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By

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ABSTRACT

THE EFFECTS OF ANXIETY, DEPRESSION, AND AGE ON COGNITIVE FUNCTIONS IN OLDER ADULTS: A LONGITUDINAL STUDY

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Terry Randyl Barclay

During the last decade, the relationship between negative affect states and cognition in older adults has become a topic of tremendous research and clinical interest. Despite a steady growth of empirical investigation in this area, however, the *long*-term impact of depression and anxiety on neuropsychological function remains poorly understood. This dissertation investigated the hypothesis that anxiety and depression contribute to age-related cognitive decline in the areas of memory, executive function, and processing efficiency. The sample included 57 community-dwelling older adults between the ages of 57 and 92 (Time 1 $\underline{M}$ = 69.86, $\underline{SD}$ = 6.36 years; Time 2 $\underline{M}$ = 75.09, $\underline{SD}$ = 6.47 years). All variables were measured using standard neuropsychological tests of performance and a short demographic questionnaire. Mood was assessed using self-report measures, including the Beck Depression Inventory (BDI), Geriatric Depression Scale (GDS), and the State-Trait Anxiety Inventory (STAI). Subjects returned for follow-up evaluation approximately 5 years after initial participation. Results of repeated measures ANCOVA analyses revealed that higher levels of depressive symptomatology were not predictive of cognitive decline above and beyond that already accounted for by age alone. However, higher levels of trait anxiety were found to predict 9.9% ($\underline{p}<.05$) of the decrement in free recall memory, 8.2% ($\underline{p}<.05$) of the decline in semantic clustering, and 8.4% ($\underline{p}<.05$) of the deterioration in executive functioning performance. Trait

anxiety was not found to predict decline in processing efficiency. Overall, these findings are consistent with a small, but growing, literature demonstrating that trait anxiety is associated with poorer verbal learning and executive dysfunction. Results suggest that even subclinical levels of anxiety may interfere with neuropsychological performance and may exacerbate the cognitive decline associated with normal aging. These findings are discussed in light of their practical, theoretical, and methodological implications and in the context of suggestions for future research.

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INTRODUCTION

During the last decade, the relationship between negative affect states and cognition in older adults has become a topic of tremendous research and clinical interest. Despite a steady growth of research in this area, however, important questions remain to be adequately addressed. Key questions as yet unanswered include (1) What is the strength of the relationship between symptoms of anxiety and depression and cognitive decline in old age? (2) Is there a difference in the extent to which mild, moderate, or severe affective symptoms impact cognitive functioning over time? (3) Which types of cognitive processes are most likely to be affected by anxiety and depression or, stated another way, do symptoms of anxiety and depression affect cognitive functioning in a global manner or are there differential effects of these symptoms on various cognitive domains? and (4) What is the nature of the interaction between age-related cognitive decline and the decline associated with symptoms of anxiety or depression? Moreover, given that the overwhelming majority of published studies in this area have been cross-sectional in nature, the *long*-term effects of emotional disturbance on cognition in old age have been ignored. From a research standpoint, as well as from the clinician's perspective, the need for longitudinal work in this line of investigation is clear.

While there has been an increase in the number of studies investigating depression, anxiety, and cognitive functioning in elderly samples, the conclusions drawn from recent research on these variables are equivocal. For example, with respect to empirical work investigating the impact of depression on cognitive functioning in older adult populations, a handful of recent studies have found significant differences in cognitive functioning between those with and without depression. Memory, verbal

fluency, and naming tasks were identified as sensitive to the effects of depression in several of these studies (King, Caine, Conwell, & Cox, 1991a, 1991b; LaRue, 1989; LaRue, D'Elia, Clark, Spar, & Jarvik, 1986; Williams, Little, Scates, & Blockman, 1987). In each of these studies, individuals with depression scored significantly lower than control subjects, but significantly higher than comparison groups of patients with Alzheimer's disease. Therefore, the researchers concluded that the effects of depression on cognition were relatively subtle. Other studies utilizing similar samples, however, have found no differences between depressives and normals, except for the subjects' perception of their memory, with depressives claiming more memory problems than controls (Freehan, Knight, & Partridge, 1991; Rohling & Scogin, 1993). Results from studies using medical patients with and without depression, and demented patients with and without depression are equally contradictory. Among medical patients, three robust studies reported no significant differences between depressives and nondepressives on a variety of cognitive tasks (Bieliauskas, Boczar, & Lamberty, 1991; Bieliauskas, Costello, & Terpenning, 1991; Bieliauskas & Glantz, 1989), whereas one longitudinal study found that those with depression performed more poorly on cognitive screening tasks alone (Parmelee, Katz, & Lawton, 1992). Similar disagreements can be found in research involving patients with dementia. In one study, demented individuals with depression displayed greater cognitive deficits than did the demented subjects without depression (Pearson, Teri, Reifler, & Raskind, 1989), whereas another study found that the rate of cognitive decline did not differ between depressed and nondepressed Alzheimer disease patients (Lopez, Boller, Becker, Miller, & Reynolds, 1990). Making sense of these conflicting reports is further complicated by the fact that almost all of the published work

has employed cross-sectional designs. Therefore, most researchers claim their results to be tentative in nature and call for longitudinal studies to confirm their findings; however, few researchers to date have conducted such studies.

The research examining the effects of anxiety on cognitive abilities is much more sparse in the literature. Those studies that do exist are, unfortunately, equally contradictory. Two studies (Costa, Fozard, McCrae, & Bosse, 1976; Schultz, Hoyer, & Kaye, 1980) have reported no age differences with respect to the effect of trait anxiety on cognition; however, one investigation (Cohen, Eisdorfer, Vitaliano, & Bloom; 1980) reported an age by trait anxiety interaction, with elderly subjects' performance more adversely affected by high anxiety than that of middle-aged subjects. An interaction between age and state anxiety has been demonstrated in one study of learning and memory (Whitbourne, 1986), and inferred in a second investigation (Ross, 1986) where older participants were more negatively influenced than younger subjects by "challenging" instructions prior to learning. However, other investigations (Martin, 1984; Monge & Gardner, 1972) have reported that the elderly are not disproportionately affected by heightened symptoms of anxiety.

The variable nature of the findings described above may be the result of methodological inconsistencies and deficiencies among the studies. Small sample sizes represent a major limitation to many of the conclusions drawn from cross-sectional studies that have been published to date. In fact, many cross-sectional studies have used fewer than 30 individuals with depressive symptoms in their investigations (Amadeo, & Self, 1990; O'Boyle, Stoudemire et al., 1991; Pearson et al., 1989; Reynolds et al. 1987; Rohling & Scogin, 1993) and this is clearly problematic. The wide variety of instruments

used to measure cognition may also help to explain inconsistent results, as some assessment tools are far more sensitive to cognitive impairments than others. For instance, a number of studies have used the Mini Mental Status Examination as their measure of cognitive functioning (O'Boyle et al., 1990; Pearson et al., 1989) even though that particular measure is known not to be sensitive to more subtle cognitive deficits in areas such as memory, executive functioning, or speed of processing. While the studies that use global measures of cognition nevertheless add to our understanding of the association between these variables, researchers need to know which cognitive processes are most readily influenced by anxiety and depression and which are relatively undisturbed. In addition to lack of measurement specificity, the variety of ages used in recent research may also explain some of the inconsistent results. Sample demographics vary widely among published articles, with a wide variation in the interpretation of "older adults". For example, in their studies of patients with major depression, Williams et al. (1987) used a sample with a mean age of 53.7 (SD = 11.1) and Rohling and Scogin (1993) reported that their "old" group had a mean age of 64.9 years (SD = 3.6). In contrast, Parmelee et al. (1992) used a sample with a mean age of 83.9 years (SD unreported). As of yet, few researchers have speculated as to how the age of their samples may be interacting with the cognitive decline they attribute to depression or anxiety.

There are other problems in this line of research that also need to be addressed. For example, the great majority of studies investigating depression or anxiety do so by dividing their samples into dichotomous groupings (i.e. anxious versus nonanxious, young versus old). The use of these variables as dichotomous has been challenged as

inappropriate, and an argument for using continuous variables has been strongly asserted (Caine, Lyness, & King, 1993). It has been noted that, by comparing extremes on a continuum, such as highly anxious versus not at all anxious, the nature of the relationship between affective states and cognition may be distorted (Pedhazur, 1982). Utilizing measures of anxiety, depression, and cognition as continuous variables thus may add to the knowledge of the relationship between these constructs.

The present study seeks to add to the existing literature on the effects of anxiety and depression on cognitive functioning in older adults by improving upon the methodological and theoretical shortcomings of earlier studies. The proposed study will do so in the following important ways: (1) examine these variables in a within-subjects longitudinal design, thereby allowing for the direct examination of the long-term effects of anxiety and depression on cognitive functioning (2) using neuropsychological measures that assess the specific cognitive functions of memory, speed of processing, and executive functioning as opposed to measures of global, non-specific types of cognition and by (3) employing statistical analyses using continuous variables of anxiety, depression, and cognitive functioning in order to avoid the distortion that may occur by falsely dichotomizing these constructs.

It should be noted that the current study has been designed to examine the potential impact of depression and anxiety on various neuropsychological functions and therefore rests on the assumption that affective states may play a causal role in cognitive decline. While it could be argued that the direction of causality is actually reversed in some cases (i.e., signs of cognitive slippage actually precipitate feelings of depression/anxiety in some individuals), the present study is limited to the examination of

whether anxiety or depression-*proneness* is related to eventual cognitive deterioration and is based on the methodological framework of most recent investigations in the field. Clearly, a great deal of further empirical work will be needed to more fully elucidate the complex, bi-directional interactions that potentially exist between negative affect states and cognition in older adulthood.

Before describing the specific methodology and hypotheses of the present study, it will be necessary to briefly review the existing literature pertaining to the prevalence and nature of depressive and anxiety-related symptoms in late life, to articulate the factors that increase an elder's risk of developing symptoms of anxiety and depression, and to then elaborate upon the conflicting evidence linking negative affective states in old age to cognitive decline.

Depression in Older Adults

Prevalence

Late-life depression is a prevalent and serious condition that causes considerable suffering for both the afflicted individual and their family. Although symptoms of depression are common among the elderly, depression is not a normal part of aging and should not be accepted as such. Depression in the elderly often coexists with other physical illnesses, as well as anxiety, and thus patients and their primary care physicians may miss or misinterpret common symptoms of depression. This comorbidity not only confounds the diagnosis and treatment of depression, but also has been shown to have detrimental effects on general health status and patient outcomes (Bruce & Hoff, 1994). Though underdiagnosed and undertreated, it is possible to distinguish late-life depression from other geriatric illnesses and then treat it accordingly (Katz, 1996). Late-life

depression, just like depression affecting younger individuals, is frequently a recurrent illness that requires long-term treatment and a number of therapies have been proven safe and effective (Kupfer, 1991). As the number of people over the age of 65 continues to increase around the world, so does the importance of diagnosing and treating this condition appropriately.

Recent research has been somewhat conflicting with regard to the prevalence of depressive disorders in late life, though most agree that the numbers are significantly greater than originally hypothesized. In fact, the prevalence of depression among older adults is now reported to be second only to dementia among persons over the age of 65 (LaRue, 1992). Among community-dwelling residents in this age range, approximately 15% exhibit depressive symptoms, 1-2% suffer from major depression, and 2% are living with dysthymia (Blandchard, 1996). Noteworthy is the fact that much higher rates, between 13-27%, are recorded for subsyndromal or "sub-clinical" depression among this group (Johnson, Weissman, & Klerman, 1992; Judd et al., 1994). Although subsyndromal depressions do no meet diagnostic criteria for major depression or dysthymia (American Psychiatric Association, 1994), the existence of depressive symptomotology is associated with an increased risk of developing major depression, a physical disability or medical illness, and an elevated use of health services. As many as 50% of the medically ill elderly who are nursing home residents suffer from subsyndromal depressions (Koenig & Blazer, 1996; Mossey, Knott, & Craik, 1990). The course of subsyndromal depression varies, but when accompanied by a severe physical disability, such as stroke, it may persist for long periods of time (Alexopoulos, Young, & Shindeckler, 1992). Many of these individuals suffering from various disabilities must

live in nursing home settings and the prevalence of depression among nursing home residents is even higher than that of the general population. Recent studies report that approximately 15-25% of nursing home residents are suffering from either minor or major depression (Lepine & Bouchez, 1998).

Although estimates of the prevalence of late-life depression may vary due to the size of the study sample, the definition of depression used in the study, and the method of diagnosis, it is clear that late-life depression is a common condition. If unrecognized and untreated, depression can interfere with physical and social functioning, as well as diminish general health and well-being, and possibly interfere with later cognitive functioning. Of particular concern is the fact that while the elderly comprise but 15% of the total population, they account for 25% of all suicides (Blazer, 1993). Additionally, it is known that depression imposes substantial economic and emotional costs on society. Indeed, the annual cost of depression in the United States is approximately 43.7 billion (Greenberg, Stiglin, Finkelstein, & Berndt, 1993).

The difficulty that researchers have encountered in gathering consistent data with respect to the prevalence of depression among older adults is complicated by the fact that those over the age of 60 are the least likely of any age group to seek out mental health services (Blazer & Williams, 1980). This underutilization of available mental health services by older adults has been attributed to a variety of potential causes, including knowledge of the service, the individual's perceived need, available transportation, affordability, and attitudes toward treatment (Krout, 1983). Most of the literature, however, has focused on the apparent negative attitudes older adults hold toward mental health services and service providers. It has been reported that older adults would rather

seek services for psychological problems from physicians and religious authorities than from mental health professionals (Priddy, Tipton, & Prohaska, 1982). It has also been suggested that older adults believe general practitioners would be most effective in treating their problems and that older adults generally lack confidence in mental health specialists' abilities to help (Waxman, Carner, Dubin, & Klein, 1982). Despite reports that the attitudes of older adults toward psychological interventions are not universally negative (Lasoski & Thelen, 1987), the general belief that older adults prefer medical interventions still prevails (Adler, 1992).

In addition to these difficulties, a number of reports show that the elderly are not always aware that they are suffering from depression, as they believe that the manifestations of the disorder are simply symptoms of aging (Blandchard, 1996; Shulman, 1989). Complicating matters is the fact that depressive disorders in the elderly also tend to show an atypical clinical picture. In fact, several studies have suggested that older people have a different pattern of depressive symptoms to that found in their younger counterparts. In particular, it has been reported that older people present with a focus on somatic symptoms while minimizing spontaneous complaints of low mood (Shulman, 1989). Sleep disturbances are more readily reported, not only in the form of early awakening, but also as frequent awakenings during the night (Shulman, 1989). Loss of appetite and fluctuations in weight may also be primary symptoms. Tebbs and Martin (1987) have reported that the ten most common somatic symptoms reported by elderly people with depression include asthenia (weakness), headache, palpitations, pain, dizziness, abdominal pain, dyspnoea (shortness of breath), localized pain, back pain, and gastrointestinal dysfunction. It is easy to see how depressive disorders among this group

are overlooked given these common physical complaints. Indeed, depression in the elderly is often hidden behind somatic symptoms, either because of somatization of the disorder or because of the accentuation of physical symptoms related to comorbid illnesses.

Etiology of Late-Life Depression

An understanding of the possible etiological factors underlying depression in old age is not only necessary to explain the relatively high overall prevalence of depressive symptomatology in this population, but can also be useful in identifying those at particularly high risk for developing the disorder. Notably, the majority of studies to date have focused on demographic and social factors, while only a few investigations have attempted to generate more integrated etiological hypotheses. Therefore, the following section will summarize the possible roles that demographic and social factors play in predisposing the elderly toward depression, in precipitating or maintaining their symptoms, or in protecting this group from affective disturbance altogether.

Demographic Factors

Gender.

Most epidemiological studies have reported a considerably greater prevalence of depressive symptomatology in elderly females than in elderly male subjects, thereby suggesting that female gender predisposes one toward depression in old age. The etiological influence of gender on depression in old age does not, however, appear to be purely direct. In fact, several studies have suggested that gender is probably best described as a moderating variable in the relationship between age and the development of depressive symptomatology. In one such study, Kennedy et al. (1989) found a highly

significant association between age and depression in women ($p<0.0002$) but no such relationship in men. Such a finding may reflect real differences in the frequency with which women and men experience symptoms of depression or, alternatively, it may be reflective of the fact that men tend to deny symptoms of depression more frequently than women. It is important to note that subsequent stepwise regression analysis in this particular study, however, showed the effects of both age and gender to be very small once health and disability variables had been properly controlled.

