive flexibility since it requires subjects to shift response set in relation to external cues. Milner (1964; 1984) demonstrated that patients with frontal lobe lesions have particular difficulty with this task, a finding confirmed by several other investigators (Drewe, 1974; Robinson et. al., 1980; Stuss et. al., 1983). It is believed that reactive flexibility depends on the proper functioning of both frontal and striatal structures (Eslinger & Grattan, 1993).

Effects of Age on Cognitive Flexibility

Severe deficits in cognitive flexibility results in an inability to perform separate tasks that make up a whole task (i.e., an entire list of errands to perform) to perfection, yet they are often unable to organize the requirements of the list to efficiently complete the task. Overall, there appears to be an impairment in the ability to perceive the big picture within its place in time or to synthesize separate behavioral sequences required for task completion (Fuster, 1989). Stuss and Benson (1987) have called this phenomenon a dissociation between self-consciousness and self-knowledge. This disorder can be observed in the patient who demonstrates full awareness of the occurrence of errors in behavior based on some external rule but is unable to modify behavior based on this knowledge. Common examples of this type of behavior are seen in a number of classic neuropsychological tests sensitive to prefrontal damage. On one common tests of cognitive flexibility, the Stroop Color-Naming Task, patients with prefrontal damage frequently report the rule requiring the naming of the color and

ignoring the word, even as the word continues to be named on successive trials (Duncan, 1995; Perret, 1974). In addition, a similar pattern is often seen another common test of cognitive flexibility (Wisconsin Card Sorting), with individuals often continuing to sort the cards using a faulty strategy, even as they express with each sort that this behavior is incorrect (Duncan, 1995; Heaton, 1981). Performance on these tasks suggests that the individual knows what to do but is unable to use that knowledge to guide his or her behavior.

To date, only a few studies have assessed spontaneous and reactive flexibility in normal aging (Shultz, Kaye, & Hoyer, 1980; Eslinger & Grattan (1993); Parkin & Lawrence, 1994), as the majority of studies have examined them in the context of Parkinson's disease (Hanes, Andrewes, and Pantelis, 1995; Rogers et al., 1998; Bowen, 1976; Cools, Van Den Bercken, & Horstink, 1984; Lees & Smith, 1983; Taylor, Saint-Cyr, & Lang, 1986; Teuber & Proctor, 1964; Teuber, 1976), Huntington's disease (Hanes et al., 1995), and schizophrenia (Cools, Brouwer, de Jong, & Slooff, 2000; Gold, Goldberg, & Weinberger, 1992; Hanes et al., 1995; Mahurin, Roderick, Velligan, 1998).

Eslinger and Grattan (1994) examined reactive flexibility, as measured by WCST, and spontaneous flexibility, as measured by the Alternate Uses Test (Guilford et al., 1978), in a group of patients with varying degrees of damage to the frontal lobes and basal ganglia. Reactive flexibility was found to be adversely affected in patients with either frontal or basal ganglia lesions whereas spontaneous flexibility was greatly impaired by frontal lesions but far less so by basal ganglia. This data strongly suggests that there may be a functional dissociation between tests of reactive and spontaneous flexibility. In addition, behavioral-anatomical studies have suggested that impairment on

spontaneous flexibility tasks is correlated most strongly with frontal lobe lesions (Benton, 1968; Jason, 1985; Jones-Gotman & Milner, 1977; Pendleton, Heaton, Lehman, & Hulihan, 1982). Patients with frontal lobe damage may fail such tasks because of their restricted ability to think beyond the conventional and literal use of objects, words, and other classes of knowledge (Perret, 1974; Zangwill, 1966).

Parkin and Lawrence (1994) examined how performance on tests of reactive and spontaneous flexibility related to performance on two tests of memory (recall and recognition). They demonstrated that aging produces a decline in memory functions considered to be sensitive to frontal dysfunction. Specifically, it was demonstrated that increased discrepancy between recall and recognition correlates with poorer performance on the WCST (reactive flexibility measure). Finally, Shultz et al (1980) found that there was an age-associated decline in as compared to younger adults (aged 18 to 23). However, the younger adults outperformed the older adults on only two of the four spontaneous flexibility tasks (Brick Uses and Hidden Pictures). It is difficult to generalize these results due to the constricted age range in both young and old samples.

The above findings suggest that age appears to negatively impact performance on neuropsychological tests of executive functioning—in particular, cognitive flexibility. However, it is still unclear as to how and why these deficits occur in normal aging. Given that neuropsychological test performance is governed by complex interactions involving both intra and inter-individual factors (Craik & Byrd, 1982; Valdois, Joanette, Poissant, Ska, & Dehaut, 1990), it has been hypothesized that age differences in executive functioning could be accounted for by unidentified medical or psychiatric problems (e.g., hypertension, depression, anxiety) which are elevated in older adults

(Boone, Miller, and Lesser (1993). Similarly, Deptula, Singh, and Pomara (1993) theorized that the disruptive effects of negative emotional states such as depression and anxiety may contribute to poorer performance by the elderly. Given the high prevalence of anxious and depressive symptoms in older adults coupled with the occurrence of lower scores on tests of executive functioning, a close analysis of the relationship between mood and cognitive flexibility is warranted.

Depression in Older Adults

Prevalence

Depression is widespread among older adults, affecting at least one of every six patients treated in general medical practice (Reynolds and Kupfer, 1999), and is considered to be the second most common mental disorder in old age, surpassed only by dementia. As the population of those aged 65 and older grows in the United States, the number of new cases of depression and subsyndromal depression should increase considerably. In fact, the World Health Organization (WHO) has recently estimated that by the year 2020, depression will be the second leading cause of disability in the world. In general, the percentage of older people in the community who meet diagnostic criteria for major depression appears to be one percent (Regier et al., 1988). If dysthymic disorder is included, the prevalence increases to three percent. An additional 1.2% suffer from a syndrome that has both depressive and anxiety features. Furthermore, several studies have found depressive symptoms in older adults to be the most prevalent in the "oldest-old" (80 years and older) and in females (Murrell et al., 1983; Berkman et al., 1986; Kennedy, Kelman, and Thomas et al., 1989). Current rates of depression in the

elderly are comparable to those in younger populations, although there may be a trend among older adults to experience milder forms of depression that do younger adults (LaRue, 1992).

Minor depression, also known as subthreshold, or subsyndromal depression, is two to four times more common than major depression in older adults (Kurlowicz & Streim1998). In fact, the prevalence of milder yet clinically significant depressive symptoms among the community-dwelling elderly is high, ranging from 15% to 20% (Blazer & Williams, 1980; Blazer et al., 1987; Regier et al., 1988). The importance of clinically significant symptoms is illustrated by the finding that depressive symptoms not meeting criteria for major depression are often associated with adverse outcomes in older adults (Gallo et al., 1997). Older adults with subsyndromal depression account for the majority of affective disorders seen in primary care (Katon & Schulberg, 1992) and can cause significant medical, functional, and social impairment (Broadhead, Blazer, George, & Tse, 1990; Mossey et al., 1989; Wells et al., 1989).

Clinical Picture of Depression

Depression has been shown to interfere with basic abilities to think, sleep, interact with others, experience gratification, maintain a sense of purpose, and maintain a sense of self-responsibility (Reynolds & Kupfer, 1999). In fact, depression contributes to as much social and physical dysfunction, including days spent in bed and general physical pain, as any other chronic medial illness (Jeste, 1997). Compared to younger depressives, older patients are more likely to have memory complaints and show greater global cognitive impairment during depressive episodes which may be severe enough to warrant a dementia diagnosis. Furthermore, depression in later life is associated with increased

patient and caregiver distress, increased disability associated with medical and cognitive dysfunction, increased health care costs, and increased mortality related to suicide and medical illness (Reynolds and Kupfer, 1999).

Depression in late life tends to be a chronic and recurrent disorder (Cole & Bellavance, 1997). In a study by Murphy (1994), it was found that during the first year of follow-up in a study of 124 subjects with late-life depression, only 35% achieved full remission of symptoms without evidence of relapse, 22% got well but relapsed, 29% remained continuously ill, and 14% died. Additionally, results showed that risk factors for poor outcome included lack of appropriate acute and maintenance treatment, severe initial symptoms, cognitive impairment, and physical illness. Given the high prevalence of depression in late life coupled with the poor outcomes associated with it, it is essential that attention be devoted to not only understanding mood disturbance in late life, but also its associations with cognitive functioning. As mentioned above, cognitive impairment serves as a risk factor for poor outcome following onset of depression in the elderly; thus understanding the quality and direction of impairment associated with mood in later life is essential.

