EFFECTS OF IMPROVED PHYSICAL FITNESS ON COGNITIVE/PSYCHOLOGICAL FUNCTIONING IN COMMUNITY-DWELLING, SEDENTARY MIDDLE-AGED AND OLDER ADULTS

By

ADRIENNE T. AIKEN MORGAN

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To my grandparents and great-grandparents, all of whom have inspired me to pursue a career in minority cognitive aging research.

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Adrienne T. Aiken Morgan

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A growing corpus of research suggests that physical exercise can improve cognition, particularly executive functioning, in older adults. However, limitations of existing research have included (a) insufficient attention to the recruitment of sedentary older adults (who would most likely benefit from exercise interventions); (b) insufficient guidance in test selection drawing on neuropsychological theory and practice; and (c) failure to elucidate the multiple pathways or components of exercise effects on cognition. The current study sought to better clarify these routes to cognitive improvement via (a) assessment of both potential physical fitness and psychosocial mediators of exercise effects on cognition, and (b) inclusion of a control group that received a comparable psychoeducational intervention, matched in study contact hours and study-related non-exercise activities, but which did not receive a physical exercise enhancement intervention. Two randomized groups of 35 (control) and 34 (invention) adults aged 50 years and older were recruited from the Gainesville/Alachua County, Florida region. Both groups underwent pre- and post-intervention cognitive, fitness, and psychosocial/socio-emotional assessment. The exercise promotion intervention group received 16 weeks of intervention (health and fitness education, weekly peer motivational coaching and group support,

etc.) in small groups with a peer mentor, while a control/comparison group received 16 weeks of "health hygiene" instruction, consisting of 16 weeks of education about general health conditions in aging (also in small groups with a peer mentor). Repeated-measures MANOVA indicated no significant between-subjects effect of the intervention ($p > .05$). There were multivariate within-subjects effects for occasion; however, there were no study group-by-occasion interaction effects. Follow-up univariate analyses revealed within-subjects effects for 9 cognitive variables. There was a modest study group-by-occasion interaction on the COWA test, with intervention group participants improving significantly more across testing occasions. Next, exploratory age group analyses revealed significant multivariate between-subjects effects of age on executive measures only. Follow-up univariate analyses demonstrated age group effects for 4 cognitive variables. For each cognitive measure, younger participants performed significantly better than their older counterparts. In addition, there were study group-by-occasion interaction effects that suggested younger control participants performed better on the One-Back Mean RT SD task, while older intervention group participants performed significantly better on LM Delayed Recall. A three-way interaction suggested that younger intervention group participants improved significantly more over time than younger controls and older participants on the Trails B test. Finally, there was modest, but inconsistent, evidence for correlated change between cognitive, physical fitness/activity, and psychosocial variables. These findings lend some support to the previous literature suggesting the benefits of physical fitness/exercise improvements on cognitive function and the frontal aging hypothesis (West, 1996; Zimmerman et al., 2006). Future research should explore the benefits of physical and cognitive interventions in diverse samples of middle-aged and older individuals. Future studies should also explore the use of alternate cognitive and physical fitness assessment tools in elucidating the cognition-fitness relationship.

CHAPTER 1
STATEMENT OF THE PROBLEM

The present study sought to extend the growing research literature that has shown physical exercise can improve cognition, particularly executive functioning, in older adults. Increased physical activity has been shown to be related to many positive outcomes, including lower mortality and morbidity rates, lower cardiac risks, improved psychological well-being (lower rates of depression), improved aerobic capacity, and improved functional capacity (e.g., McAuley et al., 2004). There is also evidence that cognitive function may be improved with improved physical fitness through increased exercise behaviors (e.g., Colcombe & Kramer 2003). Thus, there are several premises guiding the current investigation:

- Cognition in later life has plasticity, and can be improved (e.g., Jobe et al., Ball et al.)
- In addition to conventional behavioral interventions, physical exercise interventions have also been shown to be effective in improving cognition (e.g. Colcombe & Kramer, 2003)
- One route by which physical exercise interventions may improve cognition is by improving cardiovascular fitness (e.g., Haskell et al., 1992)
- Physical exercise interventions improve cognitive performance (McAuley et al., 2004).
- Physical exercise interventions may improve cognition by increasing complex activity/social participation, and by improving affective functioning (e.g., Brown, 1992; McAuley, 1993).
- Depression is another correlate of late-life cognition. Both aging and depression are independently associated with difficulties in executive functioning and high-level cognitive control processes that mediate other aspects of cognition (e.g. Hartlage, et al., 1993).
- Exercise may disproportionately affect executive control processes (Colcombe & Kramer, 2003).

A major goal of this study was to elucidate several potential pathways or components of exercise effects on cognition, separating psychosocial and activity-engagement from physical fitness changes as possible mediators of these effects. The present study sought to address this goal by examining the following specific aims:

- To investigate whether, relative to matched non-exercising control participants, sedentary adults receiving a physical exercise promotion intervention experience improvements in the primary outcome of cognitive function (particularly executive control processes).

Hypothesis: As shown in previous studies, participants receiving the exercise intervention will show improved performance on cognitive measures, particularly those assessing executive control processes, relative to control participants.

- To determine the separate and joint roles of improvement in proximal outcomes (fitness, activity, and affect) in mediating exercise intervention effects on cognition.

Hypothesis: It is further expected that changes in measures of physical fitness, physical activity engagement, and emotional well-being mediates some or all of the intervention effect on cognitive change.

CHAPTER 2
REVIEW OF THE LITERATURE

Overview

The present investigation examined the effects of improved physical fitness on cognitive functioning in a group of adults, ages 50 and older. Thus, the review of the literature will first consider the evidence for normative and non-normative cognitive declines with aging, highlighting the cognitive continuum in later life. Evidence to date suggests aging disproportionately affects executive functions and processes, and the present literature review will discuss these findings. Next, the literature review will consider evidence for the plasticity of cognition in late life. Specifically, the review will consider research that has shown cognition may be improved through cognitive training interventions, as well as behavioral interventions. Such behavioral interventions include physical exercise interventions aimed at improving cognitive function through improved physical fitness.

One route by which physical exercise interventions may improve cognition is by improving cardiovascular fitness, and this literature will be discussed. Next, additional routes by which physical exercise interventions may improve cognition (i.e., via improving mood/affect, self-efficacy, and control beliefs) will also be explored. This review is intended to set the stage for the current study, which examined pre-post exercise intervention cognitive performance changes on selected neuropsychological measures. Additionally, this study empirically investigated whether measures of executive control processes were particularly sensitive to physical exercise intervention (as a recent meta-analysis has suggested), relative to other cognitive domains. This study also explored whether physical (fitness) and psychosocial variables (mood, affect, and self-efficacy) changed as a result of an exercise promotion intervention, and whether these helped explain any pre-post changes in cognition

Conceptual model

The current study sought to confirm the effects of exercise on cognition using a more individualized approach to exercise intervention than many previous studies (this is described in greater detail below). In addition to examining exercise effects on cognition, the study also explored several possible mediators of exercise effects, including physical factors (changes in physical fitness) and psychosocial factors (changes in self-perception, depression, well-being, and self-efficacy). Thus, the conceptual model below guides the current study (Figure 2-1). As noted above, exercising may also be construed as a form of complex activity, which may increase social engagement. Many previous studies have failed to control for these social participation components of exercise, using wait-list or no-contact control groups. The present study included a "health hygiene" control group, matched with experimental participants in study contact hours and interaction with study staff and other participants and in exposure to cognitive testing. These factors were designed to reduce the confounding role of social/complex activity participation as a plausible alternative explanation of exercise effects.

Significance

The proposed study sought to further the understanding of the relationship between cognition and exercise in a middle-aged and older adult sample. It further attempted to explore the unique and combined contributions of fitness and self-perception changes as mediators of exercise effects on cognition. This is unique in that, to our knowledge, few studies have attempted to examine both process and outcome exercise-related variables as routes to cognitive improvement in an older adult population. Furthermore, this study was thought to be the first in a line of future research aimed at exploring the effect of one potentially remediable antecedent of late life cognitive decline, physical fitness.

Continuum of Late Life Cognition

Late life cognitive function appears to represent a continuum, ranging from normative age-related declines in abilities to pathological cognitive deficits. Clinical and neuroanatomical evidence support such a continuum notion of normal and pathological cognitive aging; nevertheless, there are certain factors which exacerbate (and optimize) this cognitive aging process, such that a smooth, linear transition along all points of this continuum may not be observed for all individuals. Thus, some individuals may be at a lesser or greater risk for optimal cognitive outcomes in late life.

Normal cognitive aging has been characterized in a multidimensional and multidirectional way; with aging, there is evidence for gains and loses in cognitive function (Baltes et al., 1999). Evidence suggests a gradual decline in some cognitive areas, as well as stability in other cognitive functions. Specifically, there appears to be linear declines in "fluid" abilities as well as "crystallized" abilities. Fluid abilities refer to those that are necessary for processing and learning novel information and coping with new situations in the environment (i.e., abstract reasoning, processing speed, episodic memory processing). In contrast, crystallized abilities represent the knowledge garnered from a lifetime of experience (i.e., general intelligence, verbal skills, and semantic knowledge). Interestingly, it appears fluid abilities show a more steady and accelerated decline with aging, while there is some preservation and even perhaps gain in crystallized knowledge until late life, at which point there is accelerated decline in these abilities (around 75-80 years).

Drawing from the neuropsychological literature, there are several normal aging related declines observed. For instance, while memory declines are commonly cited, executive functions also have been found to be closely related to the aging process and sustained by specific brain regions (e.g., West, 1996; Greenwood, 2000; Charot & Feyereisen, 2005; Gunstad,

et al., 2006; Zimmerman et al., 2006). Executive functions, while not conceptualized the same, are similar to fluid abilities, in that such skills are important for the successful processing and learning of novel information and coping with new situations. Specifically, executive functions, governed by the frontal brain regions, generally consist of the capacities to engage successfully in independent, purposive, self-serving behavior (Lezak et al., 2004). These behaviors include formulating goals with regard for long-term consequences, generating multiple response alternatives, choosing and initiating goal-directed behaviors, self-monitoring the adequacy and correctness of the behavior, correcting and modifying behaviors when conditions changes, and persisting in the fact of distraction (Synder & Nussbaum, 1998). With specific regard to cognitive behaviors, executive functions refer to the strategies involved in approaching, planning, or carrying out cognitive tasks, or in monitoring of performance (Lezak et al., 2004). These cognitive components, often termed "executive control" functions, of executive functions are sub-served by the dorsolateral prefrontal cortical brain regions (Synder & Nussbaum, 1998). Examples of executive control functions include working memory, mental flexibility, maintenance of task set, and task-switching.

Next, evidence has suggested frontal lobe structure and (executive) function undergo disproportionate declines with aging (e.g., West, 1996; Greenwood, 2000; Charot & Feyereisen, 2005; Gunstad, et al., 2006; Zimmerman et al., 2006); this consistent finding has been termed the "frontal aging hypothesis." The frontal aging hypothesis, which is supported by many in the literature, states frontal-executive abilities seem to be disproportionately negatively affected by aging relative to non-frontal, more posterior brain mediated abilities (West, 1996; Greenwood, 2000). Nonetheless, while with normal aging, execution of executive functions becomes inefficient (e.g., Pfefferbaum, Adalstein, & Sullivan, 2005; Brickman et al., 2006; Nordahl et al.,

2006; Sullivan & Pfefferbaum, 2006), it is not impossible. Instead, it becomes necessary for recruitment of wider-spread brain networks to perform at the same level on executive tasks (i.e., HAROLD model, Cabeza, 2002; Cherry et al., 2005).

Study in this area has shifted to not only examine cortical, or gray matter, influences on executive function, but also supporting white matter regions (Pfefferbaum et al., 2005; Brickman et al., 2006; Nordahl et al., 2006; Sullivan & Pfefferbaum, 2006). In one study (Pfefferbaum et al., 2005) using diffusion tensor imaging (DTI), the integrity of white matter fiber microstructure in a group of healthy, highly educated young and older adults was examined. DTI results indicated that older adults showed greater frontal white matter loss (anisotropy) relative to younger subjects. There were no age-related differences found for posterior white matter regions. In general, normal age-related white matter loss features degradation of myelin and microtubules and number and length of myelinated fibers, particularly in the precentral gyrus and corpus callosum. In addition, thin fibers, which are greatest in number in the frontal lobes, seem to have a predilection for loss. Axon deletion seems to also be related to the white matter normal aging process. Further study has confirmed this age-associated decline in frontal white matter, and such decline has been shown to mediate declines in neuropsychological functioning in a more general fashion (Brickman et al., 2006; Nordahl et al., 2006).

Such cortical and subcortical declines with aging have implications for the cortical-subcortical circuitry that underlies executive control processing. Specifically, there are at least five parallel circuits that connect the frontal cortex to subcortical structures. The input segment of the basal ganglia (BG) is the striatum, which consists of the caudate, putamen, and nucleus accumbens. The striatum receives input from multiple cortical areas and projects through the globus pallidus and substantia nigra to the thalamus, ultimately closing the circuit back to the

area of the cortex from which it received (Alexander, DeLong, & Strick, 1986). One circuit relevant to the present study (the associative circuit, implicated in working memory function) originates in the dorsolateral prefrontal and lateral orbitofrontal cortex and includes the caudate nucleus, part of the putamen, the anterodorsal globus pallidus internus, the substantia nigra pars reticulata, and the ventroanterior and mediodorsal thalamic nuclei. The integrity of both cortical and subcortical structures is important to the proper function of these connections, and normal aging-related brain changes may serve to jeopardize this circuitry.

These same vulnerable white matter regions that support executive control functions are those that are particularly sensitive to cardiovascular and cerebrovascular health declines (Pantoni et al., 1999; Desmond, 2002; van Boxtel et al., 2006). White matter lesions frequently occur in adults aged 60 and older, and among these older individuals, those with vascular risk factors and cerebrovascular disease are more likely to have white matter lesions (Pantoni et al., 1999; Desmond, 2002; van Boxtel et al., 2006). In normal elders without dementia, white matter lesions are associated with subtle cognitive deficits on tests of attention, speed of processing, planning, and other executive functions (Schmidt et al, 1993). Nevertheless, such white matter abnormalities may lead to the pathological aging end of the late life cognition "continuum" and results in more severe cognitive deficits. This notion is supported by evidence suggesting there are forms of vascular cognitive impairment in which the main form of neuroanatomical pathology is found in the white matter (e.g., Pantoni et al., 1999). This pathology results in the disruption of cortical-subcortical circuitry described previously (Cummings et al., 1993). Particularly, in subcortical vascular dementia, the presence of deep and periventricular white matter alterations and lacunar infarcts tends to result in difficulties on tasks involving mental manipulation, and more notably, difficulties in establishing and maintaining mental set (Libon et

al., 2004). There tends to be a relative sparing on memory and language tests (Libon et al., 2004).

These findings guide the current proposed study with regard to the hypothesized selective benefit of exercise on executive control processes. Furthermore, there is evidence to suggest that related to the selective decline in executive brain function in older individuals, physical exercise interventions work to improve these vulnerable frontal and white matter brain regions. Specifically, brain mechanisms for improvement with physical exercise include increased cerebrovascular perfusion, cerebrovascular sufficiency, and improved cortical plasticity (McAuley et al. 2004); these changes occur through increased proliferation of neurotrophin factors, dendritic branching, neurogenesis, and formation of new synaptic connections (McAuley et al. 2004). This is a fundamental premise which drives the current proposal, as the hope is to demonstrate that by improving physical, particularly cardiovascular, fitness levels through physical exercise intervention, brain cerebrovascular perfusion and sufficiency and cortical plasticity may be enhanced in a group of vulnerable individuals (due to older age and sedentary lifestyle), thereby enhancing the neurocognitive (executive control) functions dependent on the integrity of brain white matter structure and function.

Cardiovascular and Physical Health Influences on Late Life Cognition

There are certain factors that may exacerbate cognitive aging and the aging process in general. These include behavioral factors, such as diet, physical activity, and other health promoting behaviors. In addition, environmental factors, such as socioeconomic status, lifetime educational and occupational opportunity, stress, and a host of other environmental influences, interfere with "optimal" aging (Burke et al., 2001). Furthermore, the presence of cardiovascular risk factors can influence cognitive function with aging. It has been documented hypertension is a major risk for many other cardiovascular diseases (stroke, diabetes, congestive heart failure,

etc.), and its pathogenesis is linked to cognitive function (Waldstein & Katzel, 2001; Waldstein et al., 2005; Waldstein & Katzel, 2005). Hypertension is a common health problem; often, the presence of hypertension precedes and is comorbid with more serious cardiovascular diseases. Hypertension is thought to be related to increased cardiac output, increased peripheral vascular resistance, increased sympathetic nervous system activation, and compromised renal function (Waldstein & Katzel, 2001). It also has been shown to exacerbate age-related arterial stiffness (Benetos, 2002). Furthermore, hypertension has been linked to cognitive function in many ways. Mechanisms include decreased cerebral blood flow and metabolism, metabolic syndrome, atherosclerosis, white matter disease, brain atrophy, and activation of stress response systems (i.e., the hypothalamic-pituitary-adrenocortical axis) (Waldstein & Katzel, 2001; Gold et al., 2005). These mechanisms, as mentioned above, have detrimental implications for the integrity of gray (Gianaros et al., 2006) and white matter structure and executive control function.

Relationship between hypertension and cognition

In reviewing research exploring the relationship between cardiovascular illness and cognition, several common themes emerged. First, with specific regard to blood pressure, there appears to be a U-shaped association with cognition. Next, cardiovascular risk factors seem to work synergistically. Third, cardiovascular illness seems to have direct effects on the integrity and function of the brain, which in turn affects cognition and neuropsychological function. Lastly, cardiovascular illness appears highly related to genetic and environmental factors.

Historically, early studies (before the 1970's) examining the relationship between hypertension and cognitive decline have come to find strong, dramatic results (Waldstein et al., 1991). These early studies concluded hypertension was related to very severe neuropsychological and cognitive deficits; however, many of these studies were poorly controlled (Waldstein et al., 1991). They failed to account for variables such as age and

education, and many involved samples of people with more severe cardiovascular disease, and not just hypertension (Waldstein et al., 1991). More recent studies with fewer methodological flaws have come to show relationships between hypertension and cognitive decline, but these findings have often been inconsistent (Waldstein et al., 1991). While many studies have found high blood pressure and cognition to be related, others have found low blood pressure to be more related to cognition, or no relationship between blood pressure and cognition at all (Waldstein et al., 1991). Additionally, there have been inconsistencies with regard to the various neuropsychological domains most affected by high blood pressure; for instance, some studies find relationships between memory or attention and blood pressure, while others may not (Verghaegen et al., 2002; Madden et al., 2003). Furthermore, studies have found various patterns of neuropsychological and cognitive deficits, such that relative effects of blood pressure on neuropsychological domains are difficult to discern (Waldstein et al., 1991). Overall, relationships between blood pressure and cognition have been found cross-sectionally and longitudinally, while chronicity of hypertension seems to be an important predictor longitudinally (Waldstein et al., 1991). Specifically, much research suggests high blood pressure negatively affects cognitive function (Deary et al., 1998; Elias et al., 2003; Elias et al., 2004; Starr & Whalley, 2005; Waldstein et al., 1991; Waldstein & Katzel, 2001; Waldstein et al., 2005; Nilsson et al., 2004), with the effects of hypertension exacerbated by other factors, including APOE 4 allele status (Carmelli et. al., 1998) and obesity and hyperglycemia (Elias et al., 2003).

Across various studies, a common finding is the U-shaped effect of blood pressure, with regard to actual blood pressure levels and age (Carmelli, 1998; Launer, 2000; den Heijer, 2002; Madden, 2003). High blood pressure is associated with worse performance across various cognitive domains (i.e., attention, executive functions, memory and learning, processing speed,

visuospatial and perceptual functions, etc.) and higher dementia incidence, and this relationship is strongest in middle-aged adults both cross-sectionally (Madden et al., 2003) and longitudinally (Carmelli et al., 1998; Launer et al., 2000; Lopez et al., 2003). Nonetheless, low blood pressure levels are also associated with poorer cognitive performance and higher dementia risk; some work has shown that while high blood pressure at baseline may predict poor cognitive performance at baseline, low blood pressure at follow up may be more predictive of cognitive decline over time (Ruitenberg et al., 2002; Zuccala et al., 2002; Launer et al., 2000). Finally, low blood pressure seems to be more influential on cognitive decline in older adults than in younger age groups, and some suggest this is due to hypoperfusion in the brain (Waldstein & Katzel, 2001).

Underlying physiological mechanisms

A common finding in this literature is the direct effect cardiovascular disease has on brain integrity and function, which in turn negatively impacts neuropsychological function (Pantoni et al., 1999; Deary et al., 2003; Waldstein & Katzel, 2001; Raz, 2003; den Heijer, 2003; Kramer, 2001). Cardiovascular conditions, particularly hypertension, cause changes in overall weight, plus changes in brain vasculature (Raz, 2003; den Heijer, 2003; Pantoni, 1999; Kramer, 2001). There is evidence for hypertension-related atrophy in prefrontal brain regions (Raz, 2003). This finding is significant since, as aforementioned, prefrontal brain regions are responsible for executive control functions, which are vulnerable to both normal aging and poor cardiovascular health. Furthermore, there is evidence hypertension and other cardiovascular illnesses exacerbate atherogenesis, arterial stiffness, white matter changes, and there is evidence linking them to large and small vessel infarcts, silent lacunar infarcts, and ischemic attacks (Benetos et al., 2002, Patoni, 1999; Kramer, 2001; Waldstein & Katzel, 2001). These small vessel infarcts

and silent lacunar infarcts often occur in the deep and periventricular white matter brain regions, leading to observable executive control deficits (Libon et al., 2004).

Raz and colleagues (2003) and den Heijer et al. (2003) have demonstrated the relationship between high blood pressure and pathological brain changes; in their work, hypertension was related to increased brain atrophy. Even further, Raz et al. (2003) found atrophy of the prefrontal brain regions to be most related to high blood pressure, while den Heijer et al. (2003) showed there was a U-shaped relationship between hypertension and brain atrophy. Thus, both high and low blood pressure levels were both found to be associated with disproportionate amounts of brain atrophy (den Heijer, 2003). Pantoni (1999) has argued evidence demonstrates vascular cognitive impairment tends to be more frequent in individuals with white matter lesions, as well as in those with cerebral circulatory dysfunction. Similarly, Waldstein and Katzel (2001) reported evidence suggesting the effects of decreased cerebral blood flow, related to increased peripheral vascular resistance in blood vessels in the body, may also be an underlying mechanism in this relationship. It has also been reported that increased activity of both the sympathetic nervous system and hypothalamic-pituitary-adrenocortical axis is involved in stress responses to environmental challenges, and this appears to play a role in the hypertension-cognition relationship (Waldstein & Katzel, 2001).

As was explored further in the present study, these physiological brain changes have direct implications for cognitive function and declines mediated by these brain areas, which explain the pattern of neuropsychological deficits often observed (i.e., deficits in executive function, processing speed and attention, more so than deficits in memory and language) (Pantoni, 1999; Deary et al., 2003; Waldstein & Katzel, 2001; Raz, 2003; den Heijer, 2003; Kramer, 2001).

Synergistic effect of hypertension and other cardiovascular conditions

Another common finding across studies in this area is that cardiovascular risks seem to have synergistic effects on cognition (Elias et al., 2003; Martins et al., 2006; Waldstein & Katzel, 2006). One study found the effect of hypertension and obesity in some individuals, as well as the effect of hypertension and hyperglycemia in others, more negatively affected cognitive function than those with either hypertension, obesity, or hyperglycemia only (Elias et al., 2003). Furthermore, another study showed the effect of hypertension was more severe for individuals with one or more APOE 4 alleles than for those with hypertension or APOE 4 status alone, or with neither condition (Carmelli et al., 1998). Conversely, individuals with APOE 4 and treated with antihypertensive medication showed better cognitive performance those with APOE 4 and untreated by antihypertensives in another study (Hestad & Engedal, 2006). In general, it appears to be the case many cardiovascular illnesses and diseases are comorbid, and this comorbidity likely results in worse cognitive and medical outcomes. The present study explored whether an exercise promotion intervention improved these comorbid conditions either together, or in isolation, and how potential health improvements affected cognitive function.

Environmental factors

Finally, a summation resulting from various findings in this literature regarding the relationship between cardiovascular illness and cognition is the strong evidence that genetic and environmental factors seem to greatly influence cardiovascular risks (e.g., Waldstein & Katzel, 2001). Certain genetic factors include APOE 4 status, family history of cholesterol, blood pressure, and glucose/insulin (metabolic disease) levels (Waldstein & Katzel, 2001; Martins et al., 2006). These all influence the expression of cardiovascular disease and may act as predisposing factors, if conceptualizing genetic influences from a "diathesis-stress model" framework. In addition, the environment plays a role in cardiovascular risks. Dietary habits and

exercise and physical activity are all obvious influences (Waldstein & Katzel, 2001); nevertheless, social environment also plays a role (Seeman & Crimmins, 2001). Individual socioeconomic status is a major factor in mortality and morbidity rates among most diseases (Dowd & Goldman, 2006; Seeman & Crimmins, 2001; Valanis, 1999). Lastly, social relationships are important to consider in the expression of cardiovascular and other illness (Seeman & Crimmins, 2001). Studies show social relationships to be stable across the lifespan, and the more social integration one has, the lower the risk for mortality (Seeman & Crimmins, 2001). Nevertheless, to the extent social relationships cause stress in a person and cause the development of poor health habits (diet, exercise, etc), social interaction can have a negative health impact (Dowd & Goldman, 2006; Seeman & Crimmins, 2001).

The present study addressed many of these environmental factors that play a role in not only cardiovascular, but cognitive risks, and disentangle the often confounded relationship between physical fitness and psychosocial exercise outcomes.

Self-Perception Influences on Late Life Cognition

Depression and Well-Being

Depression is not a "normal" part of aging, but it is considered one of the more common mental health concerns in the elderly. In older adults, depression is the second most commonly diagnosed mental disorder (LaRue, 1992). According to the National Institutes of Health (NIH), major depression affects about 2 million of the 35 million Americans 65 and older, and another 5 million suffer from depression symptoms; however, only about 10% receive treatment (Anthony & Arboraya, 1992; Gatz et al. 1996; NIMH, 2003). While clinically diagnosed depression has lower prevalence rates in older adults, some studies have observed a curvilinear relationship with age (Haynie et al., 2001). High depression scores in young adulthood, lower in middle age, then higher in old age. Certain factors noted to account for age differences in depressive symptoms

include physical disability, chronic illness, and loss of close relatives (Haynie et al., 2001). However, chronological age is not necessarily the critical variable for depressive symptoms in late life, and instead, it has been found that negative changes in health and psychological functioning are associated with depression (Haynie et al., 2001). There is also much suggestion in the literature that depression may differ in older adults, such that diagnostic criteria may underestimate the prevalence of the disorder in the elderly. Thus, a "minor" depression category may be more appropriate to characterize depression in older adults (Knight, 2004). Minor depressive disorder is listed in *DSM-IV-TR* (American Psychological Association, 2000) as needing further study and is defined as fewer symptoms than major depression and lacking the 2 year timeframe for dysthymic disorder (Knight, 2004; Blazer et al., 1991; American Psychological Association, 2000). Furthermore, some have argued that a "depletion syndrome" of depression, marked by loss of interest and fatigue, rather than intensely depressed mood and guilt, is a better way of conceptualizing and characterizing late life depression (Newmann et al., 1991; Blazer, 2003). Complaints that physical problems have gotten worse (i.e., arthritis or headaches) is often a predominant symptom, and symptoms of anxiety or irritability may be present (Newmann et al., 1991; Blazer, 2003). Another term in the literature is "depression without sadness," such that instead of feeling sad, a depressed older adult may complain of low motivation, lack of energy, or physical problems (Newmann et al., 1991; Blazer, 2003). This may be characterized by withdrawal, apathy, and a lack of vigor.

In general, older depressed adults are less likely to admit dysphoria, guilt, or suicidal ideation compared to young depressed adults (LaRue, 1992). They instead present with somatic symptoms and a lack of positive feelings (LaRue, 1992). In addition, depression symptoms in the elderly are difficult to interpret because they may mimic or be a precursor to medical

conditions (Gatz, 2000). Late life depression is often comorbid with chronic medical illnesses or disabilities and can precede or be a risk for the development of illness (Herbst et al., 2007). Study has suggested that lifetime major depression may be associated with increased risk of coronary heart disease in older adults (Herbst et al., 2007). Depression has also been shown to increase risk of death while decreasing the ability to benefit from rehabilitation.

This suggests the prevalence of depression in the elderly may be higher than current estimates. Furthermore, older adults experiencing depression symptoms may experience similar effects of Major Depressive Disorder (the clinical diagnosis for depression), including health problems, malnutrition, disability, functional and cognitive impairment, and increased mortality rates (Gatz, 2000).

Similar to the effect of normal aging on cognitive function, depression tends to be associated with difficulties performing cognitive tasks. There have been a number of terms in the literature that refer to the potentially reversible nature of cognitive impairments associated with depression in older adults. Such terms include pseudodementia (Kiloh, 1961) and dementia syndrome of depression (Folstein & McHugh, 1978). However, these terms may not be the most appropriate, because the cognitive effects of depression can range in scope and severity and often not meet the criteria of dementia (Houston & Bondi, 2006). Instead, the term depression-related cognitive dysfunction (Stoudemire et al., 1989) has been proposed as a better alternative, since it refers to depression and is general enough to encompass a range of cognitive deficit severity.

Regarding specific cognitive deficits common with depression, depressed individuals show deficits on controlled, effortful tasks (such as those highly dependent on executive functions), while verbal skills and other automatic processes are relatively spared (e.g. Hartlage, Alloy, Vazquez, & Dykman, 1993). Overall, there is consistent evidence for neuropsychological

deficits in depression for older adult samples on tasks of executive function, visuospatial skills, and psychomotor speed (Houston & Bondi, 2006). Moreover, depression is associated with impaired performance on a number of traditional neuropsychological tests purported to measure executive functioning (i.e., Wisconsin Card Sorting Test, Stroop Color-Word Test), and various working memory measures (Moritz et al., 2002). These executive functioning deficits predict difficulty performing instrumental activities of daily living (Cahn-Weiner, Boyle, & Malloy, 2002), which is often related to functional decline in the elderly. One study sought to characterize the neuropsychological presentation of geriatric depression and to determine if depression-related executive dysfunction was more pronounced in old age (Lockwood et. al, 2002). Results indicated for measures of attention, there was no significant age-depression interaction; however, for measures of inhibitory control and focused effort there was an age-depression interaction, with depressed older adults performing worst (Lockwood et. al, 2002). They discussed two models to explain their findings: in one model, depression is viewed as unmasking executive dysfunction in patients with compromised frontal-subcortical pathways. In a second model, dysfunction of frontal-subcortical pathways is thought to be predisposing to depression and executive dysfunction. Nevertheless, not all older depressed patients have significant cognitive dysfunction. These individuals may show only mild deficits, but often continue to have subjective memory complaints (Houston & Bondi, 2006).

Vascular Depression Hypothesis

One possible explanation for such depression-related executive control deficits may be due to the fact both depression and cognitive decline share a common cardiovascular etiology (Alexopoulos et al., 1997). As such, the vascular depression hypothesis has been proposed as a way to explain the common link between depression and cardiovascular health, as well as cognitive impairment. The hypothesis originally purported that cerebrovascular disease can

"predispose, precipitate, and perpetuate" depression in the elderly and that neuropathology found in the white matter regions of the brain can be an etiological factor in the expression of depression (Alexopoulos et al., 1997; Alexopoulos, 2006). One investigation showed, through path modeling, a stronger relationship between cerebrovascular risk factors (i.e., hypertension, diabetes, and heart disease) and depression symptoms in a group of oldest-old (85 and older), after controlling for co-morbid health/medical conditions and limitations (Mast et al., 2005). Co-morbid health/medical conditions and limitations mediated the relationship between cerebrovascular risk factors and depression in individuals age 50-84 in this sample (Mast et al., 2005). Additionally, a second study examining the role of cardiovascular risk factors found that among the oldest-old living in a retirement community, both depression and number of cardiovascular risk factors at baseline predicted stroke (Krishnan et al. 2005). Depression accounted for twelve percent of the variance in stroke incidence and partially moderated the effect of cardiovascular risk factors (Krishnan et al., 2005). Furthermore, studies have shown that white matter lesion load significantly predicts depression in older samples (e.g., Godin et al. 2008; Krishnan et al. 2004). In one cross-sectional study, white matter lesion volume was significantly associated with lifetime of major depression after controlling for covariates (sex, age, hypertension, cardiovascular disease, and alcohol and tobacco consumption) (Godin et al. 2008). Furthermore, in another cross-sectional study investigating the strength of association between various vascular-related neuropathology linking to depression (i.e., cerebral white matter hyperintensities signaling periventricular and deep white matter lesions), the findings demonstrated that deep white mater lesions was more strongly correlated with depression symptoms as measured by the Geriatric Depression Scale (15-item Short-Form) (Krishnan et al., 2006).