In a complex study attempting to tease out the influence of both age and gender on social risk factors for depression, Lewinsohn et al. (1991) found that correlations between social variables and depression differed significantly between men and women in 12 of the 115 variables examined. Depression in women was associated with feelings of dissatisfaction with important life roles, with experiencing physical disease, and with marital conflict, to a much greater extent than was the case in men.

A few studies have also examined the influence of gender on the relationship between medical problems and depression. The relationship between physical illness and depression was found by Husaini et al. (1991) to be equally evident in males and females in a sample of 600 African American elderly community residents. In contrast, Cadoret and Widmer (1988) examined a group of elderly patients with a recent occurance of life-threatening or severely disabling illness with an age and sex matched control group. Depressive symptoms increased significantly in the group of ill male patients but not in the ill female patients. The increase in depression in the ill male group remained evident when controlling for other depression-linked variables, such as nursing home placement, prior history of depression, and recent stressful life events.

Family history.

There is consistent evidence that a positive family history is an important predisposing factor for depression in old age. This is particularly true for individuals with Bipolar Disorder, who suffer from both depression and mania (Shulman, 1992). Interestingly, the genetic contribution appears to be the least important in those with unipolar depression who become depressed for the first time in late life (Alexopoulos, 1989).

Financial and educational status.

Several studies have noted a relationship between poverty and depression in old age. Dean, Kolody, and Wood (1990) noted a significant direct effect of financial strain on depressive symptoms, even when controlling for the effect of negative life events and disability. Carpiniello, Carta, and Rudas (1989) found a similar relationship between poverty and depression; however, this relationship was only evident in urban residents. Kennedy et al. (1989) found that older subjects earning less than $5,000 a year had more than three times the risk of being depressed than those with incomes of more than $15,000 a year. In a follow-up study from the same sample, Kennedy et al. (1990) demonstrated that low income was significantly and independently associated with the emergence of new depression ($p<0.02$). Low income also emerged as a significant and independent correlate with depressive symptoms in a large community study by Blazer, Birchett, Service, and George (1991).

Relative to financial status, education has received somewhat less attention as a possible correlate of depression in old age. Carpiniello et al. (1989) found an association between low levels of education and depression, but this finding was restricted to women

and urban dwellers. More recently, Evans and Katona (1993) found a significant correlation between premorbid intelligence and self-rated depression scores, but not with interview-identified cases of depression.

Social Factors

Illness and disability.

An early study done by Linn et al. (1990) was one of the first to examine the link between depressive symptomatology and physical disability. They found a small but statistically significant correlation ($r = 0.23$) between level of disability and degree of depressive symptomatology. In a similar but more detailed study, Murphy (1992) compared elderly depressed subjects, the majority of whom were psychiatric outpatient referrals, with age and sex matched community controls. Severe chronic health difficulties were present in 39% of the depressed elderly compared with 26% of the normal elderly subjects.

In a much larger community sample of elderly residents, Kennedy et al. (1989) found that 30% of those with four or more medical conditions scored above the cutoff point for depression, compared with only 5% of those with no medical conditions. When depression scores were regressed against predictor variables, the contribution of health and disability to variance in depression remained highly significant ($p<0.0002$). Similarly, in a primary care based study, Evans and Katona (1993) found that depression was almost twice as common in those with significant physical health problems than in those without. Similarly, Blazer et al. (1991) found that disability and chronic illness, analyzed as separate variables, were both highly significantly associated with depression rating scores.

Several studies have examined the effects of disability as distinct from poor health. Smallegan (1989) found a significant relationship between level of disability and severity of depression, despite the fact that a very low overall rate of depression was found in their sample. In a larger stratified community sample, Dean et al. (1990) found a similar relationship between impairment in activities of daily living and depression, as did Phillips and Henderson (1991) in a nursing home cohort. In a study by Bruce and McNamara (1992), both major depression and dysthymia were more than twice as common in bed or chair-bound elderly subjects than in the non-home-bound. The relationship with dysthymia, but not that with major depression, remained statistically significant after controlling for degree of physical illness. Finally, Oxman et al. (1992) examined the influence of disability on depression prospectively through follow-up interview after a three-year interval on an institutionalized sample. Deterioration in functional ability between baseline and follow-up interviews was significantly associated with increased depression ($p<0.0001$). Physical illness thus emerges as a major factor both in precipitating and in maintaining depression in old age.

The studies described above have used cross-sectional data to examine the relationship between depression and illness and/or disability. This relationship can be examined more thoroughly with longitudinal follow-up studies involving subjects initially identified as normal (not depressed). In one such study, Murrel, Meeks, and Walker (1991) found that deterioration in health was significantly associated with the development of new symptoms of depression. They were not, however, able to demonstrate any protective effect of good physical health on depression brought on by negative life events.

Life events.

The potential for recent adverse events to precipitate depression has been well documented. An early study by Murphy (1982) replicated the studies of Brown and Harris (1978) with elderly patients and matched controls, finding that 48% of the patients, as compared with only 23% of controls, had experienced at least one severe independent event (such as bereavement, life-threatening illness of someone close) in the year prior to the onset of depression. It should be noted that the single most common life event in this study was personal physical illness, though the results remained statistically significant when this was excluded. In a more recent study using similar methodology, Emmerson et al. (1999) examined severe life events in a shorter three-month time period prior to onset of illness and found rates of 24% for the depressed patients and 7% for the controls. In a much larger stratified community sample, Dean et al. (1990) found highly significant correlations between undesirable life event scores and depression scores.

A number of studies have also examined the influence of more specific life events on the onset of depression. Linn et al. (1990) found that deaths or accidents among relatives and friends, and arguments with family members or close friends, were most closely associated with depression. In a larger community study in Canada, Stephenson-Cino et al. (1992) confirmed the overall relationship between life events and depression and found specific associations with bereavement and with recent illness of a friend or relative. Very similar associations between depression and serious illness and death among relatives and friends were reported by Pahkala, Kivela, and Laippala (1991).

The effect of moderating variables in buffering the extent to which life events trigger depression is also of considerable interest. Murphy (1982) found the association

between life events and depression to be maintained only in a subsample lacking an intimate relationship. In those who had such a relationship, rates of depression did not differ significantly between those experiencing a severe life event and those without such a stress. A similar prospective effect of intimacy against life-event-induced depression was reported by Evans and Katona (1993).

Bereavement.

Not surprisingly, bereavement is the life event most clearly implicated in the onset of depressive symptomatology in elderly subjects. Murphy (1982) found that 15% of her depressed sample, compared with only 7% of controls, had recently experienced the death of a spouse or child. Even more strikingly, Pahkala et al. (1991) found that only 2% of non-depressed community subjects, compared with 31% of depressed subjects, had recently experienced the death of a close friend or relative. Kennedy et al. (1989) found that the relative risk of depression was nearly twice as great in subjects who had experienced the death of a family member in the past six months than in those spared such an experience. In a multiple regression analysis they found that the combined variable of family illness and bereavement remained a highly significant ($p<0.0001$) associate of depression. They noted, however, that it explained only 2.9% of the variance, which was much less than the contribution of other variables, such as sleep disturbance or health and disability.

Prospective studies have also attempted to identify which bereaved subjects have higher risks of developing depression. In a two-year study of 189 widows and widowers, Zisook, Shuchter, and Lyons (1997) found that subjects who became depressed tended to be younger, to have been depressed in the past, and to have experienced sudden and

unanticipated bereavement (as opposed to bereavement following the death of a spouse who had long been ill). In contrast, an earlier study by Hill, Thompson, and Gallagher (1988) involving 95 widows found no relationship between depression at one year and the extent to which bereavement had been expected. The discrepancy between these findings may be due, in part, to the relatively small number of subjects in the Hill et al. study (1988). In another study, Dimond, Lund, and Caserta (1997) examined the role of social support in a two-year prospective follow-up study involving a group of elderly bereaved subjects. By far the strongest predictor of depression at all time points was severity of depression three weeks after bereavement. Depression at two months also showed a small but statistically significant negative relationship with age and with length of marriage, but no such effect was found between either of these variables and later depression.

Perhaps the single clearest finding from these studies is that although depressive symptomatology is very common in the weeks immediately after bereavement, most subjects experience a gradual decrease in depressive symptoms without developing a full-blown depressive illness. Persistent, clinically significant depression appears to be associated more with demographic and psychiatric variables (e.g., age, gender, past history of depression) than with bereavement alone.

Quality of relationships.

Being married appears to protect against the development of depression in old age, at least for some elderly individuals. For example, Carpiniello et al. (1989) found that only 8% of their elderly married subjects were depressed, compared with 17% of single subjects and 21% of those who were widowed. Similarily, Stephenson-Cino et al.

(1992) found that only 5.6% of their married subjects scored above the depression cut-off point on self-report measures, compared with 11.% of widows and 15.4% of separated or divorced subjects. Smallegan (1989) also found depression to be associated with lack of a spouse, as well as with the experience of marital change.

Similar relationships have been demonstrated between low levels of social interaction and a higher risk of depression. Kennedy et al. (1990) found that living alone was associated with a more than two-fold increase in the relative risk of depression. Pahkala et al. (1991) found significant relationships between depression and both living alone and decreased participation in social activities. A trend just failing to reach significance was found in the same study between depression and lack of intimate friends (19% versus 9%; $p<0.6$). Husaini et al. (1991) reported a significant relationship in women but not in men between depression and reduced frequency of contact with relatives and friends. Prospective studies by Russel and Cutrona (1991) and by Oxman et al. (1992) both suggest that lack of social support predicts the subsequent development of depression. The latter study also found that worsening levels of social support between the baseline and follow-up interviews (three years apart) predicted the emergence of depression.

Some researchers have suggested that quality of relationships may be more important in determining whether elderly subjects become depressed than "harder" variables such as marital status, whether subjects live alone, and the number of social contacts per unit of time. Interestingly, Murphy (1982) found that the absence of any close relationship was associated with rates of depression that were at least double those

found in subjects with some degree of intimate social contact, a finding replicated almost exactly in a community study by Kennedy et al. (1989).

The type of one's relationship and the nature of social interaction are clearly also relevant variables. Dean et al. (1990) found that availability of social support from spouses, friends, and adult children (in that order) were important predictors of freedom from depression, but that support from other relatives was not associated with the presence or absence of depression. In an interesting small study of nursing home residents, Rotenberg and Hamel (1988) found that depression was more common among subjects with reciprocally intimate relationships. These authors hypothesized that whereas chatting protected against depression, more emotionally laden interactions might actually cause it.

Gender and life events may be important moderating variables between intimacy and depression. Emerson et al. (1989) found the significant association between depression and the lack of a confiding relationship to be evident only in men. Fifty-seven percent of depressed men and only 3% of male controls had no confidant, whereas in women the figures were 23% and 18%, respectively. Both Murphy (1982) and Evans and Katona (1993) found that a significant association between life events and depression in elderly subjects was only present in those without any confiding relationship.

Summary of Etiological Factors

Both demographic and social factors appear to be of considerable importance in the etiology of depression in old age. A positive family history, poverty, and loneliness emerge as the most robust predisposing factors. Adverse life events, particularly bereavement, and deteriorating physical health, appear to be the most powerful

precipitants. Poor physical health also appears to be the single factor most important in impeding recovery from depression. On a more positive note, good social support, particularly in the form of an available confidant, may actually protect elderly individuals from becoming depressed even in the face of adversity.

Effects of Depression on Cognitive Functioning

Despite an increased interest in the depressive symptomatology in older adult populations, the neuropsychology of late-life depression is poorly understood. While numerous studies have examined cognitive functioning in late-life depression, only a handful have employed a comprehensive assessment of cognitive domains. Considering the studies available for review, it is clear that the reported results from these investigations have been contradictory. While a number of authors have reported severe and global cognitive deterioration related to depression (Savard, Rey, & Post, 1980; Siegel & Gershon, 1986; Siegel, Gurevich, & Oxenkrug, 1989; Siegfried, 1985), others have been unable to document abnormal cognition in this population (Bieliauskas, Costello, & Terpenning, 1991; Niederehe & Camp, 1985; Popkin, Gallagher, Thompson & Moore, 1982; Williams, Little, Scates, & Blockman, 1987). The following sections will briefly review the contradictory results obtained from cross-sectional studies examining depression and cognition in the areas of memory, speed of processing, and executive functioning. While the majority of studies in this line of research have been cross-sectional in nature, there have also been a few longitudinal studies that have been completed recently. These investigations will also be reviewed briefly.

Cross-Sectional Studies

Memory.

Although the effects of depression on several domains of cognitive function have been investigated in the elderly, the domain of memory has been the most often studied. An issue that has been the subject of increasing attention among investigators in recent years is whether depression in late life results in impairments of memory functioning compounding those observed in normal aging. Although it has been repeatedly shown that subjective memory complaints are far more common among depressed than among nondepressed older adults (Feehan, Knight, & Partridge, 1991; Williams, Little, Scates, & Blockman, 1987), researchers using neuropsychological measures have found a mixed picture concerning the effects of depression on memory functioning in old age. A host of investigators have concluded that depression can impair memory, broadly construed, in this population (Backman & Forsell, 1994; Gainotti & Marra, 1994; King, Caine, Conwell, & Cox, 1991; LaRue, Goodman, & Spar, 1992) and an equally imposing array of authorities have found little or no effect (Geffen, Bate, Wright, Rozenbilds, & Geffen, 1993; Loewenstein et al., 1991; O'Hara, Hinrichs, Kohout, Wallace, & Lemke, 1986; Rohling & Scogin, 1993). Still others have found mixed effects; that is, depressed subjects are impaired on some tasks but not others (Hart, Kwentus, Taylor, & Harkins, 1987; LaRue, D'Elia, Clark, Spar, & Jarvik, 1986).

These discrepant findings may in part be due to differences across studies with regard to which aspects of memory have been assessed. For example, there is evidence that depression-related deficits in memory may be pronounced in tasks that involve little retrieval support, as in free recall, whereas differences between those with depression and control subjects are reduced or eliminated in tasks involving a high degree of retrieval support, such as in recognition tasks (Cummings & Benson, 1983; Hart et al., 1987;

Weingartner, 1993). In a related vein, depression-related deficits in memory appear most likely to occur when there are high demands on effortful, elaborative activities at encoding the information (Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982; Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981). For example, depressed patients have difficulties in spontaneously using an appropriate organizational structure with material that is presented in a seemingly disorganized fashion (Watts, Dalgleish, Bourke, & Healy, 1990) and they do particularly poorly when long memory lists are used (Henry, Weingartner, & Murphy, 1973). By contrast, elderly depressives perform better on those memory tasks that require less effortful organization during the encoding process (Cohen et al., 1982; Norton, Houston, Johnson, & Zechmeister, 1988).

A recent meta-analysis of the effects of depression on memory has helped to clarify the relationship between these variables somewhat. The meta-analysis, published in 1995 by Burt, Zembar, and Niederehe, was based on 147 studies of depressed patients involving recall and recognition tasks. Their overall conclusion was that depression and memory impairments are significantly associated with one another. However, the effect sizes were small to moderate, depending on task and subject conditions. Average *d*s, reflecting the difference between depressed and control subjects, ranged from 0.27 to 0.6. Although depression was related to memory impairment, the strength of the relationship was smaller than that seen in degenerative conditions, where Burt and colleagues found *d*s ranging from 1.32 to 2.25. Interestingly, depression was more strongly related to memory impairment among younger than older subjects. The modest effect sizes found in the depression versus control groups, and the finding that depression has a greater impact on memory in younger than in older individuals, leaves unanswered the question

as to why so many experts believe the relationship between geriatric depression and memory is robust. Clearly there is more investigative work that needs to be done in this area.

Speed of processing.