Associated Cognitive Impairment

There is growing evidence linking depression with frontal lobe dysfunction. For instance, imaging studies of depressed patients using positron emission tomography (PET) and regional cerebral blood flow (rCBF) have shown changes in areas including the left anterior cingulate and the left dorsolateral prefrontal cortex (Bench et al., 1992; Drevets et al., 1992). In addition, computed tomography and magnetic resonance imaging studies have suggested that structural neuroanatomic factors may be related to

geriatric depression. Specifically, enlarged third and lateral ventricles, cortical atrophy, decreased caudate size, and vascular lesions in the caudate nucleus appear to be more profoundly apparent in late-life depression associated with vascular risk factors (Ohayon, Caulet, and Lemoine, 1996). Other neuroimaging evidence shows that depressed patients have reduced cerebral blood flow in different regions of the frontal cortex, but also in subcortical structures such as the caudate nucleus and amygdala (Bench et al., 1992, 1993; Drevets et al., 1992). Moreover, structural scanning using MRI ahs shown increases in subcortical white matter changes in elderly depressives (Coffey et al., 1990; Krishnan et al., 1988). However, it remains unclear to what extent such symptoms are related to damage in frontal or subcortical structures and exactly how this is related to diminished executive functioning ability.

Depressed individuals frequently complain of memory loss and poor concentration. In fact, it is not unusual to find some evidence of cognitive impairment (Johnson & Magaro, 1987; Marcopulos, 1989). Although it is unclear whether depression contributes to the onset of cognitive decline in old age, or if depression itself is a result of cognitive impairment, it is commonly accepted that depression is associated with a number of deficits in cognitive function, particularly memory (Sternberg & Jarvik, 1976; Stromgren, 1977; Weingartner et al., 1981; Cohen et al., 1982; Wolfe et al., 1987; Austin et al., 1992a, Levy-Cushman and Abeles, 1998). Impairment of frontal or executive function has been examined less frequently, although this has been reported in more severely depressed subjects (Goodwin, 1997; Raskin et al., 1982; Silberman et al., 1983; Jones et al., 1988). Bassuk et al. (1998) determined that depressive symptoms, particularly dysphoric mood, were strongly predictive of subsequent decline among

respondents with average cognitive performance scores (as measured with the Short Portable Mental Status Questionnaire (SPMSQ)). However it was unclear if the association was most prevalent in those average scorers who had already experienced a decrement in cognitive performance.

With respect to executive functioning, a few studies have shown deficits in areas such as abstract reasoning (Braff & Beck, 1974), simple perceptual discrimination (Cornell et al, 1984) and verbal fluency (Robertson & Taylor, 1985). Furthermore, aside from deficits in memory and learning, there is also impairment in speed of thinking, problem solving, and attention, which all encompass various aspects of executive function commonly associated with the syndrome of pseudodementia (Bassuk et al, 1998; Veiel, 1997; Reynolds & Kupfer, 1999). This condition represents true impairment of memory processes secondary to depression but without impairment of other aspects of the mental status. In contrast to cognitive impairment due to central nervous system changes resulting from normal aging or to the pathology of dementia, cognitive impairment associated with depression may be reversed with appropriate treatment.

More recently, neuropsychological studies in depressed patients have demonstrated impairments on neuropsychological tests of cognitive flexibility. Many studies have reported that depressed patients produce fewer words than normal control subjects on verbal fluency tasks (Caine et al, 1984; Robertson and Taylor, 1985; Wolfe et al., 1987; Dupont et al, 1990) and are impaired on the Trail Making Tests that assess set shifting more specifically (Rush et al., 1983; Fisher et al., 1986; Gray et al., 1987; Jeste et al., 1996). Cassens et al. (1990) observed dramatic impairment on the WCST in unipolar

depressed patients in terms of the number of categories completed and the number of perseverative errors.

Using the WCST, Verbal Fluency, and Cognitive Estimate (a test that requires individuals to provide reasonable answers to cognitive estimate questions), Fossati et al. (1999) found that, though depressed patients performed normally on memory tests, they were impaired with respect to cognitive flexibility and hypothesis-testing. It is important to note that this study only looked at adults between the ages of 18 and 45, thus it is largely unknown if this result generalizes to the older adult population. It was suggested by Fossati et al. (1999) that this "more general cognitive deficit" (cognitive flexibility) could contribute to the maintenance symptoms of depression, which was suggested by its association with the mean duration of depressive episodes. Finally, Beblo et al. (1999) found that neuropsychological deficits in depressed adults and older adults were most dramatic in spontaneous cognitive flexibility as measured by both design and verbal fluency measures. In addition, they found that cognitive slowing in major depression appeared to be related to the complexity of executive functioning versus a decline in psychomotor speed.

Brown et al. (1994) assessed neuropsychological functioning of depressed older adults (mean age = 58 years) in an effort to better characterize cognitive functioning seen in depression and to investigate the possibility of a continuum of impairment vs. a distinct demented subgroup. After identifying three groups using the Mini-Mental State Examination (unimpaired depressed (UD); impaired depressed (ID); and borderline depressed (BD), it was demonstrated that the UD group evidenced significant impairment of cognition on a wide range of measures despite normal-range intellectual function.

Deficits in verbal fluency, language, and memory were the most sensitive to the presence of depression. This appears to provide clear evidence that cognitive dysfunction is a real feature of depressive illness, though no causal direction can be inferred at this point. In addition, though the depressed sample appeared to perform adequately on tests of conceptual ability (Weigl test), they evidenced a clear deficit for language function, particularly verbal fluency. The authors hypothesized that it is possible that conceptual ability is relatively spared in depression with greater impairments seen in language functioning.

Fossati et al. (1999) found that depressed patients produce fewer words than control subjects on verbal fluency tasks. However, significant differences between depressed patients and control subjects were solely observed for categorical fluency. Fossati found that depressed patients were impaired on fluency tasks requiring an important demand on semantic processing. In addition, they state that it is unlikely that verbal fluency impairment is related to an effect of psychomotor retardation since a test of processing speed was not correlated with verbal fluency.

Trichard et al. (1995) assessed depressed adults on a wide range of cognitive tasks and found that one of the most significant deficits for this group occurred in tasks that tapped into verbal fluency. Why this group performed so poorly on verbal fluency is not clear. It was speculated that verbal fluency could be closely related in depressives to a particular dimension of intentional deficit and lack of initiative associated with their depressed state; however, if subjects were not motivated by the tests, their pattern of deficits would likely be widespread rather than specific. In all, depressed patients in this

particular study showed specific impairments on tasks requiring cognitive flexibility capacity and "complex integration aptitude."

Overall, depression is widespread among older adults and causes significant disability among individuals in this population demographic. Studies have shown that depression is associated with decreased performance on many cognitive functions including memory, attention, and cognitive flexibility. The question of whether depression could be the cause or the consequence of cognitive decline is receiving increased attention (Geerlings et al., 2000). Results have been mixed, though it seems that there is more support for the hypothesis that negative mood causes cognitive deficits given that older adult's perceptions of their cognitive functioning has been shown to be uncorrelated with their performance on objective tests (Deptula, 1994). An answer to this question begins with a better understanding of the interplay between negative mood and cognitive functioning.

Anxiety in Older Adults

Prevalence

In contrast to depression, anxiety in older adults has received little attention despite evidence that anxiety symptoms and disorders are fairly common in late life (Norton, Coz, Asmundson, & Maser, 1995; Small, 1997; Salzman & Lebowitz, 1991). In a review of eight random-sample community surveys, Flint (1994) found prevalence rates of diagnosed anxiety disorders ranging from 0.7 to 18.6% among older people. In addition, Epidemiological Catchment Area (ECA) data indicate that anxiety disorders are more common than major depression, dysthymia, or severe cognitive impairment among

people over the age of 65 (Regier et al., 1988). When significant anxiety symptoms that do not reach criteria for a specific disorder are considered, prevalence rates among the elderly jump to 20-25% in community samples (Copeland et al., 1987; Himmelfarb & Murrell, 1984).

Researchers have shown that there is a linear increase of anxiety symptoms, with rates in the elderly double that of adolescence (Warheit, Bell, Schwab, & Buhl, 1986). Beck and Stanley (1997) estimated that, even when taking into account the lowest published prevalence estimates, anxiety disorders were found to occur four to eight times more frequently than major depression in individuals aged 65 and older. In fact, the adverse impact of anxiety on quality of life and use of health-care resources has been shown to be equivalent to the consequences of major depression in older persons (de Beurs et al., 1999). Despite these high figures, empirical data addressing the nature and treatment of anxiety in the elderly have only begun to emerge.