Just as depression has been linked to frontal lobes and executive cognitive function, the vascular depression hypothesis has also linked vascular-related depression to frontal/executive cognition (Alexopoulos, 2006). Clinically, vascular depression has been described as a medial frontal lobe syndrome (Krishnan et al., 2004), with symptoms including psychomotor retardation, apathy, and disability. Further, cerebrovascular lesions have been associated with persisting, unstable remission of depression and increased dementia risk (Alexopoulos et al., 2002). Two streams of literature have proposed and used separate terms related to the vascular depression hypothesis. These include subcortical ischemic depression and depression-executive dysfunction syndrome, and these terms differ in their assumed etiology (Alexopoulos, 2006). First, subcortical ischemic depression (Taylor et al., 2006) asserts that the subcortical impairment and associated depression is due specifically to cerebrovascular disease; however, the etiology of depression-executive dysfunction syndrome is less defined by specific factors (Alexopoulos, 2006). Instead, this syndrome may be the result of vascular disease, general age-related changes, degenerative brain disease, or and accumulation of these and other factors (Alexopoulos, 2006). Some in this field have called for internal consistency studies to better classify vascular depression as a syndrome and to decide upon a uniform etiology (Alexopoulos, 2006; Sneed et al, 2006). Nonetheless, regardless of a specific etiology, in the present study, it is expected that exercise-related improvements in cardiovascular health will work to improve depression symptoms, which is hypothesized to lead to cognitive improvements.

Control Beliefs and Self-Efficacy

In older adults, beliefs about control and self-efficacy are importance factors with regard to cognition. Study has suggested that control beliefs in late life show a significant relationship with cognitive performance (Miller et al., 1999). These findings highlight the importance of considering the impact control beliefs and other background variables may have when

decomposing age-related variance in cognitive performance (Miller et al., 1999). Additionally, late life cognitive performance has an impact on control beliefs, suggesting a bi-directional relationship. While control beliefs have not gained as much attention in the fitness and cognition literature reviewed above, consideration of these factors is important in an exercise intervention context. Related to control beliefs is the concept of self-efficacy, which is among various theories of health behavior change (health belief model and self-efficacy theory) that include cognitive elements related to decision-making. These cognitive elements of decision-making abilities are critical determinants of engagement in health and/or exercise behaviors (Bandura, 1997). Such decision-making may reflect individuals' actual barriers to exercise behavior, lack of motivation, or ambivalence toward exercise behavior. The present study will help individuals resolve ambivalence towards exercise through techniques (goal setting and mental imagery) designed to improve self-efficacy/control beliefs. Thus, an important test of our conceptual model will be to determine whether cognitive performance also benefits from such improvement in self-efficacy and control beliefs.

Plasticity of Late Life Cognition

Despite the various, and often detrimental influences, of physical and mental health on late life cognitive function, research examining both cognitive and physical training interventions with older adults have encouragingly found some positive results for the improvement of cognitive functions in aged individuals. This evidence supports the notion of the plasticity of late life cognition, which is relevant to the current proposal, which seeks to intervene through a physical exercise promotion intervention. Even in later life, cognitive function can show positive change, and there are both environmental and physical routes to such improvement. These environmental and physical interventions will be briefly reviewed here.

Cognitive Interventions

Cognitive training (e.g., Jobe et al. 2001, Ball et al., 2002) has been shown to lead to an improvement in cognitive function. Early studies have examined the plasticity of late life cognition in laboratory settings and found these interventions to be successful in improving cognitive function (e.g., Rebok & Balcerak, 1989). In addition, these early investigations have shown cognitive interventions can lead to improved performance on specific mental and perceptual functions, as well as certain aspects of everyday cognition (Leirer, Morrow, Pariante, & Sheikh, 1998; Ball & Owsley, 2000). More recent study has continued to explore the benefits of cognitive training intervention with older adults. Specifically, the Advanced Cognitive Training for Independent and Vital Elders (ACTIVE) Clinical trial, a randomized controlled trial of three cognitive intervention approaches (speed, reasoning, and memory) for older adults, demonstrated strong, broad, and durable cognitive ability-specific training effects, comparable to or greater than the amount of cognitive decline observed in other longitudinal studies, suggesting the interventions have the potential to reverse age-related decline (Ball et al., 2002). There was minimal transfer to training effects to everyday activities (i.e., functional competence); however, through a two-year follow-up period, there was no evidence of a significant decline in ADL and IADL status (Ball et al., 2002). At a five-year follow-up period, there was evidence for transfer of cognitive training gains to IADL function, with individuals receiving cognitive intervention showing slower rates of functional decline relative to controls (Willis et al., 2006).

In general, improvements from cognitive training interventions have been found to be very specific to the cognitive skill/domain trained, with few broader cognitive improvements (Kramer & Willis, 2002). This is a major limitation of cognitive training protocols when it comes to intervening upon functional aging-related declines and reducing the risk and/or progression of

cognitive impairment disorders and dementias. Thus, the consideration of interventions with potentially broader cognitive effects is warranted.

Physical Exercise Interventions: Cardiovascular Fitness

It has well been established the effects of physical activity on health and disease (increased longevity, decreased risk of coronary heart disease, blood pressure, various cancers, type-2 diabetes, etc.; Sallis & Owen, 1999) across the lifespan. For older adults, the engagement in physical activity can improve cardiovascular health, increase metabolism, and slow declines in bone mineral density (Singh, 2002). Since there is a higher rate of chronic disease in older populations, the health benefits of physical activity and exercise may be even greater in older adults (Pescatello, & DiPietro, 1993). Specifically, older adults can benefit from participating in regular, moderate-intensity exercise (i.e., 30 minutes of brisk walking, five or more times a week) (Schiller et al., 2005). Even when physical activity is initiated late in life, health and mortality rates are still substantially affected, even after accounting for factors such as smoking, family history, weight gain, and hypertension (Blair et al., 1995). Additionally, there is evidence that regular physical activity benefits older adults who have chronic conditions as varied as cancer, cardiovascular disease, respiratory disease, and dementia (e.g., Morgan &, Bath, 1998).

Over the past several decades, research has examined the benefits of physical fitness training on cognitive function, particularly in older individuals (Colcombe & Kramer, 2003). As mentioned above, one route by which physical exercise interventions may improve cognition is by improving cardiovascular fitness. Regular physical activity can reduce adults' risk of coronary heart disease (Haskell et al., 1992) cancer (Blair et al., 1989) offer protection against non-insulin dependent diabetes (Helmrick, et al. 1991), and reduce hypercholesterolemia (Harris et al., 1991). Studies have also linked physical activity with diminished declines of functional capacity due to age and reduced risk factors associated with falls in the elderly (DiPietro, 2001;

Liu-Ambrose et al., 2004), while older adults who engage in regular physical activity reduce their risk for mortality associated with chronic disease states and premature mortality (Bean, et al., 2004).

Relationship between fitness and cognition

Early studies of the relationship between cognition and fitness made group comparisons between physically fit and unfit individuals (McAuley et al., 2004; Kramer, Erickson, & Colcombe, 2006). Like many early psychological investigations, these studies were cross-sectional in nature, and they failed to control for a host of factors that influence results observed (McAuley et al., 2004; Kramer et al., 2006). Such studies came to conclude that physically fit individuals showed better performance on cognitive measures than did physically unfit persons (McAuley et al., 2004; Kramer et al., 2006). Today, the dangers of making such conclusions are better understood. Factors such as age, education, socioeconomic status, race/ethnicity, may influence performance on cognitive measures. Thus, improving on such methodological problems of these studies, more recent studies employed longitudinal designs and compared individuals undergoing an exercise intervention to those who were not (McAuley et al., 2004; Kramer et al., 2006). These results indicated that while many people demonstrated cognitive performance improvements in these studies, other participants did not (McAuley et al., 2004; Kramer et al., 2006). These inconsistencies have left many a bit baffled and motivated to investigate the underlying truth further (Colcombe & Kramer 2003; McAuley et al., 2004). Cited methodological reasons for these inconsistencies include reliance on self-report activity data, failure to distinguish between activities that are aerobic and anaerobic in nature, poor assessment of duration, intensity, and frequency of exercise activity, non-exclusion of participants with subclinical dementia, and low statistical power (Kramer et al., 2006). Accordingly, Colcombe & Kramer (2003) conducted a meta-analysis of 18 studies that have

examined this exercise-cognition link in older adults to understand these inconsistencies better. The goal of the meta-analysis was to test several hypotheses have been proposed in the literature regarding the specific effects of exercise: atheoretical- no specific hypotheses; speed hypothesis- measures of speed of processing show the greatest improve with exercise; visuospatial hypothesis- these measures show the greatest improvements with exercise; controlled processing hypothesis- these sustained attentional tasks show the greatest improvements; and executive control hypothesis- working memory, problem solving, and inhibition type tasks show the most exercise-related gains (Colcombe & Kramer 2003). Results from the meta-analysis showed that indeed, exercise was related to the improvements in cognitive performance, with executive control processes showing the greatest exercise related improvement (Colcombe & Kramer 2003). Women and older participants showed the most cognitive gains, as combined aerobic and strength training exercise programs showed the most benefits to participants (Colcombe & Kramer 2003).

Nonetheless, there are several methodological problems with this meta-analytic study. First, the meta-analysis included 18 studies that had well over 50 different neuropsychological/cognitive measures. The authors allowed for multiple cognitive domain categorizations along the four major hypothesized cognitive domains (i.e., one measure could be considered a speed, visuospatial, and executive tasks all at once) (Colcombe & Kramer 2003). While they control for the influence of executive control processing on performance on other domains, they fail to control for the influence of the other three domains on executive control processing. This meta-analysis also suffered from including some studies that were flawed. Palleschi et al (1996) examined the effect of 12 weeks of exercise on cognitive performance (MMSE, attentional matrix, verbal span, supraverbal span tests) in a group of 15 Alzheimer's

patients. Their findings suggested that performance on neuropsychological measures was improved with exercise; nonetheless, there was no control group comparison to determine whether these patients' improvements were above mere practice effects. Also with such a small, clinical sample, these findings are difficult to generalize to other groups. Another study included in the Colcombe & Kramer (2003) meta-analysis with methodological concerns compared the effects of exercise on cognitive performance in a group of depressed older adults (Khatri 2001). This study compared depressed elders who performed 16 weeks of aerobic exercise to those prescribed anti-depressant medication and found while both groups improved in depression symptoms, only the exercise group showed improved performance on the Stroop Interference task and Visual Reproductions of the Wechsler Memory Scale (WMS) (Khatri 2001). As with the previous study, there were was no control for factors that would confound the results, particularly the mere personal contact involved in being in a study, or practice/re-test effects. Furthermore, in a study of patients with chronic obstructive lung disease (COPD), they examined whether aerobic exercise would result in improved performance on cognitive and psychological outcomes (Emery et al., 1998). These results showed that while patients did not improve lung functioning with exercise, there were improvements in depression symptoms, though not more than controls (Emery et al., 1998). Also, the only cognitive improvements above re-test effects were found for verbal fluency performance, which is thought to have a strong frontal-executive component (Emery et al., 1998). These three studies all contained clinical samples and were include in the Colcombe & Kramer (2003) meta-analysis, along with 15 other studies of normal, healthy older adults, which may obscure the clean examination of the true exercise-cognition link.

To improve upon these methodological flaws, the present study will examine a sample of community-dwelling, cognitively intact elders, without major cognitive, physical or mental health conditions. Additionally, a stream of research suggests that complex social activity participation may be a protective factor for cognition (e.g., Brown, 1992; McAuley, 1993). Elders who have had more complex, "engaged" lifestyles, higher levels of education, and who are more active in later adulthood have been shown to perform at a higher level (cognitively), and may evince attenuated rates of cognitive decline. For many adults, exercise is a possible source of complex activity; it may bring some adults out of house, provide them with complex regimens to monitor, and may increase their social participation with other exercisers ("mall walking", exercising at a fitness facility, etc.). In order to account for the effects of complex social activity, the control condition of this exercise promotion intervention will be designed to control for the influences of complex social engagement on improvements in both cognitive and affective functioning.

Psychological benefits of exercise interventions

A strong body of research suggests that along with the physical health benefits of exercise in older adults, there are also strong psychological benefits of engagement in physical activity. Particularly, there is strong evidence that participation in regular physical activity is associated with psychological health and well-being for older adults age 50 and older (Brown, 1992; Elavskey et al., 2005; Elavskey & McAuley, 2007; McAuley, 1993; Motl et al., 2005; Morgan & Bath, 1998; Pescatello & DiPietro, 1993). The engagement in structured and unstructured physical activity regimens are related to reduced symptoms of depression and anxiety and improvements in mood in older adults (Fukukawa et al., 2004). In sum, increasingly the field has come to accept the link between physical activity and a variety of positive emotional, behavioral, and physical health outcomes for older adults to be a viable one.

Furthermore, control beliefs and self-efficacy are also influenced by physical exercise interventions. One's self-perception beliefs have a critical impact on whether or not he will engage in an exercise or health intervention. The major social-cognitive theories of exercise include the basic set of social-cognitive principles: self-efficacy expectations, outcome expectations, outcome values, and intentions to change, modify, or initiate a certain behavior (Maddux, 1993). These factors are key mediators of the relationship between exercise behaviors and increased fitness, and these factors must be addressed and often intervened upon in order to ensure individuals will participate and adhere to any physical health intervention (Bandura, 1997). Thus, in the present study, these factors will be directly addressed by the exercise promotion intervention, with the aim of improving self-efficacy/control beliefs, thereby increasing exercise participation and desired physical fitness and cognitive outcomes.

Conclusion

As the preceding review of the literature has discussed, there may be multiple routes by which increasing exercise contributes to cognitive improvement. Exercise intervention may effect cognitive change through improvement of physical factors (particularly cardiovascular fitness) and/or improvement of psychosocial factors (self-perception influences, such as depression/well-being, control beliefs, and self-efficacy). Extant research has not yet paid adequate attention to disentangling these multiple pathways, thus the present study seeks to disentangle such exercise-related effects on cognition in older adults, thereby building on existing literatures, which have mostly examined these.

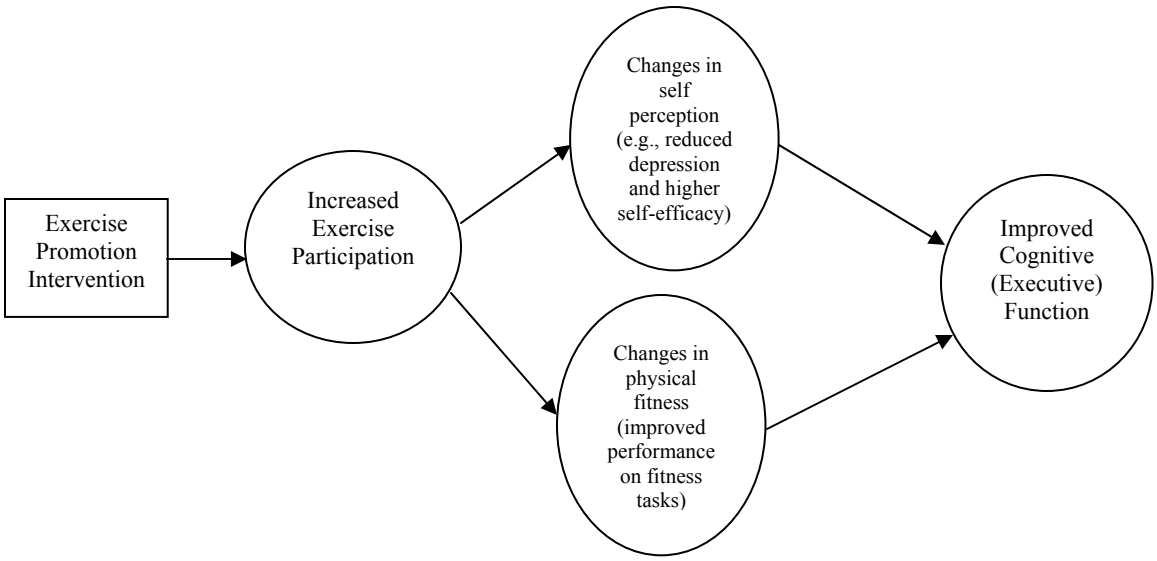


Figure 2-1. Conceptual model of the effects of exercise intervention on cognitive function

CHAPTER 3
METHODS

Overview

To achieve the specific aims outlined above, the present study was part of a larger, multi-disciplinary, pilot study examining the effects of an exercise promotion intervention on exercise behavior/beliefs and fitness in sedentary, community-dwelling adults aged 50 and older. To keep the scope of the current dissertation manageable, the present study focuses on immediate pre-test-post-test changes in cognitive functioning in adults who did and did not receive this intervention. The larger project assessed dynamic (intraindividual) cognitive and psychosocial changes throughout the period of intervention, considered other outcomes (cardiorespiratory fitness, exercise behaviors, etc.), and also has a longer-term follow-up (12-months) of the cognitive and non-cognitive outcomes in this study planned. Two randomized groups of (35 in the control group, and 34 in the intervention group) adults were recruited from the Gainesville/Alachua County, Florida region. Both the control and intervention groups underwent pre- and post-intervention cognitive, fitness, self-efficacy, sleep, and emotional well-being assessment. Additionally, the intervention was such that the control and intervention participants were divided into smaller replicates of five to twelve people, with trained peer mentors. There was quality control monitoring of the intervention and control groups to insure fidelity of protocol adherence.

The exercise promotion (experimental) group received 16 weeks of intervention (motivational interviewing, health and fitness education, weekly peer mentoring and group support session, adaptive goal setting for individual performances improvements, mental imagery, daily home-based self-monitoring, etc.). The intervention was informed by current state-of-the art interventions for sedentary adults and was customized for individual fitness goals

and avoidance of individual exercise barriers. A control/comparison group received 16 weeks of "health hygiene" instruction, consisting of education about general health issues relevant to older populations (i.e., osteoporosis, Alzheimer's disease, nutrition). There was one session devoted to non-specific discussion of exercise/physical activity. Both groups received unlimited use/access to a fitness facility; amount of exercise was monitored in both groups, such that the incremental benefits of the treatment could be carefully assessed.

Participants

The sample of participants consisted of 69 middle-aged and older adults who were sedentary and community-dwelling. Participants were recruited in the local Gainesville/Alachua County, Florida area, primarily through the use of local newspaper advertisement in the *Gainesville Sun, Senior Times,* and the Gainesville Regional Utilities (GRU) newsletter. Additionally, flyers were posted at locations around the Gainesville and University of Florida communities, and flyers were mailed to individuals on the University of Florida Older Adult participant registry. To attempt to maximize recruitment of diverse populations, an ad was also placed in the *Gainesville Guardian,* a publication targeted to the African-American community. Finally, recruitment extended to community organizations, including a seniors' group a at local community college, a local church, a retirement community affiliated with the university, a grandparents support group, local health fairs for seniors, and a direct mailing list. Figure 3-1 outlines the number of participants that were initially contacted and/or expressed interest in the study, screened by telephone, randomized to the study conditions, and included in the present analyses.

Statistical Power Considerations

Table 3-1, displays the results of a power analysis conducted to examine the study design's ability to detect effects with 32 participants per group, alpha = .05 (one-tailed;

expectations are directional), and several effect size possibilities. This is a lower-bound estimate, since it is based on a post-test-only comparison; the actual analyses were conducted as a mixed within-between analysis of variance. With expected high test-retest reliability in the primary outcome measures (executive cognitive function), under the same effect sizes, power would have exceeded that reported here. While cognition was a primary outcome in this dissertation study, it was a secondary outcome of the larger trial in which it is embedded. The study was therefore powered to affect primary outcomes of the larger trial (exercise participation, exercise self efficacy, and fitness). In the event that, as a secondary outcome of the larger trial, power is not adequate to detect significant differences on cognitive outcomes, the study will still be useful as a preliminary study for future work (specifically, to aid in effect size determination for future studies). In addition, the correlational aims of this study (to examine the extent to which fitness changes and psychosocial changes serve as proximal predictors of cognitive changes) did not require significant intervention effects to be tested and have substantially greater power.

Sample Characteristics

Table 3-2 describes participant characteristics. Overall, the sample had an average age of 63.9 years. The vast majority of the sample identified themselves as Caucasian/White (91.3%) and female (84.1%). There was an under-representation of male participants, as is customary in cognitive aging research, as well as ethnic minorities (i.e., African-American/Black, Hispanic, and Asian). On average, the sample was college-educated (16.2 years of education), and the average estimated IQ was 113, which falls in the high average range of intelligence. The sample reported a minimal level of depression and anxiety symptoms.

Cognitive Exclusions

All potential participants were screened by telephone to exclude individuals based on the following cognitive criteria: dementing illness, lifetime history of significant head injury requiring hospitalization, other neurological or major medical illnesses (Parkinson's disease, epilepsy, stroke, cancer (and/or chemotherapy and radiation above the chest)), severe uncorrected vision or hearing impairments, inpatient psychiatric treatment, extensive drug or alcohol abuse, any use of an anticholinesterase inhibitor (such as Aricept), or unavailability at future follow-up time points. Telephone screening included the 11-item Telephone Interview for Cognitive Status (TICS; Brandt, Spencer, & Folstein, 1988) for a standardized assessment of cognitive status. The TICS has a sensitively of 94% and a specificity of 100%. The cut-off score of 27 points was used to exclude demented individuals from the study (Brandt, Spencer & Folstein). The TICS, embedded in the general telephone screening protocol, is included in Appendix A.

Physician's Permission

To assure individuals were properly excluded due to cardiovascular/physical conditions/diseases and medications presented below, participants were required to submit a completed and signed nurse/physician's checklist and permission form prior to enrolling in the study. This checklist is included in Appendix B.

Physical/Cardiovascular Exclusions

Exclusion criteria for diseases or conditions likely to adversely affect the safety of elders in the exercise promotion intervention are as follows: terminal illness with life expectancy less than 12 months, cardiovascular disease (myocardial infarction in last 6 months, chronic heart failure, aortic stenosis, history of cardiac arrest, implanted cardiac defibrillator, or uncontrolled angina), pulmonary disease requiring oxygen or steroid treatment, and ambulation with assistive

devices (such as cane, walker, or wheelchair). Exclusion criteria for factors adversely affecting compliance with study protocols and interventions include: inability to read and understand English, not willing to consent to random assignment to one of the two study conditions, hospitalization for current psychiatric illness, daily alcohol consumption of more than two (for women) or three (for men) drinks, and hearing or speech impairments making verbal communication difficult.

Medication Exclusions

This study excluded individuals taking calcium channel blockers or beta-blockers. The mechanisms of these drugs are likely to adversely affect the safety of older adults' participation in an exercise intervention, since they interfere with heart rate reactivity during exercise.

Inclusion: Staging Algorithm

The Stages of Exercise Change Questionnaire (SECQ; Reed, Velicer, & Prochaska, 1997) was administered during the initial phone contact to assess participants' stages of change regarding the initiation of a physical exercise program. The SECQ contains five ordered-category items, which assess change readiness along a continuum: pre-contemplation, contemplation, preparation, action, and maintenance. Potential participants in the action and maintenance stages of physical activity were excluded, while persons in the earliest stage (i.e., pre-contemplation) were unlikely to volunteer/be compliant with an exercise intervention, by definition.

Procedure

Overview

The planned study timeline for a single participant was divided into approximately twenty weeks (Table 3-3). In the first two weeks, participants were screened, and eligible persons were enrolled into the study to undergo baseline assessment. This assessment included cognitive,

neuropsychological, psychological, and exercise/fitness testing. After randomization to either the control (health hygiene) or experimental (exercise promotion) condition, weeks three through eighteen (sixteen weeks total) of the study involved the intervention period. However, it is important to note that several factors determined the actual study timeline for each participant. The actual study timeline for each participant was influenced by his or her availability to devote sixteen weeks to participation in a group (i.e., schedule conflicts, vacation, etc), the status of participant recruitment (all the available slots of each group needed to be filled to maximize use of resources), the status of required completion and receipt of physician's permission form, and the ability of study staff to screen participants in a timely manner. Due to these factors, there was a lag of several months between initial participant contact, completion of telephone screening, and/or baseline testing and the start of the study group period for most participants. Specifically, there was an average of 5.6 weeks between baseline testing and the start of the small group sessions (range 0 to 26.6 weeks). After the 16 week intervention period, most (66 out of 69) participants were post-tested within one or two weeks following the end of the group sessions. The remaining 3 participants were tested longer than two weeks post-intervention due to scheduling conflicts (range 3 to 6.4 weeks).

During the intervention period, all participants received a free membership to either a University of Florida campus fitness facility or a community-based, church fitness center. There were a total of eight replicates of peer/support groups, with one control and intervention group each per replicate for a total of sixteen small peer/support groups. In total, 47 participants were randomly assigned to the control condition, while 44 participants were assigned to the intervention condition. In addition, study groups for replicates 1-5 were composed of people randomly assigned to peer groups including other individuals in their same age range (50-64

years or 65 and older) (Table 3-4). For replicates 6-8, the groups were age-mixed in order to meet the larger study's recruitment goals more quickly. Due to attrition of the sample, 35 control group participants and 34 intervention group participants were included in the present analyses. A detailed description of the intervention is found in a later section. Finally, following this period, participants underwent post-intervention assessment.

Table 3-5 outlines the pre-post assessment protocol (organized in a thematic grouping, not test order). Executive cognitive measures, which are thought to be more sensitive to physical fitness improvements (Colcombe & Kramer, 2003), are shown in the first row.

Rationale for Measures

Neuropsychological measures were selected to assess aspects of cognitive function expected to be affected by physical exercise intervention, as well as those expected to remain stable with exercise. Specifically, following the reviewed literature, executive control processes are hypothesized to be most positively influenced by improved aerobic fitness (Colcombe & Kramer, 2003); however, other cognitive functions, such as estimated intellectual ability and memory for structured information (story memory), are hypothesized to be remain relatively unchanged by improved aerobic fitness; these are included to help evaluate the putative *specificity* of exercise effects. Psychosocial, exercise, and aerobic fitness measures were selected to measure the physical and psychosocial components of exercise participation, as outlined by the conceptual model above, that are potential mediating variables in the relationship between exercise and cognitive function. The specific rationale for selecting each cognitive measure will be described in the section that follows.

Detailed descriptions of each measure are as follows:

Primary Outcomes

North American Adult Reading Test (NAART)

The NAART (Blair & Spreen, 1989) was used to measure pre-morbid intelligence. It was developed specifically for use with adults suspected to have compromised cognition. The NAART consisted of 61 irregular, rare words that participants were asked to pronounce. The NAART has been found to correlate between 0.40 and 0.80 with other measures of intelligence. Test-retest reliability of the NAART has been established at 0.92 within one year. The score used in was derived from using a prediction equation that took into account the number of words incorrectly pronounced. The NAART was selected as a non-executive cognitive measure in the present test battery because it is thought to be a measure of pre-morbid intellectual function (i.e., "crystallized" abilities), which would be expected to be relatively stable and less sensitive to improvements in physical fitness over time.

Logical Memory subtest of the Wechsler Memory Scale, 3rd Edition (WMS-III)

The Logical Memory subtest of the Wechsler Memory Scale-Third Edition (WMS-III) (Wechsler, 1997) measured the ability to learn and retain verbal memories for brief stories. Individuals heard a brief story of 25 propositions and were asked to recall as many story propositions as possible in an immediate recall of the story. Participants received one point for each proposition correctly recalled. Next, a second story of 25 propositions was read, followed by an immediate story recall trial. This second story was read twice to assess verbal learning slope. Delayed recall and recognition trials for each story were completed following a 25-35 minute delay interval. The LM subtest was selected as a measure of non-executive cognitive function because it measures episodic memory abilities in a structured way. Relative to word list-learning memory tasks, LM is thought to rely less on executive functioning, since individuals do

not have to impose a semantic organization on the information to maximize recall. It has been found to be a reliable memory measure for use in adult and older adult groups (Wechsler, 1997).

Control Oral Word Association (COWA)

To assess spontaneous word production, the COWA (Benton & Hamsher, 1989) was administered. Participants were read a letter ("F", "A", and "S") and asked to generate as many non-proper nouns beginning with that letter as possible within sixty seconds. Participants were not allowed to repeat previously stated words during each trial. The rationale for selecting COWA as an executive cognitive measure was due to its sensitivity to frontal lobe/executive function (Salthouse et al., 2003). Previous literature has identified the COWA as a "gold standard" of executive function (Crawford et al, 2000 & 2005).

Trail Making Test A and B (Trails A, Trails B)

The Trail Making Test (Trails A and Trails B) (Reitan, 1992) assessed attention, working memory, psychomotor speed, visual scanning and sequencing, and cognitive flexibility by requiring individuals to connect circles containing numbers (Trails A) and numbers and letters (Trails B). Trails B was conceptualized as a more demanding task, due to the increased cognitive flexibility and executive skills required for successful task completion. The rationale for inclusion of the Trail Making Test was due to both being one of the most widely used measures in clinical neuropsychological practice (Rabin et al., 2005) and due to its documented high sensitivity to brain function (Reitan & Wolfson, 1994). Trails A was categorized as a non-executive measure because it is conceptualized as a measure of attention and processing speed that is less reliant on working memory, as is Trails B. Trails B was selected as an executive cognitive measure because of the added mental flexibility and working memory required to complete this task (Lezak et al., 2004).

Letter-Number Sequencing subtest of the Wechsler Memory Scale, 3rd Edition (WMS-III)

The Letter-Number Sequencing subtest of the WMS-III (Wechsler, 1997) is a verbal working memory task that required individuals to repeat a group of numbers and letters after mental manipulation. Individuals heard a sequence of numbers and letters (ranging from 2 to 8 numbers/letters) and were then required to respond such that all numbers were stated first, in numerical order, followed by all letters stated in alphabetical order. This task was selected for inclusion in the cognitive battery as a measure of executive function, particularly working memory and attention. The Letter-Number Sequencing subtest has been found to be a reliable measure of executive function for use in adult and older adult populations (Wechsler, 1997).

N-Back task

The N-back task was the only cognitive measure administered by computer. In the N-Back task, participants saw a single letter in 48-point Arial font in the center of the computer screen and decided whether that letter matched a target letter. They were told to respond as fast and accurately as they could. The computer collected both accuracy and response time information. In the One-Back condition, participants judged whether the current letter matched the immediately preceding letter. They were instructed to push the "Yes" button indicated on the computer keyboard when that letter appeared, and the "No" button when the letter did not match the previously viewed letter. In the Two-Back condition, participants judged whether the current letter matched the letter presented two letters previously. Letters remained visible until a response was made. There was a 1 sec. inter-stimulus interval (Cohen, et al., 1994). The N-back task was selected as a measure of executive function due to previous work that has suggested its sensitivity to brain activation in dorsolateral prefrontal regions, thought to sub-serve executive control processes, in the brain (Cohen, et al., 1997).

Proximal Outcomes

Geriatric Depression Scale (GDS)

The GDS (Yesavage & Brink, 1983a) was administered to assess depressive symptomatology. The GDS is a 30- item self-report scale of yes/no questions about symptoms of depression (Do you feel that your life is empty?). A point was given for each depressive symptom endorsed. This measure has been shown to be a reliable and valid measure of depressive mood in older adults. Clinical cut-off scores for this measure are as follows: 0-10 normal depression symptoms, 11-14 mild depression symptoms, 15-21 moderate depression symptoms, and 22+ severe depression symptoms (Yesavage & Brink, 1983a). The GDS is a commonly used self-report measure of depression that has been validated for use in older adult samples (Yesavage & Brink, 1983b).