Patients suffering from depression often report the subjective experience of a slowing in mental speed (O'Connor et al., 1990). It has been recognized for some time, and makes intuitive sense, that depressed subjects' performance on cognitive tasks may be linked to the efficiency with which the task must be completed (Weingartner, 1986). For example, Hart and Kwentus (1987) found that depressed elderly patients demonstrated psychomotor slowing on two tasks requiring rapid motor performance, the Digit Symbol subtest of the Wechsler Adult Intelligence Scale and the Sternberg Memory Scanning Task. Indeed, cross-sectional studies have generally reported that both motor and cognitive speed appear to be impaired in depression (Calgiuri & Ellwanger, 2000; Sobin & Sackheim, 1997), although Elliott et al. (1996) found that middle-aged depressed patients were impaired on a measure of cognitive speed but not motor speed. Nevertheless, while numerous studies have demonstrated processing speed deficits in those with depressive symptomatology, the relationship between these variables has not been well examined outside of the traditional cross-sectional design. Therefore, the long-term effects of depression on cognitive slowing are as yet unknown.

One of the most frequently used tests to measure psychomotor slowing is the Digit Symbol Modalities Test, a very short measure in which subjects have to substitute symbols for digits according to a key shown at the top of the page. The Digit Symbol Modalities Test owes its clinical sensitivity to the fact that it makes demands on several

processes at once, including perception, working memory, sustained attention, and visuomotor coordination. For the successful completion of the task, all of the processes need to be functioning efficiently (Lezak, 1983). A number of studies that have used this test with depressed subjects have found impaired performance relative to controls (Austin et al., 1992; Wolkin et al., 1992), however, there is no published data on this measure involving depressed subjects in a longitudinal design.

Executive functioning.

Of all the cognitive domains investigated with depressed elderly subjects, executive functioning abilities have been the least studied. The few studies assessing executive skills in older depressed patients have revealed impairments on at least some tests, including word generation tasks, the Wisconsin Card Sorting Test, and the Halstead Category Test (Emery & Breslau, 1989; Geffen et al., 1993; Hart, Kwentus, Taylor, & Harkins, 1987; Savard et al., 1980). Although only a few neuropsychological studies have been conducted in this area, limited evidence from neuroimaging studies has demonstrated that individuals with depressive symptoms have reduced cerebral blood flow in different regions of the frontal cortex (Bench et al., 1992, 1993; Drevets et al., 1992), the area of the brain believed to be most associated with higher-order, executive-type abilities. Moreover, structural scanning using MRI has shown increases in subcortical white matter changes in elderly depressives (Coffey, Figiel, Djang, & Weiner, 1990; Krishnan et al., 1988), another area of the brain associated with executive abilities. This area of the brain is commonly referred to when discussing the "fronto-subcortical loop", the connections of which are thought to be necessary (that is, intact) for proper execution of executive abilities. Patients with damage to subcortical structures often

demonstrate impairment on measures sensitive to frontal lobe dysfunction, such as attentional set-shifting and planning (Owen et al., 1992; Robbins et al., 1994).

Longitudinal Studies

Very few studies have followed older depressed patients across time to document the frequency of later cognitive deterioration. Those studies that have been conducted in this area have largely focused on elderly depressives' relative risk of developing a dementing illness, such as Alzheimer's disease (AD), and have not examined the effects of depression on the cognitive functioning of normal, non-demented individuals. While few studies of this kind have incorporated the healthy elderly, studies involving dementia patients nevertheless help to shed light on the possible long-term negative effects of depressive symptoms on cognition.

One of the first longitudinal studies investigating the link between depression and the development of dementia was conducted by Kral in 1983. In that study, 22 older adults diagnosed with pseudodementia (mean age = 76.5) were followed semiannually over the course of 4 to 18 years. Twenty of the 22 patients (91%) developed Alzheimer's disease by the end of the research period. The diagnosis of AD was supported by neuropathologic evidence obtained postmortem. Of the 22 patients followed, 11 were alive at the end of the study, 9 of whom were demented and living either in hospitals or in nursing homes. While these results are quite dramatic, the interpretation of the study is made difficult because Kral (1983) used the term pseudodementia as a diagnosis and because there was inadequate description of the subjects and of how diagnoses were made. However, this study did illuminate the need for investigation into the relationship between depression and cognitive impairment in older age.

Reding, Haycox, and Blass (1985) followed 28 older patients diagnosed with a nondementing depression per DSM-III-R criteria (APA, 1987) and neurological and psychiatric evaluations. The study covered 3 years and resulted in 16 of 28 (57%) developing "frank dementia." Thirteen of those 16 patients demonstrated some sign, often subtle, of neurological disease. Reding and colleagues (1985) speculated that depression may initially present as the first sign of a developing progressive dementia.

A later study by Nussbaum, Kaszniak, Allender, and Rapcsak (1995) followed 35 older adults (70 years of age and older) diagnosed with either major depression or dysthymia, both without cognitive impairment. Two board-certified specialists, who were reported to be expert in differential diagnosis with older adults, made diagnoses independently from a review of medical and neuropsychological data. Repeat neuropsychological examinations were administered at the end of the study period (approximately 2 years), and 8 patients (22%) demonstrated progressive dementia consistent with AD. Predictors of later cognitive decline included the presence of deep white matter and subcortical lesions as measured by brain CT/MRI and abnormal electrocardiogram (EKG) conducted at the initial evaluation. These authors asserted that for older depressed individuals, an abnormal brain CT/MRI in the presence of a normal cognitive profile may represent a neurobiological marker for the development of a progressive dementia.

In contrast to the findings reported above, a longitudinal study conducted by Sachdev, Smith, Lepan, and Rodriguex (1990), found no substantial decline in a sample of 19 middle-aged adults diagnosed with pseudodementia. No decline was found in the study across a 12-year time period. However, this finding may be less relevant given the

fact that the mean age of the sample at the start of the investigation was 53. Nonetheless, Sachdev and colleagues (1990) asserted that pseudodementia was not a risk factor for later cognitive decline. Since the time of the report by Sachdev et al. (1990), at least three other investigators have also been unable to replicate the finding that depressive symptomotology increases the risk for onset of dementia (Buntinx, Kester, Berrgers, & Knottnerus, 1996; Dufouil et al., 1996; Henderson et al., 1997).

Overall, the longitudinal studies reviewed here suggest that depression in some older adults may represent an early marker for the later development of a progressive dementia. However, due to the small number of published studies and the obvious methodological concerns with this body of empirical work, researchers do not yet have enough evidence to suggest that a strong relationship exists between depression and later cognitive decline. In addition, as stated above, because these studies have focused on the risk of developing dementia, little is known about the long-term effects of depressive symptoms among the healthy elderly. The need for more research in this area is clear.

Summary of Depression-Related Cognitive Decline

While a number of authors have reported severe and global cognitive deterioration related to depression in old age, others have been unable to document abnormal cognition in this population. A handful of studies have investigated the impact of depressive symptomatology on specific cognitive abilities; however, it remains unclear whether there is a widespread impairment affecting all cognitive processes or whether separate functions such as memory, speed of processing, and executive functioning, are affected differentially.

Despite previous research, there are a number of important questions that remain unanswered in the literature. Specifically, researchers do not fully understand the strength of the relationship between depressive symptoms and cognitive decline, the degree to which varying levels of depression severity influence cognitive functioning, the types of cognitive processes most likely to be affected by depression, those least likely to be affected, or the possible interaction between age-related cognitive decline and depression-related cognitive decline. Furthermore, the vast majority of empirical work that has been done with these variables has been cross-sectional in nature and thus the long-term affects of affective disturbance on cognition have been ignored.

Anxiety in Older Adults

Prevalence

The prevalence of anxiety disorders in elderly adults is a topic that has received little attention, especially given the impressive amount of research on anxiety disorders over the past several years (Norton, Cox, Asmundson, & Maser, 1995) and the fact that symptoms of anxiety are fairly common in late life (Krasucki, Howard, & Mann, 1998; Palmer, Jeste &Sheikh, 1997). A review of the literature on affective symptoms in old age reveals that much less attention has been focused on anxiety than on depression (Raj, Corvea, & Dagon, 1993), despite the fact that both types of affective disturbance represent significant factors associated with increasing age. The serious need for more research on the impact of anxiety in the elderly population has recently been highlighted by the National Institute of Mental Health (Pearson, 1998).

Prior to the Epidemiological Catchment Area (ECA) survey (Regier et al., 1988), there was little information on population prevalence rates for anxiety disorders among

the elderly. The ECA survey documented a one-month prevalence rate of 5.5% for all of the anxiety disorders in individuals aged 65 and older, which is lower than that noted in younger adults (7.3%). However, the diagnoses of generalized anxiety disorder (GAD) and posttraumatic stress disorder (PTSD) were not included in this DSM-III-based (American Psychiatric Association, 1980) study. A later survey by Blazer et al. (1991), which included GAD but not PTSD, found a 6-month prevalence of 19.7% and a lifetime prevalence of 34.1% among an elderly community sample.

Although anxiety disorders have generally been found to be less common in the elderly than in the younger adult population, such disorders nonetheless affect a significant proportion of older adults. Flint (1994) reviewed eight random-sample community surveys and found prevalence rates of diagnosed anxiety disorders ranging from 0.7 to 18.6% among elderly people. When the prevalence rates for specific anxiety disorders are examined in this population, phobias (agoraphobia, social phobia, and simple phobia) represent the most common form of the disorder, with a rate of 4.8% (Regier et al., 1988). Lower prevalence rates have been reported for obsessive-compulsive disorder (0.8%) and panic disorder (0.1%), although generalized anxiety disorder appears to occur more frequently, with a prevalence rate of around 4.6%. Indeed, it is possible that GAD is even more prevalent than this figure indicates, given that previous studies using DSM-III-R criteria (American Psychiatric Association, 1987) did not diagnose GAD if any other disorder was present. Thus, it is possible that even higher rates of GAD will emerge from studies using current diagnostic practices.

Some experts have also argued that the significance of anxiety among older adults has been greatly underestimated by studies that have excluded subsyndromal

presentations (Himmelfarb & Murrel, 1984). This occurs when subjects, who present with symptoms of anxiety but fail to meet criteria for a specific disorder, are excluded from participation. Fortunately, while limited in number, there have been a few studies that have examined the prevalence of anxiety by focusing on symptoms, rather than disorders, and these have yielded considerably higher estimates of anxiety in elderly adults (Himmelfarb & Murrel, 1984; Magni & DeLeo, 1984). Indeed, when significant anxiety symptoms that do not reach criteria for a specific disorder are considered, prevalence rates among the elderly increase to approximately 20 to 25% in community samples (Copeland et al., 1987; Himmelfarb & Murrell, 1984). A crucial difference between these studies and research designed to assess anxiety *disorders* is the flexibility of the diagnostic criteria. In the latter situation, a person either qualifies for participation in the study or not. In the Magni and DeLeo study (1984), participants' scores on a self-report measure of anxiety were rated according to a continuum of categories. The advantage of this approach is that people who fail to qualify for a diagnosis of high anxiety were not excluded from the survey, but instead were classified as having symptoms of anxiety along a continuum from low to high. Well-defined diagnostic criteria that sharply distinguish between significant and non-significant anxiety are crucial for some types of research purposes, but it has been suggested that rigid diagnostic criteria may exclude some of the wide range of expressions of significant anxiety in elderly adults (Bliwise, McCall, & Swan, 1987).

There have been other factors complicating the assessment of anxiety in older adult populations, including difficulty with the identification of symptoms, both on the part of older adults and their treating physicians, lack of consensus regarding the

definition of anxiety as applied to the elderly, as well as the comorbidity of physical and other psychiatric illnesses that are frequently seen with symptoms of anxiety. As stated previously, mental illness in the elderly often manifests primarily as somatic complaints (Lurie, 1987) and there is evidence to suggest that anxiety, like depression, may have such a presentation in the elderly (Sallis & Lichstein, 1982; Turnball, 1989). Since many physical ailments that commonly afflict older persons have symptoms that are also considered anxiety symptoms, the problem lies in determining what the symptoms represent. Furthermore, while late-life anxiety is common and appears to have potentially serious consequences, most anxious older adults do not seek help (Himmelfarb & Murrell, 1984). Generally, older adults are believed to underutilize mental health services (Ettner & Hermann, 1997). Older people may not seek treatment for anxiety in particular because they may believe it is an inevitable part of the aging process or because they may interpret somatic anxiety symptoms as manifestations of physical illness (Turnbull, 1989). Alternatively, older adults may underutilize services because they grew up in an era when mental health treatment was considered a stigma and was reserved only for those who were "crazy". Those older adults who do seek treatment often receive care exclusively from their primary care physician. For example, in a study of older adults who received outpatient services reimbursed by Medicare for a psychiatric diagnosis, only 29% saw a psychologist or psychiatrist (Ettner & Hermann, 1997).

To complicate the problem further, physicians and mental health experts may have their own biases when it comes to evaluating symptoms of anxiety in the elderly. A study by Ford and Sbordone (1980) demonstrated that psychiatrists view elderly patients

as less likely to respond to treatment than their younger counterparts. These types of negative attitudes may influence clinical judgment and decision making and lead to improper identification and treatment of these disorders in older adults. Even when clinicians are alert to the symptoms of anxiety in this population, the coexistance of anxiety and depression makes distinguishing between the two quite difficult (Shamoian, 1991; Sheikh, 1991). For example, in a study conducted by Lindesay, Briggs, and Murphy (1989), up to 39% of phobic elderly patients experienced depression, compared with only 11% of nonphobic elderly. Similarly, Alexopoulos (1990) found a 38% prevalence of anxiety disorders in elderly outpatients with major depression. Other recent reviews have also highlighted the comorbidity of these disorders in older adults (Flint, 1994). Examination of smaller-scale studies indicates that over 90% of community-dwelling elders who received a diagnosis of GAD also met criteria for depression (Lindesay et al., 1989). In this particular sample, close to 40% of individuals who were diagnosed with a specific phobia also received a diagnosis of depression. Thus, the rates of diagnostic comorbidity between anxiety and depression appear to be substantial among the elderly. Obviously this finding has important implications for the recognition and treatment of these disorders in late life.

Etiology of Late-Life Anxiety

Although much has been written on the etiology of anxiety throughout the lifespan, relatively little literature exists on the specific causes of anxiety in the elderly. Although sparse, recent studies have attempted to identify at least some of the important risk factors associated with anxiety in older adults. These include demographic factors such as gender and race, and social factors including illness, disability, loss, and exposure

to traumatic events. This section will briefly summarize the ways in which these factors may predispose elderly subjects toward developing symptoms of anxiety, may precipitate or maintain such symptoms, or alternatively, serve as a protection against their manifestation.

Demographic Factors

Gender.

Most epidemiological studies have reported a considerably greater prevalence of anxiety symptomatology in elderly females than in elderly male subjects, thus suggesting that female gender predisposes one toward anxiety in old age. The ECA study reported earlier (Regier et al., 1988) demonstrated that older women stand a much greater chance of experiencing an anxiety disorder, relative to older men, with a documented 2:1 ratio. This finding has been extended to self-reported anxiety levels as well. More specifically, women have been documented to report higher levels of trait anxiety (Himmelfarb & Murrell, 1984), fearfulness (Liddell, Locker, & Burman, 1991), as well as specific phobias (Fredrikson et al., 1996).

Race.

Race has been another demographic characteristic reported to be of importance with respect to the development of anxiety symptoms in old age. However, despite claims that race is an important variable, it has generally been an overlooked characteristic within the literature (Kastenbaum, 1994). One of the first studies to incorporate race was the ECA study by Regier et al. (1988). However, sampling problems occurred within some minority groups that unfortunately precluded accurate prevalence data (Beidel & Turner, 1991). Although the rate of cognitive impairment was

higher among elderly African-Americans relative to elderly Caucasians in the Yale site of the ECA study (the only site that oversampled the elderly), all other DSM-III disorders, including anxiety disorders, were less prevalent in African-Americans. Unfortunately, very few reports have appeared since the ECA study was published that have tried to examine race within specific anxiety-related diagnostic categories. An exception is a study conducted by Blazer et al. (1991) in which separate prevalence rates were computed for African-American and non African-American groups by gender. This analysis indicated the highest prevalence of GAD was found in African-American women (3.7%), followed by non African-American women (2.7%), non African-American men (0.7%), and lastly, African-American men (0.3%). Although this study provides a preliminary examination into the role of race in the development of anxiety in old age, clearly more empirical work is needed to expand upon these findings.

Social Factors

Illness and disability.