Clinical Picture of Anxiety

Anxiety at even mild to moderate levels can cause significant distress. Gurian and Miner (1991) have defined anxiety as a subjective state of internal discomfort, dread, and foreboding, accompanied by autonomic nervous system arousal. There is often a subjective state of nervousness or apprehension, impaired attention, poor concentration, behavioral manifestations such as agitation or avoidance; and physical symptoms that include hyperventilation, heart palpitations, sweating, diarrhea, trembling, dizziness, headache, restlessness, and muscle aches. The intense distress associated with the symptoms of anxiety often compels the individual to some kind of action, whether it be

increased use of medical services, self-medication with substances such as alcohol, or attempts to cope through a variety of other responses, either adaptive or maladaptive.

Anxiety can be understood as two separate but related constructs. Commonly, anxiety is used to describe an emotional state characterized by distress, agitation, and worry. It is also used to describe a more enduring personality trait (Spielberger, Gorsuch, & Lushene, 1970). In 1961, Cattel and Scheier made a distinction between state and trait anxiety. State anxiety is described by more immediate and temporary feelings of tension, apprehension, nervousness, worry, and arousal of the autonomic nervous system. Trait anxiety refers to more stable anxiety levels and emphasizes individual differences in the tendency to perceive situations as stressful and dangerous. Although defined as separate constructs, state and trait anxiety are often correlated as individuals who are high in trait anxiety are more likely to experience increased state anxiety when exposed to threatening situations (Spielberger, 1970).

Although late-life anxiety is common and appears to have potentially serious consequences, most anxious older adults do not seek psychological or other help (Himmelfarb & Murrell, 1984). Many older adults prefer somatic explanations for their symptoms as they tend to view psychiatric illness as stigmatizing and embarrassing. Thus, when older adults attempt to address such concerns, they most often consult with a primary physician (Waxman, Can , Klein, 1984; Yates, 1986). Other possible reasons for underutilization of mental health services include a lack of awareness of mental health services (Lasoski & Thelen , 1987) and the tendency to attribute agitation and fear to the normal aging process (Wetherell, 1998). In addition, many mental health services were not available when today's elderly individuals were younger. Anxiety disorders were

only "officially" recognized in 1980 (Himmelfarb & Murrell, 1994), and thus many older adults have little experience with mental health services, or they may have been misdiagnosed in the past.

Cohen et al (1983) reported that higher levels of trait anxiety were associated with poorer performance on a test of reasoning in older adults, whereas trait anxiety was connected with enhanced performance in younger subjects. This indicates that there is a possibility that the characterization of anxiety may change over the lifespan and that older adults may be more vulnerable than the young to the detrimental effects of negative emotional states on cognitive performance.

Associated Cognitive Impairment

A study by Gass, Ansley, and Boyette (1994) tested the hypothesis that anxiety, as measured by MMPI-2, would show a negative relationship with performance on tests of spontaneous flexibility (FAS and Design Fluency) in adults. The findings suggested that fluency was associated with a measure of generalized anxiety (ANX from MMPI). In addition, the strongest and most consistent association emerged between the FRS scale, which assesses highly specific fears and a generally fearful disposition, with scores on this scale accounting for substantial variance in scores on FAS (13%) and Design Fluency (23%). It was concluded that elevated scores on FRS should alert the clinician to the possibility that fearfulness--perhaps test anxiety--might be a factor that contributes to lower scores on these tests, particularly in the absence of other evidence of brain dysfunction.

Hammermaster (1989) looked at levels of performance and cognitive interference in test-anxious adult subjects (mean age = 27 years) using the Test Anxiety

Scale (TAS) and the WCST as a measure of cognitive performance. In addition, subjects were asked to complete the Cognitive Interference Questionnaire (CIQ) after testing which assesses irrelevant thinking that interferes with concentration on a task. A significant positive relationship between level of test-anxiety and amount of cognitive interference experienced was established. In addition, the high-anxious subjects performed less adequately in general when compared with the low test-anxious groups. The authors of this study concluded that high test-anxiety is associated with self-preoccupations and self-devaluing cognitions during task performance. These negative preoccupations, in turn, absorbs some portion of the person's information processing capacity, thus leaving less capacity for coping with task demands. Hammermaster (1989) suggests that deterioration in performance is directly related to the competition for space between task-irrelevant information involved in worry and cognitive self-concern with task-relevant information in the processing system. She posited that highly test-anxious subjects are battling with the task of dividing their attention, as a result of critical self-focusing.

In a study by Toren et al. (2000), it was found that children and adolescents with anxiety disorders evidenced lower linguistic abilities and impaired cognitive flexibility. Using the CVLT and the WCST, it was found that the anxiety group scored lower than the control group on all measures of the CVLT and had a significantly greater number of errors, perseverative responses, and incorrect answers after negative feedback on the WCST. In addition, on the WCST, it was found that negative feedback after making a mistake induced a repetition of the mistake (perseverative erroneous responses) in the children with anxiety disorders, whereas the control children used the negative feedback

productively. This finding suggests that children with anxiety disorders displayed a rigid adherence to a specific pattern and a decreased ability to shift focus to another (Eysenck, 1990; Kendall & Chansky, 1991). Furthermore, they found that, at least in children, anxiety did not appear to be associated with nonverbal processes. Replication of this kind of study with older adults would prove useful in not only helping to delineate the differences associated with anxiety between age groups, but also assisting in qualitatively assessing the effects of anxiety on cognitive performance in the older adult population.

Similar to depression, anxiety in older adults can cause impairment in attention and memory, thus it may be mistaken for the early stages of dementia (Gurian & Miner, 1991). In fact, there is accumulating clinical, epidemiological, and biological evidence that anxiety, as the subjective manifestation of the human stress response, may cause neurotoxicity and cognitive decline (Krasucki, Howard, & Mann, 1998). Several studies have evaluated the change in physiological arousal in response to being given a cognitive task. These studies found that when challenged with a cognitive task, elderly subjects, compared with young subjects, had greater elevations in plasma norepinephrine (Raskind, Gumbrecht, & Halter, 1982) and free fatty acids (Eisdorfer, 1967), two biological indexes of autonomic nervous system arousal. Therefore, the elderly may respond to cognitive challenges with greater arousal than the young. Given the paucity of research with respect to anxiety in older adults coupled with the findings that anxiety can have a detrimental impact on neuropsychological test scores, it is imperative that additional research be conducted in this area in an effort to better characterize the relationship between anxiety and executive functioning.

Comorbidity of Depression and Anxiety

In a recent study by Beurs et al. (2000), it was found that there were high levels of comorbidity among major depressive disorders and anxiety disorders in older adults (47.5% of those with major depressive disorder had comorbid anxiety disorders; 26.1% of those with anxiety disorders had comorbid major depressive disorder). However, in a comparison of the risk factors associated with pure major depressive disorder, pure anxiety disorders, and concurrent major depressive disorder plus anxiety disorders, more differences than similarities were found. The only factor common to all three was a cognitive, trait-like marker of vulnerability called "external locus of control". Moreover, pure anxiety disorders were associated with a wide range of vulnerability and stress-related factors which were not associated with pure major depressive disorder. In addition, it was found that, although comorbidity patterns were very similar to findings in younger age groups, the data on risk factors suggest that in later life, each major grouping represent useful categories, with shared but distinct underpinnings.

In conclusion, though much attention has been paid to executive functioning as it relates to memory in older adults, few studies have directly investigated the links between executive function and normal aging, and even fewer have examined the relationship between executive function and mood in the course of normal aging. Although a great deal currently is known about the relationship of factors such as age and intellectual background to cognitive test performance, a better understanding of emotional factors as they potentially affect executive functioning would assist clinicians in formulating diagnostic impressions and making treatment recommendations. Studies are emerging that are beginning to demonstrate that even subclinical levels of depression can interfere

with neuropsychological testing in older adults. Though, much less is known about the impact of anxiety on executive functioning, more recent attention has begun to focus on the role of anxiety in neuropsychological performance.

There is a need to further understand executive functioning in older adults and the influence of mood on these processes. Understanding the relationship between aging and mood disorders in later life--even of a subclinical nature--will help researchers further elucidate the continuum between normal and pathological aging, thus allowing for a refinement of studies related to cognitive functioning. In addition to helping to clarify the commonalities and differences between normal and pathological aging, research of this kind can help guide treatment and prevention of executive cognitive decline in association with mood disturbances. It is expected that the results of this study will provide additional insight and understanding into not only changes in cognitive flexibility in older age, but also how and in what ways mood interplays.