Beck Depression Inventory- 2nd Edition (BDI-II)

The BDI-II (Beck, Brown, & Steer, 1996) was also administered to assess depressive symptomatology. The BDI-II consists of 21 groups of statements related to cognitive and somatic depression symptoms (Sadness, Changes in Sleeping Pattern, etc.). Individuals selected from one of four statements the one best describing the severity of their symptoms over the past two weeks (0 = I do not feel sad, 1 = I feel sad much of the time, 2 = I am sad all the time, 3 = I am so sad or unhappy that I can't stand it). The scale has a clinical range of 0-13 for minimal depression symptoms, 14-19 points for mild depression symptoms, 20-28 for moderate depression symptoms, and 29-63 severe depression symptoms. The BDI-II is a commonly used self-report measure of depression that has been shown to have reliability and validity with adults, both younger and older than 65 years (Beck et al., 1996; Brink et al., 1983).

State-Trait Anxiety Inventory (STAI)

The STAI (Spielberger, 1983) was administered to assess current (state) and typical (trait) anxiety symptoms. The STAI consists of 40 statements to which participants responded based on the degree to which they felt that way. For the first 20 statements (I feel calm, etc.), participants responded with respect to how they felt "right now" (i.e., not at all, somewhat, moderately so, or very much so), while for the second 20 items (I feel pleasant, etc.), they responded according to how they "generally" felt (i.e., almost never, sometimes, often, almost always). Clinical cut-offs depend on age and gender corrected normative data. The STAI has been used in numerous studies of the relationships between anxiety and various psychological constructs (Spielberger, 1983).

Exercise self-efficacy

Two measures were used to measure exercise self-efficacy expectations. Both measures were based on social cognitive theories, particularly Bandura's theory (1997) of self-efficacy. The Barriers Self-Efficacy Scale (McAuley, 1992) is a 13-item measure of perceptions of confidence to maintain regular exercise (at least three times a week), despite common exercise barriers. The Exercise Self-Efficacy Scale (McAuley, 1993) is an 8-item measure of confidence to maintain exercise (three times a week at moderate intensity for 40 minutes) consecutively for a period of 8 weeks. Both measures have been shown to be predictive of exercise behavior and have adequate internal consistency across many research studies, including those with older adults (Blissmer & McAuley, 2002; McAuley et al., 2003; McAuley et al., 2005).

Leisure Time Exercise Questionnaire (LTEQ)

The LTEQ is a three-item scale that asked individuals to rate how often they engaged in mild, moderate, and strenuous leisure-time exercise (Godin & Shephard, 1985). The LTEQ is a reliable and valid measure of adult exercise behavior, and it allows for the calculation of a total

MET score, by weighting the three intensity levels and summing: 3(mild activity) + 5(moderate activity) + 9(strenuous activity). Previous research has supported the validity and reliability of LTEQ score interpretations with adult (Godin et al., 1986; Jacobs et al., 1993) and older adult populations (Karvinen et al., 2007; Ruppar & Schneider, 2007).

Minutes of Moderate and Vigorous Physical Activity (MVPA)

Minutes of moderate and vigorous physical activity (MVPA) were computed from the LTEQ by adding the number of moderate and strenuous bouts reported and multiplying by 20. This value was then added across the week to obtain a weekly measure of MVPA. Recently, to better approximate the true level of activity needed for reliable health benefits (moderate to vigorous; Pate et al., 1995), study has suggested that minutes of mild activity be excluded from calculations of physical activity (Karvinen et al., 2007).

Pedometer

The AE 120 pedometer (Yamax SW200 engine) was used as a second measure of physical activity. The pedometer was worn on participant's hip to measure steps taken during a given day, and participants recorded the final number of steps for each day the next morning, in a log. These values were averaged across each week of the intervention period to obtain mean steps taken. This measure was included as an objective measure of physical activity. Convergent validity of pedometers with has been shown self-report measures of physical activity has been shown (Tudor-Locke, Williams, Reis, & Pluto, 2002).

Modified Balke Submax (VO_2)

Cardiorespiratory fitness was measured using a modified Balke treadmill protocol (which is widely used) to obtain VO2 max estimates. The modified Balke is a treadmill protocol that involves slope increases while speed is held constant. Participants' heart rates were monitored throughout the protocol and for two minutes prior to testing. Within the first minute, treadmill

grade was increased 1% and continued to increase at 1-minute intervals until the heart rate reached 85% of age approximated maximal heart rate. Physician's permission was a must for this test. It is important to note that the modified Balke treadmill protocol results in a less accurate estimate of cardiorespiratory fitness than a maximal graded exercise stress test (VO2max, the gold standard); however, this modified protocol is considered much safer for sedentary older adults as were studied in the present project (McAuley, 1992).

Design and Rationale for Experimental Group

The design of the exercise intervention was not the key focus of the present study; rather, it was the focus of the larger project in which this study was embedded. The exercise intervention was designed to produce individually tailored, goal-referenced exercise plans that were sustainable and situated in the everyday life contexts of middle-aged and older individuals (n = 34). The intervention was a hybrid design, which integrated various theoretical models and components of behavior change, including motivational interviewing, mentorship and social support, self-efficacy, mental imagery, and goal setting (Figure 3-2). Accordingly, the intervention was designed to maximize the probability that individuals would continue to engage in exercise once their study participation was finished. The intervention was a 13-session, 16-week psycho-educational intervention designed to enhance the adoption and maintenance of regular physical activity.

Intervention sessions were conducted by trained peer mentors, with frequent quality control observations by study investigators, and it was completely manualized, with peer mentors receiving extensive instruction in the use of the manual. Peer mentors were selected based on successful completion of the prescribed intervention previously and willingness and ability to undergo additional training related to exercise behavior change. Peer mentors that did exceptionally well at delivering the intervention protocol were asked to lead future groups, while

peer mentors who did not adhere well to study protocol were not asked to continue for future groups. In order to ensure quality control of the intervention delivery, all group sessions were either video- or audio-recorded, and peer mentors were evaluated on several criteria (supportiveness, effective communication, etc; Appendix C). Feedback was recorded and discussed with the peer mentors prior the next group session.

Each intervention group weekly session lasted between 45 and 60 minutes and began with an initial check-in regarding physical activity completed between sessions. The schedule of intervention topics for the study is listed in Table 3-6. The group discussed challenges and barriers to engaging in physical activity, as well as successful participation in exercise behaviors. The remainder of the session was devoted to introduction and discussion of the topic for the week. At the end of each session, homework was assigned to the group. Half of the participants arrived 30 minutes prior to each weekly intervention group session to complete 15-30 minutes of computerized assessments of cognition, exercise self-efficacy, and barriers to exercise self-efficacy. The second half of the participants remained 30 minutes after each group session to complete the computerized assessments.

Design and Rationale for Control Group

The 35 participants randomized to the "health hygiene" control group received a set of weekly topics based on materials from the National Institutes of Health's SeniorHealth website (NIHSeniorHealth.gov). This website featured up-to-date, easily understandable and accessible health information for seniors and their family and friends. Control participants met with a peer-mentored group each week to receive didactic instruction based on health and aging topics discussed on the NIH SeniorHealth website (Alzheimer's disease, arthritis, balance problems, COPD, diabetes, etc.). Similar to quality control practices employed for the intervention condition, control group sessions were also recorded so that specific feedback could be given to

peer mentors delivering control group didactic material. The goal was to focus on general health and overall lifestyle promotion, with little emphasis on exercise or physical activity. There was no exposure to any of the motivational, goal setting, or imagery techniques used with the experimental, exercise promotion intervention group. As mentioned previously, this group also received a free membership to a University of Florida campus fitness facility or a community-based, church fitness center. This choice was made to control for the possible confound of access to fitness facilities and to enable the isolation of the unique peer mentoring, motivational, and coaching aspects of the exercise promotion/experimental group. Table 3-7 shows an overview of the design of this health hygiene control group. Table 3-8 shows the schedule of control group topics. While no formal homework assignments were given, peer mentors often challenged control participants to independently research additional information regarding the health topics discussed.

As with the intervention group sessions, control group sessions lasted between 45 and 60 minutes each week. The first 15 minutes of the sessions consisted of a review the previous week's discussion topic and the remainder of the time (30-45 minutes) was spent on the current week's health topic. Half of the participants arrived 30 minutes prior to the group's session to complete weekly computerized assessment (15-30 minutes for cognitive and self-efficacy measures). The second half of the participants remained 30 minutes after each group session to complete computer assessments.

Table 3-1. Pre-data collection power analysis results, indicating power expected at different effect sizes with two cells of 32 participants, and alpha = .05

Effect size	Weak (d = .20)	Medium (d = .50)	Strong (d = .80)
Power (1-β)	.31	.76	.97

Table 3-2. Participants' mean characteristics

	Overall n = 69	Health Hygiene Control Group n = 35	Exercise Promotion Intervention Group n = 34	*Df*	t/X^2	*P-value*
Age	63.9 (8.7)	63.7 (9.2)	64.2 (8.3)	67	-0.21	0.838
Gender	84.1% F 15.9% M	80% F 20% M	88.2% F 11.8% M	1	0.87	0.35
Race	91.3% White/Caucasian 8.7% Other	94.3% White/Caucasian 5.7% Other	88.2% White/Caucasian 11.8% Other	3	3.46	0.326
Years of Education	16.2 (2.2)	16.5 (2.1)	15.9 (2.3)	67	1.24	0.22
GDS	4.1 (3.4)	4.4 (3.9)	3.71 (2.7)	67	0.87	0.387
BDI-2	5.4 (4.3)	5.9 (4.2)	4.8 (4.5)	67	1.06	0.293
STAI-State	29.7 (7.5)	30.3 (7.4)	29.1 (7.6)	67	0.68	0.502
STAI-Trait	30.5 (8.1)	31.9 (8.7)	29.2 (7.3)	67	1.39	0.171

Note: Mean (Standard Deviation) GDS= Geriatric Depression Scale; BDI-2 = Beck Depression Inventory- Second Edition; STAI = State Trait Anxiety Inventory

Table 3-3. Proposed study timeline for each replicate of participants

	Weeks 1-2	Weeks 3-18	Weeks 19-20
Pretesting			
Intervention			
Post-testing			

Table 3-4. Number of participants by age category, replicate, and experimental group

Age Category		Replicate Number								Total
		1	2	3	4	5	6	7	8	
Ages 50-64	Control				4	6	1	4	2	17
	Intervention				2	5	2	5	0	14
	Total				6	11	3	9	2	31
Ages 65 and older	Control	3	4	3			2	1	5	18
	Intervention	2	4	4			4	1	5	20
	Total	5	8	7			6	2	10	38
Replicate Grand Totals	Control	3	4	3	4	6	3	5	7	35
	Intervention	2	4	4	2	5	6	6	5	34
	Total	5	8	7	6	11	9	11	11	69
	Dates of active participation	6/06-1/07	3/07-6/07	3/07-6/07	4/07-7/07	4/07-8/07	6/07-10/07	12/07-4/08	1/08-5/08	

Table 3-5. Baseline and post-intervention study protocol

Domain	Measure
Primary Outcomes:	N-Back Task (Cohen et al., 1994)
Executive Control Cognitive Measures	Controlled Oral Word Association (COWA; Benton & Hamsher, 1989)
	Trail Making Test B (Trails B; Reitan, 1992)
	Letter-Number Sequencing of WMS-III (Wechsler, 1997)
Non-Executive Control Cognitive Measures	North American Adult Reading Test (NAART; Blair & Spreen, 1989)
	Trail Making Test A (Trails A) (Reitan, 1992)
	Logical Memory of WMS-III (Wechsler, 1997)
Proximal Outcomes: Psychosocial/Socio-emotional Measures: Emotional Well-being and Exercise Self Efficacy	Geriatric Depression Scale (GDS; Yesavage & Brink, 1983)
	Beck Depression Inventory- Second Edition (BDI-II; Beck, et al., 1996)
	State-Trait Anxiety Inventory (STAI; Spielberger, 1983)
	Exercise self-efficacy (EXSE; McAuley, 1993)
	Exercise barriers self-efficacy (BSE; McAuley, 1992)
Exercise Behavior Measures	Leisure Time Exercise Questionnaire (LTEQ ; Godin & Shephard, 1985)
	AE 120 pedometer (Yamax SW200 engine)
Aerobic Fitness Measure	Modified Balke Submax VO_2 (VO2)

Table 3-6. Schedule of intervention topics

Intervention week	Topic	Homework	Facilitator
1	Introduction to program and orientation to fitness facility. Computation of target heart rate.	Read *Health Benefits of Physical Activity*	Fitness center staff
2	Getting to Know You; Goal Setting Part I	What is exercise? Activity	Peer mentor
3	Goal Setting Part II	Write out your goal	Peer mentor
4	Defining exercise behavior: What is exercise? What is physical activity? How much? How hard? Types of exercise / exercise options	Read *Mental Imagery* Article Media Activity	Peer mentor
5	Barriers to Exercise	3 ways to overcome barriers to exercise	Peer mentor
6	All About Mental Imagery Define imagery Practice general example of imagery Practice exercise specific imagery	Fill out the Exercise Imagery Inventory	Peer mentor
7	Discuss Exercise Imagery Questionnaire and progress toward stated goals	None	Peer mentor
8	Getting Good at Exercise – accessing available supports to help you improve in your exercises of choice	Identifying Improvement Resources	Peer mentor
9	Revisiting barriers and goals: discuss "pesky" barriers and do status update on progress toward stated goals	Apply one suggestion for overcoming barrier(s) to exercise	Peer mentor
10	Group discussion of exercise behavior and accomplishments	None	Peer mentor
11	Sustainability: Continuing progress and maintaining success, accessing family/social support	None	Peer mentor
12	Discuss maintenance goals, long-term versus short-term goals. Review basics of goal-setting (Exercise facility membership ends)	Write out your goals for 3, 6 and 12 months	Peer mentor
13	No meeting		None
14	Share future goals; PARTY	None	Peer mentor
15	No meeting		None
16	No meeting		None

Note: (Giacobbi et al. 2008)

Table 3-7. Health hygiene control group design

Design:	Controls For:
o Didactic Information about health and disease topics (Alzheimer's disease, arthritis, balance problems, COPD, diabetes, etc.) o Free Fitness Facility Membership	o Assigns treatment effects to the unique didactic, motivation, and coaching components of the intervention o Controls for Social contact with study staff and group peers o Controls for out of house activity o Controls for retest effects o Controls for access to fitness facilities

Table 3-8. Schedule of control group topics

Control week	Topic	Facilitator
1	Introduction to program and orientation to fitness facility; Getting to know you	Fitness center staff
2	Exercise Why is exercise important? How much exercise is too much? What types of exercise are recommended?	Peer mentor
3	Osteoporosis What is osteoporosis? What are the warning signs? How do I prevent it?	Peer mentor
4	Alzheimer's Disease What do I look for? What affects the development of Alzheimer's?	Peer mentor
5	Cancer Screening What types of cancer should I be concerned about? How often should I be checked?	Peer mentor
6	Hearing Loss How common is hearing loss in older adults? How do I know if I have hearing loss?	Peer mentor
7	Arthritis What are the different types of arthritis? What can I do to treat arthritis?	Peer mentor
8	Vision Loss Is vision loss just part of getting older? Is all vision loss the same?	Peer mentor
9	Sleep Do older adults need as much sleep as young adults? How can I get a good night's sleep?	Peer mentor
10	Balance Problems What causes me to lose my balance? Is there anything I can do to prevent it?	Peer mentor
11	COPD* What is COPD? What type of exercise can I do if I have COPD?	Peer mentor
12	Heart Failure* How can I prevent heart failure? Who is most at risk for heart failure? (Exercise facility membership ends)	Peer mentor
13	No meeting	None
14	Share future goals; PARTY	Peer mentor
15	No meeting	None
16	No meeting	None

Note: (Giacobbi et al. 2008). For Replicate 8, weeks 11 and 12 included discussion of diabetes and high blood pressure health topics.

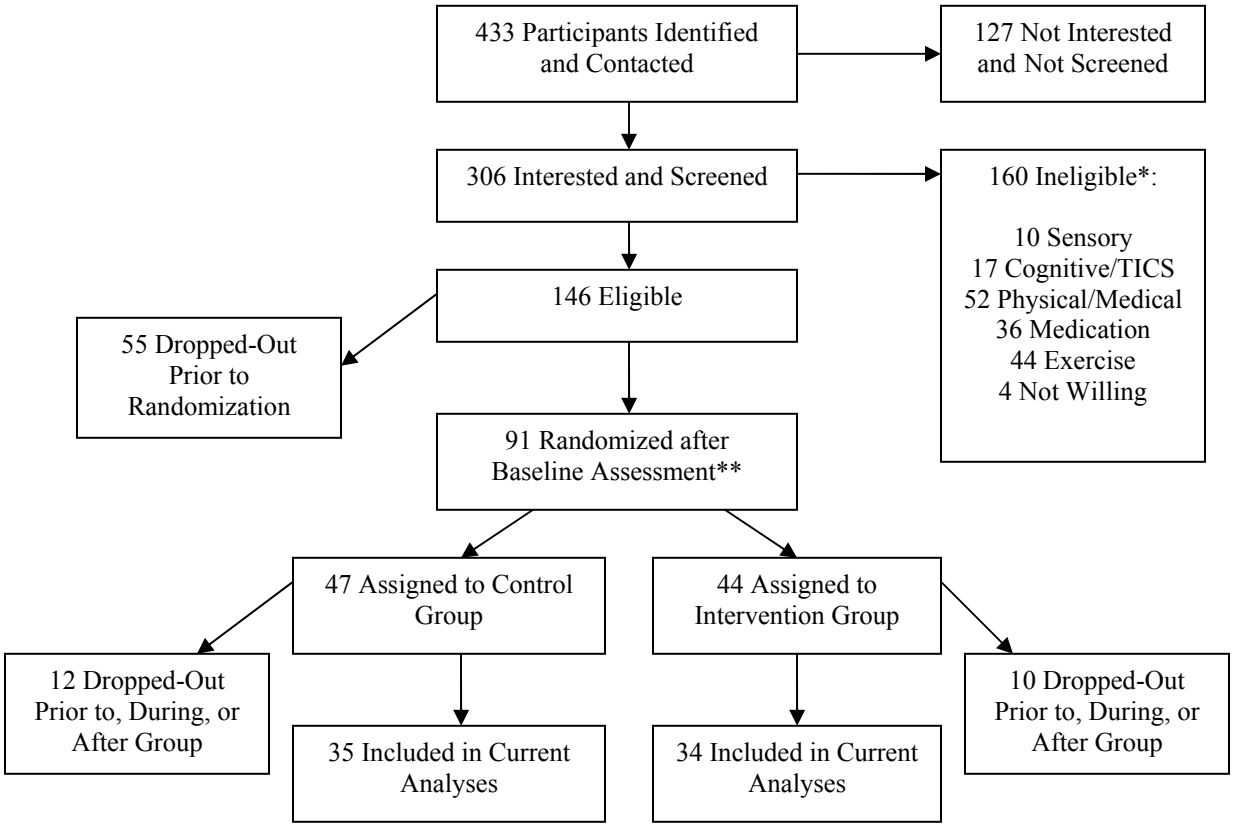


Figure 3-1. Flow chart of participants at each study phase.

Note: *Three participants met more than one exclusion criteria. **One participant was randomized and did not complete baseline assessment.

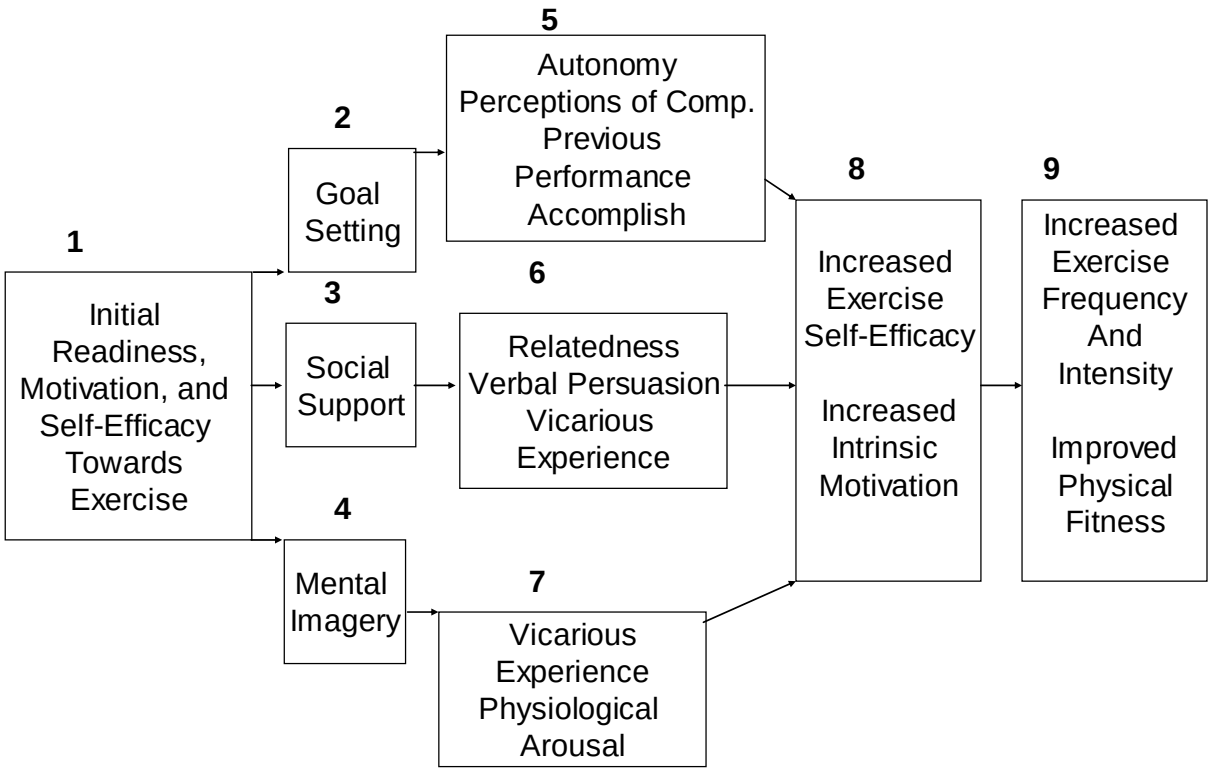


Figure 3-2. Guiding theoretical framework for exercise promotion group intervention (Buman, 2008)

CHAPTER 4
RESULTS

This study examining the effects of an exercise intervention on executive cognitive function involved a between (group assignment) and within (measure/instrument, occasion of measurement) subjects design. The primary outcome was cognitive performance, while proximal or mediating outcomes measured included physical fitness and self-perception (depression, well-being, and self-efficacy). The following results are presented with respect to two specific aims:

- To investigate whether, relative to matched non-exercising control participants, sedentary adults receiving a physical exercise promotion intervention experienced improvements in the primary outcome of cognitive function (particularly executive control processes).
- To determine the separate and joint roles of improvement in proximal outcomes (fitness, activity, and affect) in mediating exercise intervention effects on cognition.

Preliminary Analyses

Attrition of the sample

A total of 22 participants were considered "dropouts" in this study, resulting in the final sample of 69 participants. Of the 91 individuals that were randomized to either the control or intervention conditions following baseline assessment, 10 dropped out of the study prior to the 16-week study group period (Figure 3-1). One participant was randomized to a group with the expectation that pre-testing would be completed just prior to the first session; however, the participant did not undergo baseline assessment or remain in the study for the small group sessions. Post-randomization, an additional twelve participants completed part or all of the small group sessions and did not complete post-testing. As shown in Table 4-1, in comparing demographic characteristics of individuals who completed post-testing (n = 69) to those individuals who only completed pre-testing (n = 21), independent samples t-tests indicated that

the study's dropouts reported significantly higher depression symptoms. In addition, dropouts had significantly poorer performance on the Two-Back condition of the N-back task.

Missing Data

To preserve the present sample size with complete data, cell-specific mean replacement was implemented for three participants (4.3% of cases; Baltes & Mayer, 2001) on baseline and post-test cognitive variables (N-back and Logical Memory (LM)). Each missing data point for the cognitive variables was replaced with the mean value of scores obtained by individuals of the same gender, age group (i.e., 50-64 years old or 65 and older), and study group (control or intervention condition).

Each missing data point for baseline physical fitness/activity and self-efficacy variables was replaced with the mean value of those with the same gender, age group, and BMI category (i.e., BMI <18.5; 18.5-25; 25-30; and >30). This strategy was chosen as a better approximation of the physical variables, and because there were no baseline differences between the study groups on physical fitness, physical activity, or self-efficacy data (Buman, 2008). Six cases (8.7% of the sample) had missing data for the BMI variable. These data were replaced with mean BMI for individuals of the same gender and age group. Additionally, there was a total of 6 cases (8.7% of the sample) with missing baseline physical fitness data (pre-VO2 = 1 missing case), baseline physical activity (pre-LTEQ (leisure time exercise) = 1 missing case; pre-MVPA (minutes of moderate and vigorous physical activity) = 2 cases; and pre-mean pedometer steps = 4 missing cases), and/or baseline self-efficacy data (pre-EXSE (exercise self-efficacy) = 1 missing case). After BMI mean replacement, one case did not share the same gender, age group, and BMI category with anyone, thus that person's missing data (for pre-mean pedometer steps) were replaced with mean values of individuals of the same gender and age group.

Regarding post-test data, there was one case missing physical fitness (post-VO2) data at follow-up. However, for post-test physical activity and self-efficacy variables, there were substantially more missing data (post-LTEQ = 14 missing cases; post-mean pedometer steps = 17 missing cases; post-MVPA = 23 missing cases; post-BSE = 21 missing cases; and post-EXSE = 21 missing cases). Since physical activity and self-efficacy data were collected on a weekly basis, missing post-test values were replaced by the most recent available value for preceding weeks 13-16 (i.e., if week 16 was missing, this data point was replaced by the value for week 15, etc.). In the event that there were no available data, these cases were replaced with age, gender, and BMI group posttest means. There were 19 such cases without any data for weeks 13-16; thus by-cell mean replacement was completed for these individuals (post-LTEQ = 7 missing cases; post-mean pedometer steps = 10; post-MVPA = 2; post-BSE = 1 missing case; post-EXSE = 1 missing case).

Distributions of Dependent Variables and Outliers

Prior to analysis of the cognitive data, values that were ±3 standard deviations from the mean of each cognitive and psychosocial variable were set to missing and replaced with the mean value of individuals with the same gender, age group, and study group (as described above). There were 28 specific outlier values across all baseline and post-test cognitive variables. There were 12 total specific outlier values for baseline variables of LM Leaning Slope, Letter-Number Sequencing, Geriatric Depression Scale (GDS), Beck Depression Inventory-2 (BDI-II), One-Back Mean Reaction Time (RT), One-Back Mean Reaction Time (RT) Standard Deviation (SD), Two-Back Mean RT, Two-Back Mean RT SD, and Two-Back Number Correct. There were 16 total outliers on post-test measures of LM Leaning Slope, COWA, Trails B Time, GDS, BDI-II, STAI Trait Anxiety, One-Back Mean RT, One-Back Mean RT SD, One-Back Number Correct, Two-Back Mean RT, and Two-Back Mean RT SD.

Prior to outlier replacement, the distributions of all cognitive dependent variables were explored for skewness and kurtosis. Many executive cognitive were significantly skewed at baseline. Variables that were significantly positively skewed (absolute skewness values > 2) were as follows: Trails B Time (Skewness = 2.23), One-Back Mean RT SD (Skewness = 2.57), and Two-Back Mean RT (Skewness = 2.13). Variables that were significantly negatively skewed were One-Back Number Correct (Skewness = -5.48) and Two-Back Number Correct (Skewness = -3.88). Regarding kurtosis, variables that were significantly kurtotic (absolute valuates > 2) were as follows: Trails A Time (Kurtosis = 2.65), Trails B Time (Kurtosis = 6.01), One-Back Mean RT (Kurtosis = 3.35), One-Back Mean RT SD (Kurtosis = 8.25), One-Back Number Correct (Kurtosis = 33.40), Two-Back Mean RT (Kurtosis = 6.29), Two-Back Number Correct (Kurtosis = 20.67), GDS (Kurtosis = 4.77), and BDI-II (Kurtosis = 5.36).

After outlier mean replacement, the following baseline variables were significantly skewed positive: Trails B Time (Skewness = 2.23) and One-Back Mean RT SD (Skewness = 2.09). The only variable negatively skewed was One-Back Number Correct (Skewness = -5.48). Significant kurtosis values were observed for Trails A Time (Kurtosis = 2.65), Trails B Time (Kurtosis = 6.01), One-Back Mean RT SD (Kurtosis = 5.69), One-Back Number Correct (Kurtosis = 33.40), and Two-Back Number Correct (Kurtosis = 4.39). All skewness and kurtosis values (after outlier replacement) are presented in Table 4-2.

Baseline comparisons and correlations

Baseline independent samples t-tests for all cognitive, psychosocial (affect and self-efficacy), and physical fitness/activity data indicated no significant study group differences on study variables, except for EXSE ($t(67) = -3.289$; $p = .002$), with the exercise promotion intervention group reporting a higher mean level of exercise self-efficacy at baseline. However, with Bonferroni correction (.05/27 = 0.00185), this finding was no longer significant. Next,

correlations among all cognitive, psychosocial, and physical fitness/activity data were examined to determine their associations at baseline (Tables 4-3, 4-4, and 4-5). Overall, of the 153 correlations assessed, only 16 (10.5%) reached the $p < .05$ level of significance.

With regard to physical variables, bivariate correlations indicated modest significant positive relationships between the LTEQ and LM Recognition and Trails B Time, and a significant negative association between the LTEQ and LM Delayed Recall ($p < .05$). Thus, greater levels of leisure time exercise was associated with better LM Recognition scores, but worse Trails B Time and LM Delayed Recall performance, which was in the opposite direction of what would be expected. Furthermore, MVPA was significantly negatively associated with LM Immediate ($p < .05$) and Delayed Recall and LM Recognition ($p < .01$), also in the opposite direction.

Next, regarding psychosocial variables, the GDS, STAI-State Anxiety, and STAI-Trait Anxiety had significant positive correlations with the LTEQ ($p < .05$), suggesting that also counterintuitive to what would be expected, higher levels of leisure time exercise were associated with higher levels of GDS depression and state and trait anxiety symptoms. The GDS and STAI-Trait Anxiety measures were also significantly positively correlated with MVPA ($p < .01$), with higher minutes of moderate-vigorous physical activity associated with higher levels of GDS depression and trait anxiety. Furthermore, GDS depression symptoms were negatively correlated with LM Recognition scores ($p < .05$), and GDS depression symptoms were positively correlated with Trails B Time ($p < .01$). These correlations were in the directions that would be anticipated. Similarly as would be hypothesized, BDI-II depression symptoms were positively correlated with both Trails A ($p < .05$) and Trails B Time ($p < .01$). Regarding associations with anxiety scores, both STAI State and STAI Trait Anxiety variables showed negative associations

with LM Recognition at baseline ($p < .05$), indicated that, as would be expected, lower state and trait anxiety was associated with higher performance on the LM Recognition task.

Lastly, with regard to exercise-related self-beliefs, both BSE and EXSE scores were negatively associated with performance on LM Immediate Recall and LM Delayed Recall ($p < .05$). This suggested that, contrary to expectation, higher perceived self-efficacy was related to lower performance on both LM recall trials at baseline.

Next, baseline intercorrelations on all cognitive variables were computed to determine the inter-relatedness of all executive and non-executive cognitive variables (Table 4-6). Executive cognitive variables were expected to be more correlated with each other and less correlated with non-executive cognitive variables. Nevertheless, all executive cognitive measures except for One-Back Mean RT, Two-Back Mean RT, One-Back Number Correct, and Two-Back Mean RT SD were significantly correlated with two or more non-executive cognitive measures. Thus, these findings indicated that the executive cognitive variables were often more correlated with non-executive cognitive variables than with each other in this sample, suggesting the theoretical groupings of the cognitive measures was not supported empirically. It further suggested that the hypothesized separation of executive and non-executive measures with regard to treatment effects might not be supportable, given the lack of distinctiveness between the two domains.

Physical Fitness and Self-Efficacy Measures: Pre-Post Changes

Prior to examining the effect of the intervention on cognitive outcomes, the influence of the intervention on intended primary outcomes of the larger study (physical fitness/activity and exercise self-efficacy) was explored (Table 4-7 and Figure 4-1). While the present document considers the cognitive measures as primary outcomes *for this study*, the larger project in which it was embedded considered physical fitness and self-efficacy as primary outcomes and cognitive function as secondary outcomes. As such, the hypothesized improvements on cognitive

performance were expected to be dependent on the anticipated improvements in physical fitness and exercise self-efficacy.