Although a substantial amount of literature has addressed the overlap between depression and medical conditions, the same attention has not been given to anxiety disorders in this domain. Because older adults may attribute anxiety-related symptoms, such as agitation, muscle tension, and sleep problems, to physical causes, this issue is particularly important in understanding the differential diagnosis of anxiety (Gurian & Miner, 1991). Additionally, some physical conditions (e.g. cardiovascular disease, hypothyroidism) create anxiety-like symptoms, suggesting the need for thorough medical and psychological evaluation (Cohen, 1991). Although the literature is limited, several

studies have examined disability and symptoms of anxiety in both community-dwelling populations as well as in elderly patients presenting to medical clinics and hospitals.

It is no surprise that patients with chronic physical illnesses have been shown to be particularly vulnerable to the development of psychiatric disorders, and anxiety disorders in particular. At least one community-based survey found that persons with a chronic medical condition had a 41% increased risk of developing a psychiatric disorder, and more than 11% of the individuals with chronic medical conditions had suffered from a recent anxiety disorder (Wells, Golding, & Burnam, 1988). Another study conducted by Stein, Heuser, Juncos, and Uhde (1990) found that 38% of patients with Parkinson's disease met criteria for an anxiety disorder. Similarly, panic disorder has been diagnosed in 8% of outpatients with stable chronic obstructive pulmonary disease (Karajgi, Rifkin, Diddi, & Kolli, 1990). Of patients with early dementia, 38% have been shown to have a comorbid anxiety disorder (Wands et al., 1990). In another study by Magni et al. (1988), involving a group of elderly medical inpatients, 40% of their sample was reported to have experienced significant symptoms of anxiety. In this study, the severity of anxiety symptoms was not associated with whether the individual had chronic versus acute medical problems, but symptoms of phobia were notably higher in patients with disorders of the central nervous system.

While this brief review cannot be considered an exhaustive list of the available research conducted in this area, it is clear that the interrelationship between specific medical problems and symptoms of anxiety is an important one that deserves greater attention.

Loss and exposure to traumatic events.

Anecdotal reports, as well as empirical research on anxiety in late life, suggest a major etiological role in the life changes associated with old age, many of which involve some form of loss. This includes loss of, or decline in, both mental and physical abilities, together with the approaching reality of death, loss of a spouse, loss of health, loss of independence, and loss of self-esteem upon retiring (Banazak, 1997; O'Brien-Counihan, 1997; Spar & LaRue, 1997). Changing roles within the family and society, financial constraints associated with a limited income and potential medical costs, and feelings of helplessness and loss of control are other potential precipitants of anxiety in older adults (Zisook, Schneider, & Shuchter, 1990).

Anxiety disorders in the elderly are also frequently associated with traumatic events. Lindesay (1991) reported that the onset of agoraphobia in old age frequently occurs after a traumatic event such as acute physical illness, falls, or muggings. The documented presence of anxiety disorders such as PTSD (Sembi et al., 1998), agoraphobia (Burvill et al., 1995), and GAD (Castillo et al., 1993) after a stroke suggests that the trauma of this medical event, and/or specific neurobiological changes, may play an important role in the onset of anxiety disorders in old age. One special population in this category that has received little attention is individuals at risk for posttraumatic stress disorder. In particular, individuals exposed to extreme traumas, such as combat, the Holocaust, or being taken as a prisoner of war, may function adequately until they begin to age, at which point PTSD symptomatology may begin (Averill & Beck, 2000). Many explanations exist for this phenomenon, including increased psychological and physical vulnerability associated with aging, exposure to an event that resembles the initial trauma, and the reflective nature of many older adults (Averill & Beck, 2000). Thus,

older adults who have been exposed to various types of traumatic events may represent an underserved anxiety-disordered population. These findings, in combination with evidence from other studies reported here, are important in shedding light on the ways in which trauma and loss in old age may be associated with the development anxiety-related symptomatology.

Effects of Anxiety on Cognitive Functioning

Given the lack of attention that past research has given to symptoms of anxiety in old age, it is not surprising that the neuropsychology of late-life anxiety is poorly understood. While much research has focused on age-associated cognitive decline in the older adult population, emphasis has begun to be placed on other factors, such as heightened anxiety, that may contribute to decrements in an older person's performance. Oberleder (1967) recognized the importance of examining affective status in older adults and summarized this line of reasoning in the following way: "Perhaps the greatest difference in working with the older subject is that physical, psychosocial, and anxiety factors increase with age; tests become more threatening generally, and performance more likely to be affected by non-test factors ... The effects of both longstanding and situational anxiety are particularly important" (p. 189). However, despite this early recognition that emotional factors may play a key role in the cognitive changes associated with age, researchers have yet to fully explore this hypothesis. Furthermore, research that has examined these variables has been mostly concerned with the negative effects of anxiety and depression on the performance of elderly subjects at the time of the emotional disturbance (cross-sectional designs), and there exists virtually no empirical investigations on the long-term effects of negative emotional states, particularly that of

trait anxiety. With this in mind, the following section will briefly summarize the limited body of research pertaining to late-life anxiety and cognition in the areas of memory, speed of processing, and executive functioning.

Memory

Of all the cognitive domains investigated with respect to the effects of anxiety on performance, that of learning and memory has been the most extensively evaluated. An early study conducted by Leon (1989) suggests that anxious subjects may generate internal task-irrelevant information, which may strain memory capacity or otherwise interfere with the allocation of attentional resources to the task at hand. Indeed, the most recent studies of anxiety and memory have tended to focus on subjects' biases with respect to attentional focus rather than on general deficits in actual memory performance. For example, Broadbent, Broadbent, and Jones (1986) found that highly anxious subjects experienced more difficulty in focusing attention during experimental tasks, regardless of the content of presented material. In a similar study, Mathews et al. (1990) found that individuals suffering from generalized anxiety disorder had more difficulty detecting a letter at an unexpected location when words were appearing elsewhere on the screen, suggesting a general difficulty with attentional control.

In additional to the difficulty that highly anxious older adults may have with maintaining attentional focus, there is also literature to suggest that working memory and initial encoding of information are the most affected by anxiety, while more automatic tasks are the least affected (Bradley, Mogg, & Williams, 1994; Darke, 1988; Hill & Vandervoort, 1992; MacLeon & Donnellan, 1993). One specific hypothesis that has gained considerable attention with respect to these findings has been that anxiety biases

people away from encoding information in terms of semantic features and makes them more susceptible to encode information in terms of more superficial features, such as the temporal order in which information is presented. For example, when a list is constructed of words that are capable of being grouped into several semantic categories, but are presented to the subject in random order, normal subjects tend to recall them in semantic clusters. It seems that this may be disrupted in individuals with high levels of anxiety. This has been reported in patients with sub-threshold anxiety disorders by Schwartz (1985), and with highly anxious subjects by Mueller (1986). In general, it appears that both state and trait anxiety are particularly associated with significantly poorer performance in verbal learning tasks during late life (Deptula et al., 1993; Paterniti, Dufouil, Bisserbe, & Alperovitch, 1999; Whitbourne, 1976).

Speed of Processing

Unfortunately, there is very little information concerning the effects of anxiety on processing speed among older adults and the literature concerning the long-term effects of anxiety on information processing is non-existent. Nevertheless, those studies that have been conducted have reported results consistent with the idea that anxiety negatively impacts processing efficiency. For example, King, Hannay, Masek, and Burns (1978) found a significant inverse correlation between self-reported symptoms of anxiety and performance on two timed tasks, the finger tapping and form board tests. However, this finding pertained only to female subjects. Although this result may be related to actual gender performance, they note that only the female subjects in their study endorsed significant anxiety symptoms. In another study, Hockey (1986) found that experimentally-induced anxiety resulted in increased speed of processing performance

paired with significantly poorer accuracy. In an elaboration of these findings, some researchers have proposed that heightened anxiety restricts an individual's ability to efficiently process relevant information. This explanation has been proposed in the processing efficiency theory put forward by Eysenck and Calvo (1992). This theory predicts that, especially in stressful conditions or in the case of high demands on information processing, high trait-anxious individuals are at a disadvantage. An excess of worries and other anxious thoughts preempt part of the processing resources they would otherwise have available and therefore negatively impact accuracy of performance. While this represents an interesting theory in the literature, little evidence has been collected in support of its claims.

Executive Functioning

Given the scant research associated with anxiety and the elderly, it is not surprising that very little information exists with respect to the specific impact of anxiety on executive functions within this population. One of the only studies that has investigated these variables was conducted by Cohen et al. (1980). Cohen and his colleagues reported that higher levels of trait anxiety were associated with poorer performance on a test of abstract reasoning in the elderly. Although other evidence in this domain is lacking, some writers have suggested that the anxious elderly may be at a disadvantage when it comes to solving abstract, novel problems because of an increase in cognitive rigidity that may occur (Kuhlen, 1964). Rigidity has been defined by Kuhlen as the adherence to a previously established behavior pattern in situations in which response change would be optimal. Flexibility is characterized as the ability to appropriately modify established behaviors in response to situational demands. Kuhlen (1964)

proposed the idea that increasing anxiety leads to cognitive ridigity in the elderly, which in turn may negatively impact their ability to make use of novel problem-solving skills when performing tasks of executive functioning. This theory has yet to be thoroughly investigated.

Summary of Anxiety-Related Cognitive Decline

At present, the effects of anxiety in the aged have not been thoroughly investigated. In addition, it seems that researchers have not yet well connected the symptoms of anxiety to specific types of tasks or cognitive processes, such as memory, speed of processing, or executive functioning. Rather, anxiety is most often discussed in the literature as a nonspecific mediator of age-related cognitive decline. Presumably the effects of anxiety are not ubiquitous with respect to cognitive functioning, however little empirical work has been conducted to make the necessary distinctions between the domains of functioning most impacted by anxiety-related symptoms in old age. Furthermore, all reports on anxiety and cognition published to date have been limited to cross-sectional data. The existing literature has so far not presented studies relating to the relationship between anxiety and normal age-related cognitive decline (Wetherell, Reynolds, Gatz, & Pedersen, 2002). Therefore, an important question remains as to whether general anxiety proneness is related to accelerated rates of decline relative to those who are less anxious. Exploring the relationships between anxiety and cognitive performance and decline in the older adult population is important for several reasons, as indicated by previous authors (Wetherell et al, 2002). First, because neuropsychological tests are used in diagnosing dementia, there are serious consequences for our deficient knowledge with respect to which tests may be vulnerable to the effects of anxiety in older

adults. Additionally, learning more about the effects of anxiety on cognition across the lifespan can lead to a better understanding of cognitive processes associated with anxiety and aging.

Specific Hypotheses to be Tested

The present study seeks to add to the existing literature on the effects of anxiety and depression on cognitive functioning in older adults by improving upon the methodological and theoretical shortcomings of earlier studies. This investigation will do so in the following important ways: (1) examine these variables in a within-subjects longitudinal design, thereby allowing for the direct examination of the long-term effects of anxiety and depression on cognitive functioning (2) using neuropsychological measures that assess the specific cognitive functions of memory, speed of processing, and executive functioning as opposed to measures of global, non-specific types of cognition and by (3) employing statistical analyses using continuous variables of anxiety, depression, and cognitive functioning in order to avoid the distortion that may occur by falsely dichotomizing these constructs.

Based on the literature reviewed above, the specific hypotheses of the current study include the expectation of the following results:

1. Confirmation of an age-related decline in cognitive functioning between Time 1 and Time 2 in the areas of memory, speed of processing and executive functioning.
2. Together, age and depression will be more predictive of cognitive decline between Time 1 and Time 2 than age alone in the following domains:
    a. memory (free recall and semantic clustering)
    b. speed of processing

c. executive functioning

3. The Geriatric Depression Scale (GDS), which was designed specifically for an older adult population, will be a stronger predictor of the decline in these domains than the Beck Depression Inventory (BDI), which was not developed exclusively for use with the elderly.

4. Together, age and trait anxiety will be more predictive of cognitive decline between Time 1 and Time 2 than age alone in the following domains:

a. memory (free recall and semantic clustering)

b. speed of processing

c. executive functioning

5. Measures of depression and anxiety will <u>not</u> be significant predictors of cognitive decline between Time 1 and Time 2 above and beyond that of age when examined with the Mini Mental Status Exam, a measure of gross (non-specific) cognitive functioning.

METHOD

Participants

The present investigation included 57 community dwelling older adults from the Lansing, Michigan area, between the ages of 57 and 87 (Time 1 $\underline{M}$ = 69.86, $\underline{SD}$ = 6.36 years; Time 2 $\underline{M}$ = 75.09, $\underline{SD}$ = 6.47 years). Public advertisements in local newspapers and other mailings were used in 1997 and 1998 to solicit willing participants for the study procedures of Time 1. Subjects who expressed an interest in the study were then screened using the Mini Mental Status Exam (MMSE). Individuals who demonstrated cognitive impairment with a score of 23 or below on the MMSE were excluded from the sample in order to ensure that all participants were healthy and functioning at a level consistent with normal aging. Those excluded from participation in the study at Time 1 were also referred to other agencies in the community for more comprehensive assessments. Subjects who met screening requirements and participated in the study were given the opportunity to attend mood and memory training workshops at Michigan State University.

In 2002 and 2003, approximately five years after their initial participation, subjects were sent letters and received telephone calls to solicit their involvement in the Time 2 portion of the study. Informed consent was obtained from each participant prior to the completion of the study procedures described below. Participation in the study was completely voluntary.

Procedures

The procedures and instruments used during Time 1 and Time 2 of the study were identical in order to assess the cognitive changes that occurred over the 5-year time

period. Participants completed detailed questionnaires yielding demographic and life history information before completing a set of neuropsychological tests. The total length of testing for each individual was approximately 2 hours. The order of measures was systematically varied across participants. All testing sessions were conducted individually. The administered procedures assessed, among other things, the individual's mood, memory, executive functioning, speed of processing, and general cognitive ability. The protocol used in this investigation was formally approved by the Michigan State University Committee on Research Involving Human Subjects (UCRIHS) under IRB # 03-912.

Instruments

The measures used in this investigation included the following:

Screening Instrument

Mini-Mental State Exam (MMSE)

The MMSE (Folstein & McHugh, 1975) is a 30-item measure used to screen for cognitive impairment and document changes in intellectual functioning over time. The measure is easily administered and scored in approximately 5-10 minutes. The MMSE requires the examiner to ask questions and record responses given by the subject. The test consists of items that assess orientation to time and place, attention and concentration, language, constructional ability, and immediate and delayed memory recall. Each correct response is awarded one point and the total score is the number correct out of 30. Scores below 24 are considered abnormal when screening for dementia or delirium and this cut-off is recommended for most populations (Lezak, 1995).

The psychometric properties of the MMSE are well documented in the literature. Inter-rater reliability has been shown to be above .65 (Foster et al., 1988) and test-retest reliability estimates for intervals of less than two months generally fall between .80 and .95 (O'Connor et al., 1989). Most studies report that the MMSE has adequate specificity and sensitivity for detecting the presence of dementia, particularly in cases where there exists moderate to severe forms of cognitive impairment (Spreen & Strauss, 1998). The MMSE also shows modest to high correlations with measures of intelligence, memory, attention and concentration, and executive functions (Axelrod et al., 1992). In addition, Crum et al. (1993) has published extensive norms for the MMSE by age (18-85) and education based on probability sampling of more than 18,000 community-dwelling individuals.

Demographic Questionnaire

Information on age, gender, level of education, occupation, and health status were obtained by a self-report style demographic questionnaire. Level of education was measured in the conventional manner and based on responses to the following questions: a.) 'What is the highest grade of school you completed?' and b.) 'If you attended college/graduate school, please specify how many years and what degree(s) you obtained'. Occupational level was assessed by asking participants, 'What kind of work did you do most of your working life?'. Answers to this question were then coded according to the Eppinger Demographic Formula (Eppinger et al., 1987) in which unskilled labor = 1; semi-skilled labor = 2; not in labor force (including homemakers) = 3; skilled labor = 4; managerial/official/clerical/sales/ self-employed = 5; professional/technical/artistic = 6. Finally, health status was measured using the

following questions: a.) 'How would you rate your overall health at the present time?' in which excellent = 1; good = 2; fair = 3; poor = 4 and b.) 'Do your health problems stand in the way of doing things you want to do?' in which not at all = 1; a little = 2; a great deal = 3.