Primary Aims and Hypotheses

Based on the research reviewed above, the specific hypotheses of the present study include the expectation of the following results:

Aim 1: A structural equation model has been developed that will allow for a test of prediction of cognitive flexibility from four latent variables: age, depression, trait anxiety, state anxiety and depression. Essentially, a simultaneous test of which variable(s) most significantly predicts cognitive flexibility in older adults will be performed.

- Hypothesis 1: Each variable is expected to account for a significant

proportion of variance in cognitive flexibility, with age being the strongest predictor.

Aim 2: Older adults may be more depressed and anxious because they perceive their memory as deteriorating (poorer memory functioning causes the individual to be more anxious and depressed), rather than mood affecting cognitive flexibility. To assess this possibility, a reciprocal interaction in structural equation modeling will be performed.

- Hypothesis 2: It is hypothesized that the strength of the path from mood to cognitive flexibility will be stronger than the reciprocal path of cognitive flexibility to mood.

Aim 3: A model that will assess the breakdown of cognitive flexibility into reactive and spontaneous flexibility will be tested.

- Hypothesis 3: It is hypothesized that including both reactive and spontaneous flexibility into the model will better fit the data than the original model utilizing cognitive flexibility as a unitary construct.

Aim 4: Anxiety and depression will be evaluated in terms of its impact on both reactive and spontaneous flexibility. In line with previous research that has demonstrated that there appears to be a decrement in verbal/linguistic abilities in depression that may result from a lack of initiation (Beblo et al., 1999; Brown et al., 1994), and

that anxiety seems to be associated with decreased reasoning and problem solving abilities, the following relationships are hypothesized to exist among the data:

- Hypothesis 4a: It is expected that a depressed individuals will evidence a greater decrement in tests of verbal fluency (spontaneous flexibility) as compared to those who are primarily anxious and those who are neither anxious nor depressed.
- Hypothesis 4b: It is expected that anxious subjects will demonstrate greater impairment in tests of reactive flexibility versus controls and those who are depressed.

METHOD

Participants

One hundred and twenty community-dwelling older adults (aged 55 and older) will be recruited for this study. All participants will be recruited through local newspaper advertisements and local talks given to community groups in the greater Lansing, Michigan area. Participants will be screened for dementia using the Mini Mental Status Examination; those who score 24 or better and report no significant history of severe neurological or medical problems likely to have adversely affected their cognitive abilities (e.g., stroke, major ischemia, significant traumatic brain injury) will be included in the study. Proper screening will ensure that the final sample will be most representative of the normal aging population.

Measures

Cognitive Flexibility will be assessed with five different measures including the Stroop Color and Word Test (Stroop, 1935), the Wisconsin Card Sorting Test (Heaton, 1981), the Trailmaking Test Part B (Reitan, 1958), the FAS Controlled Oral Verbal Fluency Test, and the Category Fluency Test.

a. Stroop Color and Word Test (Stroop, 1935)

This test provides a general measure of cognitive flexibility. It specifically addresses the ease with which a person can shift perceptual set in keeping with changing demands; in addition it assesses abilities to suppress a habitual response in favor of an unusual one. This test yields three basic scores including Word Naming, determined by the number of words (written in black type) read in 45 seconds, Color Naming, determined by the number of items (red, green, and blue "X's") correctly read in 45 seconds, and Color-Word naming, determined by the number of incongruent items read in 45 seconds. This final incongruence section is particularly challenging because the participant must suppress the well-learned response of reading words and instead must focus on the incongruent color of ink rather than the word itself. The tendency to read the word may intrude upon the color naming, thus decreasing the interference effect score, or the "Stroop effect" (Lezak, 1995; Spreen & Strauss, 1998). Performance is assessed by the time required to complete each naming trial and the difference between the color naming and interference condition. Recently, the Stroop effect has attracted the interest of aging researchers because it appears to be a strong measure of inhibitory processes (reference), a construct central to cognitive flexibility

Golden (1976) found the Stroop was 88.9% effective in differentiating between normal controls and brain-damaged clients and 82.9% accurate in discriminating between psychiatric controls and brain damaged clients. Regard (1981) reported that the Stroop effect was greater for patients with left frontal lobe damage than for other patients or control groups. Uchiyama, Mitrushina, D'Elia, Satz, and Mathews (1994) used the Stroop and other measures to assess frontal lobe functioning in geriatricc and non-geriatric samples. Stroop color naming and Stroop color-word naming were found to be two of the most sensitive measures for prediciting frontal lobe functioning. This test is also sensitive to severity of dementia (Koss et al., 1984). Spreen and Strauss (1991) reported a one-month test-retest reliability for each page of the Stroop to be.90, .83, and .91, respectively. Validity evidence for the Stroop as a measure of frontal lobe functioning includes observe dhigh correlations with other tests of frontal lobe functioning including a verbal fluency test (r = .580 and a version of the Tower of London (r - .65) (Spreen & Strauss, 1998).

b. The Trail Making Test (TMT)--Reitan, 1958, 1992.

The TMT (part B) is an efficient and sensitive instrument of cognitive flexibility that is easily administered and reliably discriminates between normal individuals and those with brain impairment (Lezak, 1995; Stuss, Stethem, Hugenholtz, & Richard, 1989). The test is given in two parts: Trail Making, Part A (TMT-A) involves drawing a line connecting consecutive numbers form 1 to 25. Part B (TMT-B) involves drawing a similar line, connecting alternating numbers and letters in sequence (i.e., 1-A-2-B and so on). Only Part B will be analyzed in this study.

Slowed performance on Part B is sometimes thought to be indicative of executive dysfunction (Pontius & Yudowitz, 1980). More specifically, it is thought to indicate impaired ability to modify a plan of action or to maintain two trains of thought simultaneously (Lezak, 1995; Reitan, 1971). Mitrushina et al. (1999) described Part B as assessing the "ability to alternate between sets of stimuli, an executive function" (p. 33). The cognitive skills required for completing the TMT Part B demands mental flexibility for satisfactory performance (Spreen & Strauss, 1991). There is substantive validity evidence suggesting that this test is also a measure of frontal lobe functioning (Reitan, 1968,6; Picton et al., 1986). Reliability of the difference score is reported to be .71 (Lezak, 1995).

c. Wisconsin Card Sorting Task (WCST)—Berg, 1948; Grant and Berg, 1948.

The WCST is the most widely recognized test of executive functioning because it has consistently showed more marked impairment after frontal lobe lesions that after posterior brain lesions (Grant and Berg, 1948; Milner and Petrides, 1984; Stuss and Benson, 1986; Drewe, 1974; Milner, 1963; Malmo, 1974; Nelson, 1976; Robinson, Heaton, Lehman and Stilson, 1980; Stuss and Benson, 1986). Primarily a test of reactive flexibility, this test has been described as a measure of hypothesis formation and set-shifting (Moscovitch & Winocur, 1995) and as a measure of concept formation processes and inhibition of inappropriate responses. The WCST has been used primarily as a test of perseveration, (state what type of cognitive flexibility it measures beyond perseveration) defined as continuing a class of response previously labeled incorrect, but it also can be used to assess abstract thinking, defined as the ability to sort cards according to a principle of class membership (Robinson, Heaton, Lehman, & Stilson, 1980). Four

stimulus cards are placed in front of the subject, the first with a red triangle, the second with two green starts, the third with three yellow crosses, and the fourth with four blue circles on them. The task if for the subject to sort the cards into piles below the stimulus cards. The response cards have designs similar to those in the stimulus cards, varying in color, geometric form, and in number. The subject completes the first category (color) when 10 successive cards have been sorted correctly, at which point, without warning, the sorting category is changed to form. Testing is terminated when the patient completes six categories (color, form, and number, each twice), or has depleted the supply of response cards. Subjects' responses are recorded and analyzed for type of errors (e.g., perseverative), as well as for number of errors.