First, the critical group-by-occasion effect was examined in univariate analyses of variance (ANOVA). This effect would address whether there were experimental group differences in exercise change over time, assessing the hypothesis of disproportionate improvement in the experimental group. The critical group-by-occasion interaction did not reach significance for any of the larger study's primary outcomes, suggesting that the intervention was not effective in producing disproportionate improvements in intended outcome variables. This suggested that a core assumption in the study's conceptual model (that the intervention would affect fitness and activity) was not supported. Despite this, the planned primary analyses were completed, to assess whether there was, nonetheless, a direct association between exercise group assignment and secondary cognitive outcomes.

It should be noted that there was a single between subjects effect for one measure: exercise self-efficacy (EXSE) ($F(1, 67) = 7.30$; $p = .01$; Partial $\eta^2 = 0.10$). The exercise promotion intervention group had significantly higher exercise self-efficacy than the control group, but this was true across both pre-test and post-test (i.e., it did not indicate the presence of group differences in self-efficacy change, which was hypothesized).

Independent of group assignment, significant within-subjects effects (i.e., occasion effects, or significant changes from pretest to posttest) were found for four measures. For the LTEQ ($F(1, 67) = 17.78$; $p < .001$; Partial $\eta^2 = 0.21$), MVPA($F(1, 67) = 17.98$; $p < .001$; Partial $\eta^2 = 0.21$), and VO2 ($F(1, 67) = 7.97$; $p = .006$; Partial $\eta^2 = 0.11$), participants across both groups improved across time on these measures. Somewhat unexpectedly, for exercise self-efficacy, the significant occasion effect ($F(1, 67) = 5.82$; $p = .02$; Partial $\eta^2 = 0.08$) indicated that, across

groups, participants experienced self-efficacy reductions over time. Cohen's *d* estimates of the time effects were as follows: LTEQ, $d = 0.51$; MVPA, $d = 0.51$; mean pedometer steps, $d = 0.22$; VO2, $d = 0.34$; BSE, $d = -0.12$; EXSE, $d = -0.29$, with the latter two coefficients reflecting poorer mean self-efficacy at post-test. It should be noted that all of these effects were small to medium (Cohen, 1992). Examining the means in Figure 4-1, with a baseline V02 of 28, and an average 2 point increase, the mean level fitness increase in this study represented only 7%. The self-reported gain in metabolic equivalents, based on self-reported activity, was an increase of 4 points over a baseline value of 6, which represented a more sizeable 67% increase.

Anxiety and Depression: Pre-Post Changes

Since changes in psychosocial variables of anxiety and depression were hypothesized to be one route by which changes in executive cognitive function would occur, these variables were included in a repeated-measures MANOVA to determine intervention effects on these variables (Table 4-8 and Figure 4-2). Again, the critical occasion-by-group interaction did not reach significance (Wilks' $\Lambda = 0.94$; $F(4, 64) = 1.00$, $p < 0.41$; Partial $\eta 2 = 0.06$). Turning to the main effects, while there was no overall significant between subjects effect (Wilks' $\Lambda = 0.97$; $F(4, 64) = 0.41$, $p = .80$; Partial $\eta 2 = 0.03$), there was a significant overall within subjects effect of occasion (Wilks' $\Lambda = 0.71$; $F(4, 64) = 6.68$, $p < .001$; Partial $\eta 2 = 0.29$). Post-hoc univariate ANOVAs (Table 4-9) on each anxiety and depression measure revealed significant reductions over time for the STAI Trait Anxiety ($F(1, 67) = 11.58$; $p < .001$; Partial $\eta^2 = 0.15$) and BDI-II measures ($F(1, 67) = 22.68$; $p < .001$; Partial $\eta^2 = 0.25$), suggesting that there were improvements in anxiety and depression symptoms for the combined groups. Cohen's *d* estimates of effect sizes for anxiety and depression within-subjects effects were small to medium

and are as follows: GDS, $d = 0.01$; BDI-II, $d = 0.58$; STAI State Anxiety, $d = 0.13$; and STAI-Trait Anxiety, $d = 0.41$.

Aim 1: Exercise Promotion Intervention-Related Improvements in Cognition

Study Group Comparisons

To address the first aim, repeated-measures multivariate analyses of variance (MANOVA) was employed to determine whether there were cognitive changes with time, across instruments, both within and between groups. Of critical interest was the occasion-by-group interaction, which addressed whether subjects receiving the exercise promotion intervention improved more than "health hygiene" controls. As shown in previous studies, it was hypothesized that participants receiving the exercise intervention would show improved performance on cognitive measures, particularly those assessing executive control processes, relative to control participants.

Two repeated-measures MANOVAs were conducted to test this hypothesis, one for non-executive cognitive measures and a second for executive cognitive tests (Table 4-10). First, for the non-executive cognitive measures, there was no significant occasion-by-group interaction as hypothesized, suggesting that the exercise promotion intervention group did not improve significantly more than the health hygiene control group across time (Wilks' $\Lambda = 0.92$; $F(6, 62) = 0.51$, $p = .80$; Partial $\eta^2 = 0.08$). Next, there was no significant between subjects (intervention group) effect (Wilks' $\Lambda = 0.90$; $F(6, 62) = 1.12$; $p = 0.36$; Partial $\eta^2 = 0.10$). This suggests there were no overall mean group differences in cognitive performance on non-executive measures. There was a significant within-subjects effect of occasion (Wilks' $\Lambda = 0.55$; $F(6, 62) = 8.48$, $p < .001$; Partial $\eta^2 = 0.45$), suggesting that overall, there was an effect of time on performance. Specifically, the entire sample, regardless of random group assignment, changed significantly in

their overall performance on non-executive measures from baseline to post-test. The direction of this finding will be explored in follow-up univariate analyses presented below.

Next, for the executive cognitive measures, a second repeated-measures MANOVA again showed no significant occasion-by-group interaction, suggesting that the exercise promotion intervention group did not improve significantly more or less than the health hygiene control group over time (Wilks' $\Lambda = 0.86$; $F(9, 59) = 0.96$, $p = .48$; Partial $\eta^2 = 0.14$). There was no significant between subjects effect (Wilks' $\Lambda = 0.97$; $F(9, 59) = 0.31$, $p = .97$; Partial $\eta^2 = 0.03$), but there was a significant within-subjects effect of occasion. (Wilks' $\Lambda = 0.65$; $F(9, 59) = 4.01$, $p < .001$; Partial $\eta^2 = 0.35$). Follow-up univariate analyses were conducted to ascertain the direction of the effect and are explored below.

The presence of significant overall occasion effects required follow-up univariate analyses to ascertain their directions in each measure (Tables 4-11 and 4-12). Beyond occasion analyses, these follow-up analyses also examined the between-group effect and interactions; while these effect examinations were not "protected" by the omnibus test, they were still useful as follow-up, exploratory analyses. The analyses that follow are reported with their original, uncorrected probabilities, since there is little consensus on alpha adjustment for these follow-up tests (Tabachnick & Fidell, 2007). However, with six non-executive measures, a simple Bonferroni correction would require probabilities of $p = 0.05/6 = 0.0083$ to be labeled as significant. Similarly, with nine executive measures, a simple Bonferroni correction would require probabilities of $p = 0.05/9 = 0.0056$ to be labeled as significant. The implications of these adjustments will be considered in the discussion chapter that follows.

While there were no significant between-subjects effects, there was an occasion-by-group interaction for one cognitive measure (COWA), suggesting intervention group participants

improved significantly more on this measure than did controls over time ($F(1, 67) = 4.15$; $p < .046$; Partial $\eta^2 = 0.06$); Table 4-12 and Figure 4-4). To examine this further, a pre-post COWA mean change score was computed for each participant and used as the dependent variable in a follow-up, one-way analysis of variance to examine whether there was a significant group difference in the magnitude of change on this measure. The results indicated a significant difference in the magnitude of mean change in COWA scores over time, with the intervention group demonstrating greater improvement in COWA scores over time than controls (mean (SD) change for intervention group: 4.44 (6.46) ; for control group: 0.58 (9.02); $F(1, 67) = 4.15$; $p = .046$, Partial $\eta^2 = 0.06$).

Next, there were within-subjects effects of occasion for several cognitive variables. Tables 4-10 and 4-11 show *F* – statistics, degrees of freedom, and significance values for the within-subjects effects of each repeated-measures ANOVA. These cognitive variables include NAART, Trails A, and LM Immediate and Delayed recall (non-executive measures) and COWA, One-Back Mean RT, Two-Back Mean RT, and Two-Back Mean RT SD (executive measures). Estimated marginal means for each group are presented in Table 4-13, and Figures 4-3 and 4-4 display line graphs of estimated marginal means by group. Overall, there were significant improvements on these cognitive variables for the entire sample, regardless of study group, over time. These practice/occasion effects were of small to medium size for most of the cognitive variables (Cohen, 1992). Cohen's d estimates of effect were as follows: NAART, $d = 0.40$; Trails A, $d = 0.32$; LM Immediate Recall, $d = 0.61$; LM Delayed Recall, $d = 0.67$; LM Recognition, $d = 0.27$; LM Learning Slope, $d = 0.13$; COWA, $d = 0.31$; Trails B, $d = 0.09$; Letter Number Sequencing, $d = 0.05$; One-Back Mean RT, $d = 0.41$; One-Back Mean RT SD, $d = 0.49$;

One-Back Number Correct, d = 0.20; Two-Back Mean RT, d =0.17; Two-Back Mean RT SD, d = 0.35; and Two-Back Number Correct, d = 0.34.

Reliable Change

As follow up analyses, reliable change index scores were calculated to examine intraindividual trajectories of change. The goal was to determine the proportion of the sample that experienced sizable gains above the usual effect of practice that would be observed in an untreated control group. This analysis allowed the subset of participants who experienced especially large improvement to be identified to determine whether the proportion of such participants differed by study group.

Traditional reliable change index scores (RCI) use published normative data to determine a confidence interval (90% or 95% are commonly used) that indicates the expected, normal variation of change scores. In this calculation, the average post-test score for each measure was subtracted from the average baseline scores and divided by the standard error of the difference between scores ($[(SD2x + SD2y)(1-rxy)]^{1/2}$). The baseline change, around which the RCI confidence interval is computed, is traditionally zero.

A modified reliable change score, employed in this study, assumes that the mean level of practice-related gain in the control group serves as the baseline around which the reliable change confidence interval should be established. This is done because even untreated participants typically experience some gain or change. When RCI scores are adjusted for practice (RCI-P), performance of a control condition is taken into account by adjusting the RCI for the average change in scores experienced by the control group. The RCI-P scores were calculated for the present sample by adding and subtracting the average change in scores to each RCI-P. Because of the conservative nature of the RCI-P scores, an alpha criterion of p = .10 was used. Table 4-14 displays, for each measure, the pre-test and post-test standard deviations, correlations between

pre-test and post-test scores, the standard error of the difference, and the standard error of the difference times 1.64 (the criterion of significance at $p = .10$). Also, the control group's mean change scores and the thresholds for reliable decline and reliable improvement, adjusted for the control group's mean change, are presented.

Upon examination of the present sample's scores to determine how many individuals reliably changed from pre- to post-test, there was little evidence for reliable improvement across cognitive measures (Tables 4-15 and 4-16). Logical Memory Immediate Recall was most sensitive to reliable improvement (24.6% of the sample); however, relative to controls, fewer intervention participants experienced reliable improvement (28.6% of controls improved versus 20.6% of intervention participants), although this difference was not significant (nor was any other tested) using chi-square analysis. Overall, most participants showed no reliable change between baseline and post-test. In addition, a small number of participants demonstrated reliable decline across measures.

Exploratory Analyses: Age Group Comparisons

As additional exploratory, follow-up analyses, repeated-measures MANOVAs were conducted, using age group as a second between subjects factor, to determine whether intervention effects might be moderated by age (i.e., Did younger participants experience more improvement than older participants? Did the occasion-by-group interaction exist for one age group, but not for the other?). This time, two three-way repeated-measures MANOVAs were conducted (Table 4-15). The repeated-measures MANOVA for non-executive cognitive measures demonstrated, as with the previous analyses, no significant interactions for occasion-by-study group (Wilks' $\Lambda = 0.92$; $F(4, 60) = 0.83$, $p = .55$; Partial $\eta^2 = 0.08$), occasion-by-age group, (Wilks' $\Lambda = 0.97$; $F(4, 60) = 0.34$, $p = 0.91$; Partial $\eta^2 = 0.03$), or occasion-by-study group-

by age (Wilks' Λ= 0.94; F(4, 60) = 0.63, p = .70; Partial η^2 = 0.06). There was also no study group-by-age interaction (Wilks' Λ= 0.90; F(6, 60) = 1.17, p = .33; Partial η^2 = 0.10). In addition, overall study group (Wilks' Λ= 0.89; F(6, 60) = 1.20, p = .32; Partial η^2 = 0.11) and overall age group effects (Wilks' Λ= 0.83; F(6, 60) = 2.09, p = 0.07; Partial η^2 = 0.17) did not reach significance. Although, it is worth nothing that the age group effect approached our criterion of significance (p = .05). As with the initial multivariate analyses for study aim one, there was a significant within-subjects effect (Wilks' Λ= 0.55; F(6, 60) = 8.11, p <.001; Partial η^2 = 0.45).

Next, for the executive cognitive measures there were no significant multivariate occasion-by-study group (Wilks' Λ= 0.86; F(9, 57) = 1.01, p = 0.44; Partial η^2 = 0.14), occasion-by-age group (Wilks' Λ= 0.89; F(9, 57) = 0.80, p = 0.62; Partial η^2 = 0.11), or occasion-by-study group-by-age group (Wilks' Λ= 0.83; F(9, 57) = 1.34, p = 0.24; Partial η^2 = 0.17) interactions. A study group-by-age group interaction effect approached significance (Wilks' Λ= 0.77; F(9, 57) = 1.91, p = 0.07; Partial η^2 = 0.23). While there was no effect of study group (Wilks' Λ= 0.96; F(9, 57) = 0.27, p = .98; Partial η^2 = 0.04), there was a significant age group effect (Wilks' Λ= 0.73; F(9, 57) = 2.33, p = 0.03; Partial η^2 = 0.27). Additionally, there was a significant within-subjects effect for occasion (Wilks' Λ= 0.64; F(9, 57) = 3.58, p < .001; Partial η^2 = 0.36), suggesting that overall, there was an effect of time on performance.

Follow-up univariate ANOVAs are presented in Tables 4-16 and 4-17, and estimated marginal means are displayed in Table 4-18. The only non-executive cognitive variable showing an age group effect was Trails A Time, indicating that the younger age group (50-64 year olds) performed this task significantly faster than the 65 and older group (score was number of seconds). The non-executive variables showing significant within-subjects effects included the

NAART, Trails A Time, LM Immediate Recall, LM Delayed Recall, and LM Recognition, indicating all participants significantly improved across occasions on these measures, regardless of study group random assignment (Figure 4-5). There was a study group-by-age group interaction effect for LM Delayed Total Recall, with the older individuals in the exercise promotion intervention group performing significantly better than younger individuals (regardless of study group) and older controls.

There was a study group-by-age group interaction for the One-Back Mean RT SD (Figure 4-6). Younger control group participants were significantly less inconsistent in their mean reaction time on this measure than younger intervention participants and all older participants. Additionally, there was an age-by-occasion interaction effect for the One-Back Mean RT task. This interaction effect suggested that younger participants improved significantly more than older participants in mean reaction time over time. Finally, there was a study group-by-age group-by-occasion interaction for Trails B Time. Younger intervention group participants improved significantly more on this task than their younger control group counterparts and older participants of both study groups.

Executive cognitive variables with significant age group effects were the One-Back Mean RT, Two-Back Mean RT, and Two-Back Mean RT SD. These findings indicate that the younger age group had significantly faster mean reaction time and were significantly less inconsistent in their mean reaction time across occasions, when compared to their older counterparts. Further, within-subjects effects for executive cognitive variables were the COWA, One-Back Mean RT, One-Back Mean RT SD, Two-Back Mean RT SD, and Two-Back Number Correct. All participants improved significantly on these measures across time (i.e., there was improvements

in mean reaction time on the One-Back Mean RT and less inconsistency on the One-Back and Two-Back RT SD variables).

Aim 2: Cognitive changes correlated with activity, fitness and psychosocial changes

In the aforementioned specific aims, examination of whether intervention-affected changes in fitness, activity, and self-perception variables might mediate expected intervention-related changes in cognitive variables (particularly executive outcomes) was originally planned. However, the general absence of intervention effects on cognitive outcomes renders this mediator analysis unwarranted (per Baron & Kenney, 1986).

Nonetheless, since the study results indicated at least practice-related improvement on most cognitive variables, and since participants in both exercise groups did have access to facilities and support to increase their activity, the associations between observed changes in cognition and changes in activity, fitness, or self-belief variables were nonetheless investigated. This was done to determine whether there was any support that improvements in exercise-related outcomes would be associated with cognitive improvements, regardless of group membership (since there was evidence that both groups experienced VO2 and leisure time activity improvements).

Consequently, to address the second aim, pre-post change scores across cognitive, self-perception, and fitness variables were computed and correlated (Tables 4-21, 4-22, and 4-23). Bivariate correlations between physical fitness and activity and cognitive performance indicated a significant positive correlation between change in LM Immediate Recall score and change in VO2 ($r = 0.31$, $p < .05$). This suggests that improvements in immediate recall of the LM stories were positively associated with improvements in cardiorespiratory fitness over time. There were significant negative correlations between the LTEQ and One-Back Mean RT SD ($r = -0.25$, $p < .05$) and mean pedometer steps and LM Learning Slope ($r = -0.29$, $p < .05$). These findings

indicate inverse relationships between these sets of variables, such that improved leisure time exercise was associated with decreased inconsistency in mean reaction time on the One-Back task, and improved mean pedometer steps was related to reduced learning slope across testing occasions. It is important to note that these significant relationships represent the minority of those tested. For the 60 correlations tested between physical fitness/activity and cognition, only 3 (5%) reached significance.

Next, in correlating changes in physical fitness/activity and psychosocial variables, the only significant association was between the GDS and VO2 ($r = -0.25$, $p < .05$). This inverse relationship indicated that a decrease in GDS depression symptoms was associated with an increase in cardiorespiratory fitness. Of the 24 correlations between physical fitness/activity and self-perception, only 1 (4.2%) was significant. Finally, correlations between psychosocial changes and cognitive score changes over time revealed three significant negative correlations between changes in GDS depression and LM Delayed Recall ($r = -0.24$, $p < .05$), BDI-II depression and Two-Back Mean RT SD ($r = -0.26$, $p < .05$), and BSE and Two-Back Number Correct ($r = -0.28$, $p < .05$). These negative correlations indicated that reduced GDS depression symptoms was associated with improved delayed recall of the LM stories over time, reduced BDI-II depression symptoms was related to increased reaction time inconsistency on the Two-Back task over time, and higher self-efficacy on the BSE was related to an improvement in the number correct on the Two-Back task over time. As with fitness variables, only the minority of associations tested reached the $p < .05$ criterion of significance. Of the 90 correlations tested between self-perception and cognition, only 3 (3.3%) reached significance. It should also be noted that, given the total number of correlations examined, if the *p*-values for these correlations were Bonferroni-corrected, the critical value of alpha would be $p = .05/176 = 0.00029$.

Table 4-1. Baseline mean comparison of study completers and dropouts

	Overall n = 90	Completers n = 69	Dropouts n = 21	*Df*	t/X^2	*p*-value
Age	63.6 (8.5)	63.9 (8.7)	62.3 (7.7)	88	-0.755	0.452
Gender	82.2% F 17.8% M	84.1% F 15.9% M	76.2% F 23.8% M	1	0.682	0.409
Race	90.0% White/Caucasian 10.0% Other	91.3% White/Caucasian 8.7% Other	85.7% White/Caucasian 14.3% Other	4	4.752	0.314
Years of Education	16.1 (2.2)	16.2 (2.2)	15.9 (2.4)	88	-0.504	0.615
VO2	28.1 (7.9)	27.9 (7.5)	29.0 (9.5)	88	0.544	0.588
GDS	4.8 (4.0)	4.1 (3.4)	7.1 (5.1)	88	3.228	<.001
BDI-II	6.3 (4.8)	5.4 (4.3)	9.3 (5.2)	88	3.469	<.001
STAI-State	30.3 (7.9)	29.7 (7.5)	32.2 (9.1)	88	1.282	0.200
STAI-Trait	31.5 (8.8)	30.5 (8.1)	34.9 (10.2)	88	2.010	0.470
NAART	113.2 (6.5)	113.1 (6.5)	113.5 (6.6)	88	0.293	0.770
LM Immediate Recall	41.0 (8.8)	41.0 (8.6)	41.0 (9.9)	88	-0.026	0.979
LM Delayed Recall	25.9 (7.5)	25.8 (7.5)	26.4 (7.7)	88	0.317	0.752
LM Learning Slope	4.01 (2.5)	4.1 (2.7)	4.2 (1.8)	88	0.194	0.847
LM Recognition	26.4 (2.4)	26.4 (2.3)	26.7 (2.5)	88	0.531	0.597
Trails A Time	31.6 (10.7)	32.5 (11.1)	28.4 (8.6)	88	-1.583	0.117
COWA	39.5 (10.4)	39.5 (10.8)	39.5 (9.2)	88	0.017	0.986
Trails B Time	78.1 (38.1)	81.0 (39.9)	68.5 (30.1)	88	-1.322	0.190
Letter-Num. Sequencing	10.7 (2.6)	10.5 (2.6)	10.9 (2.5)	88	0.582	0.562
One-Back Mean RT	866.4 (203.5)	847.4 (199.8)	928.6 (207.8)	88	1.616	0.110

Note: Mean (Standard Deviation). [a]There were unequal variances for these measures. LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-1 Continued.

	Overall n = 90	Completers n = 69	Dropouts n = 21	*Df*	t/X^2	*p*-value
One-Back Mean RT SD	361.1 (313.1)	330.3 (224.9)	462.4 (500.4)	88	1.712	0.090
One-Back Number Correct	96.1 (12.4)	95.5 (14.1)	98.0 (3.3)	88	0.799	0.427
[a]Two-Back Mean RT	1521.2 (796.7)	1756.0 (748.2)	749.9 (325.7)	77.2	-8.769	<.001
[a]Two-Back Mean RT SD	1058.9 (713.3)	912.8 (538.1)	1538.9 (981.7)	23.8	2.797	0.010
[a]Two-Back Number Correct	71.8 (34.8)	89.6 (14.3)	13.3 (3.3)	85	-40.843	<.001

Note: Mean (Standard Deviation). [a]There were unequal variances for these measures. LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-2. Distributions of dependent variables at baseline

n= 69	Mean	Std. Deviation	Skewness	Std. Error	Kurtosis	Std. Error
NAART	113.06	6.46	-0.33	0.29	-0.36	0.57
LM Immediate Recall	41.01	8.57	-0.08	0.29	-0.19	0.57
LM Delayed Recall	25.78	7.52	-0.08	0.29	-0.10	0.57
LM Learning Slope	4.07	2.67	0.18	0.29	0.62	0.57
LM Recognition	26.35	2.34	-0.46	0.29	-0.37	0.57
COWA	39.48	10.84	0.32	0.29	0.22	0.57
Trails A time	32.55	11.10	*1.39	0.29	*2.65	0.57
Trails B time	81.00	39.90	*2.23	0.29	*6.01	0.57
Letter-Number Sequencing	10.53	2.62	*0.87	0.29	0.98	0.57
One-Back Mean RT	837.28	175.35	*0.76	0.29	*1.32	0.57
One-Back Mean RT SD	315.41	184.63	*2.09	0.29	*5.69	0.57
One-Back Number Correct	95.52	14.05	*-5.48	0.29	*33.40	0.57
Two-Back Mean RT	1667.01	530.85	*0.83	0.29	0.18	0.57
Two-Back Mean RT SD	888.84	492.85	*1.33	0.29	*1.66	0.57
Two-Back Number Correct	90.87	9.51	*-1.82	0.29	*4.39	0.57
GDS	4.06	3.36	*1.28	0.29	*1.51	0.57
BDI-II	5.35	4.33	*1.07	0.29	1.06	0.57
STAI State	29.71	7.51	0.51	0.29	-0.50	0.57
STAI Trait	30.54	8.09	*1.07	0.29	0.78	0.57
VO2	27.89	7.45	-0.28	0.29	-0.66	0.57
LTEQ	6.08	5.86	*2.56	0.29	*9.82	0.57
MVPA	76.23	126.59	*4.22	0.29	*23.86	0.57
Pedometer Steps	6145.41	2860.41	*0.83	0.29	0.88	0.57
BSE	67.57	23.48	*-0.65	0.29	-0.50	0.57
EXSE	74.14	27.03	*-0.96	0.29	-0.03	0.57

Note: *$p < .05$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation; GDS = Geriatric Depression Scale; BDI-2 = Beck Depression Inventory- Second Edition; STAI = State Trait Anxiety Inventory; VO2 = Modified Balke Submax; BSE = Barriers Self Efficacy Scale; EXSE = Exercise Self-Efficacy Scale

Table 4-3. Baseline correlations between physical activity, physical fitness, and cognitive variables

	VO2	LTEQ	Mean Pedometer Steps	MVPA
NAART	-0.107	0.100	-0.111	0.128
Trails A Time	-0.111	0.150	0.010	0.140
LM Immediate Recall	0.105	-0.199	-0.122	*-0.306
LM Delayed Recall	0.168	*-0.240	-0.171	**-0.317
LM Recognition	0.085	*0.295	-0.122	**-0.365
LM Learning Slope	-0.004	-0.206	-0.226	-0.105
COWA	-0.090	0.036	0.130	0.093
Trails B Time	-0.171	*0.269	-0.015	0.177
Letter Number Sequencing	0.072	-0.073	0.101	-0.111
One-Back: Mean RT	0.008	0.018	-0.036	-0.001
One-Back: Mean RT SD	0.020	-0.039	-0.024	-0.001
Two-Back: Number Correct	0.010	-0.013	0.018	-0.039
Two-Back: Mean RT	-0.132	-0.087	-0.063	-0.160
Two-Back: Mean RT SD	-0.189	-0.066	-0.062	-0.122
Two-Back: Number Correct	0.136	-0.032	0.125	0.005

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation; VO2 = Modified Balke Submax; LTEQ = Leisure Time Exercise Questionnaire; MVPA = Minutes of Moderate and Vigorous Physical Activity

Table 4-4. Baseline correlations between physical fitness, physical activity, and psychosocial variables

	VO2	LTEQ	Mean Pedometer Steps	MVPA
GDS	-0.128	*0.286	0.014	**0.397
BDI-2	0.137	0.154	0.086	0.120
STAI State Anxiety	-0.136	*0.257	0.092	0.215
STAI Trait Anxiety	0.017	*0.291	0.064	**0.372
BSE	-0.004	0.005	0.071	-0.012
EXSE	0.059	0.144	0.146	0.111

Note: *$p < .05$; **$p < .01$; GDS = Geriatric Depression Scale; BDI-2 = Beck Depression Inventory- Second Edition; STAI = State Trait Anxiety Inventory; BSE = Barriers Self Efficacy Scale; EXSE = Exercise Self-Efficacy Scale; VO2 = Modified Balke Submax; LTEQ = Leisure Time Exercise Questionnaire; MVPA = Minutes of Moderate and Vigorous Physical Activity

Table 4-5. Baseline correlations between psychosocial and cognitive variables

	GDS	BDI-2	STAI State Anxiety	STAI Trait Anxiety	BSE	EXSE
NAART	0.185	0.086	-0.005	0.124	-0.130	-0.197
Trails A Time	0.146	*0.249	0.166	0.059	0.129	-0.001
LM Immediate Recall	-0.017	-0.003	-0.139	0.001	*-0.307	*-0.248
LM Delayed Recall	-0.033	-0.028	-0.144	-0.010	**-0.320	*-0.288
LM Recognition	*-0.239	-0.187	*-0.253	*-0.251	-0.074	-0.055
LM Learning Slope	-0.009	-0.117	0.050	-0.040	0.008	-0.048
COWA	0.080	-0.012	-0.002	0.125	-0.018	0.005
Trails B Time	**0.341	**0.314	0.142	0.169	0.112	-0.065
Letter Number Sequencing	0.095	0.131	-0.031	0.066	0.023	-0.065
One-Back: Mean RT	0.008	0.017	0.022	-0.101	-0.063	-0.138
One-Back: Mean RT SD	0.023	0.014	-0.010	-0.031	0.074	-0.019
One-Back: Number Correct	-0.036	-0.057	-0.136	-0.039	0.210	0.099
Two-Back: Mean RT	-0.069	-0.005	0.099	-0.133	0.137	0.034
Two-Back: Mean RT SD	0.057	0.029	0.068	-0.101	0.174	0.052
Two-Back: Number Correct	0.024	-0.123	-0.071	-0.008	0.002	0.054

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation; GDS = Geriatric Depression Scale; BDI-2 = Beck Depression Inventory- Second Edition; STAI = State Trait Anxiety Inventory; BSE = Barriers Self Efficacy Scale; EXSE = Exercise Self-Efficacy Scale

Table 4-6. Cognitive intercorrelations at baseline

	COWA	Trails B Time	Letter-Number Sequencing	One-Back: Mean RT	One-Back: Mean RT SD
COWA	1				
Trails B Time	-0.115	1			
Letter Number Sequencing	**0.488	*-0.252	1		
One-Back: Mean RT	-0.181	0.155	-0.182	1	
One-Back: Mean RT SD	-0.138	0.236	-0.210	**0.727	1
One-Back: Number Correct	-0.062	-0.136	-0.047	0.034	-0.050
Two-Back: Mean RT	-0.093	0.095	-0.105	0.204	0.157
Two-Back: Mean RT SD	-0.156	0.104	-0.114	*0.241	0.219
Two-Back: Number Correct	*0.252	*-0.289	0.205	*-0.270	*-0.237
NAART	**0.394	-0.031	0.178	-0.023	-0.234
Trails A Time	*-0.282	**0.519	*-0.381	0.128	0.045
LM Immediate Recall	0.210	-0.225	*0.238	-0.177	**-0.381
LM Delayed Recall	*0.280	**-0.359	*0.243	-0.141	**-0.367
LM Recognition	0.214	*-0.293	0.077	-0.119	-0.205
LM Learning Slope	-0.053	-0.138	0.076	0.062	0.117

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-6 Continued.

	One-Back: Number Correct	Two-Back: Mean RT	Two-Back: Mean RT SD	Two-Back: Number Correct	NAART
One-Back: Number Correct	1				
Two-Back: Mean RT	0.173	1			
Two-Back: Mean RT SD	0.142	*0.841	1		
Two-Back: Number Correct	*0.277	0.057	0.001	1	
NAART	-0.033	-0.109	-0.227	0.195	1
Trails A Time	-0.005	0.048	0.002	*-0.300	0.055
LM Immediate Recall	-0.083	-0.142	-0.182	0.182	**0.335
LM Delayed Recall	0.011	-0.197	-0.190	*0.273	**0.337
LM Recognition	0.106	-0.038	-0.063	*0.295	0.180
LM Learning Slope	0.164	0.008	0.015	-0.054	-0.006

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-6 Continued

	Trails A Time	LM Immediate Recall	LM Delayed Recall	LM Recognition	LM Learning Slope
Trails A Time	1				
LM Immediate Recall	-0.068	1			
LM Delayed Recall	-0.204	**0.840	1		
LM Recognition	-0.118	**0.578	**0.654	1	
LM Learning Slope	-0.191	-0.145	-0.115	-0.031	1

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-7. Univariate analyses of variance for physical activity and fitness

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
Between Subjects: Study Group					
Mean Pedometer Steps	1	67	0.00	0.96	0.00
LTEQ	1	67	0.45	0.50	0.01
VO2	1	67	0.68	0.41	0.01
MVPA	1	67	0.37	0.54	0.01
BSE	1	67	3.37	0.07	0.05
EXSE	1	67	7.30	**0.01	0.10
Within-Subjects: Occasion					
Mean Pedometer Steps	1	67	3.19	0.08	0.05
LTEQ	1	67	17.78	**0.00	0.21
VO2	1	67	7.97	**0.01	0.11
MVPA	1	67	17.98	**0.00	0.21
BSE	1	67	1.05	0.31	0.02
EXSE	1	67	5.82	*0.02	0.08
Interactions: Study Group by Occasion					
Mean Pedometer Steps	1	67	0.95	0.33	0.01
LTEQ	1	67	0.00	1.00	0.00
VO2	1	67	0.36	0.55	0.01
MVPA	1	67	0.59	0.45	0.01
BSE	1	67	0.25	0.62	0.00
EXSE	1	67	1.71	0.19	0.02

Note: *$p < .05$; **$p < .01$; LTEQ = Leisure Time Exercise Questionnaire; VO2 = Modified Balke Submax; BSE = Barriers Self Efficacy Scale; EXSE = Exercise Self-Efficacy Scale; MVPA = Minutes of Moderate and Vigorous Exercise

Table 4-8. Repeated-measures multivariate analysis of variance on anxiety and depression measures[a]

Effects	Wilks' Lambda	*F*	Hypothesis df	Error df	*P*-value	Partial η^2
Between Subjects: Study Group	0.97	0.41	4	64	0.80	0.03
Within Subjects: Occasion	0.71	6.68	4	64	**0.00	0.29
Interaction: Study Group by Occasion	0.94	1.00	4	64	0.41	0.06

Note. *$p < .05$; **$p < .01$; [a]There were unequal variance-covariance matrices for these measures.