Measures of Mood

Beck Depression Inventory (BDI)

The BDI (Beck, 1987) is a self-report measure consisting of 21 multiple-choice statements presented on four pages of paper. Each item is concerned with a particular aspect or symptom of depression, including cognitive symptoms, vegetative signs, mood changes, as well as somatic complaints, and asks subjects to rate their experience on a scale of graded severity. The approximate time required to complete the inventory is 5-10 minutes. A total score is obtained by adding the highest score circled for each of the 21 items. The total score can range from 0-63, with higher scores indicating more severe levels of depression. The instrument has classified depression severity in the following manner: 0-9 = normal range; 10-15 = mild depression; 16-19 = mild/moderate depression; 20-29 = moderate/severe depression ; 30+ = severe depression (Spreen & Strauss, 1998).

The BDI is a well researched instrument with strong psychometric properties. In addition, it has been used with a wide range of populations due to the ease of administration and scoring. In the original norming sample, Beck (1970) reported that test-retest reliability was above .90 and that change scores tended to parallel changes in depression severity for individual subjects. Reynolds and Gould (1981) have reported Spearman-Brown reliability to be .93 and internal consistency of test items to be .86.

Other authors have reported a coefficient alpha of .88 (Steer et al., 1989). Furthermore, the BDI is known to have good concurrent validity with instruments such as the MMPI Depression Scale (.75; Reynolds and Gould, 1981) and the Hamilton Rating Scale (.85; Brown et al., 1995) as well as clinical ratings of depression (.66; Schaefer et al., 1985).

Geriatric Depression Scale (GDS)

The GDS (Brink et al., 1982) is a self-report measure consisting of 30 yes/no questions pertaining to potential symptoms of depression. The instrument was designed specifically for the elderly population and is recommended for use by Spreen and Strauss (1998) due to the simplicity of administration and the fact that it contains somatic items which are more suitable for older adults than those presented on the BDI. The GDS requires subjects to read statements and answer each by circling the yes/no response that most accurately reflects their experience. Alternatively, the items can be read to subjects if there is any concern regarding their ability to read or respond using paper-and-pencil. The estimated time required for completion of the GDS is approximately 5-10 minutes. A total score is obtained on the instrument by adding the point values assigned to each response (0 or 1). The following cutoff points are used to determine level of depression: 0-9 normal range; 10-19 = mild depression; 20-30 = moderate/severe depression (Spreen & Strauss, 1998).

The psychometric properties of the GDS warrant the instrument's wide-spread use among older adult populations. Brink et al. (1982) reported that in the original norming sample, internal consistency (alpha) and split-half reliability were both .94. Retest reliability over a one-week time span has been reported to be .85 (Koenig et al., 1988). In addition, concurrent validity has been established with other self-report measures

including the BDI (.73; Hyer & Blount, 1984), Hamilton Rating Scale (.83; Yesavage et al., 1986), MMPI Depression Scale (.72; Bielauskas & Lamberty, 1992), and the DMS-based Symptom Checklist for Major Depressive Disorders (.77; Bielauskas & Lamberty, 1992). Criterion validity as measured against the Research Diagnostic Criteria has been reported as .82 (Yesavage & Brink, 1983).

State-Trait Anxiety Inventory (STAI)

The STAI (Spielberger et al., 1979) is a self-report measure consisting of 40 items pertaining to potential symptoms of anxiety. The first 20 items measure state anxiety and subjects are asked to read each question and indicate how they feel at the current moment according to a scale that includes the responses "Not at All", "Somewhat", "Moderately So", and "Very Much So". The second half of the test measures trait anxiety and subjects are asked to read each question and indicate how they feel generally (not necessarily at the current moment) according to a similar scale of responses.

A great deal of data has accumulated to attest to the utility of this measure with older populations. In two recent studies, psychometric properties of the STAI were examined in samples of participants carefully selected via semi-structured diagnostic interviews. In one report, the STAI was administered to 50 community dwelling older adults with GAD diagnosed according to the DSM-IV and 94 normal control participants with no psychiatric diagnoses (Stanley et al., 1996). Internal consistency for both STAI subscales was good, as was test-retest reliability of the Trait subscale. Stability of the State subscale over time was only moderate, as would be expected. High levels of convergent validity were also evident in the control group. For both GAD patients and control group participants, the State and Trait subscales correlated strongly ($r = .74$). In

another recent report, Kabacoff, Segal, Hersen, and Van Hasselt (1997) administered the STAI to over 200 older adult psychiatric outpatients diagnosed according to the SCID. Similar to findings from Stanley et al. (1996), internal consistency for the measure was strong, and the State and Trait subscales correlated strongly with one another (r = .72). In this study, the STAI-Trait significantly discriminated patients with an anxiety disorder from those without, although the STAI-State did not, as would be expected.

Memory

California Verbal Learning Test (CVLT)

Memory performance is frequently assessed with the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987). The CVLT is a measure of verbal learning and memory consisting of five oral presentations of a list of 16 shopping items (Monday's list) followed by periods of free-recall. The words on the list are presented verbally at the rate of one word per second. The list contains 4 items that belong to 4 different categories, including fruits, clothing, tools, and spices/herbs. After 5 trials of Monday's list, the examiner presents a second, interference, list of 16 shopping items (Tuesday's list) and a period of free-recall. Immediately after the presentation and free-recall of Tuesday's list, the subject is asked to recall all of the items from Monday's list in a period of free-recall as well as in a period of cued-category recall (Short Delay). This same procedure is undertaken 20 minutes later (Long Delay). Finally, a recognition trial is completed involving the oral presentation of 44 shopping items in which the subject attempts to identify those that appeared on Monday's list. There are no time limits on any of the memory tasks undertaken with this test.

The authors of this test report split-half reliability coefficients ranging from .77-.86. Analyses on this tests's normative sample also revealed a coefficient alpha of .74 (Delis, Kramer, Freeland, & Kaplan, 1988). The correlations between the CVLT and other tests commonly used to measure the intended construct are said to be quite good (Lezak, 1995).

Speed of Processing

Symbol Digit Modalities Test (SDMT)

The SDMT (Smith, 1982) is a measure of speed of processing, complex scanning, and visual tracking that allows comparison between oral and written performance (Lezak, 1995). The oral and written portions of the test each have a time-limit of 90 seconds. During the first trial, subjects are presented with a sheet of paper that includes a key displaying pairs of digits and symbols. Beneath the key are rows of double boxes in which symbols are presented without their corresponding numbers. The participants are then asked to use the key to determine which number is associated with each symbol and to write as many numbers as possible in the empty boxes in the 90 second period. During the second trial, the subject is asked to call out the missing numbers for each symbol, which are then recorded by the examiner. The score obtained from this test is the number of correctly placed numbers in each portion of the test with a maximum score of 110 on each. The norms for the SDMT are well established and reliability is satisfactory (Spreen & Strauss, 1998). More specifically, test-retest reliability correlations have been reported to be .80 for the written portion and .76 for the oral portion (Spreen & Strauss, 1998).

Trail Making Test (TMT), Part A

The TMT (Reitan, 1958) consists of two parts, A and B. Trails A requires the drawing of lines to connect numbered circles in chronological order and is thought to be a good measure of processing speed and visual scanning (Spreen & Strauss, 1998). Subjects are asked to connect the dots as rapidly as possible without making any mistakes, and scores on the TMT are recorded in total seconds to complete the task. On both parts of this test, mistakes made by the subject concerning the order of circles connected are corrected by the examiner. The normative data for this test have been well established. Interrater reliability estimates of the measure have been reported to be .90 (Spreen & Strauss, 1998).

Executive Functioning

Wisconsin Card Sorting Test (WCST)

The WCST (Heaton, 1993) assesses the ability of an individual to use abstraction and to shift cognitive strategies in response to changing environmental cues (Lezak, 1995). It is widely accepted as a measure of executive function, as individuals must employ strategic planning, organized searching, the ability to use environmental cues to shift cognitive sets, and must act in a way that is goal-oriented (Spreen & Strauss, 1998). Subjects are asked to match a set of cards, which vary according to the number and color of different shapes, with one of 4 "key" cards. The individual is asked to complete the task in the absence of explicit matching guidelines. Instead, they must rely solely on the feedback given after each trial indicating whether they have made a correct or incorrect match. In addition, once the subject correctly matches 10 cards consecutively, the sorting principle is changed. Subjects are not aware of this change and must modify their

responses accordingly. A great deal of empirical work on executive functioning has been done using the WCST as both the construct validity and interrater reliability of the instrument have been well established (Lezak, 1995). In fact, interrater reliability estimates have been reported to range from .88 to .96 (Lezak, 1995).

Trail Making Test (TMT), Part B

Part B of the TMT (Reitan, 1958) is used to assess aspects of executive functioning including mental flexibility, divided attention, and sequencing (Spreen & Strauss, 1998). The measure has enjoyed a long history of use as it is easy to administer and takes little time to complete (Lezak, 1995). Subjects are asked to connect a series of circles in a "connect-the-dot" fashion, alternating between a sequence of numbers and letters. The instructions ask participants to complete the task as fast as they can without removing the pencil from the paper. Interrater reliability of the test has been reported to be .90 (Spreen & Strauss, 1998).

Stroop Test (ST)

This test measures components of executive processes including concentration, the ability to shift attentional resources, and response inhibition (Golden, 1978). There are 3 trials administered to the subject and each has a 45 second time-limit. During the first trial, subjects read words aloud (either "red", "blue", or "green") printed in black ink. Twenty words are printed in each of 5 columns on the page and appear in random order. In the second trial, these words are replaced with X's which are printed in different color inks (either red, blue, or green) and the subject is asked to name these randomly listed colors as quickly as possible. The final trial combines both of the preceding stimuli and requires the subject to read the color of ink in which the words

"red", "blue", and "green" are printed. This last trial is known as the interference condition because individuals must suppress a more instinctual response (i.e., reading words) in favor of a less common way of processing information (i.e., naming colors). Lezak (1995) reports that the ST has satisfactory reliability and more specific test-retest reliabilities have been reported by Spreen & Strauss (1998) to be .90, .83, and .91 for the three portions of the test.

RESULTS

Descriptive Statistics

In all, a total of 57 of the 81 subjects who participated in Time 1 of the study were successfully recruited to participate in Time 2. Of the 24 subjects who were lost to follow-up, 11 could not be reached by mail or by phone, 6 refused participation (3 for medical/health reasons, 1 due to scheduling complications, and 2 did not comment on the reason for refusal), 5 had moved out of the Lansing area, and 2 passed away. The sample retention rate over the 5-year period was therefore 71%. This group of 57 subjects was designated as the final sample for the present study.

Comparisons between those subjects who were lost to follow-up and those who completed Time 2 procedures revealed that the two groups did not differ significantly on any of the key demographic variables, including age, gender, education, occupational status, marital status, or self-perceived health rating. Cross-tab comparisons involving dependent variables revealed that subjects who dropped out of the study scored significantly better on one measure of processing speed, Trails A ($M=30.08$, $p=.047$). No other significant differences between the two groups were found.

Sample characteristics, including age, gender, premorbid IQ, years of education, occupation, marital status, and health ratings are displayed in Table 1. Twenty-eight of the subjects were men (49.1%) and 29 were women (50.9%). All participants were Caucasian. The average age of the subjects at Time 1 was 69.86 (SD = 6.36 years) and ranged from 57 to 87. At the time of follow-up, the average age of participants was 75.09 (SD = 6.47 years), with a range of 61 to 92. Although there was some variability between participants in the amount of time that elapsed between measurement at Time 1

and Time 2, the majority of subjects completed follow-up within 2 months of their five-year anniversary (M=4.73 weeks, SD=2.01). Most subjects were married at the time of data collection (55.4% at Time 1 and 59.6% at Time 2). The sample was well educated with an average of 17.22 years (SD = 2.47) of formal schooling. With respect to occupational status, 84.2% of the subjects reported occupational attainment at or above the managerial level, with a modal rating at the rank of professional (52.6%). Finally, on a scale of 1 to 4, modal self-reported health status was 'good' (as opposed to 'excellent', 'fair', or 'poor') at the time of initial data collection (55.4%) and five years later (71.9%).

Means and standard deviations for subjects' Time 1 performance on all dependent measures, including the MMSE, CVLT, TMT, SDMT, WCST, and ST, appear in Table 2. Means and standard deviations for the same variables at Time 2 of the investigation are presented in Table 3. Correlation matrixes between age and all dependent variables are presented in Tables 4 and 5 for data collection at Time 1 and Time 2, respectively.

Average depression scores as measured by the Beck Depression Inventory (BDI) were 9.25 (SD = 4.56, range 0-23) at Time 1 and 8.12 (SD = 4.27, range 0-24) at Time 2. Depression was also measured with the Geriatric Depression Scale (GDS), a tool designed specifically for use with an older adult population. Average depression scores as measured by the GDS were 11.69 (SD = 4.98, range 0-26) at Time 1 and 11.02 (SD = 5.86, range 0-24) at the second wave of data collection. Table 6 summarizes the frequencies at which subjects scored at various levels of depression, (i.e., normal, mild/moderate, moderate/severe) as defined by cut-off scores for each of the measures. It is important to note, however, that although this table implies a categorical approach to the operationalization of depression, these scores were kept as continuous variables

during data analysis to improve upon the methodological limitations of earlier investigations. Trait anxiety scores, as measured by the State-Trait Anxiety Inventory (STAI) were 36.35 (SD = 19.23, range 20-72) at Time 1 and 35.98 (SD = 19.64, range 20-75) at Time 2. When compared to the normative sample of older adults with similar levels of education (Spielberger et al., 1979), these scores represent a range from the 3rd to >99th percentile ranking. As expected, trait anxiety remained stable across time points. The Pearson Product Moment correlation between scores at Time 1 and Time 2 was .94 ($p<.01$). Interestingly, there were no group differences in rates of depression or anxiety among men and women or among those with higher or lower levels of education.

Power Analysis

A power analysis was conducted to ensure adequate statistical power in detecting small, medium, and large effect sizes with the present sample. In this investigation, the program G-Power (Faul & Erdfelder, 1992) was used to estimate an appropriate sample size that would allow proper examination of the proposed hypotheses. For significance tests of Pearson Product Moment Correlations, Cohen (1992) defines small, medium, and large effect sizes as .10, .30, and .50 respectively. For a desired power of .80 and with alpha set at .05, G-Power (Faul & Erdfelder, 1992) indicates that 414 individuals are needed to detect small effect sizes, 48 to detect medium effect sizes, and 21 to detect large effect sizes of .50 and above. Therefore, the present study has adequate power to detect medium and large effects for correlational analyses but is inadequate for effects smaller than .30.

For significance tests involving analysis of variance, Cohen (1992) defines small, medium, and large effect sizes as .10, .25, and .40 respectively. For a desired power of

.80 and with alpha set at .05, G-Power (Faul & Erdfelder, 1992) indicates that 322 individuals are needed to detect small effect sizes, 52 to detect medium effect sizes, and 21 to detect large effect sizes of .40 and above. Therefore, the present study has adequate power to detect medium and large effects using analysis of variance techniques, but is inadequate for effect sizes smaller than .10.

Missing Data Analysis

Analyzing data with missing cases can lead to biased results and incorrect inferences. Although there were very few cases of missing data in the present study, a procedure for data imputation was used to lay the groundwork for later statistical analyses. In order to run a missing data analysis, the dataset was first imported into SYSTAT v. 10.2. Variables that contained missing cases were then analyzed and data was imputed using the EM estimation method. The expectation maximization (EM) method uses a two-step process to impute missing data (Little & Schenker, 1995). First, in the expectation (E) step, expected values are generated based on the algorithym's "best guess" as to what a complete dataset would look like given the parameters of existing data. After filling in missing data with these estimates, the maximization (M) step is conducted to recalculate the regression coefficients. These new coefficients are then substituted back into the E step and a new M step is performed. This process is repeated over and over again until the changing parameter estimates from iteration to iteration become negligible. Once finished, the process yields a complete dataset that preserves correlations between the variables.

After completing the data imputation process in SYSTAT, the output was examined to determine whether missingness was random. Results from the analysis did

not generate evidence that missingness followed a systematic pattern and therefore data was found to be missing in random.