Validity evidence for this test as a measure of frontal lobe functioning includes research findings that individuals with prefrontal lesions tend to display perseverative responses on this test (Milner, 1964; Heaton, 1981; Anderson, Jones, Tranel, Tranel, & Damasio, 1990). In addition, neuro-imaging technique research has also provided evidence that the WCST is a measure of frontal lobe function. Specifically, magnetic resonance imaging (MRI) studies have found that the volume of the dorsolateral prefrontal cortex is significantly correlated with the number of perseverative errors made on the WCST in normal young and normal old adults (Raz, Gunning, Head, Briggs, Dupuis, McQuain, Loken, Thornton, & Ackers, 1995; Raz, Head, Gunning, & Acker, 1996). Both intra-scorer and inter-scorer reliability estimates range from ,88 to .96 (Lezak, 1995).

d. Controlled Oral Word Association Test (FAS--Benton & Hamsher, 1976)

This test is measures verbal fluency and is one of the most commonly used tests of spontaneous flexibility. Subjects are asked to give orally as many words as they could beginning with the letters F, A, and S in that order, allowing 1 minute per letter. Fluency performance is thought to reflect the executive function of strategic retrieval search (Bryan & Luszcz, 2000; Parker & Crawford, 1992). Verbal fluency has been used routinely in neuropsychological studies and is considered one of the more sensitive measures of incipient dementia (Ober, Dronkers, Koss, Delis, & Friedland, 1986). In addition, lesions in the frontal lobe of right-handed patients have been shown by Milner (1963) to produce a decrement of word fluency. Benton (1976) further confirmed the dependence of word fluency on the integrity of the left frontal lobe with the finding that bilateral frontal lesions do not entail a greater impairment of performance than unilateral, left frontal lesions, despite the larger mass of cerebral tissue destroyed. One-year retest reliability in older adults has been reported as .70 (Snow et al., 1988). Concurrent validity has also been established for the FAS (Coelho, 1984).

e. Category Fluency

This instrument is a well-known test of verbal fluency (spontaneous flexibility) that requires a fundamental process of retrieval of lexically or semantically associated items. As opposed to letter fluency where individuals must name words that begin with a specified letter, subjects are not limited by having to generate words that begin with a specified letter. Instead, subjects must move within semantic categories while naming. In this test, subjects are asked to name as many animals, fruits, and vegetables they can in

60 seconds. Scores take into account perseverations, intrusions, and variations and are summed within categories.

Measure of Mental Status

a. Mini-Mental State Exam (MMSE; Folstein, Folstein, & McHough, 1975)

The MMSE is a 30-item form created to briefly screen for gross cognitive functioning. It assesses orientation, immediate recall, attention and calculation, intermediate recall, language abilities, and constructional dyspraxia. Scores can range from zero, indicating severe impairment, to 30, indicating no impairment in mental status. This measure will be used to screen participants to ensure that the sample is representative of a normal aging population. A cut-off score of 24 will be used to identify scores more suggestive of impairment (Lezak, 1983).

Measures of Depression

a. Geriatric Depression Scale (GDS)

The GDS is a 30-item self-report sale that is specifically designed to measure the number of depressive symptoms in older adults (Yesavage, Brink, Rose, Lum, Huang, Adey, & Leirer, 1983). This measure utilizes a binary response option (yes/no) that makes it easily comprehended by older adults, a short completion time, and a limited number of potentially confounding somatic symptoms. Additionally, there are more features characteristic of late-life depression as compared to other scales and has been shown to be a valid measure of depressive symptoms in older patients with mild to moderate cognitive impairment (O'Neill, Rice, Bladke, Walsh, & Oakley , 1992). It includes somatic items which are thought to be more appropriate for older populations

(Spreen & Strauss, 1998). The total score for this test is calculated by adding the point values assigned to each response with the following cutoff scores identifying varying levels of depressive symptomotology: 0-9 – normal; 10-19 = mild depression; 20-30 = moderate/severe depression (Spreen & Strauss, 1998)..

The item-total correlations of the GDS range from .32 to .83, the split-half correlation was .94 and the internal consistency was also found to be .94 (Koenig et al., 1988). The GDS correlates with other self-report measures of depression including the Beck Depression Inventory (Beck & Beck, 1972; r = .73), the MMPI Depression Scale (Bielauskas & Lamberty, 1992; r - .72), and the Hamilton Depression Scale (Hamilton, 1967; r = .83) (Yesavage, Brink, Rose, & Adey, 1986). Factor analyses revealed a major factor of dysphoria (unhappiness, dissatisfaction with life, emptiness, downheartedness, worthlessness, and helplessness). In addition two minor factors were revealed: one of worry, dread, and obsessive thought and the other one of apathy and withdrawal (Parmelee, Lawton, & Katz, 1989). Criterion validity has been measured against the Research Diagnostic Criteria and found to be .82 (Yesavage et al., 1983).

b. Beck Depression Inventory (BDI)—Beck et al., 1961)

The BDI is a 21-item self-report scale designed to measure both number and degree of affective, cognitive, and somatic features of depression. Many of the BDI items are thought to be representative of depression in late life, including increased guilt, loss of self-esteem, and reduced sense of life-long accomplishment (Kurlowicz and Streim, 1998). The subject is asked to rate their experience on a scale of graded severity. The total score for this test is determined by adding the highest number circled for each

of the 21 items. Higher scores indicate higher levels of depression. The authors of this test identified the following cutoff scores: 0-9 = normal; 10-15 = mild depression; 16-19 = mild/moderate depression; 20-29 = moderate/severe depression; 30+ = severe depression (Spreen & Strauss, 1998).

There is an extensive amount of psychometric data for the BDI. The authors of this test reported a test-retest reliability estimate above .90 (Beck, 1970). Other research has revealed a Spearman-Brown reliability estimate of .93 and an inter-item internal consistency estimate of .86 (Reynolds & Gould, 1981). Other researchers have reported a coefficient alpha for this measure of .88 (Steer et al., 1989). In addition, concurrent validity estimates between the BDI and the MMPI Depression Scale (Reynolds & Gould, 1981), the Hamilton Rating Scale (Brown, Schulberg & Madonia, 1995) and clinical ratings of depression (Schaefer, Brown & Watson, 1985) have been reported to be .75, .85, and .66, respectively.

Measures of Anxiety

a. State-Trait Anxiety Inventory (STAI)—Spielberger et al., 1970

The STAI is a well-known 40-item self-report scale that is widely used in research and clinical settings. This instrument includes both state and trait scales, measuring respectively transient and enduring levels of anxiety. Strong psychometric support is available for the STAI with younger adults (Spielberger et al ., 1970), and preliminary data from community and psychiatric samples of older adults suggest adequate internal consistency and convergent validity (Himmelfarb & Murrell, 1983; Patterson et al., 1980).

b. Penn State Worry Questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990) PSWQ; Meyer et al, 1990)

The PSWQ a 16-item scale designed to assess a trait-like tendency to worry. Participants are asked to rate their responses on a 5-point Likert-type scale, and higher scores indicate greater worry. Although a relatively new scale, research using the scale indicates it shows good psychometric characteristics (J. G. Beck, Stanley, & Zebb, 1995; Brown, Antony, & Barlow, 1992; Meyer et al., 1990).

Beck, Stanley, and Zebb (1995) examined the psychometric properties of the PSWQ in older adults. The scale was administered to 47 patients with generalized anxiety disorder (GAD) and 94 normal control Ss, ages 55-82 yrs. The PSWQ appeared to have good internal consistency and adequate convergent validity in both samples. Factor analyses indicated a 2-factor structure in both the GAD and the control sample. Items loading on the 1st factor appeared to assess a tendency to worry, while those loading on the 2nd factor seemed to assess an absence of worry. Item-total correlations, as well as item-scale correlations resulting from the 2-factor solutions of each sample were examined. Overall, the PSWQ showed adequate psychometric properties in these two samples of older adults which suggested that this measure is particularly suited for use with geropsychology populations.

Statistical Modeling

Refer to Figure 1 for an illustration of the detailed model that will be used to test hypotheses 1 and 2.

A model has been constructed using structural equation modeling in Amos that will simultaneously test hypothesized relationships using cognitive flexibility as a unitary

construct.

Identification

Preliminarily, Anxiety will be divided into two constructs: state and trait anxiety (both latent variables), with state anxiety being measured by STAI-S and trait anxiety measured by both STAI-T and the PSWQ. However, if there are identification problems associated with the model, these three measures (STAI-S, STAI-T, and PSWQ) will need to be combined for parsimony and to increase degrees of freedom. The indicators for Depression are the Beck Depression Inventory (BDI) and the Geriatric Depression Scale (GDS). Age is another latent variable that will be used to predict cognitive flexibility. Finally, the latent variable for cognitive flexibility (Cog Flex) is indicated by the WCST (Wisconsin Card Sorting), Stroop (Stroop Color-Word Test), TrailsB (Trailmaking Part B), FAS (Conrolled Oral Verbal Fluency), and Cat (Category Fluency).

For the first aim of the study, an exploratory analysis will be performed to assess which of the following four latent variable will be significant in the prediction of cognitive flexibility: age, trait anxiety, state anxiety, and depression. Inferences will be made based on the rank order of the standardized path coefficients.