Table 4-9. Follow-up univariate analyses of variance for depression and anxiety measures

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
Between-Subjects: Study Group					
STAI State Anxiety	1	67	0.04	0.85	0.00
STAI Trait Anxiety	1	67	1.10	0.30	0.02
GDS	1	67	1.33	0.25	0.02
BDI-2	1	67	1.46	0.23	0.02
Within-Subjects: Occasion					
STAI State Anxiety	1	67	1.07	0.31	0.02
STAI Trait Anxiety	1	67	11.58	**0.00	0.15
GDS	1	67	0.01	0.91	0.00
BDI-2	1	67	22.68	**0.00	0.25
Interactions: Study Group by Occasion					
STAI State Anxiety	1	67	1.53	0.22	0.02
STAI Trait Anxiety	1	67	2.14	0.15	0.03
GDS	1	67	0.30	0.58	0.00
BDI-2	1	67	0.03	0.87	0.00

Note: *$p < .05$; **$p < .01$; GDS = Geriatric Depression Scale; BDI-2 = Beck Depression Inventory- Second Edition; STAI = State Trait Anxiety Inventory

Table 4-10. Repeated measures multivariate analyses of variance for cognitive domains with study group as between-subjects factor

Effects	Wilks' Lambda	*F*	Hypothesis df	Error df	*P*-value	Partial η^2
Non-Executive Measures						
Between Subjects: Study Group	0.90	1.17	6	62	0.33	0.10
Within Subjects: Occasion	0.55	8.35	6	62	**0.00	0.45
Interaction: Study Group by Occasion	0.92	0.89	6	62	0.51	0.08
Executive Measures[a]						
Between Subjects: Study Group	0.97	0.23	9.00	59.00	0.99	0.03
Within Subjects: Occasion	0.65	3.59	9.00	59.00	**0.00	0.35
Interaction: Study Group by Occasion	0.86	1.09	9.00	59.00	0.38	0.14

Note: *p< .05; **p < .01; [a]There were unequal variance-covariance matrices for these measures.

Table 4-11. Follow-up univariate analyses of variance for non-executive measures

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
Between-Subjects: Study Group					
NAART	1	67	1.31	0.26	0.02
Trails A Time	1	67	0.02	0.88	0.00
LM Immediate Total Recall	1	67	2.52	0.12	0.04
LM Delayed Total Recall	1	67	1.49	0.23	0.02
LM Recognition	1	67	0.55	0.46	0.01
LM Learning Slope	1	67	1.14	0.29	0.02
Within-Subjects: Occasion					
NAART	1	67	10.68	**0.00	0.14
Trails A Time	1	67	6.89	*0.01	0.09
LM Immediate Total Recall	1	67	24.85	**0.00	0.27
LM Delayed Total Recall	1	67	30.69	**0.00	0.31
LM Recognition	1	67	4.82	*0.03	0.07
LM Learning Slope	1	67	1.15	0.29	0.02
Interactions: Study Group by Occasion					
NAART	1	67	0.01	0.93	0.00
Trails A Time	1	67	0.44	0.51	0.01
LM Immediate Total Recall	1	67	0.12	0.73	0.00
LM Delayed Total Recall	1	67	0.61	0.44	0.01
LM Recognition	1	67	1.78	0.19	0.03
LM Learning Slope	1	67	0.88	0.35	0.01

Note. *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test

Table 4-12. Follow-up univariate analyses of variance for executive measures

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
Between Subjects: Study Group					
COWA	1	67	0.06	0.80	0.00
Trails B	1	67	0.56	0.46	0.01
Letter-Number Sequencing	1	67	0.15	0.70	0.00
One-Back: Mean RT	1	67	0.94	0.34	0.01
One-Back: Mean RT SD	1	67	0.32	0.57	0.00
One-Back: Number Correct	1	67	0.01	0.92	0.00
Two-Back: Mean RT	1	67	0.04	0.84	0.00
Two-Back: Mean RT SD	1	67	0.01	0.91	0.00
Two-Back: Number Correct	1	67	0.20	0.66	0.00
Within-Subjects: Occasion					
COWA	1	67	7.04	**0.01	0.10
Trails B	1	67	0.50	0.48	0.01
Letter-Number Sequencing	1	67	0.15	0.70	0.00
One-Back: Mean RT	1	67	11.28	**0.00	0.14
One-Back: Mean RT SD	1	67	16.80	**0.00	0.20
One-Back: Number Correct	1	67	2.61	0.11	0.04
Two-Back: Mean RT	1	67	1.87	0.18	0.03
Two-Back: Mean RT SD	1	67	8.28	**0.01	0.11
Two-Back: Number Correct	1	67	7.95	**0.01	0.11
Interactions: Study Group by Occasion					
COWA	1	67	4.15	*0.05	0.06
Trails B	1	67	0.52	0.47	0.01
Letter-Number Sequencing	1	67	1.33	0.25	0.02
One-Back: Mean RT	1	67	0.51	0.48	0.01
One-Back: Mean RT SD	1	67	2.16	0.15	0.03
One-Back: Number Correct	1	67	0.18	0.67	0.00
Two-Back: Mean RT	1	67	0.15	0.70	0.00
Two-Back: Mean RT SD	1	67	0.68	0.41	0.01
Two-Back: Number Correct	1	67	0.84	0.36	0.01

Note: *$p < .05$; **$p < .01$; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-13. Raw cognitive estimated marginal means by study group

Measure	Control Group				Exercise Promotion Group			
Non-Executive	Baseline		Post-Test		Baseline		Post-Test	
	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error
NAART	113.88	1.09	115.14	1.05	112.22	1.11	113.42	1.06
Trails A Time	32.32	1.89	30.06	1.30	32.78	1.30	28.98	1.32
LM Immediate Recall	39.37	1.43	44.51	1.54	42.71	1.45	47.18	1.57
LM Delayed Recall	25.09	1.27	28.54	1.22	26.50	1.29	31.09	1.24
LM Recognition	26.03	0.40	26.95	0.29	26.69	0.40	26.91	0.30
LM Learning Slope	4.48	0.45	3.60	0.38	3.65	0.46	3.59	0.38
Executive	Baseline		Post-Test		Baseline		Post-Test	
	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error
COWA	40.14	1.84	40.73	1.67	38.79	1.87	43.24	1.69
Trails B Time	85.68	6.75	78.48	6.33	76.19	6.84	76.27	6.42
Letter Number Sequencing	10.56	0.45	10.20	0.38	10.50	0.45	10.68	0.39
One-Back: Mean RT	860.36	29.59	776.51	25.01	813.52	30.02	759.09	25.37
One-Back: Mean RT SD	339.04	31.17	218.51	17.20	291.09	31.63	234.15	17.45
One-Back: Number Correct	95.09	2.39	98.44	0.46	95.97	2.43	97.93	0.47
Two-Back: Mean RT	1663.50	90.39	1588.65	102.79	1670.62	91.71	1535.99	104.29
Two-Back: Mean RT SD	916.13	83.79	715.60	70.90	860.75	85.02	749.45	71.93
Two-Back: Number Correct	90.81	1.62	94.36	1.01	90.94	1.64	92.76	1.02

Note: LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-14. Calculation of reliable change index scores adjusted for control group practice effect

	Pre SD	Post SD	r.t1.t2	SE of Difference	Reliable Change	Control Group Mean Change	Reliable Decline	Reliable Improvement
NAART	6.46	6.21	0.85	3.47	5.70	1.26	-4.44	6.95
LM Immediate Recall	8.57	9.16	0.58	8.16	13.38	-2.27	-15.64	11.11
LM Learning Slope	2.67	2.22	-0.25	3.88	6.36	5.14	-1.22	11.50
LM Delayed Recall	7.52	7.29	0.59	6.68	10.95	3.46	-7.50	14.41
LM Recognition	2.34	1.71	0.51	2.04	3.34	0.92	-2.42	4.27
COWA	10.84	9.86	0.64	8.76	14.36	-0.89	-15.25	13.47
Trails A Time	11.10	7.68	0.70	7.45	12.22	0.58	-11.64	12.80
Trails B time	39.90	37.16	0.48	39.21	64.30	-7.19	-71.49	57.10
Letter-Number Sequencing	2.62	2.25	0.71	1.86	3.04	-0.36	-3.40	2.69
One-Back Mean RT	175.35	147.10	0.48	165.68	271.72	-83.86	-355.58	187.87
One-Back Mean RT SD	184.63	101.31	0.45	156.47	256.61	-120.52	-377.13	136.08
One-Back Number Correct	14.05	2.74	0.01	14.28	23.42	3.35	-20.07	26.77
Two-Back Mean RT	530.85	604.22	0.31	669.54	1098.05	-74.84	-1172.89	1023.21
Two-Back Mean RT SD	492.85	416.68	0.52	449.00	736.35	-200.53	-936.88	535.83
Two-Back Number Correct	9.51	5.97	0.57	7.39	12.11	3.56	-8.56	15.67

Note: NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation; SE = Standard Error; Reliable Change = Standard Error of Difference * 1.64

Table 4-15. Participants who reliably changed on non-executive cognitive measures

	Control (n= 35)	Intervention (n = 34)	Total (n = 69)
NAART			
Reliable Decline	2 (5.7%)	1 (2.9%)	3 (4.3%)
No Reliable Change	31 (88.6%)	32 (94.1%)	63 (91.3%)
Reliable Improvement	2 (5.7%)	1 (2.9%)	3 (4.3%)
LM Immediate Recall			
No Reliable Change	25 (71.4%)	27 (79.4%)	52 (75.4%)
Reliable Improvement	10 (28.6%)	7 (20.6%)	17 (24.6%)
LM Delayed Recall			
Reliable Decline	3 (8.6%)	0 (0%)	3 (4.3%)
No Reliable Change	31 (88.6%)	33 (97.1%)	64 (92.8%)
Reliable Improvement	1 (2.9%)	1 (2.9%)	2 (2.9%)
LM Learning Slope			
Reliable Decline	13 (37.1%)	14 (41.2%)	27 (39.1%)
No Reliable Change	22 (62.9%)	20 (58.8%)	42 (60.9%)
LM Recognition			
Reliable Decline	3 (8.6%)	2 (5.9%)	5 (7.2%)
No Reliable Change	29 (82.9%)	31 (91.2%)	60 (87.0%)
Reliable Improvement	3 (8.6%)	1 (2.9%)	4 (5.8%)
Trails A Time			
Reliable Decline	3 (8.6%)	6 (17.6%)	9 (13%)
No Reliable Change	31 (88.6%)	27 (79.4%)	58 (84.1%)
Reliable Improvement	1 (2.9%)	1 (2.9%)	2 (2.9%)

Note: NAART = North American Adult Reading Test; LM = Logical Memory

Table 4-16. Participants who reliably changed on executive cognitive measures

	Control (n= 35)	Intervention (n = 34)	Total (n = 69)
COWA			
Reliable Decline	1 (2.9%)	0 (0%)	1 (1.4%)
No Reliable Change	32 (91.4%)	31 (91.2%)	63 (91.3%)
Reliable Improvement	2 (5.7%)	3 (8.8%)	5 (7.2%)
Trails B Time			
Reliable Decline	2 (5.7%)	1 (2.9%)	3 (4.3%)
No Reliable Change	30 (85.7%)	32 (94.1%)	62 (89.9%)
Reliable Improvement	3 (8.6%)	1 (2.9%)	4 (5.8%)
Letter-Number Sequencing			
Reliable Decline	3 (8.6%)	1 (2.9%)	4 (5.8%)
No Reliable Change	32 (91.4%)	29 (85.3%)	61 (88.4%)
Reliable Improvement	0 (0%)	4 (11.8%)	4 (5.8%)
One-Back Mean RT			
Reliable Decline	2 (5.7%)	1 (2.9%)	3 (4.3%)
No Reliable Change	31 (88.6%)	30 (88.2%)	30 (88.2%)
Reliable Improvement	2 (5.7%)	3 (8.8%)	3 (8.8%)
One-Back Mean RT SD			
Reliable Decline	4 (11.4%)	2 (5.9%)	6 (8.7%)
No Reliable Change	31 (88.6%)	30 (88.2%)	61 (88.4%)
Reliable Improvement	0 (0%)	2 (2.9%)	2 (2.9%)
One-Back Number Correct			
No Reliable Change	34 (97.1%)	32 (94.1%)	66 (95.7%)
Reliable Improvement	1 (2.9%)	2 (5.9%)	3 (4.3%)

Note: COWA= Controlled Auditory Word Association; RT=Reaction Time; SD=Standard Deviation

Table 4-16. Continued

	Control (n= 35)	Intervention (n = 34)	Total (n = 69)
Two-Back Mean RT			
Reliable Decline	2 (5.7%)	2 (5.9%)	4 (5.8%)
No Reliable Change	31 (88.6%)	30 (88.2%)	61 (88.4%)
Reliable Improvement	2 (5.7%)	2 (5.9%)	4 (5.8%)
Two-Back Mean RT SD			
Reliable Decline	3 (8.6%)	1 (2.9%)	4 (5.8%)
No Reliable Change	31 (88.6%)	30 (88.2%)	61 (88.4%)
Reliable Improvement	1 (2.9%)	3 (8.8%)	4 (5.8%)
Two-Back Number Correct			
Reliable Decline	1 (2.9%)	2 (5.9%)	3 (4.3%)
No Reliable Change	32 (91.4%)	30 (88.2%)	62 (89.9%)
Reliable Improvement	2 (5.7%)	2 (5.9%)	4 (5.8%)

Note: COWA= Controlled Auditory Word Association; RT=Reaction Time; SD=Standard Deviation

Table 4-17. Multivariate analyses of variance for cognitive domains with study group and age group as between-subjects factors

	Wilks' Lambda	*F*	Hypothesis df	Error df	*P*-value	Partial η^2
Non-Executive Measures						
Between Subjects: Study Group	0.89	1.20	6	60	0.32	0.11
Age Group	0.83	2.09	6	60	0.07	0.17
Study Group by Age Group	0.90	1.17	6	60	0.33	0.10
Within Subjects: Occasion	0.55	8.11	6	60	**0.00	0.45
Interaction: Study Group by Occasion	0.92	0.83	6	60	0.55	0.08
Age Group by Occasion	0.97	0.34	6	60	0.91	0.03
Study Group by Age Group by Occasion	0.94	0.63	6	60	0.70	0.06
Executive Measures						
Between Subjects: Study Group	0.96	0.27	9	57	0.98	0.04
Age Group	0.73	2.33	9	57	*0.03	0.27
Study Group by Age Group	0.77	1.91	9	57	0.07	0.23
Within Subjects: Occasion	0.64	3.58	9	57	**0.00	0.36
Interaction: Study Group by Occasion	0.86	1.01	9	57	0.44	0.14
Age Group by Occasion	0.89	0.80	9	57	0.62	0.11
Study Group by Age Group by Occasion	0.83	1.34	9	57	0.24	0.17

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

Table 4-18. Follow-up univariate analyses of variance for non-executive measures: Study group and age group as between subjects factors

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
NAART					
Study Group	1	65	1.76	0.19	0.03
Age Group	1	65	2.42	0.12	0.04
Study Group by Age Group	1	65	0.48	0.49	0.01
Occasion	1	65	10.50	**0.00	0.14
Study Group by Occasion	1	65	0.00	0.95	0.00
Age Group by Occasion	1	65	0.29	0.59	0.00
Study Group by Age Group by Occasion	1	65	0.05	0.83	0.00
Trails A Time					
Study Group	1	65	0.08	0.77	0.00
Age Group	1	65	7.50	**0.01	0.10
Study Group by Age Group	1	65	0.48	0.49	0.01
Occasion	1	65	7.74	**0.01	0.11
Study Group by Occasion	1	65	0.73	0.40	0.01
Age Group by Occasion	1	65	0.36	0.55	0.01
Study Group by Age Group by Occasion	1	65	2.16	0.15	0.03
LM Immediate Total Recall					
Study Group	1	65	2.12	0.15	0.03
Age Group	1	65	0.08	0.78	0.00
Study Group by Age Group	1	65	1.80	0.18	0.03
Occasion	1	65	23.11	**0.00	0.26
Study Group by Occasion	1	65	0.17	0.68	0.00
Age Group by Occasion	1	65	0.21	0.65	0.00
Study Group by Age Group by Occasion	1	65	0.14	0.71	0.00
LM Delayed Total Recall					
Study Group	1	65	1.03	0.31	0.02
Age Group	1	65	0.08	0.77	0.00
Study Group by Age Group	1	65	3.17	0.08	0.05
Occasion	1	65	29.03	**0.00	0.31
Study Group by Occasion	1	65	0.49	0.49	0.01
Age Group by Occasion	1	65	0.00	0.95	0.00
Study Group by Age Group by Occasion	1	65	0.43	0.52	0.01

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test

Table 4-18. Continued

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
LM Recognition					
Study Group	1	65	0.45	0.51	0.01
Age Group	1	65	0.36	0.55	0.01
Study Group by Age Group	1	65	0.01	0.91	0.00
Occasion	1	65	5.16	0.03	0.07
Study Group by Occasion	1	65	1.45	0.23	0.02
Age Group by Occasion	1	65	0.60	0.44	0.01
Study Group by Age Group by Occasion	1	65	0.23	0.63	0.00
LM Learning Slope					
Study Group	1	65	0.90	0.35	0.01
Age Group	1	65	0.07	0.79	0.00
Study Group by Age Group	1	65	0.55	0.46	0.01
Occasion	1	65	1.05	0.31	0.02
Study Group by Occasion	1	65	1.02	0.32	0.02
Age Group by Occasion	1	65	0.25	0.62	0.00
Study Group by Age Group by Occasion	1	65	1.35	0.25	0.02

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test

Table 4-19. Follow-up univariate analyses of variance for executive measures: Study group and age group as between subjects factors

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
COWA					
Study Group	1	65	0.04	0.84	0.00
Age Group	1	65	0.15	0.70	0.00
Study Group by Age Group	1	65	0.50	0.48	0.01
Occasion	1	65	7.00	*0.01	0.10
Study Group by Occasion	1	65	4.03	*0.05	0.06
Age Group by Occasion	1	65	0.51	0.48	0.01
Study Group by Age Group by Occasion	1	65	0.23	0.64	0.00
Trails B Time					
Study Group	1	65	0.57	0.45	0.01
Age Group	1	65	3.46	0.07	0.05
Study Group by Age Group	1	65	1.93	0.17	0.03
Occasion	1	65	0.92	0.34	0.01
Study Group by Occasion	1	65	0.20	0.65	0.00
Age Group by Occasion	1	65	0.29	0.59	0.00
Study Group by Age Group by Occasion	1	65	5.93	*0.02	0.08
Letter-Number Sequencing					
Study Group	1	65	0.16	0.69	0.00
Age Group	1	65	0.86	0.36	0.01
Study Group by Age Group	1	65	0.19	0.67	0.00
Occasion	1	65	0.10	0.75	0.00
Study Group by Occasion	1	65	1.39	0.24	0.02
Age Group by Occasion	1	65	0.33	0.57	0.01
Study Group by Age Group by Occasion	1	65	0.01	0.94	0.00
One-Back: Mean RT					
Study Group	1	65	1.17	0.28	0.02
Age Group	1	65	6.54	*0.01	0.09
Study Group by Age Group	1	65	1.19	0.28	0.02
Occasion	1	65	11.21	**0.00	0.15
Study Group by Occasion	1	65	0.57	0.45	0.01
Age Group by Occasion	1	65	5.45	*0.02	0.08
Study Group by Age Group by Occasion	1	65	2.26	0.14	0.03

Note: *$p < .05$; **$p < .01$; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-19 Continued.

Effects	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
One-Back: Mean RT SD					
Study Group	1	65	0.16	0.69	0.00
Age Group	1	65	0.62	0.43	0.01
Study Group by Age Group	1	65	5.32	*0.02	0.08
Occasion	1	65	17.71	**0.00	0.21
Study Group by Occasion	1	65	1.74	0.19	0.03
Age Group by Occasion	1	65	0.24	0.63	0.00
Study Group by Age Group by Occasion	1	65	3.51	0.07	0.05
One-Back Number Correct					
Study Group	1	65	0.01	0.91	0.00
Age Group	1	65	0.28	0.60	0.00
Study Group by Age Group	1	65	0.26	0.61	0.00
Occasion	1	65	2.64	0.11	0.04
Study Group by Occasion	1	65	0.18	0.67	0.00
Age Group by Occasion	1	65	0.62	0.44	0.01
Study Group by Age Group by Occasion	1	65	0.38	0.54	0.01
Two-Back: Mean RT					
Study Group	1	65	0.07	0.79	0.00
Age Group	1	65	5.55	*0.02	0.08
Study Group by Age Group	1	65	1.12	0.29	0.02
Occasion	1	65	1.76	0.19	0.03
Study Group by Occasion	1	65	0.11	0.74	0.00
Age Group by Occasion	1	65	0.18	0.67	0.00
Study Group by Age Group by Occasion	1	65	0.72	0.40	0.01
Two-Back: Mean RT SD					
Study Group	1	65	0.08	0.77	0.00
Age Group	1	65	5.78	*0.02	0.08
Study Group by Age Group	1	65	0.00	0.99	0.00
Occasion	1	65	7.78	**0.01	0.11
Study Group by Occasion	1	65	0.66	0.42	0.01
Age Group by Occasion	1	65	0.31	0.58	0.00
Study Group by Age Group by Occasion	1	65	0.11	0.74	0.00

Note: *$p < .05$; **$p < .01$; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-19 Continued.

	Hypothesis df	Error df	*F*	*P*-value	Partial η^2
Two-Back: Number Correct					
Study Group	1	65	0.07	0.79	0.00
Age Group	1	65	2.12	0.15	0.03
Study Group by Age Group	1	65	0.48	0.49	0.01
Occasion	1	65	7.43	**0.01	0.10
Study Group by Occasion	1	65	0.83	0.37	0.01
Age Group by Occasion	1	65	0.20	0.65	0.00
Study Group by Age Group by Occasion	1	65	0.03	0.86	0.00

Note: *$p < .05$; **$p < .01$; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-20. Raw cognitive estimated marginal means by age and study group

	Younger Group							
	Control Group				Exercise Promotion Group			
Non-Executive	Baseline		Post-Test		Baseline		Post-Test	
	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error
NAART	113.07	1.56	114.63	1.50	110.19	1.71	111.53	1.65
Trails A Time	28.38	2.66	27.16	1.75	31.87	2.93	25.25	1.93
LM Immediate Recall	41.00	2.06	46.06	2.22	42.00	2.27	45.50	2.44
LM Delayed Recall	26.06	1.83	30.06	1.73	24.79	2.02	28.86	1.91
LM Recognition	25.88	0.57	26.88	0.42	26.31	0.63	26.93	0.47
LM Learning Slope	4.76	0.65	3.12	0.54	3.71	0.72	4.00	0.60
Executive	Baseline		Post-Test		Baseline		Post-Test	
	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error
COWA	40.88	2.68	42.65	2.40	38.21	2.95	42.93	2.65
Trails B Time	68.01	9.29	70.40	8.97	82.68	10.24	65.52	9.88
Letter Number Sequencing	10.88	0.65	10.65	0.55	10.57	0.71	10.93	0.60
One-Back: Mean RT	760.93	39.14	755.63	36.15	776.21	43.13	741.20	39.84
One-Back: Mean RT SD	268.29	43.28	200.10	24.83	333.60	47.69	241.58	27.36
One-Back: Number Correct	92.88	3.46	98.65	0.68	95.79	3.82	98.07	0.74
Two-Back: Mean RT	1518.78	128.37	1341.44	143.18	1566.19	141.46	1470.00	157.78
Two-Back: Mean RT SD	773.49	117.76	623.45	100.35	720.47	129.76	623.33	110.58
Two-Back: Number Correct	91.78	2.33	94.71	1.44	93.21	2.57	94.71	1.58

Note: LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-20 Continued.

	Older Group							
	Control Group				Exercise Promotion Group			
Non-Executive	Baseline		Post-Test		Baseline		Post-Test	
	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error
NAART	114.64	1.51	115.62	1.46	113.64	1.43	114.74	1.38
Trails A Time	36.04	2.59	32.79	1.70	33.42	2.45	31.60	1.61
LM Immediate Recall	37.83	2.00	43.06	2.16	43.20	1.90	48.35	2.04
LM Delayed Recall	24.17	1.78	27.11	1.68	27.70	1.69	32.65	1.59
LM Recognition	26.17	0.56	27.01	0.41	26.95	0.53	26.90	0.39
LM Learning Slope	4.21	0.63	4.05	0.53	3.60	0.60	3.30	0.50
Executive	Baseline		Post-Test		Baseline		Post-Test	
	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error	Mean	Std. Error
COWA	39.44	2.60	38.91	2.34	39.20	2.47	43.45	2.22
Trails B Time	102.36	9.03	86.12	8.72	71.65	8.57	83.79	8.27
Letter Number Sequencing	10.25	0.63	9.78	0.53	10.45	0.60	10.50	0.51
One-Back: Mean RT	954.28	38.04	796.23	35.13	839.64	36.09	771.61	33.33
One-Back: Mean RT SD	405.85	42.06	235.91	24.13	261.34	39.90	228.94	22.89
One-Back: Number Correct	97.17	3.37	98.24	0.66	96.10	3.19	97.83	0.62
Two-Back: Mean RT	1800.17	124.75	1822.14	139.15	1743.72	118.35	1582.19	132.01
Two-Back: Mean RT SD	1050.84	114.44	802.63	97.52	958.95	108.57	837.74	92.52
Two-Back: Number Correct	89.89	2.26	94.04	1.40	89.35	2.15	91.39	1.33

Note: LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation

Table 4-21. Correlations between physical activity, physical fitness, and cognitive change scores

	VO2	LTEQ	Mean Pedometer Steps	MVPA
NAART	-0.09	-0.01	0.06	-0.02
Trails A Time	-0.03	-0.13	-0.02	-0.18
LM Immediate Recall	*0.31	0.01	0.14	0.01
LM Delayed Recall	0.14	0.03	0.09	0.06
LM Recognition	0.09	-0.01	0.06	-0.09
LM Learning Slope	-0.02	-0.13	*-0.29	-0.16
COWA	-0.18	0.03	-0.05	0.08
Trails B Time	-0.13	0.23	-0.06	0.16
Letter Number Sequencing	0.09	-0.01	-0.15	-0.10
One-Back: Mean RT	0.21	-0.07	0.13	0.04
One-Back: Mean RT SD	0.11	*-0.25	0.04	-0.21
Two-Back: Number Correct	-0.11	0.00	0.09	-0.06
Two-Back: Mean RT	-0.21	-0.01	0.11	-0.03
Two-Back: Mean RT SD	-0.21	-0.13	0.02	-0.08
Two-Back: Number Correct	-0.20	0.09	0.02	0.10

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation; VO2 = Modified Balke Submax; LTEQ = Leisure Time Exercise Questionnaire; MVPA = Minutes of Moderate and Vigorous Physical Activity

Table 4-22. Correlations between physical fitness, physical activity, and psychosocial change scores

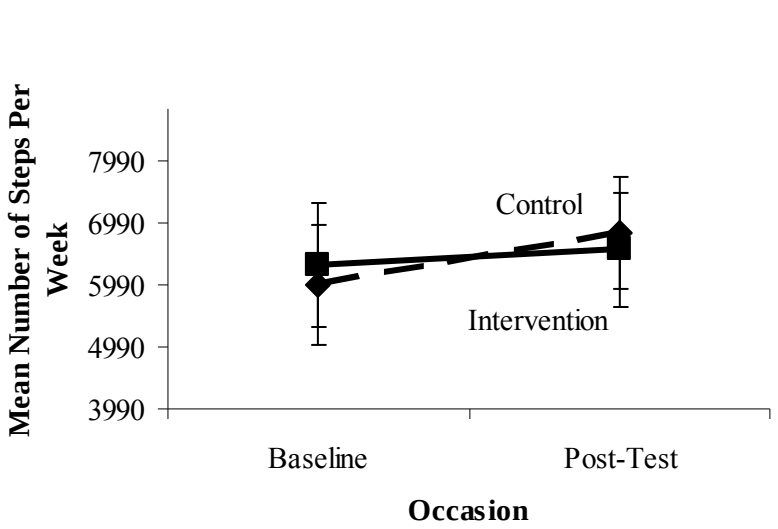
	VO2	LTEQ	Mean Pedometer Steps	MVPA
GDS	*-0.25	0.05	-0.14	0.08
BDI-2	0.02	0.02	-0.06	-0.04
STAI State Anxiety	-0.04	0.18	0.08	0.20
STAI Trait Anxiety	-0.05	-0.11	0.14	-0.11
BSE	0.04	0.12	-0.06	0.08
EXSE	-0.15	0.11	0.05	0.09

Note: *$p < .05$; **$p < .01$; GDS = Geriatric Depression Scale; BDI-2 = Beck Depression Inventory- Second Edition; STAI = State Trait Anxiety Inventory; BSE = Barriers Self Efficacy Scale; EXSE = Exercise Self-Efficacy Scale; VO2 = Modified Balke Submax; LTEQ = Leisure Time Exercise Questionnaire

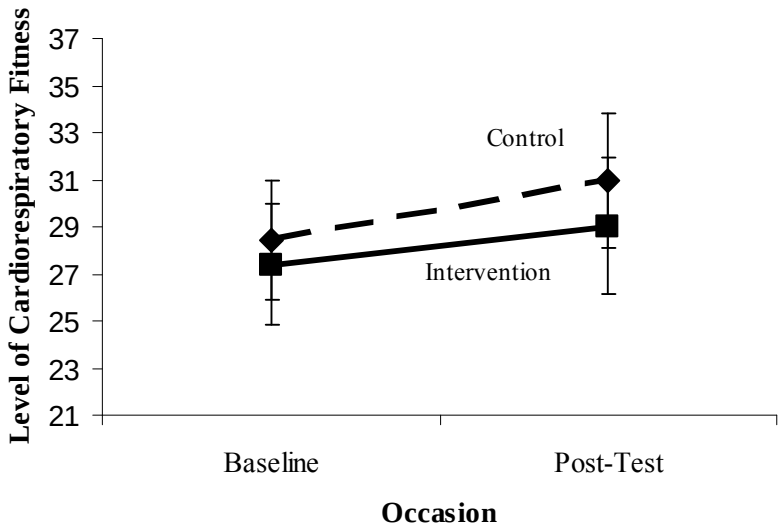
Table 4-23. Correlations between psychosocial and cognitive change scores

	GDS	BDI-2	STAI State Anxiety	STAI Trait Anxiety	BSE	EXSE
NAART	-0.01	-0.14	-0.01	-0.06	0.06	-0.15
Trails A Time	0.03	0.05	-0.06	-0.11	0.18	0.12
LM Immediate Recall	-0.22	-0.09	-0.05	0.15	0.11	0.08
LM Delayed Recall	*-0.24	0.01	0.03	0.21	-0.01	-0.02
LM Recognition	-0.02	-0.03	-0.12	0.16	0.07	-0.12
LM Learning Slope	-0.03	-0.03	0.04	-0.20	0.13	0.04
COWA	-0.17	-0.01	-0.10	0.00	0.22	0.02
Trails B Time	-0.09	0.21	0.18	-0.02	0.04	-0.09
Letter Number Sequencing	0.08	0.19	0.08	0.15	0.08	-0.16
One-Back: Mean RT	0.04	-0.08	0.01	-0.08	-0.10	-0.13
One-Back: Mean RT SD	-0.02	-0.03	0.00	0.01	-0.11	-0.14
One-Back: Number Correct	0.00	-0.20	0.03	-0.17	0.15	0.09
Two-Back: Mean RT	0.12	-0.19	0.20	0.01	0.12	-0.01
Two-Back: Mean RT SD	0.02	*-0.26	0.21	-0.08	0.18	-0.03
Two-Back: Number Correct	0.06	-0.11	0.15	-0.01	*-0.28	-0.02

Note: *$p < .05$; **$p < .01$; LM = Logical Memory; NAART = North American Adult Reading Test; COWA = Controlled Oral Word Association; RT = Reaction Time; SD = Standard Deviation; GDS = Geriatric Depression Scale; BDI-2 = Beck Depression Inventory-Second Edition; STAI = State Trait Anxiety Inventory; BSE = Barriers Self Efficacy Scale; EXSE = Exercise Self-Efficacy Scale

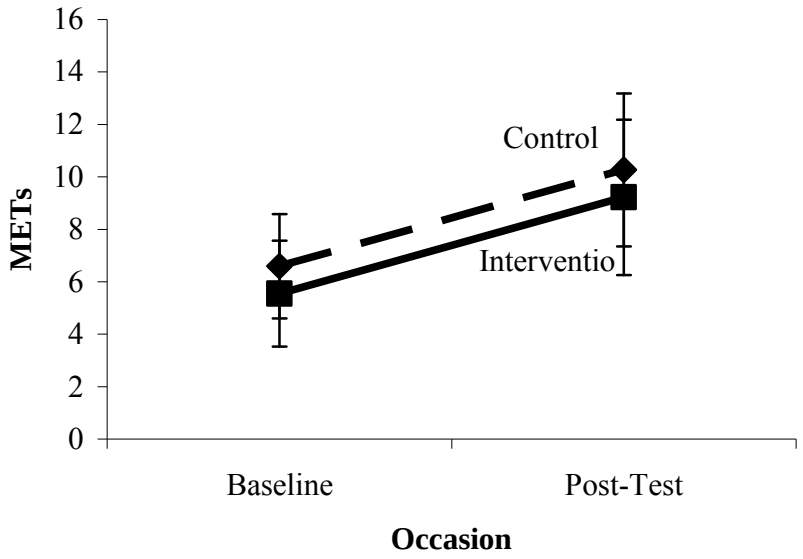


A



B

Figure 4-1. Mean scores by study group and occasion for physical fitness, activity, and self-efficacy variables A) Mean pedometer steps. B) Cardiorespiratory fitness (VO2). C) Leisure Time Exercise (LTEQ). D) Mean minutes of moderate or vigorous physical activity. E) Barriers of Self Efficacy (BSE). F) Exercise Self-Efficacy.