Hypothesis 1: Age and cognitive decline

The first hypothesis of the present study sought a confirmation of an age-related decline in cognitive functioning between Time 1 and Time 2 on measures of memory, executive functioning, and speed of processing. As expected, age was significantly correlated with measures of cognitive functioning at Time 1 and Time 2. With respect to memory, increasing age was associated with poorer performance on the CVLT in both the areas of free recall (Time 1 r=-.432, p<.01; Time 2 r=-.518, p<.01) and semantic clustering (Time 1 r=-.300, p<.05; Time 2 r=-.399, p<.05). Increasing age was also shown to be associated with poorer performance on measures of executive functioning, including the WCST (Time 1 r=.303, p<.05; Time 2 r=.458, p<.01), the Stroop Test (Time 1 r=-.475, p<.01; Time 2 r=-.400, p<.01), and Trails B (Time 1 r=.332, p<.05; Time 2 r=.594, p<.01). Lastly, age was associated with slower processing speed efficiency on the SDMT (Time 1 r=-.445, p<.01; Time 2 r=-.489, p<.01) and Trails A (Time 1 r=.292, p<.05; Time 2 r=.576, p<.01).

The relationship between age and cognitive *decline* on these measures over the 5-year time period was examined using repeated measures analysis of covariance (ANCOVA). In each analysis, the dependent variables were Time 1 and Time 2 scores for the cognitive domain in question (e.g., memory, processing efficiency, executive functioning) and age at Time 1 was entered as a covariate. Results revealed a significant main effect for time in each area, including free recall (Wilks' =.817, F(1, 55)=6.659, p=.018, partial 2=.183), semantic clustering (Wilks' =.913, F(1, 55)=2.639, p=.041,

partial 2=.087), processing speed (Wilks' =.878, F(1, 55)=6.947, p=.010, partial 2=.122), and executive functioning (Wilks' =.744, F(1, 55)=7.044, p=.012, partial 2=.256). Similarly, a covariate effect for age was found in each of the models, including free recall (Wilks' =.910, F(1, 55)=5.241, p=.025, partial 2=.090), semantic clustering (Wilks' =.906, F(1, 55)=2.880, p=.035, partial 2=.094), processing speed (Wilks' =.890, F(1, 55)=5.016, p=.019, partial 2=.110), and executive functioning (Wilks' =.744, F(1, 55)=7.044, p=.012, partial 2=.256). Overall, these results provide support for an age-related decline in neuropsychological function across a wide-range of cognitive processes.

Factor Scores

An advantage of the present study is that multiple measurements were used with each subject to examine cognitive performance in the areas of processing speed and executive functioning. As can be seen in Tables 4 and 5, correlations between the two measures of processing speed, the Symbol Digit Modalities Test (SDMT) and the Trailmaking Test Part A (TMT), were significant both at Time 1 (r=-.524, p<.01) and Time 2 (r=-.549, p<.01). Likewise, the correlations between measures of executive functioning were significant at each time point, including those between the Wisconsin Card Sorting Test (WCST) and the Stroop Test (ST) (Time 1 r=-.336, p<.05; Time 2 r=-.442, p<.05), the WCST and Trails B (Time 1 r=.644, p<.05; Time 2 r=.691, p<.05), and between Trails B and the ST (Time 1 r=-.390, p<.01; Time 2 r=-.485, p<.01).

In order to combine the information contained in the measurements of these two constructs (and to avoid overmodelling the data), factor scores were computed using a principle components analysis. As can be seen in Tables 7 and 8, these new composite

scores correlated significantly with age and were therefore used in subsequent analyses as overall indicators of processing speed and executive functioning.

Hypothesis 2: Age and BDI depression predicting cognitive decline

Repeated measures analyses of covariance (ANCOVA) were conducted to determine the effects of age and BDI depression scores on various domains of cognitive functioning over a 5-year time period. More specifically, it was hypothesized that age and scores on the Beck Depression Inventory (BDI) would be associated with a decline in performance on measures of free recall memory, semantic clustering, processing speed, and executive functioning. Prior to conducting the analyses to test this hypothesis, the relationships between covariates and dependent variables were examined to determine whether or not there were any non-linear components (e.g., quadratic functions). Linearity was analyzed by creating bivariate scatterpots of the variables in question. Scatterplots revealed that none of the relationships deviated significantly from linearity, which simplified the analyses.

The dependent variables used in each repeated measures ANCOVA were Time 1 and Time 2 scores for the type of cognitive functioning in question (i.e., free recall memory, semantic clustering, processing speed, executive functioning). Covariates in each model were age at Time 1, BDI score at Time 1, and BDI score at Time 2. Results from the analyses appear in Table 9.

Free Recall Memory

With respect to free recall memory, results indicate a main effect for time (Wilks' =.803, F(1, 53)=6.580, p=.022, partial 2=.197) as well as a covariate effect for age (Wilks' =.908, F(1, 53)=5.279, p=.026, partial 2=.092). However, the effects of BDI

depression scores at Time 1 (Wilks' =.1.000, F(1, 53)=005, p=.944, partial 2=.000) and Time 2 (Wilks' =.988, F(1, 53)=623, p=.434, partial 2=.012) were shown to be non-significant.

Semantic Clustering

When examining semantic clustering, results indicate a main effect for time (Wilks' =.899, F(1, 53)=2.789, p=.037, partial 2=.101) and a covariate effect for age (Wilks' =.922, F(1, 53)=2.582, p=.046, partial 2=.078). However, BDI depression scores failed to reach significance in predicting change in semantic clustering performance between time points. As covariates in the model, BDI depression at Time 1 (Wilks' =.1.000, F(1, 53)=.025, p=.876, partial 2=.000) and Time 2 (Wilks' =.967, F(1, 53)=1.766, p=.190, partial 2=.033) were non-significant.

Processing Speed

When processing speed factor scores were entered as dependent variables in the repeated measures ANCOVA, results indicate a main effect for time (Wilks' =.876, F(1, 53)=6.998, p=.009, partial 2=.124) and a covariate effect for age (Wilks' =.883, F(1, 53)=6.651, p=.012, partial 2=.117). Similar to previous analyses, however, the effects of BDI depression scores at Time 1 (Wilks' =.991, F(1, 53)=.467, p=.498, partial 2=.009) and Time 2 (Wilks' =.999, F(1, 53)=.044, p=.834, partial 2=.001) were shown to be non-significant.

Executive Functioning

Finally, with respect to executive functioning, results indicate a main effect for time (Wilks' =.752, F(1, 53)=6.021, p=.019, partial 2=.248) as well as a covariate effect for age (Wilks' =.805, F(1, 53)=4.192, p=.034, partial 2=.195). However, BDI

depression scores failed to reach significance in predicting change in semantic clustering performance between time points. As covariates in the model, BDI depression at Time 1 (Wilks' =.989, F(1, 53)=.557, p=.459, partial 2=.011) and Time 2 (Wilks' =.983, F(1, 53)=.909, p=.345, partial 2=.017) were non-significant.

Overall, results from repeated measures analysis of variance procedures indicate that this hypothesis was not supported as BDI depression was not shown to be a significant predictor of cognitive decline in this sample of older adults.

Hypothesis 3: Age and GDS depression predicting cognitive decline

Repeated measures analyses of covariance (ANCOVA) were conducted to determine the effects of age and GDS depression scores on various domains of cognitive functioning over a 5-year time period. More specifically, it was hypothesized that age and scores on the Geriatric Depression Scale (GDS) would be associated with a decline in performance on measures of free recall memory, semantic clustering, processing speed, and executive functioning. Since the GDS was designed specifically for use with an older adult population, it was anticipated that this measure would be a stronger predictor of cognitive deterioration than the BDI, which was not developed exclusively for use with the elderly. Prior to conducting the analyses to test this hypothesis, the relationships between covariates and dependent variables were examined to determine whether or not there were any non-linear components (e.g., quadratic functions). As before, linearity was analyzed by creating bivariate scatterpots of the variables in question. Scatterplots revealed that the relationships did not deviate significantly from linearity, with the exception of the GDS score as plotted against free recall memory. Therefore, the natural

log transformation of the GDS score was used in the analysis in which free recall memory was the dependent variable.

The dependent variables used in each repeated measures ANCOVA were Time 1 and Time 2 scores for the type of cognitive functioning in question (i.e., free recall memory, semantic clustering, processing speed, executive functioning). Covariates in each model were age at Time 1, GDS score at Time 1, and GDS score at Time 2. Results from the analyses appear in Table 10. Since the effects of time and age on each of the dependent variables have been described earlier, only the results pertaining to the covariates GDS Time 1 and GDS Time 2 will be reported here.

Free Recall Memory

Results indicate that GDS depression scores failed to reach significance in predicting change in free recall memory performance between time points. As covariates in the model, GDS depression at Time 1 (Wilks' =.962, F(1, 53)=2.058, p=.157, partial 2=.038) and Time 2 (Wilks' =.951, F(1, 53)=2.666, p=.109, partial 2=.049) were non-significant.

Semantic Clustering

When examining semantic clustering, the effects of GDS depression scores at Time 1 (Wilks' =.978, F(1, 53)=1.181, p=.282, partial 2=.022) and Time 2 (Wilks' =.957, F(1, 53)=2.355, p=.131, partial 2=.043) were shown to be non-significant.

Processing Speed

When processing speed factor scores were entered as dependent variables in the repeated measures ANCOVA, results indicate that GDS depression scores at Time 1

(Wilks' =.969, F(1, 53)=1.660, p=.203, partial 2=.031) and Time 2 (Wilks' =.999, F(1, 53)=.029, p=.866, partial 2=.001) were non-significant as covariates in the model.

Executive Functioning

Finally, results indicate that GDS depression scores failed to reach significance in predicting change in executive functioning performance between time points. As covariates in the model, GDS depression at Time 1 (Wilks' =.948, F(1, 53)=2.851, p=.097, partial 2=.052) and Time 2 (Wilks' =.956, F(1, 53)=2.389, p=.128, partial 2=.044) were non-significant.

Overall, results from repeated measures analysis of variance procedures indicate that this hypothesis was not supported as GDS depression scores were not shown to be significant predictors of cognitive decline in this sample of older adults.

Hypothesis 4: Age and trait anxiety predicting cognitive decline

Repeated measures analyses of covariance (ANCOVA) were conducted to determine the effects of age and trait anxiety on various domains of cognitive functioning over a 5-year time period. More specifically, it was hypothesized that age and scores on the State-Trait Anxiety Inventory (STAI) would be associated with a decline in performance on measures of free recall memory, semantic clustering, processing speed, and executive functioning. Prior to conducting the analyses to test this hypothesis, the relationships between covariates and dependent variables were examined to determine whether or not there were any non-linear components (e.g., quadratic functions). Linearity was analyzed by creating bivariate scatterpots of the variables in question. Scatterplots revealed that none of the relationships deviated significantly from linearity, which simplified the analyses.

The dependent variables used in each repeated measures ANCOVA were Time 1 and Time 2 scores for the type of cognitive functioning in question (i.e., free recall memory, semantic clustering, processing speed, executive functioning). Covariates in each model were age at Time 1, STAI score at Time 1, and STAI score at Time 2. Results from the analyses appear in Table 11. Since the effects of time and age on each of the dependent variables have been described earlier, only the results pertaining to the covariates STAI Time 1 and STAI Time 2 will be reported here.

Free Recall Memory

Results indicate that trait anxiety scores were significantly related to the change in free recall memory performance between time points. As covariates in the model, STAI scores reached significance at both Time 1 (Wilks' =.901, F(1, 53)=6.271, p=.025, partial 2=.099) and Time 2 (Wilks' =.938, F(1, 53)=4.953, p=.039, partial 2=.062).

Semantic Clustering

When semantic clustering scores were entered as dependent variables in the repeated measures ANCOVA, results indicate that the trait anxiety score at Time 1 (Wilks' =.918, F(1, 53)=2.019, p=.051, partial 2=.082) was significant as a covariate in the model. Trait anxiety at Time 2 failed to reach significance, although it came very close to the .05 level (Wilks' =.939, F(1, 53)=1.997, p=.057, partial 2=.061).

Processing Speed

With respect to processing speed, results indicate that trait anxiety scores failed to reach significance in predicting change in performance between time points. As covariates in the model, trait anxiety at Time 1 (Wilks' =.986, F(1, 53)=.727, p=.398,

partial 2=.014) and Time 2 (Wilks' =.991, F(1, 53)=.463, p=.499, partial 2=.009) were non-significant.

Executive Functioning

Finally, results indicate that trait anxiety scores were significantly related to the change in executive functioning performance between time points. As covariates, STAI scores reached significance at both Time 1 (Wilks' =.916, F(1, 53)=3.682, p=.041, partial 2=.084) and Time 2 (Wilks' =.945, F(1, 53)=3.125, p=.052, partial 2=.055).

Overall, results from repeated measures ANCOVA analyses indicate that this hypothesis was partially supported. Trait anxiety was shown to be a significant predictor of cognitive decline in the areas of free recall memory, semantic clustering, and executive functioning. However, trait anxiety was not found to be predictive of processing speed efficiency in this sample of older adults.

Hypothesis 5: Anxiety, depression, and global cognitive functioning

Repeated measures analyses of variance (ANCOVA) were conducted to test the hypothesis that depression and anxiety would not be significant predictors of cognitive decline between Time 1 and Time 2 above and beyond that of age when examined with the Mini Mental Status Exam, a measure of gross (non-specific) cognitive functioning. Prior to conducting the analyses to test this hypothesis, the relationships between covariates and dependent variables were examined to determine whether or not there were any non-linear components (e.g., quadratic functions). As in earlier hypotheses, linearity was analyzed by creating bivariate scatterpots of the variables in question. Scatterplots revealed that none of the relationships deviated significantly from linearity,

which simplified the analyses. The results of repeated measures ANCOVA analyses appear in Table 12.

MMSE and BDI depression

The dependent variables used in this analysis were Time 1 and Time 2 scores on the Mini-Mental Status Exam (MMSE). Covariates in the model were age at Time 1, BDI depression at Time 1, and BDI depression at Time 2. Results indicate a main effect for time (Wilks' =.838, F(1, 53)=3.105, p=.043, partial 2=.162) as well as a covariate effect for age (Wilks' =.851, F(1, 53)=2.649, p=.051, partial 2=.149). However, as predicted, the effects of BDI depression scores at Time 1 (Wilks' =.990, F(1, 53)=.503, p=.481, partial 2=.010) and Time 2 (Wilks' =.991, F(1, 53)=.473, p=.495, partial 2=.009) were shown to be non-significant.

MMSE and GDS depression

MMSE scores at Time 1 and Time 2 were used as the dependent variables in this analysis. Covariates in the model were age and GDS depression scores at Time 1 and Time 2. Results indicate that GDS depression failed to reach significance in predicting change in MMSE scores between time points. As covariates in the model, GDS depression at Time 1 (Wilks' =.996, F(1, 53)=.214, p=.646, partial 2=.004) and Time 2 (Wilks' =.987, F(1, 53)=.698, p=.407, partial 2=.013) were non-significant.

MMSE and trait anxiety

The dependent variables used in this analysis were Time 1 and Time 2 scores on the Mini-Mental Status Exam (MMSE). Covariates in the model were age, STAI scores at Time 1, and STAI scores at Time 2. As predicted, the effects of trait anxiety at Time 1

(Wilks' =.975, F(1, 53)=1.312, p=.257, partial 2=.025) and Time 2 ((Wilks' =.981, F(1, 53)=.998, p=.322, partial 2=.019) were shown to be non-significant.

Overall, results from repeated measures ANCOVA analyses indicate that this hypothesis was supported, as measures of depression and anxiety were not shown to be significant predictors of decline when using a global, non-specific measure of cognitive functioning.

DISCUSSION

Hypothesis 1 of the present study predicted that age would be significantly associated with a decline in measures of processing speed, executive functioning, and memory. This hypothesis was supported. As predicted, age was correlated with measures of processing speed, the Symbol Digit Modalities Test (SDMT) and the Trailmaking Test (TMT) Part A, such that increasing age was associated with slower performance. This finding is consistent with previous research (Cerella, 1990; Hartley, 1986, 1993; Hultsch, Hertzog, & Dixon, 1990; Kail & Salthouse, 1994; Salthouse 1985a, 1992; Schaie, 1989, 1994; Van Gorp, Satz, & Mitrushina, 1990) and demonstrates that normal aging is indeed associated with a general slowing of cognitive processing. This finding lends support to proponents of the general slowing hypothesis (Nettelbeck and Rabbitt, 1992) who argue that the decrease in performance demonstrated by older adults on a variety of cognitive tasks is related, in part, to a general decrease in processing rate with age. It is also important to point out that the size of the correlation between age and decreased processing speed in the present study is consistent with other research. Salthouse (1985) has demonstrated that the median correlation between age and measures of speed across a wide range of behavioral activities is .45. The correlation between age and processing speed efficiency in the present study was .445 ($p<.01$) at Time 1 and .489 ($p<.01$) at Time 2. Furthermore, when entered as a covariate in the repeated measures ANCOVA analysis, age accounted for 11.0% ($p<.05$) of the variance in processing speed decline between Time 1 and Time 2.