A reciprocal interaction predicting S-Anx, T-Anx, and Dep (mood) from Cog Flex will be evaluated (hypothesis 2). As mentioned above, it may be necessary to combine S-Anx and T-Anx into a latent variable measuring general anxiety. If this action is necessary, general anxiety will be named Anx and it, along with Dep, will be used to test the reciprocal interaction.

Hypotheses 3, 4a, and 4b will be evaluated using a divided model represented in Figure 2. In this model, Cog Flex will be broken down into spontaneous flexibility (SCF) and reactive flexibility (RCF) and no changes will be made to any of the other latent variables. SCF has two indicators represented by FAS and Cat. RCF is indicated by WCST, Stroop, and TrailsB. This model may be simplified if necessary for model identification.

Figure 1

Cognitive Flexibility: One-Factor Model

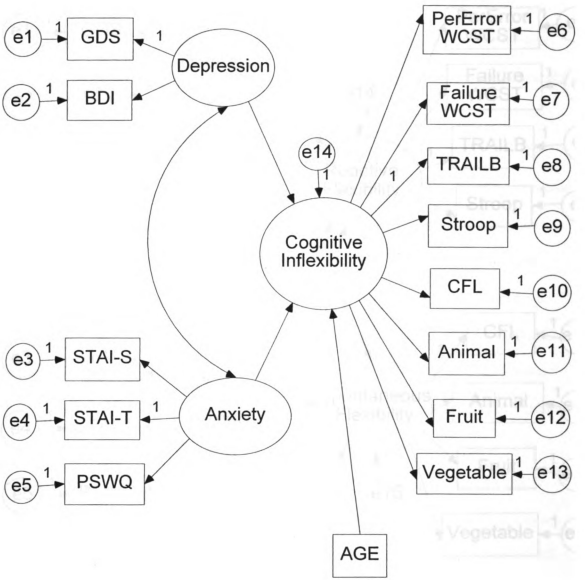
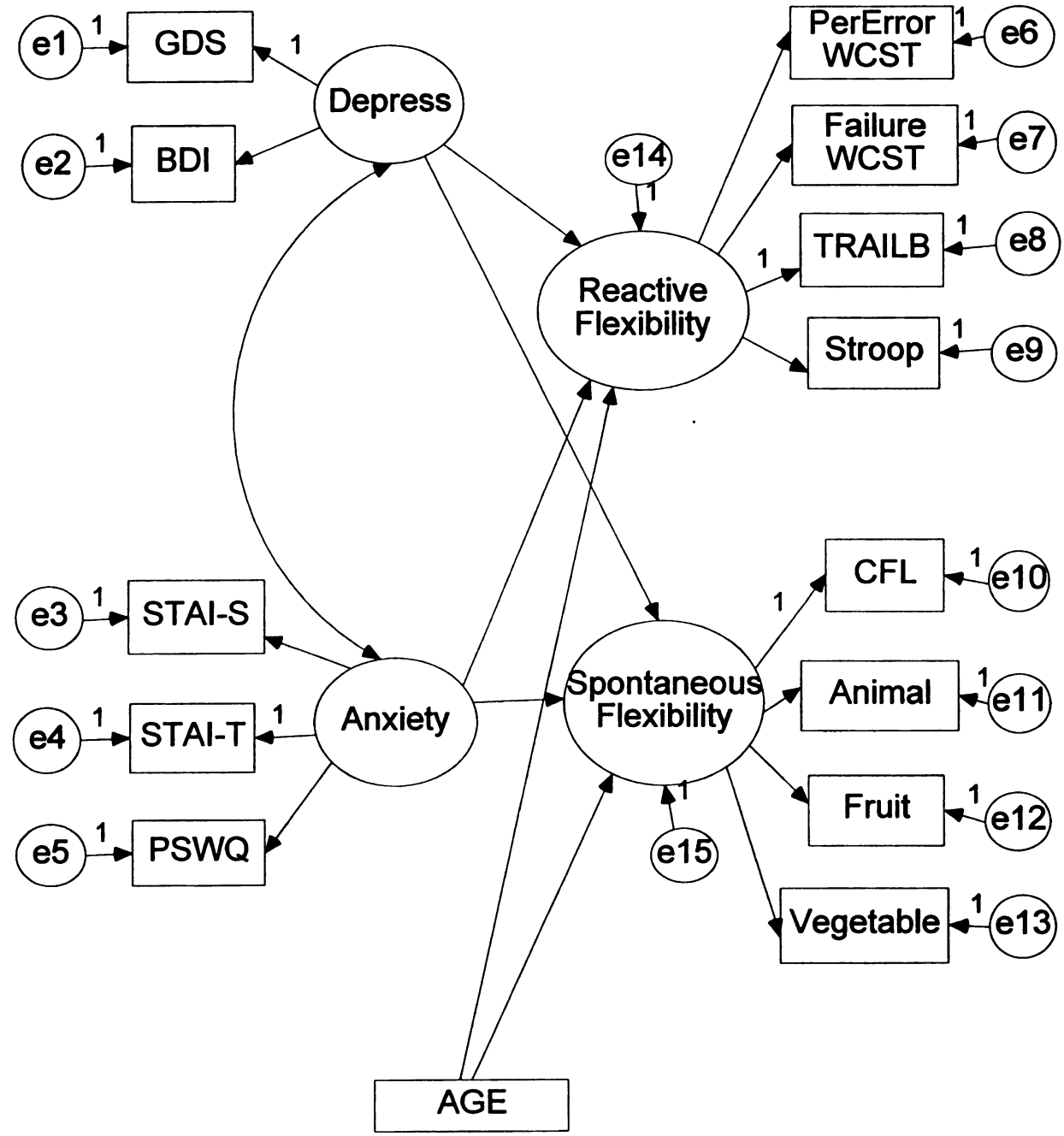


Figure 2

Cognitive Flexibility: Two-Factor Model



RESULTS

Hypothesized Models

Using structural equation modeling, relationships between Depression, a latent variable with two indicators (the GDS and BDI) and Anxiety, a latent variable with three indicators (the State portion of the STAI, the Trait portion of the STAI, and the PSWQ), were examined. Also included in the analysis was a measured indicator of Age. The hypothesized one-factor model is presented in Figure 1. Circles represent latent variables and rectangles represent measured variables. Absence of a line connecting variables implies lack of a hypothesized direct effect. Predicting latent variables for mood are correlated because, though anxiety and depression have been shown to be distinct (i.e. physiological arousal is unique to anxiety; and anhedonia, is unique to depression) (Clark & Watson, 1991; Burns & Eidelson, 1998), anxiety and depression represent negative affect and correlations between the constructs are moderately high (presented in Table 1).

Figure 1 illustrates the hypotheses that Depression, Anxiety, and Age will be directly associated with Cognitive Inflexibility; thus high levels of Depression and Anxiety will predict high levels of Cognitive Inflexibility as measured by the following variables: total time to complete Trails B (TrailsB); Perseverative Errors on the WCST (PerError--WCST); Failure to Maintain Set on the WCST (Failure--WCST); Stroop Color/Word trial total (Stroop); ratio of total perseverative responses to total word output on the COWAT (CFL); and ratio of total perseverative errors to total word output on the Category fluency task (Fruit, Vegetable, and Animal). In addition, it is hypothesized that

age will directly affect Cognitive Inflexibility with older individuals performing more poorly on tasks measuring cognitive flexibility than younger individuals.

Figure 2 depicts cognitive flexibility as a two-factor construct (latent variables include Reactive Flexibility and Spontaneous Flexibility). Reactive Flexibility is indicated by TrailsB, PerError (WCST), Failure (WCST), and the Stroop; Spontaneous Flexibility is measured by both fluency tasks (COWAT and Category Naming). It was hypothesized that the two-factor model would be better fitting and thus more accurately reflect the data than the one-factor model.

Statistical Analyses

For SEM techniques, Amos (Analysis of Moment Structures), Version 4.0 was used (Arbuckle, 1994, 1995) and robust maximum likelihood (ML) estimation was employed. Several fit indices are reported in addition to the standard chi-square test, including the comparative fit index (CFI; Bentler, 1990), the Tucker-Lewis Index (TLI; Bentler & Bonett, 1980; Bollen, 1989; Marsh, Balla, & McDonald, 1988), the Goodness of Fit Index (GFI; and the root mean square error of approximation (rmsea; Browne & Cudeck, 1993). The CFI is identical to McDonald and Marsh's (1990) relative noncentrality index but is scaled to fall in the range from 0 to 1. The GFI is analogous to R^2 and indexes the relative amount of the observed variances and covariances accounted for by the model. CFI, TLI, and GFI values close to 1 (higher than .90) indicate a good fit. Browne and Cudeck (1993) have advised that rmsea values below .06 indicate a good fit of a model in relation to the degrees of freedom, whereas values greater than .1 indicate that a model should not be accepted. Goodness of fit of any individual model

parameter was determined by examining its critical ratio, a statistic comparable with a t statistic with infinite degrees of freedom. In both models, a covariance matrix was used for estimation.