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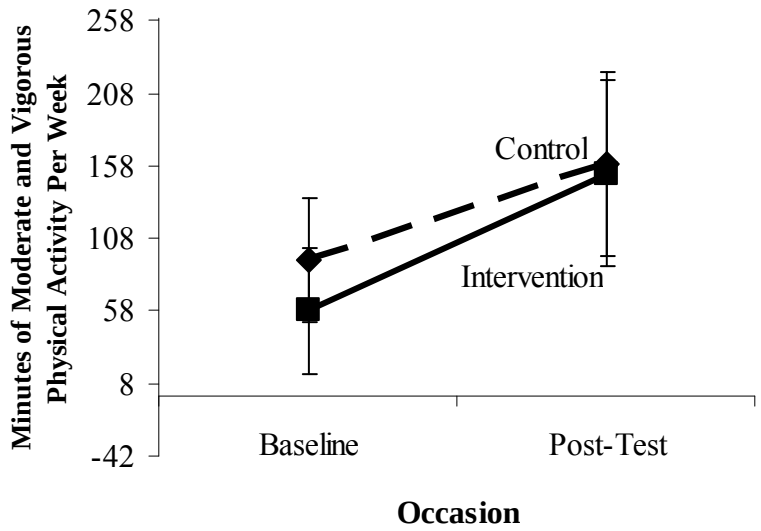
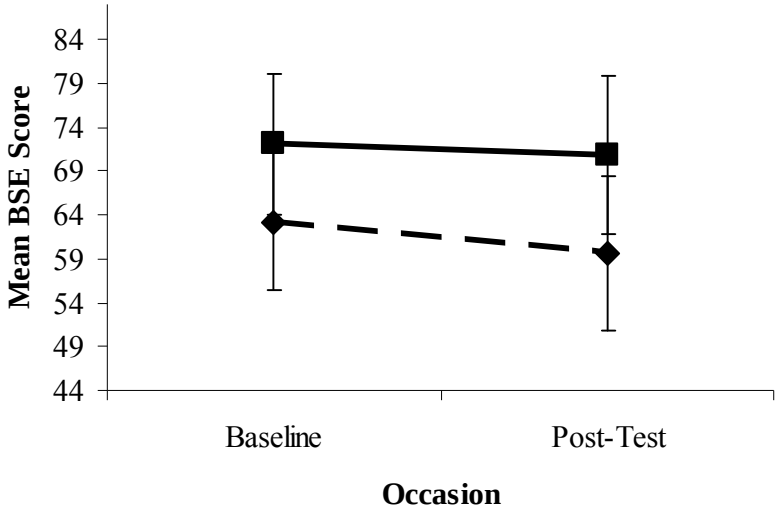
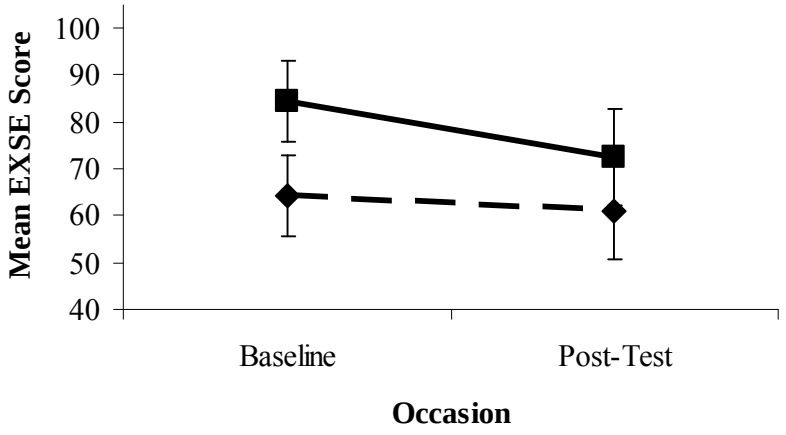


D

Figure 4-1 Continued.

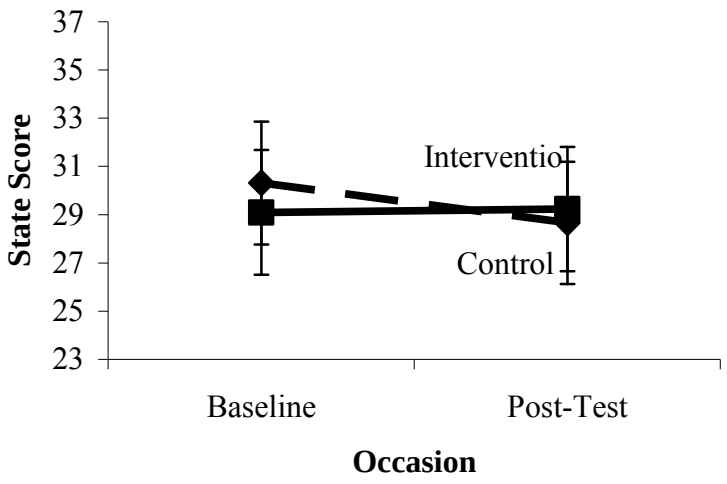


E

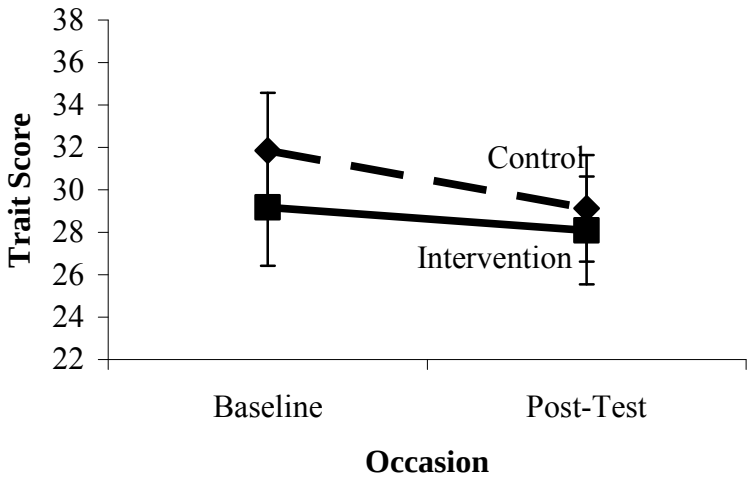


F

Figure 4-1 Continued.

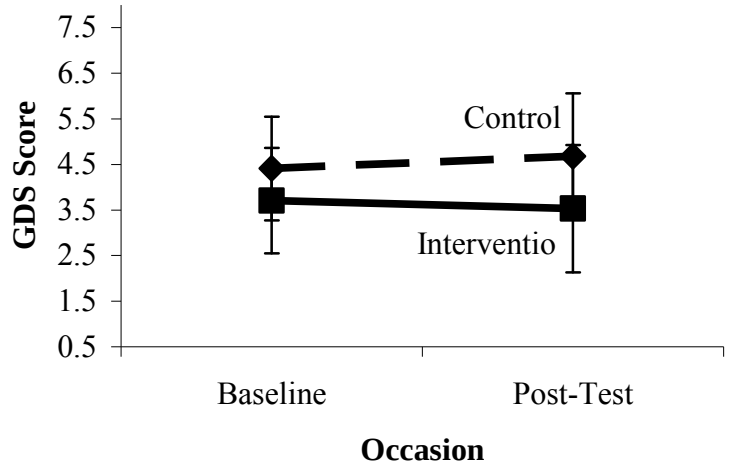


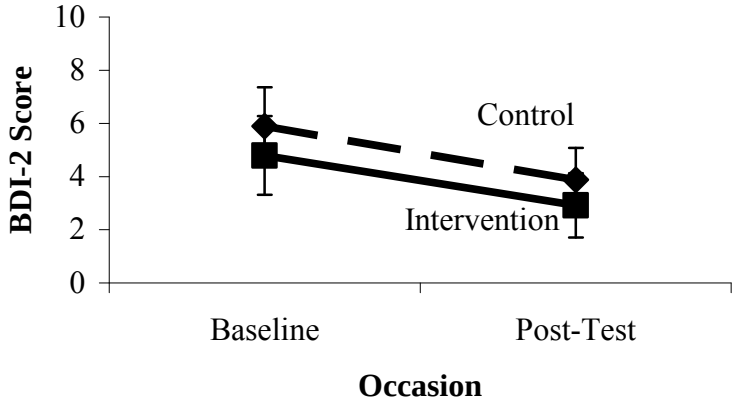
A



B

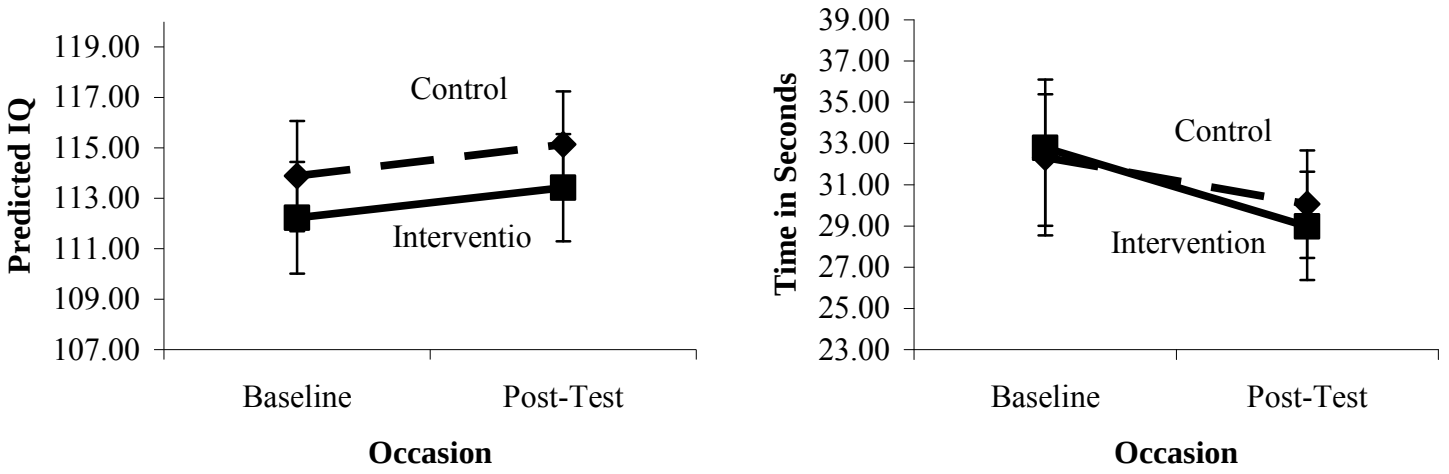
Figure 4-2. Mean anxiety and depression scores by study group and occasion A) STAI-State Anxiety. B) STAI-Trait Anxiety. C) GDS. D) BDI-2.





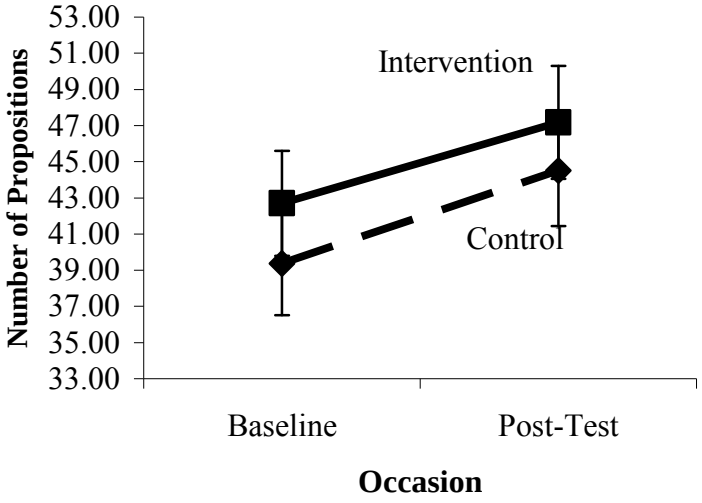
C D

Figure 4-2 Continued.



A B

Figure 4-3. Mean cognitive scores by study group and occasion for non-executive cognitive variables A) NAART. B) Trails A. C) LM Immediate Recall. D) LM Delayed Recall. E) LM Recognition. F) LM Learning Slope.



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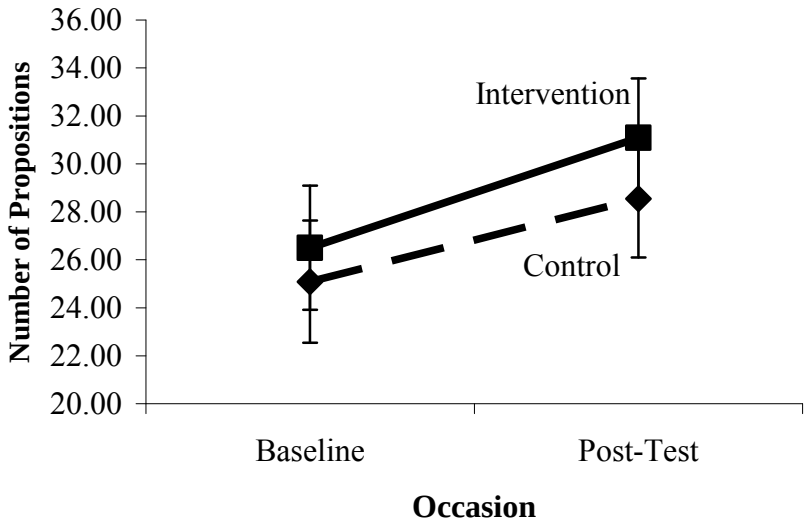

D

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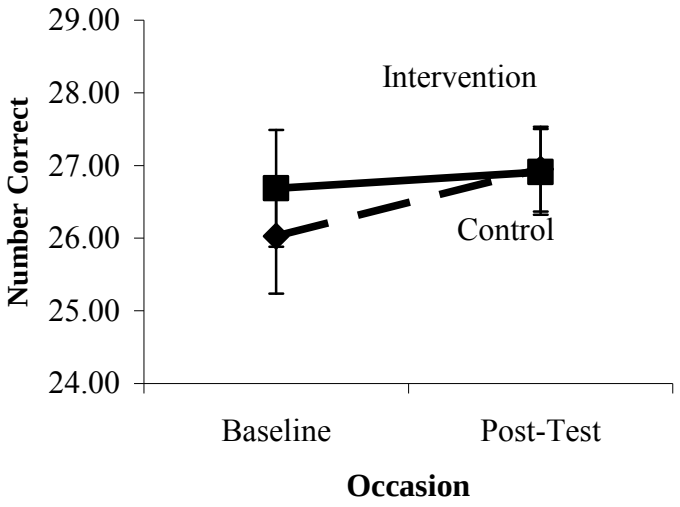

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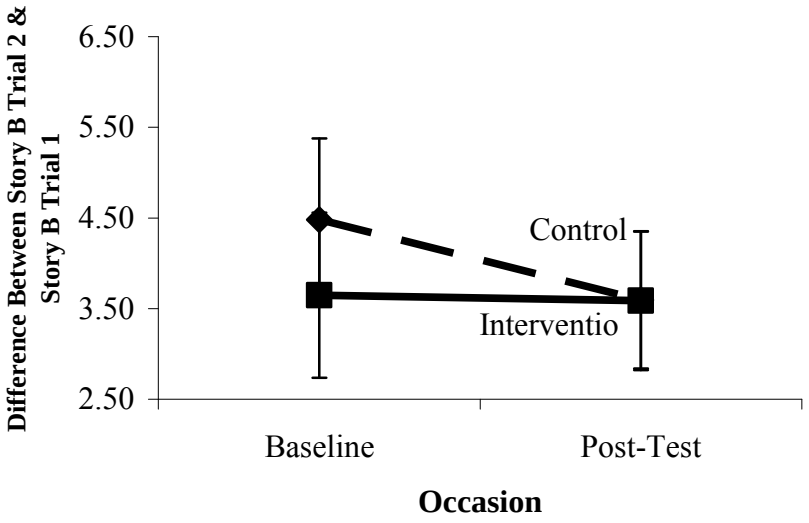
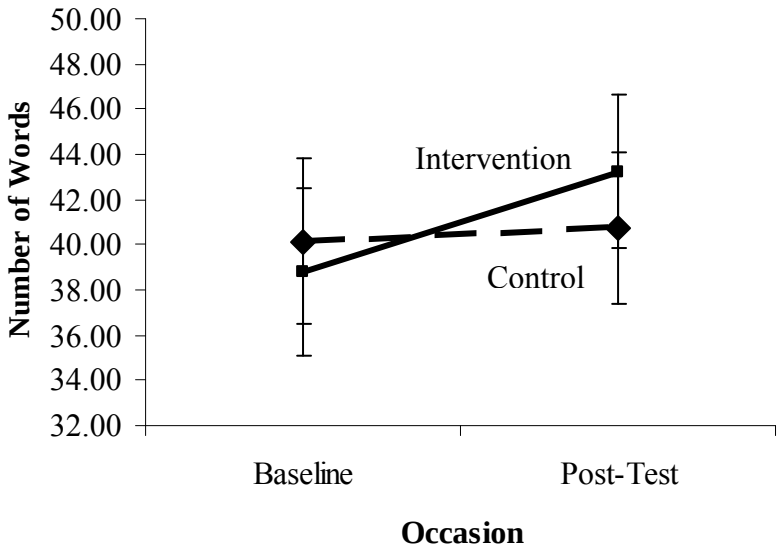
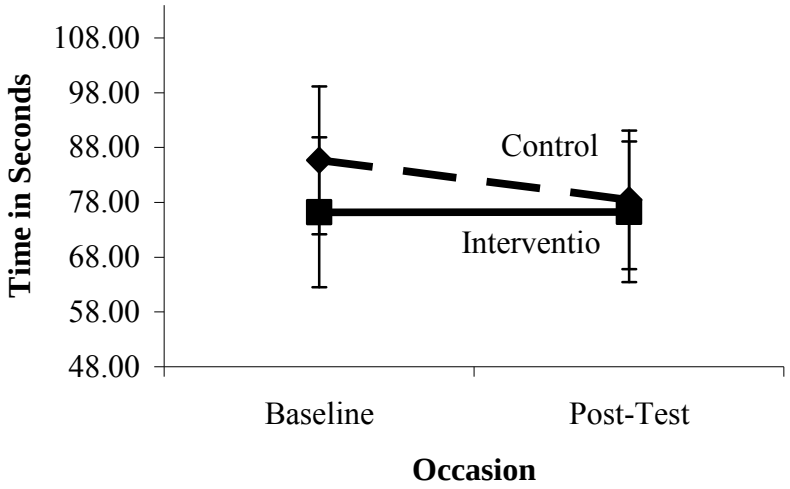


F

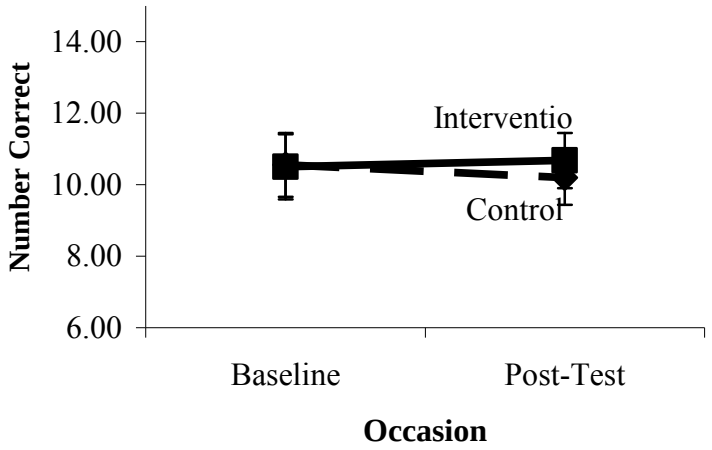
Figure 4-3 Continued.





A B

Figure 4-4. Mean cognitive scores by study group and occasion for executive cognitive variables A) COWA. B) Trails B. C) Letter-Number Sequencing. D) One-Back Mean RT. E) One-Back Mean RT SD. F) One-Back Number Correct. G) Two-Back Mean RT. H) Two-Back Mean RT SD. I) Two-Back Number Correct.



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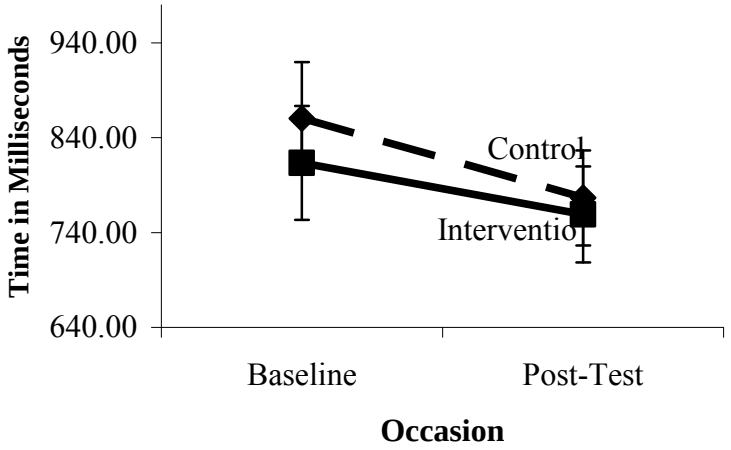

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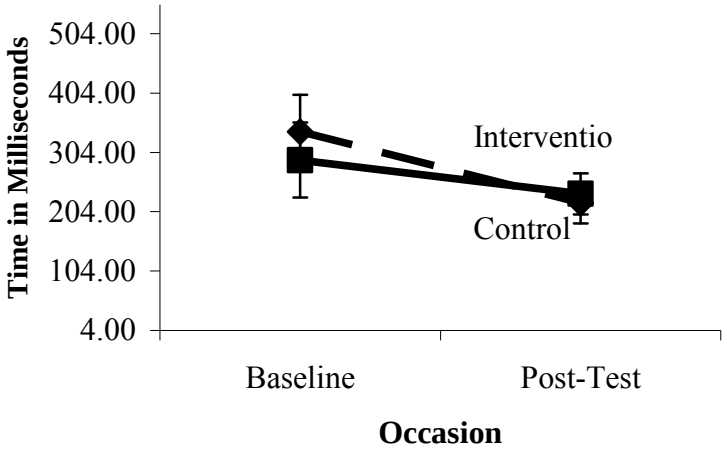

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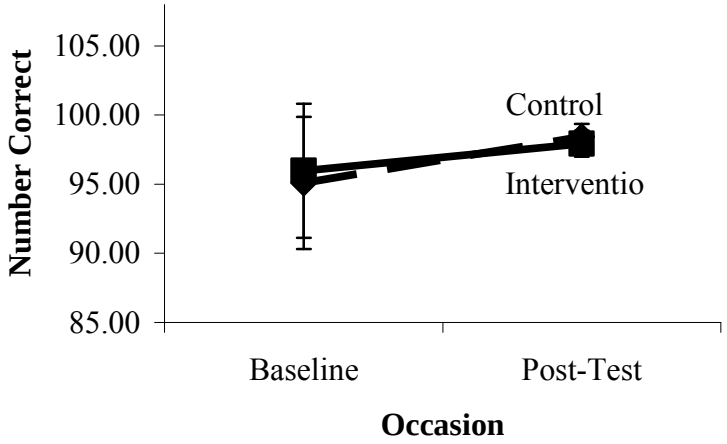

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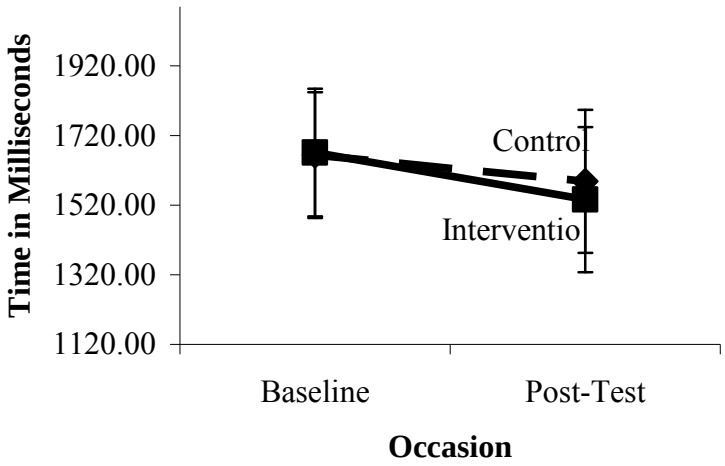

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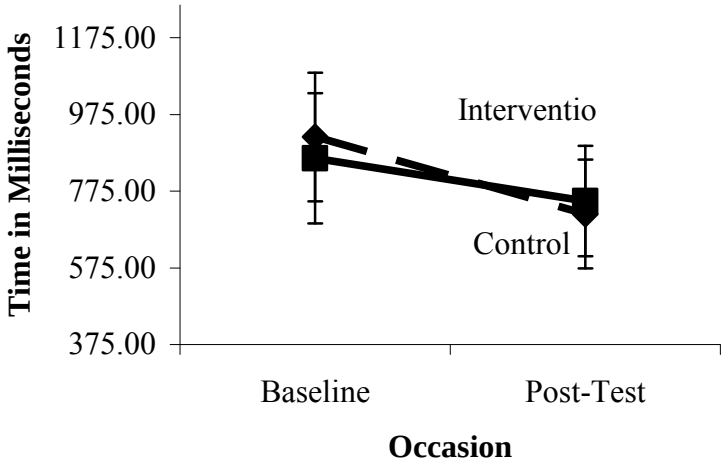

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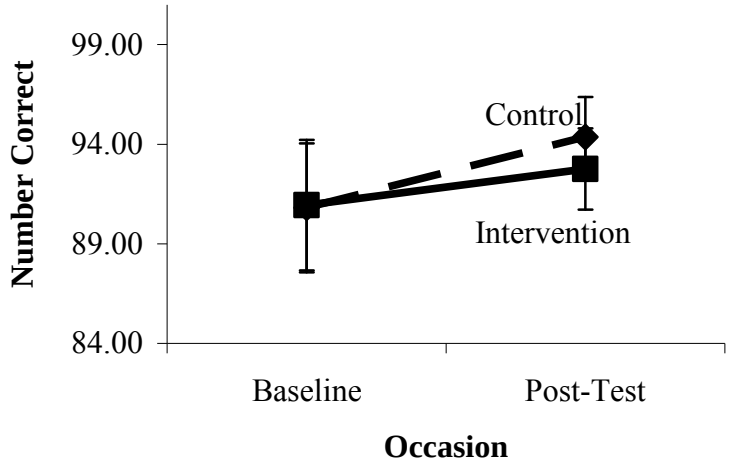

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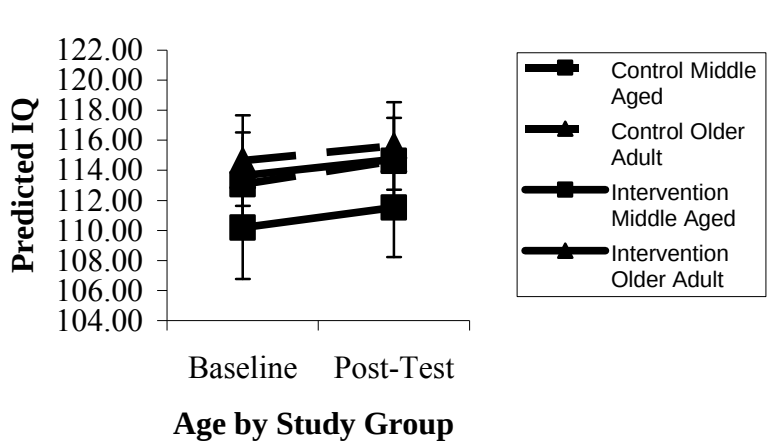

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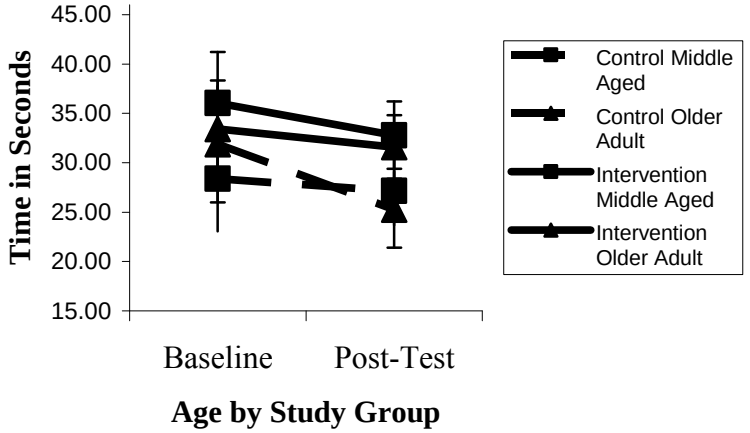
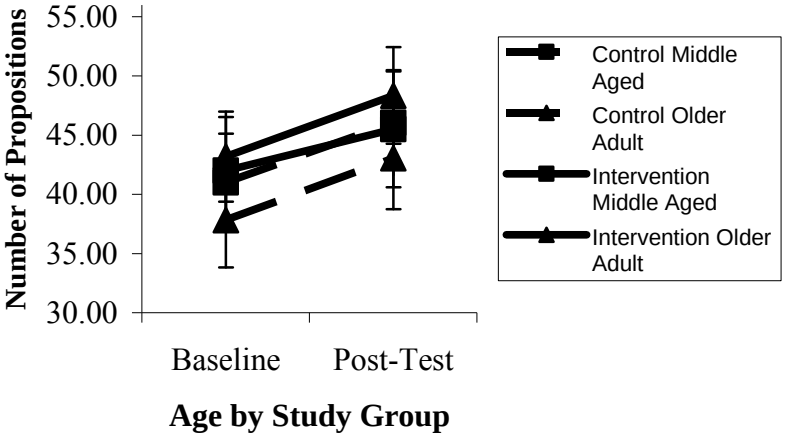


B

Figure 4-5. Mean cognitive scores by study group, age group, and occasion for non-executive cognitive variables A) NAART. B) Trails A. C) LM Immediate Recall. D) LM Delayed Recall. E) LM Recognition. F) LM Learning Slope