As predicted, age was also associated with measures of executive functioning, including the Wisconsin Card Sorting Test (WCST), the Trail Making Test (TMT) Part

B, and the Stroop Test (ST). This is consistent with a vast amount of research indicating that executive abilities deteriorate with age (Ardila & Rosselli, 1989; Barr & Giambra, 1990; Burke, 1972; Daigneault, Braun, & Whitaker, 1992; Haaland et al, 1987; Salthouse & Mitchell, 1990; Shimamura & Jurica, 1994; Pierce et al, 1989; Whelihan & Lesher, 1985). When entered as a covariate in the repeated measures ANCOVA analysis, age accounted for 25.6% ($p<.01$) of the variance in executive functioning decline between time points.

Finally, this study was successful in demonstrating a significant association between age and decline in overall memory functioning. As predicted, age was associated with a decrement in memory performance on the California Verbal Learning Test (CVLT) such that increasing age was associated with a decline in both free recall ability and the use of appropriate encoding strategies to learning new information (i.e., reduced semantic clustering). This finding is consistent with an extensive amount of empirical work suggesting that memory functions decline with age (Backman & Forsell, 1994; Gainotti & Marra, 1994; Geffen et al., 1993; Loewenstein et al., 1991; O'Hara et al., 1986; Rohling & Scogin, 1993). In the current study, repeated measures ANCOVA analyses revealed that age predicted 9.0% ($p<.05$) and 9.4% ($p<.05$) of the variance in free recall memory and semantic clustering, respectively.

Hypotheses 2 and 3 predicted that, like age, depression would be predictive of cognitive decline in the areas of processing speed, executive functioning, and memory. Although there has been very little research on the long-term impact of depression on cognitive abilities, single episodes of depression have been shown to have negative effects on many cognitive functions (Backman & Forsell, 1994; Calgiuri & Ellwanger,

2000; Emery & Breslau, 1989; Gainotti & Marra, 1994; Geffen et al., 1993; Hart et al., 1987; LaRue et al., 1992; King, et al., 1991; Sobin & Sackheim, 1997). It stands to reason that recurrent or persisting depressive symptomatology may have even greater deleterious effects on processing efficiency and overall cognitive functioning, especially in an older adult population which has already suffered cognitive decline associated with increased age. However, results from the current study failed to support such a hypothesis. More specifically, scores on the BDI and GDS did not result in statistically significant covariate effects in repeated measures ANCOVA analyses in which processing speed, executive functioning, or memory were entered as dependent variables. Therefore, higher levels of depressive symptomatology were not predictive of cognitive decline above and beyond that already accounted for by age alone. Although it was hypothesized that depression would be identified as a risk factor for cognitive decline in old age, these results were not all-together surprising given the conflicting findings with respect to these variables in the literature (Bieliauskas et al., 1991; Geffen et al., 1993; Hart et al., 1987; Niederehe & Camp, 1985; Popkin et al., 1982; Williams et al., 1987). Taking into account results from the current study, it would appear that symptoms of depression do not, in fact, contribute to age-related cognitive decline in older adults.

Another possible explanation for the lack of association between depression scores and cognitive decline in the present study may be related to restricted variance among the independent variables. It is important to keep in mind that the group of subjects used in this investigation was not a clinical sample. Although a range of depressive symptoms was detected by both the BDI and GDS for subjects across time points, the average level of depression as reported by participants was actually quite low

overall. More specifically, average depression scores at Time 1 and Time 2 were consistent with only mild levels of depressive symptomatology according to the established criteria for each self-report questionnaire. Perhaps if the sample had included a greater number of individuals suffering from more severe depression, the increased variability would have made it easier to detect differences in cognitive decline among the participants. In a similar vein, it is worth noting that the sample as a whole was generally high functioning and may therefore have encompassed selection biases not well controlled for in this study. For example, the sample was a fairly homogeneous group of older adults with higher than average education (M=17.22, SD=2.47), IQ scores (M=111.95, SD=5.8), and occupational attainment (i.e., 84.2% of subjects were at or above the managerial level). This lack of variation in the sample may have contributed to an attenuation of the correlations between the variables, making it difficult to detect differences in the ANCOVA analyses. Therefore, future research that investigates the relationship between depression and cognitive functioning in older adults should make attempts to include a more diverse sample.

A third explanation for the lack of association between depression and cognitive functioning may have to do with inadequate power. A power analysis was conducted prior to examining the data. This analyses revealed that the present sample size of 57 subjects was sufficient for detecting medium and large effect sizes, .25 and .40 respectively (Cohen, 1992), but was inadequate for effects smaller than .10. In order to detect smaller effects, a sample size of 322 subjects would be needed (G-Power; Faul & Erdfelder, 1992). Therefore, it may be that depression indeed has a significant, but

relatively small, impact on cognitive functioning in older adults but the present sample size was inadequately powerful to detect such an effect.

Hypothesis 4 of the present study stated that trait anxiety would be predictive of cognitive decline above and beyond that of age alone in the areas of memory, speed of processing, and executive functioning. This hypothesis was partially supported as trait anxiety was indeed found to impact the rate of decline in memory and executive functioning, but was not shown to have an effect on processing efficiency. More specifically, scores on the State-Trait Anxiety Inventory (STAI) resulted in statistically significant covariate effects in ANCOVA analyses in which memory and executive functioning were entered as dependent variables, while scores on the STAI failed to reach significance in the model in which speed of processing scores were used as dependent measures. Trait anxiety scores at Time 1 were found to predict 9.9% ($p<.05$) of the variance in free recall memory, 8.2% ($p<.05$) of the variance in semantic clustering, and 8.4% ($p<.05$) of the variance in executive functioning. At Time 2, anxiety scores accounted for 6.2% ($p<.05$) of the variance in free recall memory and 5.5% of the variance in executive functioning performance. Although Time 2 scores on the STAI failed to reach significance in predicting decline in semantic clustering, the covariate effect came very close to the .05 level (Wilks' =.939, F(1, 53)=1.997, p=.057, partial 2=.061). Overall, these findings are consistent with a small, but growing, literature demonstrating that trait anxiety is associated with poorer verbal learning (Deptula, Singh, & Pomara, 1993; Paterniti, Dufouil, Bisserbe, & Alperovitch, 1999; Whitbourne, 1986) and executive dysfunction (Cohen et al, 1980). The fact that the current study failed to show an association between anxiety and speed of processing, while unexpected, is

consistent with at least one study currently published in the literature (Schultz et al., 1980). Taken as a whole, these data suggest a significant relationship between trait anxiety and cognitive decline and provide support that, like age, anxiety-proneness is a risk factor for intellectual deterioration. In other words, higher levels of trait anxiety are related to accelerated rates of cognitive decline relative to those who are less anxious.

To account for these findings, researchers have proposed that heightened anxiety restricts an individual's ability to efficiently process relevant information. This explanation has been articulated most clearly by Eysenck and Calvo (1992) in descriptions of their processing efficiency theory. This theory predicts that, especially in stressful conditions or in the case of high demands on information processing, high trait-anxious individuals are at a disadvantage. An excess of worries and other anxious thoughts preempt part of the processing resources they would otherwise have available and therefore negatively impact accuracy of performance. Given that storage and processing resources are presumed to be limited, the "space" taken up by anxiety results in reduced capacity for processing new information. Furthermore, because older age is already associated with reduced working memory and processing resources, this population may be more vulnerable than younger adults to the effects of anxiety across multiple cognitive domains. It may be that the elderly have less reserve capacity to compensate for the adverse effects of anxiety than the young. In particular, older adults with higher levels of anxiety would be expected to demonstrate poorer performance on tasks involving memory and executive functioning skills, as was shown in the present investigation. Since neuropsychological tests designed to measure psychomotor speed do not generally make high demands on information processing (i.e., the tests rely on more

familiar, over-learned skills), this theory may help explain why anxiety had no effect on declines in processing speed in the current sample. For example, because a task like connecting dots on the Trail Making Test requires very little working memory capacity to complete, the task can still be done adequately even in cases where anxiety has occupied much of the "space" required for information processing. Taken together, findings from the current study are consistent with research suggesting that working memory and initial encoding of information are the most affected by anxiety, while more automatic tasks are among the least affected (Bradley, Mogg, & Williams, 1994; Darke, 1988; Hill & Vandervoort, 1992; MacLeon & Donnellan, 1993).

While the processing efficiency theory (Eysenck and Calvo, 1992) offers an explanation as to why anxiety may interfere with working memory processes in older adulthood, there is at least one other theory that has been proposed to specifically address the negative impact of anxiety on executive functioning skills. Some writers in the field have suggested that the anxious elderly may be at a particular disadvantage when it comes to solving abstract, novel problems because of an increase in cognitive rigidity that may occur (Kuhlen, 1964). Rigidity has been defined by Kuhlen as the adherence to a previously established behavior pattern in situations in which response change would be optimal. Flexibility is characterized as the ability to appropriately modify established behaviors in response to situational demands. Kuhlen (1964) proposed the idea that increasing anxiety leads to cognitive ridigity in the elderly, which in turn may negatively impact their ability to make use of novel problem-solving skills when performing tasks of executive functioning. While this theory has not been well examined in the literature to date, it may well help to account for the finding that trait anxious individuals suffer a

greater loss in executive functioning abilities over time relative to those who are less anxious.

In addition to the theories set forth by Eysenck and Calvo (1992) and Kuhlen (1964), it is important to consider that the cognitive impairment resulting from trait anxiety in some individuals may also be secondary to CNS dysfunction. In other words, the neuropsychological impairments of highly trait anxious subjects may reflect subtle, undetected pathological processes that profoundly affect brain integrity. For example, an emerging body of research in the neuroscience literature describes how the neuroendocrine system responds to anxiety by stimulating the hypothalamic-pituitary-adrenal (HPA) axis to release corticosteroids, namely cortisol, which has been known for some time to affect cognitive function (de Quervain, Roozendaal, & McGaugh, 1998; Kirschbaum, Wolf, May, Whippich, & Hellhammer, 1996; Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; Newcomer, Selke, Melson, Hershey, & Craft, 1999). In fact, Mitchell (1995) in a careful review of 17 studies in which cognitive impairment was examined in relation to measures of HPA axis activity, concluded that a significant correlation between these two variables was found consistently. It should be noted that the specific deficits observed with increased levels of cortisol are quite similar to those noted in the context of normal aging (Newcomer et al, 1999). That is, subjects appear to direct more attention to irrelevant stimuli, which results in decreased processing of relevant information and poorer performance. Increased cortisol levels have been shown to cause particular deficits in memory performance (Lupien et al., 1994; Seeman, McEwen, Singer, Albert, & Rowe, 1997; Squire, 1992) and executive functioning (Carpenter & Gruen, 1982).

The precise mechanisms by which corticosteroids may alter cognition are unknown at this time. However, a few studies have shown that cortisol reduces the average evoked potential response to relevant, but not irrelevant, stimuli (Kopell, Wittner & Lunde, 1980; Sarrieau, Dussaillant & Agid, 1986). This in turn causes impairment in selective attention by increasing the level of inappropriate "noise" in the system (e.g, irrelevant stimuli). Interestingly, numerous studies have also shown that increased levels of cortisol may impair cognitive functioning as a result of toxic effects to a region of the brain that controls memory function, namely the hippocampus. The hippocampus appears to be particularly sensitive to the effects of increased cortisol because it contains the highest concentration of corticosteroid binding sites in the brain (McEwen, Davis & Parsons, 1997; Sarrieau et al., 1986). Corticosteroids are reportedly damaging to hippocampal cells because they act to inhibit regional cerebral glucose metabolism (de Leon, McRae, Rusinek, Convit, & De Santi, 1997) and are associated with hippocampal atrophy (Lupien et al., 1998; Sapolsky, 1996). By adversely affecting glucose uptake, cortisol increases the neuronal vulnerability of hippocampal cells to a variety of neurotoxic insults. In fact, corticosteroid levels appear to predict the magnitude of hippocampal neuronal loss as well as the extent of cognitive impairments (Landfield, 1991).

Interestingly, the effects of corticosteroids on neuronal activity tend to observe an inverted U-shaped curve, with extremely low and high cortisol levels suppressing neuronal activity (Joels & de Kloet, 1994). This seems to fit nicely with the Yerkes-Dodson theory (1908) which describes the now classic, U-shaped pattern of association between arousal and cognitive performance. According to this well-known theory, low

and high levels of arousal lead to poor performance, whereas mid-range arousal promotes optimal cognitive functioning. The biological underpinnings of the Yerkes-Dodson theory may therefore be explained, at least in part, by the effects of corticosteroids. The research reviewed above suggests that individuals who are anxiety-prone tend to demonstrate higher levels of arousal, which is accompanied by increased release of cortisol in the brain. Prolonged cortisol release is in turn associated with neurotoxic effects, neuronal loss, and cognitive impairment. Clearly, the accumulation of research in this area strongly suggests that increased HPA activity in the brains of anxiety-prone individuals is related to accelerated rates of cognitive decline. While cortisol levels were not measured in the current study, it would make sense that such neuroendocrine responses to increased levels of anxiety may have contributed to the cognitive deterioration seen in the areas of memory and executive functioning.

Hypothesis 5 stated that measures of depression and anxiety would be predictive of cognitive decline when examined in relation to particular domains of functioning (e.g., memory, executive functioning, speed of processing), but would not predict decline on a measure of gross, non-specific cognition such as the MMSE. Repeated measures ANCOVA analyses revealed that this hypothesis was supported, as anxiety and depression did not result in statistically significant covariate effects when MMSE scores were used as dependent measures. This finding is important because it suggests that measures of global cognitive functioning, like the MMSE, are not sensitive enough to pick up changes in cognition that are negatively impacted by chronic affective disturbance. In the present study, trait anxiety was clearly demonstrated to have an adverse affect on cognition, with higher trait anxiety predicting declines in memory and

executive functioning. However, had this investigation not used such domain-specific measures of neuropsychological functioning, this finding would not have been detected.

A review of the literature suggests that many researchers in the field are still using the MMSE as the primary measure of cognition in studies examining affective disturbances among older adults. While studies that use such non-specific measures of cognitive functioning nevertheless add to our understanding of the association between these variables, researchers need to know which cognitive processes are most readily influenced by anxiety and depression and which are left relatively undisturbed. It seems that so far, investigators have not yet well connected the symptoms of anxiety and depression to specific types of neuropsychological functioning, such as memory, processing speed, or executive skills. Rather, these variables are most often discussed in the literature as nonspecific mediators of age-related cognitive decline. Presumably the effects of anxiety and depression are not ubiquitous with respect to cognition, however little empirical work has been conducted to investigate the extent to which these variables differentially affect specific domains of brain functioning. Findings from the current study suggest that future research in this area should examine cognition in a way that will allow these distinctions to be made clear.

While the results from this investigation suggest that depression and anxiety do not predict decline on measures of gross cognitive functioning such as the MMSE, a few alternative explanations must also be considered. First, the lack of significance associated with anxiety/depression and the MMSE may be related to inadequate statistical power. A power analysis conducted prior to examining the data revealed that the present sample size of 57 subjects was sufficient for detecting medium and large

effect sizes, .25 and .40 respectively (Cohen, 1992), but was inadequate for effects smaller than .10. In order to detect smaller effects, a sample size of 322 subjects would have been needed (G-Power; Faul & Erdfelder, 1992). Therefore, it may be that anxiety and depression indeed have a significant, but relatively small, impact on global cognitive functioning that could not be detected with a sample size of 57 subjects.