Assumptions for Both Hypothesized Models

The assumptions of multivariate normality and linearity were evaluated through SPSS and Amos. Only one outlier in the data set was omitted: one male was found to have an extremely low score on the PSWQ ($z = 6.68$). The analysis was thus performed on 283 participants. Missing data were deleted pairwise in SPSS before the resulting covariance matrix was analyzed in Amos. Eleven measured variables (BDI, GDS, CFL, TrailsB, Failure, Fruit, Vegetable, Animal, STAI-S, STAI-T, AND WCST (PerError) were significantly positively skewed (all p's $< .001$). This is indicative of the high-functioning nature of this sample. Indeed, the average subject completed roughly 16 years of education ($SD = 3.04$) and the average estimated Verbal IQ for the sample was also high ($M = 118.3$, $SD = 8.47$). The correlation matrix is shown in Table 1. In addition, the means, standard deviations, and kurtosis/skewness estimates are depicted in Table 2. Finally, parameter estimates for both models are displayed in Figures 3 and 4.

Model Estimation for One-Factor Hypothesized Model

Figure 1 presents a one-factor model of cognitive inflexibility. The two-headed arrow between the anxiety and depression latent variables reflect their correlation. To establish a metric for the unobserved variables in the model (including the error terms E1-E14), one path coefficient from each latent variable was fixed at 1.0 (e.g., Arbuckle, 1995; Bollen, 1989). The four two-headed arrows linking specific error terms reflect the assumption of some shared method variance among scales derived from the same

instrument. That is, the three Category fluency scales may share a common source of measurement error, as may the two scales derived from the WCST.

The fit of the one-factor model to the covariance matrix was shown to adequately fit the sample covariance matrix, $\chi 2$ (70, N = 166) = 134, p = .000), GFI = .937, CFI = .932, TLI = .911, rmsea = .058. Figure 1 displays the unstandardized factor loadings, R2 values, and correlations for the sample. Three of the four error term correlations were significant. The SRMR (root mean square residual), the average difference between the sample variances and covariances and the estimated population variances and covariances, was = 0.059 which indicates adequate fit. According to the descriptive measures of fit, this hypothesized one-factor model does appear to fit the data well.

This model demonstrates that both DEP (C.R. = 2.885, p = .004), and Age (C.R. = 8.390; p = .000) significantly predict Cognitive Inflexibility. The standardized regression weights for these predictors were: DEP = .273; and Age = .619. It was found that the fluency measures do not seem to hold up as measures of Cognitive Inflexibility for this sample. Standardized regression weights demonstrate little prediction (all p's > .05): CFL = 0.122; Vegetable = 0.034; Fruit = 0.107; and Animal = 0.104. Overall, results demonstrate that cognitive flexibility (at least for this sample) may not be best characterized as one factor and, instead, may be best predicted by two constructs.

Model Estimation for Two-Factor Hypothesized Model

Due to the poor prediction on behalf of the fluency measures (which comprised the Spontaneous Flexibility latent variable), it was thought that a divided, or two-factor, model might better fit the data than the one-factor model. Thus, a two-factor model was also examined with Depression, Anxiety, and Age predicting Reactive and Spontaneous

Flexibility. The hypothesized two-factor model is presented in Figure 2. Again, as with the one-factor model, the Depression and Anxiety latent variables were correlated in the model as well as the error variances from those indicators that were derived from the same measures.

Overall, the two-factor model demonstrates slightly better fit of the cognitive inflexibility measures, $\chi 2$ (67, N = 166) = 121, p = 0.000), GFI = .942, CFI = .944, TLI = .924, rmsea = .054. Figure 2 displays the unstandardized factor loadings, R2 values, and correlations for the sample. The SRMR was = 0.057 which indicates adequate fit. According to the descriptive measures of fit, this hypothesized two-factor model fits the data and is better fitting than the one-factor model.

This model demonstrated that Depression significantly predicted Reactive Flexibility (C.R. = 3.025, p = .002); standardized regression weights for this predictor was 0.275. In addition, Age predicted Reactive Flexibility (C.R. = 8.534; p = .000) and Spontaneous Flexibility (C.R. = 2.456; p = 0.014) with standardized regression weights being .620 and .279, respectively.

Results of the reciprocal interaction analyses demonstrate no significant results and model fit was particularly poor. This indicates that it appears as if the statement that mood better predicts cognitive flexibility is more plausible than the reverse. Results thus indicate that we can be more confident that the relationship flows through negative mood. In addition, an interaction between anxiety and depression was also unable to be assessed due to pairwise deletion of data; due to missing data, a nonpositive covariation matrix was produced.

Table 1

Correlation Matrix for Sample

	BDI	GDS	STAI State	STAI Trait	PSWQ	Trail B	WCST PerErr	WCST Failure
Age	-.005	-.077	-.066	-.101	-.194**	.402**	.271**	.114
BDI	1.000	.810**	.375**	.441**	.367**	.212**	.082	.094
GDS		1.000	.390**	.535**	.533**	.165**	.118	.066
STAI State			1.000	.566**	.394**	.119	.102	.167*
STAI Trait				1.000	.580**	-.041	.142	.166*
PSWQ					1.000	-.050	.153	.120
TrailB						1.000	.304**	.185**
WCST Per Err							1.000	.189**
Fail WCST								1.000

Note: * Correlation significant at the .05 level—2-tailed
** Correlation significant at the .01 level—2 tailed

Table 1 (cont'd)

	Stroop	CFL	Animals	Fruits	Vegetables
Age	.457**	.144*	.087	.050	.012
BDI	.108	-.032	-.072	.049	-.003
GDS	.161**	-.005	-.078	.068	-.035
STAI State	.084	.021	.013	.071	-.117
STAI Trait	.062	.050	-.024	-.003	-.134
PSWQ	.035*	-.083	.018	.210**	-.111
TrailB	.497**	.063	.036	.086	.061
WCST PerError	.339**	.023	.015	-.009	-.019
WCST Failure	.145*	.025	.054	.066	-.042
Stroop	1.000	-.092	.152*	-.089	-.060
CFL		1.000	.163**	.140**	.040
Animals			1.000	.210**	.164**
Fruits				1.000	.316**
Vegetables					1.000

Table 2

Means and Standard Deviations for Sample

				Skewness		Kurtosis	
	Mean	SD	N	Statistic	SE	Statistic	SE
Age	69.68	7.87	281	-.02	.15	-.76	.29
BDI	7.05	5.82	267	1.74	.15	4.91	.30
GDS	6.23	5.51	268	1.29	.15	1.60	.30
Stroop	32.87	9.60	270	.24	.15	0.00	.30
CFL	.041	.081	281	-6.37	.15	89.76	.29
Trails B	96.16	45.07	283	1.96	.15	6.35	.29
WCST Per Error	16.05	12.43	264	1.78	.15	5.13	.30
WCST Failure	.97	1.26	264	1.67	.15	3.43	.30
Animal	.40	.025	283	3.05	.15	13.54	.29
Fruit	.41	.062	283	2.25	.15	5.5	.29
Vegetable	.26	.057	282	3.23	.15	12.54	.36
STAI State	33.36	10.24	178	1.12	.18	3.07	.36
STAI Trait	35.20	10.38	177	1.61	.18	6.08	.36
PSWQ	38.12	11.75	166	.54	.19	-.527	.38

Note: N's vary from 166 to 283 due to missing data.