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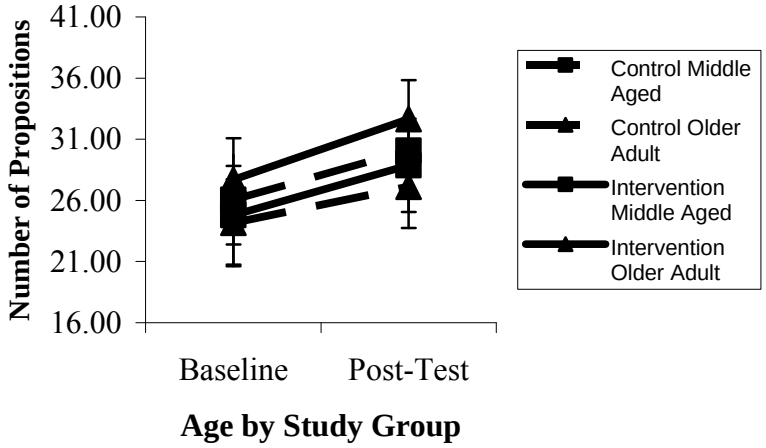

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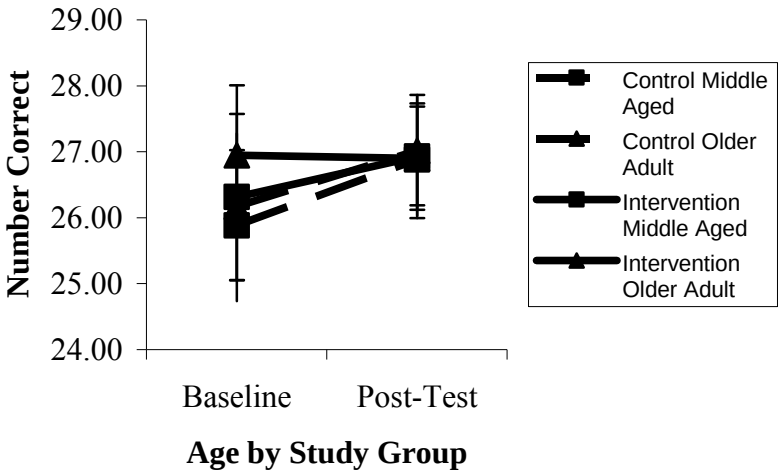

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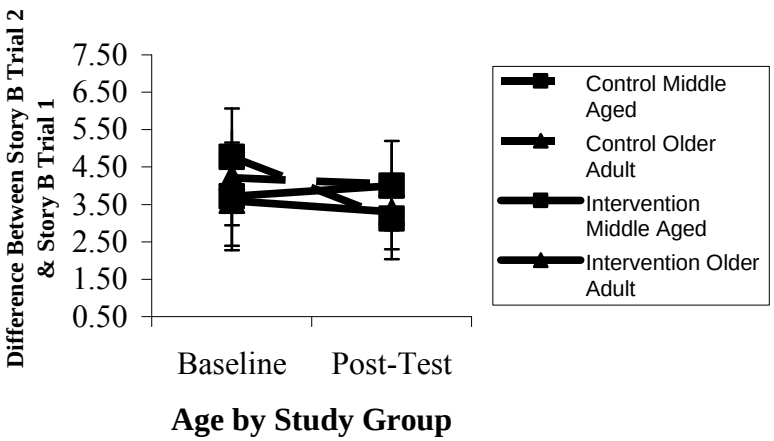

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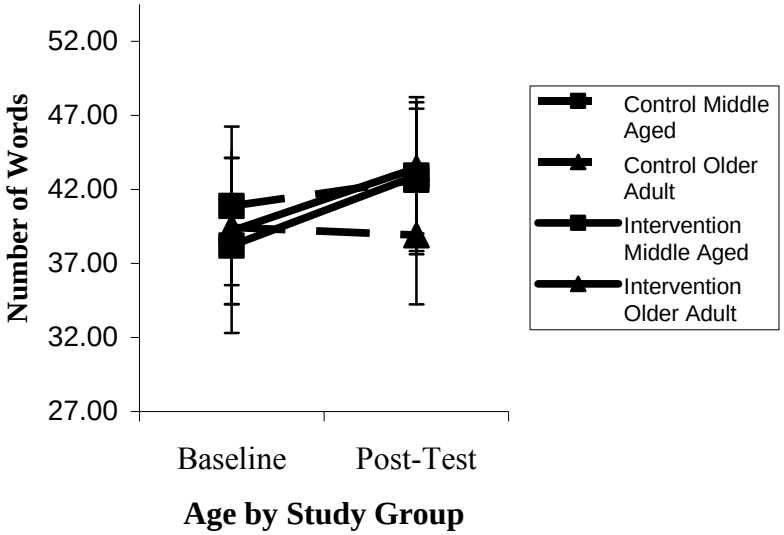

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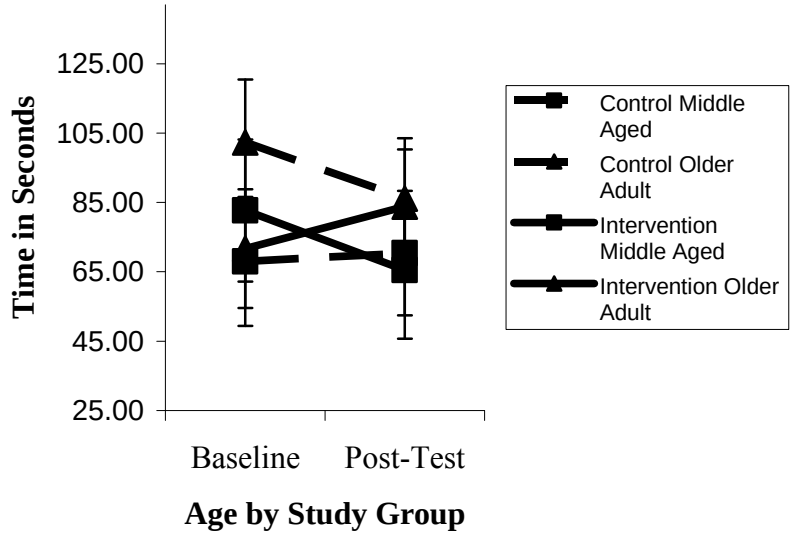
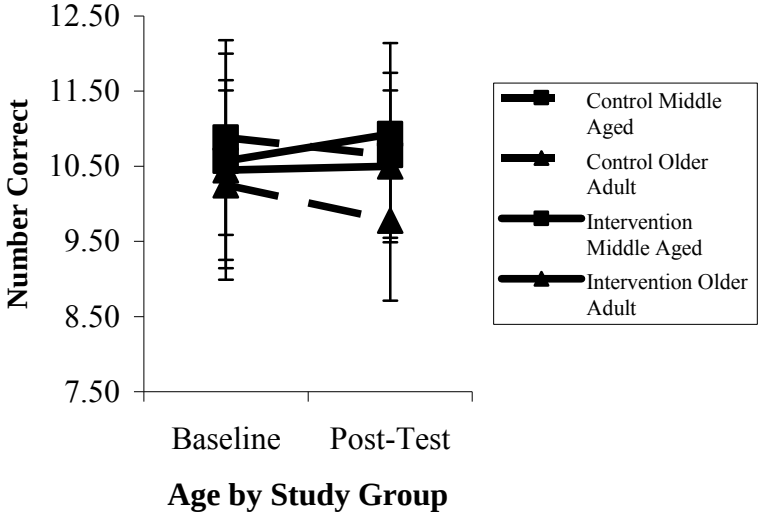


B

Figure 4-6. Mean cognitive scores by study group, age group, and occasion for executive cognitive variables A) COWA. B) Trails B. C) Letter-Number Sequencing. D) One-Back Mean RT. E) One-Back Mean RT SD. F) One-Back Number Correct. G) Two-Back Mean RT. H) Two-Back Mean RT SD. I) Two-Back Number Correct.



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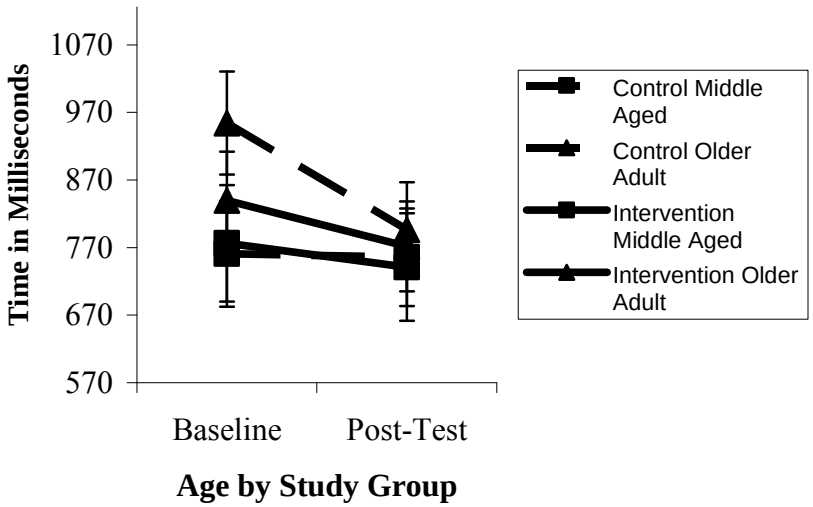

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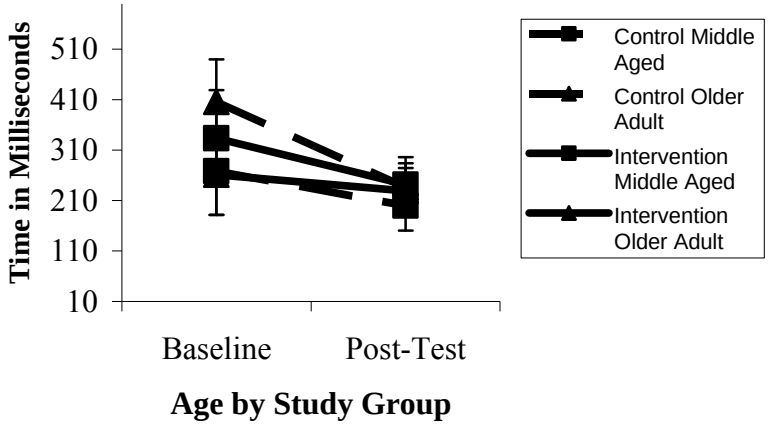

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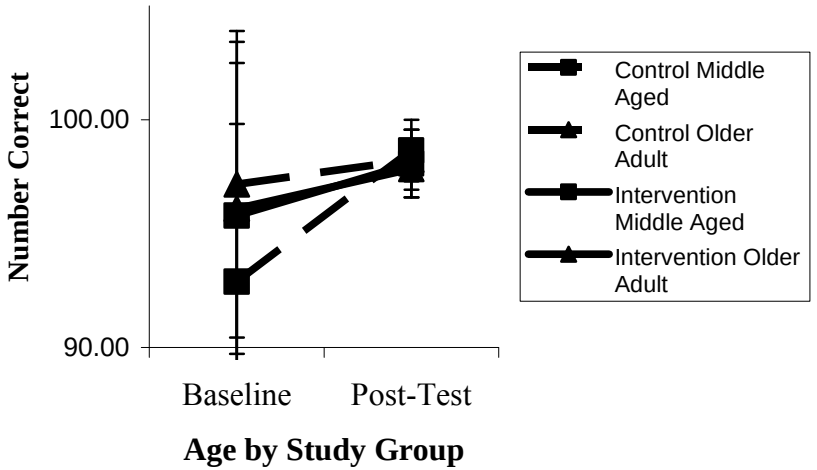

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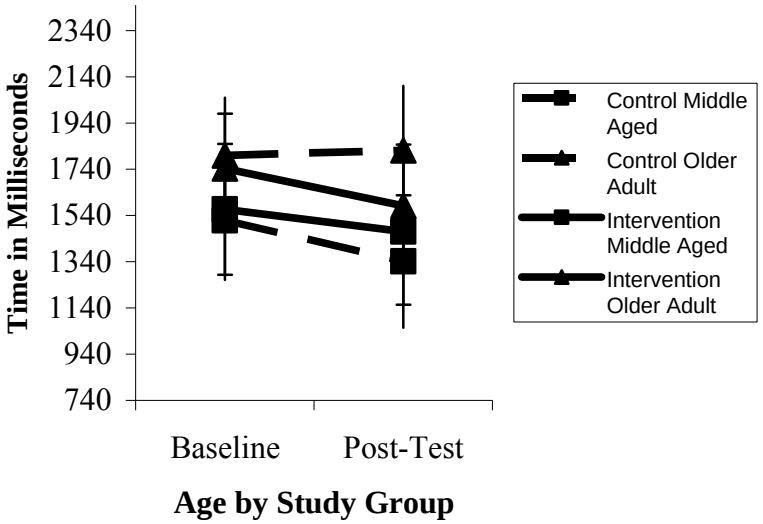

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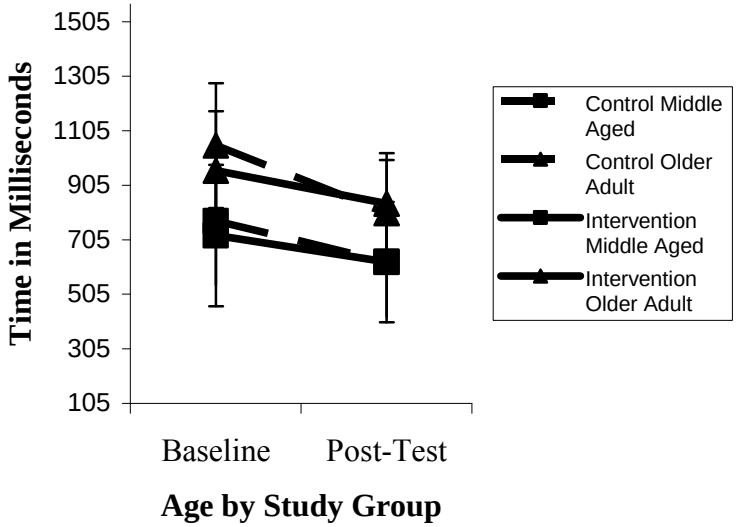

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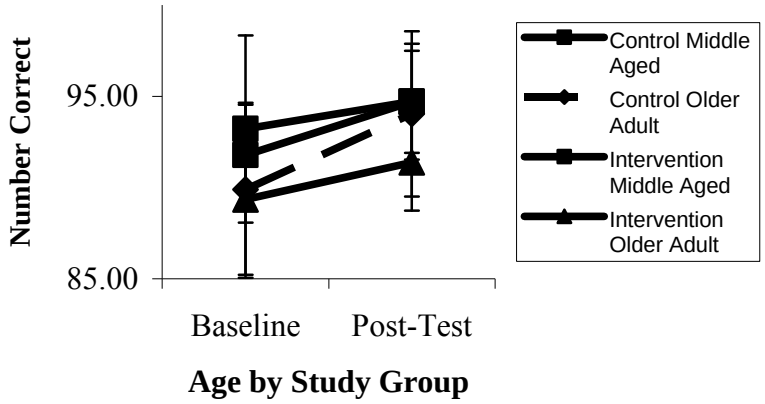

I

Figure 4-6 Continued.

CHAPTER 5
DISCUSSION

The purpose of this study was to examine the effects of an exercise promotion intervention on executive cognitive function in middle-aged and older adults. The present chapter will review the major study findings and their implications, discuss the study's methodological limitations, discuss key conceptual issues that emerged from these results, and explore future directions regarding the improvement of cognitive function through physical exercise intervention.

The current investigation followed several major premises in the existing literature regarding the plasticity of cognitive function in late life (e.g., Jobe et al., Ball et al.) and the utility of physical exercise interventions in improving cognition and cognitive performance (e.g. Colcombe & Kramer, 2003; McAuley et al., 2004). Specifically, prior research has shown that cognitive function in older adults can be improved through both cognitive and physical training. In addition, the literature inconsistently suggests that executive control processes may be disproportionately improved when cardiovascular fitness levels increase (e.g. Colcombe & Kramer, 2003; McAuley et al., 2004). Where this research has fallen short is in drawing connections between the multiple routes by which cognitive function may be improved in late life. Most of this work has concerned physical/cardiovascular fitness improvements; however, a second route to affecting late life cognitive function is through improving complex activity/social participation and mood (e.g., Brown, 1992; McAuley, 1993). Specifically, the vascular depression hypothesis accounts for the common link between depression and cardiovascular health, as well as cognitive impairment (Alexopoulos, 2006).

Review of Study Findings

The present study sought to examine two routes, a cardiovascular/physical fitness pathway and a psychosocial pathway, by which an exercise promotion intervention could result in

improvements in executive cognitive function. This study examined cognition, physical fitness and activity, and mood and affective functioning prior to and after a 16-week exercise promotion intervention and was organized with respect to the following specific aims:

- To investigate whether, relative to matched non-exercising control participants, sedentary adults receiving a physical exercise promotion intervention experience improvements in the primary outcome of cognitive function (particularly executive control processes).
- To determine the separate and joint roles of improvement in proximal outcomes (fitness, activity, and affect) in mediating exercise intervention effects on cognition.

The 69 participants included in the present analyses had an average age of 63.9 years (SD = 8.7). The majority of the sample self-identified as Caucasian/White (91.3%) and female (84.1%). In addition, participants were on average, college educated (16.2 years of education) with high average estimated IQ (NAART Predicted IQ = 113). The sample reported a minimal level of depression and anxiety symptoms.

Preliminary Analyses

Baseline correlations among measures

Prior to addressing the specific aims of the study, preliminary analyses were conducted to determine whether there was an intervention effect on the larger study's primary outcomes of physical activity and fitness. First, at baseline, there were no significant differences between the exercise promotion and control groups on cognitive, physical fitness/activity, and psychosocial variables. In addition, only baseline self-reported leisure time exercise (LTEQ) was significantly correlated with 3 of the 15 cognitive variables (two non-executive and one executive). This pattern of correlational analyses was unexpected, given the extant literature regarding the association between physical fitness/activity and cognitive performance. One explanation for this may be the significant skewness and kurtosis of many of the cognitive variables included. Even with by-cell mean replacement of outliers, non-normality among these variables likely

reduced the likelihood that significant correlations between variables would be found. Further, though power analyses indicated the present sample size provided sufficient power for the larger study, it may be the case that it was not large enough to detect the small effects of the cognitive variables, which were conceptualized as secondary outcomes in the larger study. Thus, a larger sample size would increase the power of the present study to detect these small cognitive effects.

Also of importance to note was the lack of substantial baseline association of age and education with cognition. Age showed significant positive correlations with only two (out of 15; 13.3%) baseline cognitive variables: One-Back Mean RT (r = 0.36, p = .002) and Two-Back Mean RT SD (r = 0.28, p = .022). Education was significantly correlated with only four (out of 15; 26.7%) cognitive baseline variables: NAART Estimated IQ (r = 0.30, p = 0.013), LM Immediate Recall (r = 0.37, p = .002), LM Delayed Recall (r = 0.31, p = .009), and COWA (r = 0.24, p = 0.046). This lack of expected baseline association of age and education with cognitive variables was likely due to the smaller age range than the typical extreme group design that compares young and old. The middle-aged (50-64 years) and older (65 and older) age groups were likely much closer in cognitive performance than would be needed to detect significant effects of normal cognitive aging. Further, as will be explored greater in the limitations section of this chapter, this sample was highly-educated and advantaged, with restricted variability in educational attainment. Again, a larger sample size may have improved the power to detect significant baseline relationships between these variables.

Pre-post changes in fitness and activity

Preliminary pre-post analyses of the effects of the study's intervention on the parent study's primary outcomes revealed that there were no significant intervention effects on physical fitness or physical activity outcomes. Both the intervention and the control groups demonstrated significant improvement in leisure time exercise, cardiorespiratory fitness, and minutes of

moderate and vigorous physical activity, suggesting the sample improved over time; nevertheless, there were no group-by-time interactions to suggest the intervention group improved significantly more than controls. Cohen's *d* effect sizes for the significant effects of time were small to medium (LTEQ, $d = 0.51$; MVPA, $d = 0.51$; mean pedometer steps, $d = 0.22$; VO2, $d = 0.34$). In this study, there was an average 7% increase in VO2, and a 67% increase in self-reported metabolic equivalents of activity (LTEQ). As a meta-analysis (reviewed below) suggests, these VO2 improvements did not reach the threshold of clinical significance. The more improved self-reported measure has not been studied with regard to cognitive outcomes, thus the clinical significance of the improvement on this measure is harder to judge.

Previous studies have shown that improvement in aerobic capacity by 11% was unrelated to cognitive improvement above a practice effect (Madden et al., 1989); nevertheless, a 27% increase in aerobic capacity was associated with significant cognitive improvement in a prior study (Dustman et al., 1984). A more recent meta-analysis studying the aerobic capacity/fitness-cognition link reviewed eleven randomized, controlled trials and found that aerobic fitness interventions resulted in an approximate 14% increase in cardiorespiratory fitness (Angevaren et al., 2008). This 14% increase was associated with significant improvement in cognitive function: The largest effects on cognition were found for motor function (1.17), auditory attention (0.52), and delayed memory functions (0.50). When compared to effect sizes of the current cognitive variables, these published effects are generally much larger than those here.

Pre-post changes in self-efficacy, anxiety, and depression

With regard to psychosocial outcomes, for one self-efficacy measure (EXSE) there was a between-subjects effect, suggesting the intervention group had significantly higher mean exercise self-efficacy scores at both baseline and post-test; nevertheless, a within-subjects effect for occasion on this measure indicated there was reduced self-efficacy over time for both groups.

As discussed later in the study limitations of this chapter, it may be the case that participants became more realistic regarding their personal goals for physical activity change and felt less confident in their ability to make such changes. Cohen's *d* effect sizes were small for these significant occasion effects (Cohen, 1992; BSE, *d* = -0.12; EXSE, *d* = -0.29). There were no group-by-occasion interactions. Examination of changes in anxiety and depression symptoms indicated that there was only a multivariate effect of occasion and univariate within-subjects effects for the STAI-Trait Anxiety and BDI-II Depression measures. Overall, the sample improved in trait anxiety and depression symptoms over time; however these were small to medium effects (Cohen, 1992; GDS, *d* = 0.01; BDI-II, *d* = 0.58; STAI State Anxiety, *d* = 0.13; and STAI-Trait Anxiety, *d* = 0.41).

Given the results of these preliminary analyses, it is important to note that the core assumption of this study's conceptual model was that improvements in physical activity, fitness, and self-efficacy and affect would mediate or moderate any changes in cognitive, particularly executive, function. Thus, the likelihood that the present study would observe an effect of the intervention on cognitive improvements over time was greatly reduced. Despite this, the planned analyses were completed to assess for any direct associations between the exercise promotion intervention and cognitive variables.

Primary Analyses

Aim One

The goal of the first specific aim of the study was to examine the effect of the exercise promotion intervention on cognitive performance. It was hypothesized that participants receiving the exercise promotion intervention would show improved performance on cognitive measures, particularly those assessing executive control processes, relative to control participants. However, contrary to this hypothesis, the present findings indicated no significant

multivariate effect of the intervention on cognitive performance for either executive or non-executive measures. There was a multivariate effect of occasion, suggesting there was change in overall performance on non-executive and executive cognitive measures across time. However, there was no evidence of the critical group-by-occasion interactions at the multivariate level. This indicated that the intervention was not effective in improving cognitive outcomes. Reasons for the failure to find intervention effects will be discussed in the limitations section that follows.

Despite the lack of multivariate between-subjects or interaction effects, exploratory follow-up univariate analyses were conducted to determine the directions of significant multivariate within-subjects effects observed for both non-executive and executive measures. Univariate results revealed no between-subjects effects; however, there was a modest group-by-occasion interaction for the COWA test indicating intervention group participants improved significantly more than controls over time. This interaction represented a small effect (Partial $\eta^2 = 0.06$) and was no longer significant with Bonferroni correction.

Additionally, univariate within-subjects effects were found for several executive and non-executive measures: NAART, Trails A Time, and LM Immediate and Delayed recall (non-executive measures), and COWA, One-Back Mean RT, Two-Back Mean RT, and Two-Back Mean RT SD (executive measures). The findings suggested improvements on these measures for the entire sample across time, which was likely a practice effect. With Bonferroni correction, these occasion effects only reached significance for the NAART and LM Immediate and Delayed recall. There was little evidence that above and beyond this expected practice effect, the intervention group improved to a greater degree than controls. As mentioned above, examination of effect sizes for each cognitive change score revealed small to medium effects of time (Cohen's d = 0.049 to 0.668; Cohen, 1992). The largest effects were for the LM Immediate

and Delayed recall trials (0.605 and 0.668, respectively), while the smallest were for Letter-Number Sequencing and Trails B (0.049 and 0.087, respectively).

As a follow-up to these analyses, reliable change index scores (adjusted for the practice effect demonstrated by the control group) were calculated to examine intraindividual trajectories of change. The goal was to determine the proportion of the sample that experienced sizable gains above the typical effect of practice. Results of these analyses indicated little evidence for reliable improvement for either the control and intervention groups. The LM Immediate Recall variable showed the largest percentage of the sample experiencing improvements across time (24.6% of the sample); however, relative to controls, there were fewer intervention participants who reliably improved (28.6% of controls improved versus 20.6% of intervention participants), although this difference (nor any other) was significantly different between study groups. This suggested little evidence for reliable cognitive improvement, not due to change alone, for our sample.

Next, as additional follow-up analyses, the repeated-measures MANOVAs described above were re-run with age group as a second between-subjects factor. This permitted analysis of whether age moderated intervention effects (did one age group improve more than the other?). Further, it allowed exploration of potential two-way (study group-by-age group) and three-way (study group-by-age group-by-occasion) interactions. At the multivariate level, there were no significant effects of age group for non-executive measures, though this effect did approach significance. There were also no two-way or three-way interactions involving age group. Univariate analyses demonstrated an age group effect for Trails A Time (the younger age group was significantly faster on this task), as well as study group-by-age group interaction effect for LM Delayed Recall (older individuals in the intervention group performed significantly better

than younger individuals, regardless of study group, and older controls). This finding for LM Delayed Recall was contrary to what would be expected given the consistent findings in the literature that older adults tend to perform more poorly on measures of episodic memory. It may be the case that there was a failure of randomization. The older intervention participants were likely a selective group of individuals that did not represent the general population.

Next, for executive cognitive measures, there was a significant multivariate effect of age group, but no multivariate two-way or three-way interactions. Univariate analyses revealed significant age group effects for One-Back Mean RT, Two-Back Mean RT, and Two-Back Mean RT SD. These findings suggested the younger group (middle-aged) had significantly faster mean reaction time and were significantly less inconsistent in mean reaction time. Further, an age group-by-study group interaction for the One-Back Mean RT SD variable indicated younger control group participants were significantly less inconsistent in mean reaction time than younger intervention group and older participants. This latter finding suggested that contrary to our hypothesis, the intervention was not effective in improving cognitive function on executive measures. An age group-by-occasion interaction effect revealed younger participants improved significantly more in their mean reaction time on the One-Back Mean RT variable than their older counterparts. Furthermore, there was a three-way interaction for Trails B Time, which demonstrated that younger intervention group participants improved significantly more on this task than younger controls and all older participants. This three-way interaction provides modest support for the effect of the intervention on executive cognitive function. While, with Bonferroni correction, this effect no longer reached the criterion for significance, this finding offered trend support for the study's hypothesis of differential effects of exercise promotion on cognitive function.

Interestingly, only executive measures were sensitive to the inclusion of age as a second between-subjects variable in multivariate models. These findings indicated that there was a stronger effect of age than for the intervention in the sample. While this is, in part, contrary to the study hypotheses, these age effects were consistent with the broader cognitive aging literature indicating that older adults show age related declines in processing speed and fluid cognitive abilities. In addition, these findings supported the frontal aging hypothesis (West 1996; Greenwood, 2000; Zimmerman et al., 2006), which suggests that frontal-executive abilities undergo disproportionate declines with aging as compared to non-frontal, more posterior-mediated brain abilities (West, 1996; Greenwood, 2000; Zimmerman et al., 2006).

Aim Two

For the second specific aim of the study, it was proposed that physical fitness/activity, self-efficacy, and affect variables would be used as covariates in follow-up ANCOVAs; however, given the lack of significant intervention effects or group-by-occasion interactions, these analyses were not appropriate. Thus, instead, change scores were computed for each cognitive, physical fitness/activity, and psychosocial variable and correlated in order to determine whether changes in the cognitive variables were associated with changes in the proposed covariates. Overall, there was modest support for correlated change. Improved physical fitness was associated with improved LM Immediate Recall performance and reduced depression on the GDS, while improved leisure time exercise (LTEQ) was associated with less inconsistency on the One-Back Mean RT SD. However, contrary to expectations, improvement in mean pedometer steps was correlated with reduced LM Learning Slope across testing occasions. Furthermore, reduced GDS depression was associated with improved LM Delayed Recall, and improved self-efficacy on the BSE was correlated with improved mean performance on the Two-Back Number Correct variable. Contrary to what would be expected, reduced BDI-II depression

was related to increased reaction time inconsistency across occasions on the Two-Back Mean RT SD. It is important to note that with Bonferroni correction, these correlations between change scores were no longer significant.

Study Limitations

This study has many limitations that are important to briefly consider.

Sampling issues

First, the present study utilized a relatively small, convenience sample of predominately Caucasian/White, female, healthy, highly educated adults who were recruited from the Gainesville/Alachua County Florida area. While the current sample size proved adequate power to detect certain within-subjects effects, a larger sample size would have increased the study's power to detect between-subjects and/or interaction effects that were trends in the present data and that apparently obtained effect sizes lower than those expected a priori.

Diversity of the sample was an additional concern. Recruitment strategies (i.e., use of an older adult registry, newspaper advertisement, community flyers, and presentations at health and research fairs, etc.) were not specifically focused to permit oversampling of underrepresented racial/ethnic minorities, who traditionally suffer from health disparities and presumably would benefit from inclusion in a health/exercise promotion, lifestyle program. The present study's timeline and resources did not allow for recruitment strategies more appropriate for collecting racial/ethnic minority samples, such as identifying and collaborating with community gatekeepers (i.e., church pastors, community organizers), to be properly instituted. Furthermore, when attempts were made to form community partnerships with African-American churches and other community liaisons, competing research studies (with similar health promotion messages) that were in the field at the same time challenged the successful recruitment of participants.

Thus, as will be discussed in the future directions section of this chapter, modification of the recruitment strategies used in this study would be necessary to recruit a more diverse sample.

Exclusion and inclusion criteria may further have constrained sample heterogeneity. While it was important for the present study to have various cognitive, physical, health/medical exclusionary criteria to prevent various confounding variables from being introduced into the study and to ensure the safety of participants undergoing cardiorespiratory fitness assessment, the implementation of such exclusions may have excluded racial/ethnic minorities, particularly African-Americans, who disproportionately suffer from cardiovascular and other health conditions. The sample also suffered from a low number of male participants. While men are generally less represented in cognitive aging research due to higher mortality rates, the current study's findings regarding physical exercise intervention-related cognitive improvements are less generalizable to non-female populations. In general, this study's strict exclusionary criteria likely reduced normal variation among participants and decreased the study's power to detect major intervention effects on physical and cognitive outcomes. Future studies might consider fewer exclusionary criteria. In particular, utilizing alternative measures of physical fitness (versus VO2) would change the need for exclusion of individuals taking beta-blocker and calcium-channel blocker medications, as was done in the present study. The use of these medications is very common among this study population, but the current sample was a highly select group of people who were in good cardiovascular health and not in need of such medications.

One potential limitation concerns the inclusion of middle-aged adults (along with older adults) in the present sample, which may have introduced more "noise" in the present analyses due to the increased between person heterogeneity. However, this is unlikely given that the two

age groups had similar mean years of education and percent gender, and when follow up exploratory analyses controlled for age group assignment, the overall pattern of results remained unchanged. In fact, the inclusion of the age group variable revealed significant relationships that would have otherwise gone unobserved. Specifically, the three-way interaction showing greater improvements in performance over time on the Trails B task for younger intervention group participants was an interesting finding and one of few significant study findings that was in the expected direction. This finding was considered a small effect (Partial $\eta^2 = 0.08$) and was no longer significant with Bonferroni correction; however, this trend is consistent with study hypotheses.