A third explanation for the lack of association between mood measures and global cognitive functioning may have to do with restricted variance among MMSE scores. In order to ensure that all participants were healthy and functioning at a level consistent with normal aging, subjects were excluded from participation in the study if their performance on the MMSE fell below the 23-point cutoff. Clearly, this procedure acted to restrict variance on this measure as those who performed the most poorly were not included in the analyses. It is possible that, if these subjects had been included in the study, the increased variance may have led to significant results. In a related vein, it should be noted that the change in MMSE scores between Time 1 and Time 2 was actually quite small overall (M=1.49 points). It may simply be that this small decrement represents unsystematic change that could not be predicted in the analyses.

Methodological Considerations

This investigation sought to add to the existing literature on the effects of anxiety and depression on cognitive functioning in older adults by improving upon the methodological and theoretical shortcomings of earlier studies. This was accomplished in the following important ways: (1) by examining the variables in a within-subjects longitudinal design, thereby allowing for the direct exploration of the *long-term* effects of anxiety and depression on cognitive functioning (2) controlling for cohort effects, which

has not been accomplished in earlier investigations using cross-sectional data (3) using neuropsychological measures that assessed the specific cognitive functions of memory, speed of processing, and executive functioning as opposed to measures of global, non-specific types of cognition and by (4) employing statistical analyses using continuous variables of anxiety, depression, and cognitive functioning which avoided the distortion that can occur by falsely dichotomizing these constructs.

Despite these improvements in methodology, important shortcomings of this investigation must also be noted. First, operationally defining the constructs of interest poses an exceptionally troublesome limitation in the research of neuropsychological test performance. What is the most accurate way of assessing constructs such as executive functioning or speed of processing? Unfortunately, experts in the field disagree as to what constitutes an accurate or appropriate definition of such cognitive abilities and often debate about which instruments should be used to adequately measure them. For example, the on-going dispute between Parkin (1998) and Baddeley (1998) regarding the construct of executive functioning poses empirical questions that need to be considered when designing any study involving the measurement of such abilities. These disagreements in the field highlight the need to use multiple measures when attempting to assess constructs of the sort examined in the present study. Indeed, one strength of this study is that three instruments were used to measure executive functioning, including the Wisconsin Card Sorting Test (WCST), Trail Making Test (TMT) Part B, and the Stroop Test (ST). Likewise, two measures were used to assess speed of processing, including the Symbol Digit Modalities Test (SDMT) and the Trail Making Test (TMT) Part A. However, it should be noted that a limitation exists with regard to the measurement of

memory, as only one instrument was utilized to assess this construct in the present study. Future research should make every attempt possible to include more than one measure of each variable. While the use of multiple measures does not completely solve the issue of construct validity, it can be argued that doing so contributes to a more accurate estimation of the constructs of interest.

A second limitation of this study is concerned with the generalizability of the research findings. The sample consisted of fairly well-functioning, normal, older adults and was not particularly representative of the socioeconomic, educational, or cultural variation in the general population. While there is undoubtedly interest in understanding the processes of normal aging as it pertains to such individuals, it is important to point out that the conclusions drawn from the present study may not generalize to the larger population of older adults.

Future Directions

As already mentioned, future research would do well to include samples of individuals that are more heterogeneous with respect to socioeconomic, educational, and cultural characteristics and therefore more representative of the population as a whole. A diverse sample not only helps to avoid the problems associated with restricted variance and attenuated correlations, it also increases the likelihood that results will be generalizable to a more extensive population of older adults.

In addition, future research would likely benefit from the inclusion of a younger comparison group. The comparison of extreme age groups, as opposed to examining data solely from a group of older adults, would be more likely to pick up on and emphasize age-related decline in cognitive performance. Including a younger comparison group

would serve to increase the variation in performance assessed and, in turn, more clearly highlight age-related differences in the areas of memory, executive functioning, and speed of processing.

Future research may also consider the inclusion of both normal, healthy adults as well as those who are functioning at a level inconsistent with normal aging, such as those with Alzheimer's disease or other dementing illnesses. This would not only allow an investigation of the relative impact of anxiety and depression on the processes of normal aging, but could also assess their impact on the progression of pathological diseases processes in later life. As the present study points out, anxiety accounts for a significant amount of variance in cognitive decline among older adults and may therefore contribute to decline associated with certain types of dementia. Such empirical questions could not be answered by this study and warrant further investigation.

Finally, results from this study suggest that even subclinical levels of anxiety may interfere with cognitive functioning in older adults. Clearly, this variable can no longer be dismissed as unimportant or irrelevant in an older adult population and must be controlled for in future research. The creation of new diagnostic tools as well as better treatment methods must also be encouraged. Likewise, more research is needed to investigate whether or not behavioral and/or pharmacological interventions aimed at reducing anxiety will be capable of attenuating the negative effects of this variable on cognitive decline in an older adult population.

Summary

It appears that age is significantly associated with cognitive deterioration in the areas of memory, executive functioning, and speed of processing. Contrary to

expectation, depressive symptomatology did not contribute to degeneration in these domains and is therefore not considered a risk factor for cognitive decline in old age. However, trait anxiety was found to predict a significant and unique amount of the variance in reduction of memory and executive functioning skills. As a result, anxiety-proneness appears to be related to accelerated rates of cognitive decline. These findings highlight the need for future research in this area in order to more clearly elucidate the impact of such variables on the process of normal aging.

APPENDIX

Table 1. Sample characteristics at Time 1 and Time 2.

Participant Variable		Value	N	
			Valid	Missing
Gender			57	0
	Male	28 (49.1%)		
	Female	29 (50.9%)		
Estimated Premorbid IQ		M=111.95 (SD=5.8)	57	0
Years of Education		M=17.22 (SD=2.47)	57	0
Primary Occupation			57	0
	Not in Labor Force	3 (5.3%)		
	Unskilled Labor	0		
	Semi-skilled Labor	2 (3.5%)		
	Skilled Labor	4 (7.0%)		
	Managerial	18 (31.6%)		
	Professional	30 (52.6%)		
Age			57	0
	Time 1	M 69.86 (SD=6.36)		
	Time 2	M=75.09 (SD=6.47)		
Marital Status				
Time 1	Never Married	3 (5.4%)	54	3
	Married	31 (55.4%)		
	Widowed	13 (23.2%)		
	Separated	0		
	Divorced	7 (12.5%)		
Time 2	Never Married	2 (3.5%)	57	0
	Married	34 (59.6%)		
	Widowed	16 (28.1%)		
	Separated	1 (1.8%)		
	Divorced	4 (7.0%)		
Health Rating				
Time 1	Excellent	18 (32.1%)	54	3
	Good	31 (55.4%)		
	Fair	4 (7.1%)		
	Poor	1 (1.8%)		
Time 2	Excellent	7 (12.3%)	57	0
	Good	41 (71.9%)		
	Fair	6 (10.5%)		
	Poor	3 (5.3%)		

Table 2. Means and standard deviations for subjects' Time 1 performance on all dependent measures, including the MMSE, CVLT, SDMT, TMT, WCST, and ST.

Dependent Measure		Time 1 Mean (SD)	N	
			Valid	Missing
MMSE	Total Score[1]	28.50 (1.54)	57	0
CVLT	Free Recall Total[1]	44.30 (9.83)	54	3
	Semantic Clustering Score[1]	2.13 (0.79)	54	3
SDMT	Total Number Correct, Oral Trial[1]	53.19 (12.51)	55	2
TMT, Part A	Total Time in Seconds[2]	34.36 (12.32)	57	0
WCST	Perseverative Errors[2]	16.19 (12.05)	52	5
ST	Interference Score[1]	-4.39 (7.44)	54	3
TMT, Part B	Difference Score in Seconds[2]	53.95 (31.08)	57	0

[1] Higher score indicates better performance
[2] Lower score indicates better performance

Table 3. Means and standard deviations for subjects' Time 2 performance on all dependent measures, including the MMSE, CVLT, SDMT, TMT, WCST, and ST.

Dependent Measure		Time 2 Mean (SD)	N	
			Valid	Missing
MMSE	Total Score[1]	27.01 (2.15)	56	1
CVLT	Free Recall Total[1]	41.04 (13.40)	55	2
	Semantic Clustering Score[1]	1.61 (0.96)	55	2
SDMT	Total Number Correct, Oral Trial[1]	49.72 (14.00)	52	5
TMT, Part A	Total Time in Seconds[2]	48.99 (38.60)	55	2
WCST	Perseverative Errors[2]	18.22 (13.22)	54	3
ST	Interference Score[1]	-6.75 (6.56)	57	0
TMT, Part B	Difference Score in Seconds[2]	65.47 (33.97)	56	1

[1] Higher score indicates better performance
[2] Lower score indicates better performance

Table 4. Correlation matrix between age and Time 1 measures of global cognition, processing speed, executive functioning, and memory.

	Age	MMSE	SDMT	TMT (A)	WCST	STROOP	TMT (B)	Recall	Semantic
Age	1.00	-.351**	-.489**	.576**	.303*	-.475**	.332*	-.432**	-.300*
Global Cognition:									
MMSE	-.351**	1.00	.545	-.390*	-398**	.300*	-.333*	.376**	.248
Processing Speed:									
SDMT	-.489**	.545	1.00	-.524**	-398**	.270	-.447*	.372**	.183
TMT (A)	.576**	-.390*	-.524**	1.00	.210	-.258	.415**	-.422**	-.140
Executive Functioning:									
WCST	.303*	-.336*	-.398**	.210	1.00	-.336*	.644*	-.238	-.312*
STROOP	-.475**	.300*	.270	-.258	-.336*	1.00	-.390**	.285*	.259
TMT (B)	.332*	-.333*	-.447*	.415**	.644*	-.390**	1.00	-.320*	-.204
Memory:									
Recall	-.432**	.376**	.372**	-.422**	-.238	.285*	-.320*	1.00	.633**
Semantic	-.300*	.248	.183	-.140	-.312*	.259	-.204	.633**	1.00

Note. Pearson Product Moment Correlations by listwise comparison N=57. * p value < .05; ** p value < .01

Table 5. Correlation matrix between age and Time 2 measures of global cognition, processing speed, executive functioning, and memory.

	Age	MMSE	SDMT	TMT (A)	WCST	STROOP	TMT (B)	Recall	Semantic
Age	1.00	-.384**	-.445**	.292*	.458**	-.400**	.594**	-.518**	-.399*
Global Cognition:									
MMSE	-.384**	1.00	.443	-.783**	-.269*	.411**	-.305*	.537**	.176
Processing Speed:									
SDMT	-.445**	.443	1.00	-.549**	-467**	.254	-.425*	.499**	.167
TMT (A)	.292*	-.783**	-.549**	1.00	.257	-.231	.760**	-.468**	-.095
Executive Functioning:									
WCST	.458**	-.269*	-.467**	.257	1.00	-.442*	.691*	-.416**	-.147
STROOP	-.400**	.411**	.254	-.231	-.442*	1.00	-.485**	.406**	.128
TMT (B)	.594**	-.305*	-.425*	.760**	.691*	-.485**	1.00	-.406**	-.214
Memory:									
Recall	-.518**	.537**	.499**	-.468**	-416**	.406**	-.406**	1.00	.772**
Semantic	-.399*	.176	.167	-.095	-.147	.128	-.214	.772**	1.00

Note. Pearson Product Moment Correlations by listwise comparison N=57. * p value < .05; ** p value < .01

Table 6. Frequency of depression at Time 1 and Time 2 as measured by the BDI and GDS.

Level of Depression	Time 1	Time 2
BDI:		
Normal (0-9)	27	29
Mild/Moderate (10-19)	26	23
Moderate/Severe (20-63)	4	5
GDS:		
Normal (0-9)	20	17
Mild/Moderate (10-19)	28	33
Moderate/Severe (20-30)	9	7

Table 7. Correlation matrix between age, executive functioning (EF) factor, and processing speed (PS) factor at Time 1.

	Age	EF Factor	PS Factor
Age	1.0	.483**	.610**
EF Factor	.483**	1.0	.573**
PS Factor	.610**	.573**	1.0

Table 8. Correlation matrix between age, executive functioning (EF) factor, and processing speed (PS) factor at Time 2.

	Age	EF Factor	PS Factor
Age	1.0	.544*	.492**
EF Factor	.544**	1.0	.574**
PS Factor	.492**	.574**	1.0

Table 9. Repeated measures ANCOVA results showing covariate effects of age and BDI depression scores on free recall memory, semantic clustering, processing speed, and executive functioning.

DV	Covariate	F	Wilks'	*p* Value	Partial Eta Squared
Free Recall Memory		6.580	.803	**.022***	.197
	Age	5.279	.908	**.026***	.092
	BDI Time 1	.005	1.000	.944	.000
	BDI Time 2	.623	.988	.434	.012
Semantic Clustering		2.789	.899	**.037***	.101
	Age	2.582	.922	**.046***	.078
	BDI Time 1	.025	1.000	.876	.000
	BDI Time 2	1.766	.967	.190	.033
Processing Speed		6.998	.876	**.009****	.124
	Age	6.651	.883	**.012***	.117
	BDI Time 1	.467	.991	.498	.009
	BDI Time 2	.044	.999	.834	.001
Executive Functioning		6.021	.752	**.019***	.248
	Age	4.192	.805	**.034***	.195
	BDI Time 1	.557	.989	.459	.011
	BDI Time 2	.909	.983	.345	.017

Note: * $p<.05$ **$p<.01$

Table 10. Repeated measures ANCOVA results showing covariate effects of age and GDS depression scores on free recall memory, semantic clustering, processing speed, and executive functioning.

DV	Covariate	F	Wilks'	*p* Value	Partial Eta Squared
Free Recall Memory		6.407	.809	**.024***	.191
	Age	5.222	.911	**.027***	.089
	GDS Time 1	2.058	.962	.157	.038
	GDS Time 2	2.666	.951	.109	.049
Semantic Clustering		3.014	.880	**.027***	.120
	Age	2.792	.915	**.036***	.085
	GDS Time 1	1.181	.978	.282	.022
	GDS Time 2	2.355	.957	.131	.043
Processing Speed		3.235	.908	**.025***	.092
	Age	2.858	.926	**.037***	.074
	GDS Time 1	1.660	.969	.203	.031
	GDS Time 2	.029	.999	.866	.001
Executive Functioning		5.954	.761	**.020***	.239
	Age	4.330	.789	**.031***	.202
	GDS Time 1	2.851	.948	.097	.052
	GDS Time 2	2.389	.956	.128	.044

Note: * $p<.05$ **$p<.01$

Table 11. Repeated measures ANCOVA results showing covariate effects of age and trait anxiety on free recall memory, semantic clustering, processing speed, and executive functioning.

DV	Covariate	F	Wilks'	*p* Value	Partial Eta Squared
Free Recall Memory		7.049	.881	**.010****	.119
	Age	7.449	.875	**.009****	.125
	STAI Time 1	6.271	.901	**.025***	.099
	STAI Time 2	4.953	.938	**.039***	.062
Semantic Clustering		2.615	.901	**.040***	.099
	Age	2.580	.904	**.047***	.096
	STAI Time 1	2.019	.918	**.051***	.082
	STAI Time 2	1.997	.939	.057	.061
Processing Speed		6.944	.882	**.011***	.118
	Age	6.602	.887	**.013***	.113
	STAI Time 1	.727	.986	.398	.014
	STAI Time 2	.463	.991	.499	.009
Executive Functioning		4.610	.787	**.027***	.213
	Age	4.534	.791	**.028***	.209
	STAI Time 1	3.682	.916	**.041***	.084
	STAI Time 2	3.125	.945	**.052***	.055

Note: * $p<.05$ **$p<.01$

Table 12. Repeated measures ANCOVA results showing covariate effects of age, BDI depression, GDS depression, and trait anxiety on MMSE scores.

DV	Covariate	F	Wilks'	*p* Value	Partial Eta Squared
MMSE		3.105	.838	**.043***	.162
	Age	2.649	.851	**.051***	.149
	BDI Time 1	.503	.990	.481	.010
	BDI Time 2	.473	.991	.495	.009
		3.619	.819	**.036***	.181
	Age	3.502	.824	**.037***	.176
	GDS Time 1	.214	.996	.646	.004
	GDS Time 2	.698	.987	.407	.013
		4.973	.709	**.023***	.291
	Age	4.448	.813	**.030***	.187
	STAI Time 1	1.312	.975	.257	.025
	STAI Time 2	.998	.981	.322	.019

Note: * $p<.05$ ** $p<.01$

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