Table 3

One-Factor Hypothesized Model Parameter Estimates

Parameters		Estimate	S.E.	C.R.	P
Cog Inflex	← ANX	0.033	0.349	0.328	0.743
Cog Inflex	← AGE	0.619	0.286	8.390	0.000
Cog Inflex	← DEP	0.273	0.524	2.885	0.004
BDI	← DEP	0.807	0.060	14.25	0.000
GDS	← DEP	1.000			
PSWQ	← ANX	0.703	0.088	10.79	0.000
STAI-T	← ANX	0.838			
Animal	← Cog Inflex	0.104	0.000	1.488	0.137
Fruit	← Cog Inflex	0.107	0.000	1.536	0.125
Failure	← Cog Inflex	0.231	0.003	3.209	0.001
TrailsB	← Cog Inflex	0.671			
STAI-S	← ANX	0.633	0.076	9.820	0.000
Vegetable	← Cog Inflex	0.034	0.000	0.485	0.627
CFL	← Cog Inflex	0.122	0.000	1.747	0.081
Stroop	← Cog Inflex	0.670	0.027	7.933	0.000
Per Error	← Cog Inflex	0.462	0.031	6.108	0.000

Note: Regression estimates are standardized

Table 4

Two-Factor Hypothesized Model Parameter Estimates

Parameters	Estimate	S.E.	C.R.	P
Reactive ← DEP	0.275	0.504	3.025	0.002
Spontaneous ← DEP	-0.103	0.001	-0.702	0.483
Spontaneous ← AGE	0.279	0.001	2.456	0.014
Reactive ← AGE	0.620	0.284	8.523	0.000
Spontaneous ← ANX	0.158	0.001	0.967	0.333
Reactive ← ANX	0.004	0.335	0.040	0.968
BDI ← DEP	0.806	0.060	14.18	0.000
GDS ← DEP	1.005			
PSWQ ← ANX	0.701	0.088	10.80	0.000
STAI-S ← ANX	0.632	0.076	9.83	0.000
STAI-T ← ANX	0.839			
PerError ← Reactive	0.461	0.030	6.31	0.000
Failure ← Reactive	0.223	0.003	3.184	0.001
TrailsB ← Reactive	0.678			
Stroop ← Reactive	0.746	0.027	8.72	0.000
CFL ← Spontaneous	0.514			
Animal ← Spontaneous	0.327	0.243	1.615	0.106
Fruit ← Spontaneous	0.246	0.248	1.466	0.143
Vegetable ← Spontaneous	0.033	0.150	0.308	0.758

Note: Regression estimates are standardized

DISCUSSION

It was hypothesized that anxiety and depression would predict cognitive flexibility in a community-dwelling, older adult population. Using structural equation modeling, two hypothesized models illustrating cognitive flexibility as one- and two-factor models were analyzed. Results demonstrated that, in both paradigms, depression and age both significantly predicted performance on tests of cognitive flexibility. Though the one-factor model fit reasonably well, it was shown that a two-factor model better fit the data.

The results of this study indicate that, even with the addition of age and anxiety in the model, depression predicts over and beyond these variables. This is interesting given that the sample from which this data was drawn was not significantly depressed. In fact, the majority of participants demonstrated only mild to moderate levels of depression. Thus, this study illustrates that even the presence of symptoms of depression can interfere with at least one component of executive functioning, cognitive flexibility.

It was found that depression appeared to specifically affect reactive flexibility in this study. This relationship may exist due to a general slowing often present in depression and/or the possibility that greater distraction was present in those subjects who reported higher levels of depression. Specifically, reactive flexibility requires an ability to freely shift cognition and behavior in accordance with the distinctive demands of a situation and it may be that that the ability to organize cognition and behavior in this way is characterized by a linear relationship with depression. Thus, this may explain why higher rates of depressive symptoms lead to greater difficulty on tasks of cognitive flexibility. Indeed, these symptoms are consistent with the DSM-IV criteria which

indicates that depression is often characterized by distraction, fatigue, and interference with basic abilities to think, sleep, and maintain a sense of purpose (Reynolds & Kupfer, 1999). It seems likely that an individual with depressive symptoms may be affected by an inability to attend to more than one source of stimuli at a time, difficulty with problem solving, and trouble remembering information due to inability to actively choose and employ optimal strategies.

It was expected that, in addition to depression, anxiety, too, would significantly predict poorer performance on tasks assessing cognitive flexibility. However, results demonstrated that anxiety poorly predicted cognitive flexibility in this study. It may be that, for this sample, anxiety served as a focusing factor which may have helped increase valuable vigilance and attention. It is known that anxiety produces autonomic nervous system arousal (Gurian & Miner, 1991); however, it may be that cognitive flexibility becomes significantly impaired at only very high levels of anxiety. It is indeed possible that this sample did not experience anxiety at severe enough levels to produce negative effects on test performance. As an alternative, it may be that anxiety exerts its effects in a different manner. For instance, as many in the field have posited (i.e. Hammermaster, 1989) but few have tested, anxiety may function as an inverted-U such that an intermediate amount of anxiety may be optimal for best performance (originally hypothesized by Yerkes-Dodson (1908)). Conversely, in this paradigm, values at the very low and high end of the continuum would be most detrimental to neuropsychological performance.

It was also expected that negative mood would be predictive of spontaneous flexibility but, due to low correlations between the predictors and the small portion of

variance accounted for by the spontaneous flexibility factor itself, this relationship could not be adequately investigated. Thus, apart from previous studies that have found fluency measures to be predictive of cognitive flexibility, this study was unable to demonstrate any strong relationship. The poor measurement of cognitive flexibility by the spontaneous flexibility factor seems to be due to the lack of variance in the sample for total word output and perseverative errors. Specifically, 34.5% of the sample made no perseverative errors on the CFL fluency task. In addition, on the other fluency measure (Category Fluency), roughly 74% of the participants made no perseverative errors on average across each task. Thus, for this high-functioning sample, the fluency tasks appear to discriminate poorly, leading to reduced prediction. It may be that these tasks are of little utility with high-functioning samples and that the reactive flexibility measures better discriminate within this population.

It is important to note that several limitations were present in this study. First, the data collected stemmed from a cross-sectional design which considerably limits the ability to reveal important age changes, or true changes that occur with advancing age. Accordingly, the design limits any discussion about individual patterns of change. Second, measures of mood state were assessed via self-report; replication with scales administered by trained interviewers would be useful. Furthermore, as stated above, the sample in general was particularly high-functioning and homogeneous which likely limits the ability of these results to generalize to the aging population at large. This limitation, however, is ever-present in aging research as it is difficult to gain access to lower-functioning aging populations.

CONCLUSION

It has been hypothesized that the disruptive effects of negative mood states may contribute to poorer performance by older adults (Deptula & Singh, 1995). Yet, while the relationship between emotional states and performance on cognitive tasks has been well studied in the young, there is a paucity of research of this type in older adult populations (Rankin, Gilner, Geller, & Katz, 1994; Salzman & Lebowits, 1991). In fact, impairment of frontal or executive function as a result of negative mood has been examined less frequently, although this has been reported in more severely depressed subjects (Goodwin, 1997; Raskin et al., 1982; Silberman et al., 1983; Jones et al., 1988).

In this study, the relationship between cognitive flexibility (one aspect of executive functioning), anxiety, and depression in older adults was explored. While no relationship was found between anxiety and cognitive flexibility, results indicate that even low to moderate depression can be predictive of performance after controlling for age; this result is most striking since it was ascertained in a high-functioning aging population. Given that this sample scored quite high (mean = 28.25/30) on the Mini-Mental Status Examination (MMSE) and tests of memory, it is possible that executive functioning skills are most sensitive to the effects of aging. In fact, it may be that executive functioning is among the first cognitive capacities to be affected by negative mood. This has important implications because it may be that, for higher-functioning samples, tests of executive functioning more accurately identify inception of true cognitive decline. In addition, these results may point to the greater use of executive

functioning tasks to identify depressed patients who may or may not be demented or to monitor progression of impairment.

Failure to take into account executive skills such as cognitive flexibility with regard to performance on neuropsychological examinations may result in incorrect conclusions about a patient's true deficits, regardless of the presence or absence of depression. Since diminished cognitive flexibility has been shown to be one of the earliest deficits associated with dementia, it is important that health service professionals understand the contribution that depression may add to the clinical picture (Lezak, 1995; La Rue, 1992). Indeed, it has been found that older adults are more likely to have memory complaints and show greater global cognitive impairment during depressive episodes which may be severe enough to warrant a dementia diagnosis (Zarit & Knight, 1996). Thus, it is important that the underlying depression be treated before proceeding with a diagnosis of dementia. This is especially important since it has been shown that, once depressive symptoms have been treated, cognitive capacity typically returns to normal in non-demented individuals. Finally, it is important to understand the relationship between cognitive flexibility and mood because, as suggested by Trichard et al. (1995), cognitive inflexibility may be a deficit that could contribute to the maintenance symptoms of depression.

It is hoped that the results of this study provides additional insight and understanding into the interplay between negative mood, age, and cognitive flexibility. Understanding the relationship between aging and mood disorders in later life—even of a subclinical nature—will help researchers further elucidate the cognitive changes that occur in later life as well as help guide researchers and clinicians in the treatment and

prevention of executive cognitive decline in association with mood disturbance. Moreover, it will help researchers further clarify the continuum between normal and pathological aging, thus allowing for a refinement of studies related to cognitive functioning.

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