Retention issues

A second study limitation involves retention of the sample. The larger, 16-week intervention study, in which the present study is embedded, required a considerable time commitment from participants, which made recruitment and retention of participants challenging. Of the 433 participants identified through various recruitment methods, 90 were randomized to a study group and pre-tested. This amounted to a 20.9% recruitment yield. While only twelve participants dropped out of the study during the intervention period, there were nine participants who underwent baseline neuropsychological testing and did not attend any study group sessions. Had the study retained these 21 participants that were randomized and pre-tested, but not post-tested, this would have equated to roughly a 30% increase in the present sample's numbers. Other retention issues involve the collection of complete data for each participant. As noted in the Methods chapter, there was a substantial amount of missing physical activity and self-efficacy data towards the end of the study, due to participant non-adherence to study protocols (i.e., completing daily and weekly questionnaires on physical activity and self

efficacy). This may have been in part due to the tapering of group sessions towards the end of the 16-week intervention period.

Interestingly, the current study drop-out rate was higher than that of various previous physical activity/exercise training studies (reporting attrition rates), which had retention rates ranging from 83 to 92 percent (Madden et al., 1989; Blumenthal et al. 1991; Hawkins et al., 1992; Emery et al., 1998; Elavskey et al., 2005; Motl et al., 2005; Elavsky & McAuley, 2007). In these studies, participants were either highly motivated to participate (Madden et al., 1989; Blumenthal et al. 1991) or the interventions employed a structured exercise training protocol that followed American College of Sports Medicine (ACSM) guidelines for physical activity intensity and duration minimums to improve self-efficacy, self-esteem, and quality of life. Employing more structure in the exercise promotion lifestyle intervention may have helped participants to feel more confident in the physical activity changes they were making. Using ACSM guidelines also may have ensured sufficient exercise behavior changes to effect physical fitness at the magnitude needed to impact cognitive function significantly. Furthermore, as mentioned previously, participants that dropped out of the present study differed significantly form those completing the study only in self-reported depression and anxiety symptoms. Perhaps, initially intervening upon these symptoms would have resulted in a higher rate of retention. Finally, having more financial resources to recruit and retain participants may have resulted in a higher final sample size. Katula and colleagues (2007) used numerous methods (brochures, newspaper, radio, television, etc.) to recruit participants during the course of a four-site randomized, controlled pilot study of the benefits of a physical activity intervention for immobility prevention in older adults. In the end, the study successfully randomized 424 older adults (a 13.5% recruitment yield) to two intervention conditions after spending approximately

$439 in direct recruiting costs per randomized participant. While the numbers involved in Katula et al.'s study were beyond the scope of the present study, their findings give some insight into the feasibility of recruiting geographically and ethnically diverse populations of older community-dwelling adults for a physical activity intervention trial.

Measurement issues

A third limitation involves the selection neuropsychological battery of tests employed. Since the current study was designed to be embedded within a larger research protocol, it was necessary to limit the breath of neuropsychological assessment to be undertaken at baseline and post-intervention. Traditional neuropsychological evaluation is comprehensive in nature, and it may be the case that with a greater breadth of cognitive measures, intervention effects on cognitive performance would have been better detected. Nonetheless, the present selection of cognitive measures was guided by prior research that has distinguished the effects of improved cardiovascular fitness on executive cognitive function; particularly executive control processes (Colcombe & Kramer, 2003). While the present investigation relied primarily on clinical and experimental measures of one aspect of executive cognitive function, working memory, it may be the case that inclusion of other measures of working memory and/or other aspects of executive function (such as response inhibition) would have increased the likelihood of significant cognitive effects. Noteworthy is the fact that there was a near-ceiling effect on the N-back working memory task accuracy. This was one example of the need for more extensive neuropsychological assessment in the present study.

The present study used a modified treadmill protocol that estimated VO2 after 85% of maximal heart-rate was reached, which, while safe for an older sample, was a less sensitive estimation of cardiorespiratory fitness. It may be the case that the use of a VO2 max stress test instead would have resulted in stronger relationships between cardiorespiratory fitness and

cognition; nevertheless, VO2 max may not be a direct indicator of cerebral profusion. Neuroimaging should be employed in the future to better assess the direct effect of cardiorespiratory fitness on the brain.

As mentioned previously, the timing of the post-test interval was variable across participants. Most participants were tested at varying intervals following the intervention period. While efforts were made to ensure each participant was post-tested within two weeks of study completion, scheduling conflicts precluded this from being accomplished for some participants (3 out of 69; range 3 to 6.4 weeks post-intervention). This variability in post-test intervals may have introduced error into our analyses by not capturing each participant at the same point in the study timeline. Since it might be expected that the strongest physical activity/fitness intervention effects would be found just after the completion of the intervention program, all participants ideally would have been tested at the same point in their individual timelines.

Intervention issues

Fourth, the exercise promotion intervention was a "novel" approach to exercise intervention that combined various theoretical approaches. The larger study utilized a 16-week intervention period (with 13 weeks of small peer group sessions), which may not have been extensive enough impact exercise behaviors, and in turn, fitness and cognitive improvements. Additionally, the program was a lifestyle intervention that was not designed to prescribe specific time-periods or intensity of exercise behavior, with the rationale that this type of intervention would result in increased maintenance of lifestyle changes and more favorable long-term outcomes. The lack of specific behavioral targets for intervention participants, while consistent with the intervention's theoretical underpinnings, likely resulted in this group failing to meet minimum physical activity levels that would effect physical fitness improvements sufficient enough to mediate cognitive change (Buman, 2008). It may be the case that without the

concrete, structured program of other exercise studies (Madden et al., 1989; Blumenthal et al. 1991; Hawkins et al., 1992; Emery et al., 1998; Elavskey et al., 2005; Motl et al., 2005; Elavsky & McAuley, 2007), participants simply did not exercise enough or at a level of intensity for physical fitness and cognitive improvements to be detected. Effect sizes for these physical fitness/activity variables were small for both between-subjects effects and group by occasion interaction effects (Partial Eta^2 ranging from 0.0 to 0.1), and this suggests that there was little association between group membership and fitness/activity levels. Another consideration is the tapering of group sessions in the final weeks of the intervention period. While this decision was made to slowly wean participants from the influence of social contact and peer-mentorship afforded by study participation, the strongest physical fitness/activity effects may have been found at week 12, which was last week of real group instruction.

Also, the decline (instead of improvement) in self-efficacy over time may suggest that participants became more realistic regarding the difficulties associated with implementing behavioral changes in physical activity and thus, felt less confident in their ability to make such changes after gaining a better understanding the actual work involved. This sample may have been so highly-advantaged and accustomed to being very goal- and self-directed and highly motivated that they initially underestimated the difficulty of making physical activity changes at this stage in their lives. Their decreased self-efficacy may be driven by some comparison between their current ability to make significant activity changes and the ease with which they may have made them in the past. Furthermore, the use of multiple peer coaches may have affected treatment delivery. While quality control procedures were implemented, individual differences between the peer coaches (i.e., interactions with participants) likely differentially impacted the success of the small peer groups.

Finally, one limitation inherent in the larger study's design was permitting the health hygiene control group access to an exercise facility in exchange for study participation. This decision was made to avoid the potential confound of the anticipated intervention effects and access to a fitness facility and also mounted a very stringent test of the added value of this motivational intervention. Nonetheless, this decision precluded complete study manipulation of exercise/physical activity, as there was no way to limit the amount or intensity of exercise in the control group. Also, the social contact and use of a peer-mentorship model may have increased motivation in the control group to make changes in physical activity. It may be the case that the combination of these factors made for an uncharacteristic control group, instead of what would be expected from a more traditional, no-contact control condition.

Conceptual Issues

A number of broader conceptual issues were introduced in the Introduction Chapter, and several others emerged as a result of the analyses. This section considers several of the key issues that emerged.

Selection of the correct intervention

A first issue to consider is whether a 16-week intervention period, with only 12 weeks of actual instruction, is sufficient enough to reasonably expect adequate physiological change. Unfortunately, inconsistencies in the existing literature provide less concrete guidance in the types and lengths of exercise intervention protocols to ensure consistent effects on both physical and cognitive outcomes (Kramer et al., 2006). Review of various exercise intervention trials, with cognitive outcomes, shows that the length of training programs may range from as few as 10 weeks (Hawkins et al., 1992; Emery et al., 1998) to as many as 14 months (Blumenthal et al., 1991) and provide mixed evidence for the relationship between physical fitness and cognition. Methodological reasons for such inconsistent findings that have been discussed include

differences in the nature, intensity, and duration of aerobic fitness interventions, as well as, differences in sample characteristics, measurement of fitness, and the nature of control groups (Colcombe & Kramer, 2003). There have been several studies of self-efficacy and affective function (without inclusion of cognitive outcomes) that have employed a 6 month exercise training program (Elavskey et al., 2005; Motl et al., 2005; Elavsky & McAuley, 2007) and found positive results. Additionally, a more recent randomized, controlled fitness intervention (walking) study found that after 6 months, intervention group participants performed better on a focused attention task and had increased frontal and parietal brain activation than participants in a stretching/toning control group condition (Colcombe et al., 2004).

What is important to note is that for these studies, there was some level of structure regarding the types, duration, frequency, and often, intensity that was expected of participants. While for theoretical reasons, participants in the present study were not given specific duration or intensity prescriptions or a guideline for minimum exercise goals, it may be the case that sedentary older adults require greater level of instruction that includes specific procedural and declarative knowledge when it comes to implementing physical activity changes. Drawing from the literature on cognitive strategy training and use among older adults, studies suggest that older adults suffer from a metacognitive deficit, such that they do not spontaneously use cognitive compensation strategies (e.g., Dixon & Hultsch, 1983), even following instruction in such strategies. The present study relied more heavily on cognitive-behavioral strategies and likely held to traditional motivational interviewing techniques too closely (wherein goals are defined by the individual). A modification might well have been to establish an objective, normative target for intervention intensity, duration and frequency (e.g., ACSM guidelines), and then use the motivational interviewing strategies to help move participants from their baseline state to this

final state. In other words, helping to more actively shape the goals of participants seems a likely ingredient for higher success. Consequently, an intervention that is more behavioral in nature, with individualized targets more clearly defined a priori, may be more suitable for older groups.

Selection of the correct follow-up interval

Next, as mentioned above, the present exercise promotion was designed with long-term behavioral lifestyle outcomes in mind. As such, it may be the case that our lack of significant findings at post-test may not tell the whole story. Longer-term (e.g., 6-, 12-, or 18-month) longitudinal follow up may be necessary to detect a delayed effect of the intervention. A true test of the effectiveness of the intervention may be its effect at long-term follow up, rather than at an immediate post-test interval.

One hypothesis to consider is that intervention effects might cumulate. Specifically, the present study tried to introduce health habits and personal goal setting that would lead to an altered fitness lifestyle. If successful, the intervention would produce small incremental gains that would continue long after the study is completed. (This is admittedly idealistic; most follow-ups of exercise studies find that effects dissipate after cessation of treatment).

Another hypothesis is that intervention effects are delayed because they are revealed downstream, with a separation of the decline trajectories of those with and without the intervention. There is a recent example of such a delayed effect, albeit in the cognitive domain. In the ACTIVE clinical trial of cognitive interventions for older adults (Willis et al., 2006), ACTIVE study investigators did not find a transfer effect of cognitive (i.e., memory, reasoning, and speed) training on daily function (i.e., IADLs, self-ratings, everyday problem solving) until a 5-year follow-up assessment. Two reasons cited for this delayed intervention effect were previous work suggesting a lag between the onset of cognitive decline and the onset of functional

declines, as well as the advantaged nature of the ACTIVE sample. Specifically, since cognitively and functionally impaired individuals were initially excluded from the study, only after normative cognitive and functional declines occurred in the control group could the protective effect of cognitive training be detected (Willis et al., 2006). Thus, in the present sample, in addition to any cumulative and continuous gain that the intervention might promote, the protective effect of participation in a lifestyle physical activity intervention may not be clearly manifested until the onset of normative aging-related decline, at which point, different rates of physical decline between the study groups may be observable.

On the relative importance of physical vs. mental exercise

This study was designed to evaluate the secondary effect of an exercise promotion intervention on cognitive function. In contrast to many other studies with longer and more structured interventions, the study failed to find an exercise effect. Despite this, the majority of participants (regardless of group assignment) experienced cognitive improvements on most measures from pre-test to post-test. Interestingly, despite the lack of an intervention effect as hypothesized, the most consistent finding in the present study was the significant effect of occasion, with improvements noted across time on cognitive, physical, and psychosocial measures. It appears these within-subjects effects constituted the classic "practice effect" that has been widely shown, due to retest, across the lifespan. In fact, RCI scores (adjusted for the average change in cognitive scores for the control group) indicated few cognitive gains, above and beyond the average practice effect, for the majority of both control and intervention group participants. This is not surprising, given the lack of significant difference between the groups. Of course, using the control group as the baseline for defining practice may be problematic if our health-hygiene + gym membership control group constituted a *real* intervention. In future work with this data set, it may be important to use published test-retest and published practice effects

for our dependent variables to better gauge, for each group, whether the improvements observed exceeded what one might normally expect due to retest alone.

However, it is not clear whether these occasion effects represent "pure" practice or something more. Since both groups experienced fitness and activity gains, there is some possibility that these gains might have been activity-related for both groups. Given the weak association between improvements in physical fitness, declines in self-efficacy, and improved cognitive performance, it may be the case that, unlike in the conceptual model presented (Figure 2-1), the physical fitness and psychosocial pathways were not the important routes for cognitive improvements in this study, as hypothesized.

However, another inadvertent intervention was occurring throughout the study. Co-investigators of the larger study included weekly assessments of cognition (not the measures in this study, but Letter Series, Number Copy, Reaction Time, and Symbol Digit) in the larger study's protocol. When considering pre-test and post-test assessments, this means most participants had up to 18 weekly practice sessions with cognitive measures. Thus, this repeated assessment constituted a kind of intensive practice-based cognitive intervention and likely contributed to some the time effect observed on cognitive measures (Because of the known specificity of practice effects, and the lack of overlap between weekly and pre-post cognitive measures, the magnitude of transfer from the weekly practice may have been fairly small). Furthermore, there was no age group difference in this practice effect, such that both age groups benefited equally from practice.

These findings raise questions about the "best" approach for obtaining cognitive improvements in future work. It appears that relatively simple cognitive practice was a far simpler route to cognitive improvement than physical exercise promotion. Given this, one

wonders if the best "first-line" approach for improving cognition in the future might not be direct cognitive intervention. Of course, there are many questions to be answered. For example, the literature suggests that cognitive training effects may be highly specific to the domains studied (e.g., Willis et al., 2006), whereas physical training effects may be more broadly global (e.g., Kramer et al., 2006). Thus, one possibility is that exercise improves general brain health, and with it, promotes broad, low-magnitude cognitive gain. However, to improve specific functions to a higher magnitude, those specific functions need to be practiced and trained.

Dealing with mood and affect

This study experienced almost 30% attrition. In examining the predictors of attrition, the variables that emerged as most uniquely predictive were pre-test depression symptoms. Although the exercise promotion intervention in this study was self-efficacy focused, it did not specifically target mood issues. One wonders if better sample retention, and larger intervention effects, might have been obtained if a mood intervention component had been included in this study.

One could imagine an altered multi-step intervention model in future work. If individuals with higher levels of depression and anxiety symptoms had poor coping skills, and found it especially challenging to engage in a lifestyle physical activity program (the extreme form of which was dropout), then perhaps these depression and anxiety symptoms should have been addressed first.

It is important to note that participants were not, for the most part, at clinical levels of depression and anxiety. Rather, the argument is that subsyndromal depression and anxiety may interfere with full participation in the intervention. If this is true, then future research might first employ cognitive-behavioral approaches (either in the full group, or individually in one-on-one sessions with persons experiencing elevated levels) for dealing with mood issues. The idea is

that mood interventions become the "pre-intervention." One rationale of such affective pre-intervention would be to prepare the individual to subsequently maximize his or her ability to receive and benefit from subsequent physical and cognitive lifestyle intervention.

Future Directions

Longitudinal follow-up

There are several directions that the present line of research may take in the future. First, longitudinal follow up of current sample is planned in the larger study to determine whether any lifestyle changes in physical activity and exercise are maintained over time. Despite the lack of significant between-group findings for physical fitness and activity, there were within-subjects effects for VO2, leisure time exercise, and minutes of moderate and vigorous physical activity. Thus, it would be informative to assess longitudinally whether any of these effects remain, and/or whether there is group separation in maintenance or subsequent improvements. From a cognitive standpoint, longitudinal assessment also would be interesting to determine whether any long-term gains in cognitive performance. Furthermore, the larger study as a broader goal of using the present physical, psychosocial, and cognitive data as pilot data for a future, large-scale study examining these relationships. It will be important for future study in this area to improve upon the present study's methodological limitations in order to increase the likelihood that an intervention effect would be detected.

Enhanced sampling

A second future direction that is of particular significance is improving the cultural diversity of physical exercise promotion studies in the future. Not only would improving cultural diversity increase the heterogeneity of future samples, such improvements would also allow for more interesting questions regarding the generalizablity of physical exercise interventions and study findings to various populations of middle-aged and older adults. This is

of importance from a public heath perspective, since cardiovascular disease, obesity, hypertension, and other related chronic health conditions are among the leading causes of morbidity and/or mortality in the US (Mokdad et al., 2004) and are related to poorer performance on neuropsychological and cognitive measures (e.g., Waldstein, 1995; Breteler, van Swieten, Bots, & Grobbee, 1994). Racial/ethnic minority groups, particularly African Americans, tend to suffer from vast disparities in health status relative to the majority population, and in turn, have higher rates of these cardiovascular conditions and poorer cognitive performance (National Center for Health Statistics (NCHS), 1990). Thus, the implementation of a physical exercise promotion intervention in culturally diverse samples could begin to remediate prevalent cardiovascular health conditions in underserved groups. In addition, examining physical fitness-related improvements in cognitive performance in a diverse sample could further augment the literature that has attempted to both characterize and explain racial/ethnic group differences in late life cognitive function (e.g., Aiken-Morgan et al. 2008; Manly, Jacobs, Touradji, Small, & Stern, 2002). This work might focus on whether physical fitness improvements resulted in similar cognitive gains for multiple racial/ethnic groups.

To accomplish this goal of examining a culturally diverse sample in future work, recruitment methods must be modified. Specifically, partnerships with community gatekeepers, (such as church pastors, heads of community organizations, etc.) would need to be formed and maintained in a manner that would be equally beneficial to both the researcher and the community being targeted. Retention also would need to be a key focus of future work, and study operations would likely need to take place in the community at a convenient location, or even the participants' homes, to ensure continued participation throughout the study timeline.

Finally, collaboration with researchers in the field with expert experience in the recruitment and retention of diverse samples would be ideal.

Further conceptualization of the target cognitive domains

A third future direction, further conceptualization of executive cognitive outcome measures, has theoretical implications for the continued examination of the differential effect of improved cardiovascular fitness on executive cognitive function. The term "executive function" is used as an umbrella term to describe an individual's ability to engage successfully in independent, purposive, self-serving behavior and involve strategies necessary to approach, plan, or carry out cognitive tasks, or the strategies needed in the monitoring of performance (Lezak et al., 2004). Given such a broad definition of this construct, it is understandable that previous literature has shown little consensus regarding appropriate measures of executive function or its underlying factor structure (Kemper & McDowd, 2008). Throughout the literature, there have been many measures of executive function used, with both macro and micro approaches to assessment (Kemper & McDowd, 2008). This variation has contributed to differing factor structures observed for executive function based on the sample studied (i.e., young adults, normal elders, and Alzheimer's patients). One example is how Royal et al. (2003) found one three-factor structure of executive function in older adults (abstraction, procedural control, and attention switching), while others have found a different three-factor structure (shifting, updating, and inhibition) in younger samples (Miyake et al, 2000; Friedman et al., 2004). In general, convergent validity and discriminant validity among executive measures have not been well supported (Luszcz & Lane, 2008). Salthouse (2005) has posited that executive function tests are essentially tests of fluid ability or general intelligence ("g").

Two specific problems with executive function tests that have been cited include a "task impurity" problem and the fact that performance on executive measures may reflect many

executive processes (Luszcz & Lane, 2008). Nonetheless, verbal fluency (COWA) has been identified by previous studies as a "gold standard" measure of executive function, due to its sensitivity to frontal lobe function (Crawford et al., 2000 & 2005; Salthouse et al., 2003). The present study's findings that COWA was most sensitive to the intervention effect (albeit very modestly) are consistent with this previous work.

This lack of consensus in conceptualization and assessment of executive cognitive function likely contributes to the inconsistencies in the extant literature regarding the relationship between physical/aerobic fitness and cognitive function. To date, the studies that have concluded that executive cognitive function is disproportionately affected by improvements in cardiovascular/physical fitness have examined predominately one type of executive function: executive control processes or working memory (Colcombe & Kramer, 2003). However, abstract reasoning, conceptualization, and problem-solving are also considered executive functions that are sub-served by frontal lobe-subcortical connections in the brain via white matter tracts, which are disproportionately affected by both normal brain aging (e.g., Sullivan & Pfefferbaum, 2006) and are particularly sensitive to cardiovascular and cerebrovascular health declines (e.g., van Boxtel et al., 2006). Nevertheless, little attention in the literature has been given to these other types of executive cognitive functions, which tend to depend on conceptualization and abstraction and less on one's ability to mentally hold and manipulate information or rapidly shift/alternate between competing mental sets, as do working memory tasks. This distinction is important because working memory tasks rely more on intact attentional abilities, and these tasks often have a speed component to them (such as the Trail Making Test and N-back task). Executive measures of conceptualization and abstract reasoning/problem-solving abilities do not require the same level of attentional control and future

research should examine these other types of executive functions to gain a more complete understanding of the effects of cardiovascular/physical fitness.

It may be the case that the fitness-cognition relationship is mediated primarily by the attentional system, such that this is the mechanism for improved executive control processing. In fact, in Colcombe & Kramer's (2003) meta-analysis, there was no control for the overlap between executive measures that could also be categorized as measuring a related cognitive function (i.e., speed, visuospatial ability, or sustained attention). Their "executive" category included the largest number of studies; thus, this category may have had the most variance, and that could be the explanation for why executive measures appeared to be most-improved by exercise interventions. Very concretely, almost every measure in their meta-analysis was classified as executive *and* something else. Thus, Colcombe & Kramer's conclusion that executive control processes show the most exercise-related improvements may require further examination.

Future research should examine more than executive tasks that are highly dependent on the attentional system in the quest to better understand the link between fitness and executive cognitive function. Including measures of higher-order conceptual abilities would provide a deeper level of analysis of all of the executive functions that are supported by frontal lobe and subcortical brain function.

Future work should also pay attention to sensitivity and specificity of measures in the selection of cognitive batteries. More sensitive measures might be selected in a highly-advantaged sample, such as the present one, to maximize the likelihood of detecting individual differences in baseline performance, which could then be more sensitive to a physical

fitness/exercise effect. In other words, there must be sufficient variability in our instruments so that they can be sensitive to improvements.

However, if a sample included a less healthy sample of individuals (e.g., those with cardiovascular disease) specificity of measures might be more important to consider. Instruments that are sensitive specifically to cardiovascular impairment, and that therefore might respond to improvements in cardiovascular condition would be desired. In order to identify such cardio-sensitive measures, preliminary measurement work might need to be conducted in an age-homogenous sample (to eliminate age-related variability) or in a sample more representative of the lifespan (so that age can be covaried out before identifying measures that have high degrees of unique cardiovascular variance).

Further conceptualization of fitness and activity measurement

In addition to exploring other measures of executive cognitive function, future studies in this regard would benefit from the use of alternative measures of cardiovascular health/fitness. The present study used a VO2 estimate, which is a measure of cardiorespiratory fitness or the circulatory and respiratory systems' ability to efficiently supply oxygen to the body during sustained physical activity. However, in order to include this measure, participants taking beta-blockers and/or calcium-channel blockers had to be excluded due to the negative health risk of pushing these individuals to 85% of their maximal heart rate. This exclusionary criterion excluded a larger number of potential participants. One approach in the future may be to include a physician and/or nurse (i.e., trained clinicians) on the study team to perform this test in a safe manner.

Nevertheless, despite VO2 being considered a gold-standard in the field (the present study used a modified treadmill protocol that estimated VO2 after 85% of maximal heart-rate was reached), future research might consider other approximations of physical fitness that would

exclude fewer older participants from this type of study. Colcombe et al (2004) used the Rockport 1-mile walk test as a more appropriate measure of aerobic fitness for use with older adults and reported a high correlation between the Rockport test and treadmill VO2 ($r = .88$, $p < .01$) (Colcombe et al., 2004). Furthermore, measures of general lung function might approximate an individual's fitness, specifically their ability to efficiently use oxygen necessary for meaningful physical activity. In particular, measures that might be considered are forced expiratory volume (FEV; a measure of amount of air that can be forcibly blown one seconds) and forced vital capacity (FVC; the total amount of air that can forcibly be blown out after full inspiration). These spirometry measures are considered primary indicators of lung function, and they may be useful predictors of cognitive function in an aging sample. For instance, one literature has investigated the relationship between lung function and cognitive performance and found positive relationships across various cognitive measures (e.g., Allaire, Tamez, Whitfield, 2007). Less work has been done to determine whether executive cognitive function might show disproportionate associations with spirometry measures.

Relative effect size of exercise and combinatorial interventions

Finally, another future direction of the present research is implementing interventions in older populations that combine physical and cognitive training protocols. The literature suggesting the benefits of both physical fitness intervention (Colcombe & Kramer, 2003) and cognitive training (Jobe et al., 2001) on cognitive performance in older adults helped to build the premises for this study that cognition in late life is plastic and may be improved through intervention. Beyond the scope of this study was the implementation of other experimental conditions (i.e., cognitive and combined physical and cognitive) that could be manipulated to see if cognition, particularly executive function might be improved. While there is little previous work in this regard, a novel intervention approach would allow for the comparison of the

efficacy of various types of interventions, as well as exploration of the optimal combination of cognitive and physical training that would result in reliable cognitive outcomes for aging populations.

Conclusion

As the aging population in the US and world continues to rapidly grow, the call for lifestyle interventions to prevent the onset of both physical disease and cognitive impairment and dementia will also grow stronger. While the present study found weak effects of an exercise promotion program, as well as only modest support for association between physical fitness/activity and psychosocial improvements and cognitive performance improvements over time, it is critical that future intervention work in the field of cognitive aging build upon the present study. This work has significant public health implications, as the societal burden of caring for older adults with disabling medical diseases, including Alzheimer's disease and other dementias, is great and threatens to become even greater in the future without effective, preventive lifestyle intervention.

APPENDIX A
TELEPHONE SCREENING PROCEDURE

ELIGIBLE: _____
SCHEDULED: _____
INELIGIBLE: _____

THE AAMP STUDY

TELEPHONE SCREENING PROCEDURE

This telephone screener includes (a) telephone consent, (b) exclusionary criteria, (c) basic demographic questions, (d) the Telephone Interview of Cognitive Status (TICS), and (e) the Exercise Staging Algorithm (ESA)

Participant ID Number: _____

Date of Screening: _____

Name of AAMP Researcher conducting the Screening:

_____

TELEPHONE INTRODUCTORY SCRIPT:

Hello, may I speak with (NAME)?-------->**WAIT TO SPEAK WITH PARTICIPANT.**

My name is (TESTER) and I'm calling for the AAMP Study at the University of Florida. You recently indicated your interest in participating in this peer-mentored exercise promotion research study for older adults. Do you recall your previous interest in this study?

IF PARTICIPANT RECALLS PRIOR CONTACT:	**IF PARTICIPANT DOES NOT RECALL PRIOR CONTACT:**
↓ Do you recall how you heard of the AAMP Study? _____ ↓ Do you have any questions?	↓ The purpose of this program is to promote health and exercise behaviors in older adults through the use of a peer-mentoring program. Do you have any questions?

IF PARTICIPANT HAS QUESTIONS. ANSWER APPROPRIATELY AND STATE:

I am calling today to see if you are eligible for this program. I would like to ask you a few questions which will take about 20 minutes. Is this a good time to talk with me?

IF YES: BEGIN TELEPHONE SCREENER.

IF NO: GET CALL BACK TIME AND STATE: Thank you. A member of our staff will call you then.

Call back date and time: _____

[If you will be unable to make this call-back appointment yourself, please make arrangements with another researcher to do so.]

Informed Consent to Participate in Research - Telephone Screening

1. Name of Participant ("Study Subject")

_____

2. Title of Research Study

Trial of a multi-component exercise promotion intervention for older adults

3. Principal Investigator and Telephone Number(s)

Peter A. Giacobbi, JR., Ph.D.

THE FOLLOWING CONSENT/CONFIDENTIALITY STATEMENT MUST BE READ TO ALL POTENTIAL PARTICIPANTS AND INITIALED.

"Before we begin, I'd like to mention a few things. All of your responses are completely confidential, and will only be seen or heard by people directly associated with the study. No information about any specific individual will ever be reported. Your name will never appear in any report about this study. You may refuse to answer any questions at any time. Do you have any questions before we begin?"

I HAVE READ THIS STATEMENT TO THE PARTICIPANT.

Signature/Initials of Telephone Interviewer: _____

Print Name: _____ Date: _____

First, I would like to confirm some information about you.

1. Can you please give me your full name, address, and phone number?

Name: _____

Address: _____

_____

Phone: _____

E-mail (if applicable):_____

ASK GENDER (2) ONLY IF NOT KNOWN OR UNABLE TO DETERMINE.
OTHERWISE, CODE QUEST. 2 AND NOW ASK PREFERRED TITLE QUESTION.

2. Are you male or female?

MALE..... 1 [Is that Mr., Dr., Rev., or other?]
RECORD PREFERRED TITLE ON CONTACT RECORD.

FEMALE.....2 [Is that Mrs., Miss, Ms., Dr., Rev., or other?]
RECORD PREFERRED TITLE ON CONTACT RECORD.

3. What is your date of birth? .___/____/._____

IS PARTICIPANT'S AGE WITHIN 6 WEEKS OF 50th BIRTHDAY OR OLDER TODAY?

YES 1

NO 2	**INELIGIBLE: READ SCRIPT BELOW AND END INTERVIEW**

AGE INELIGIBILITY CLOSE-OUT SCRIPT:
These are the only questions I need to ask. This research study is designed for people who are age 50 or older. I would like to thank you for the time you have taken to speak with me. We will not need to contact you again for this study, but could we contact you in the future for other studies? Thank you.

3a. Is English your first language? YES ……1
NO ……2

4. What is your marital status?
[READ RESPONSE CATEGORIES IF UNABLE TO ANSWER]

MARRIED, ...1

LIVING AS MARRIED, ...2

SEPARATED, ...3

DIVORCED, ...4

WIDOWED, ...5

SINGLE, OR NEVER MARRIED? 6

5. Does anyone live in the home with you?

YES.....1 NO.....2

6. What is the highest grade of school or level of education that you completed?[CODE ONLY ONE RESPONSE]

DID NOT GO TO SCHOOL	00	VOCATIONAL/TRAINING/ SOME COLLEGE AFTER HS GRAD	13
GRADE 1	01		
GRADE 2	02	ASSOCIATE DEGREE	14
GRADE 3	03		
GRADE 4	04	COLLEGE GRAD/BA-BS	16
GRADE 5	05	SOME PROFESSIONAL SCHOOL AFTER COLLEGE GRAD	17
GRADE 6	06		
GRADE 7	07	MASTER'S DEGREE	18
GRADE 8	08		
GRADE 9	09	DOCTORAL DEGREE (PhD, MD, DVM, DDS, JD, etc.)	20
GRADE 10	10		
GRADE 11	11		
GRADE 12/GED	12		

7. What race do you consider yourself? (PROBE: White, Black/African American, Asian, Native Hawaiian/Pacific Islander, American Indian/Alaskan Native, or another race?)

WHITE/CAUCASIAN 1

BLACK/AFRICAN AMERICAN 2

ASIAN 3

NATIVE HAWAIIAN/PACIFIC ISLANDER.....4

AMERICAN INDIAN/ALASKAN NATIVE.....5

BIRACIAL.....6

* SPECIFY:_____

OTHER 7

* SPECIFY: _____

DON'T KNOW 8

IF PARTICIPANT IS UNABLE TO ANSWER 7, PROBE: Which race do you most identify with or consider yourself to be?

8. Are you Hispanic or Latino?

YES.....1 NO.....2

9. The next questions are about your vision. Do you wear glasses or contact lenses to read?
YES.....1 NO.....2

BEGINNING WITH ITEM 10a, AND FOR ALL OTHERS, DO NOT TERMINATE INTERVIEW IF INELIGIBLE. COLLECT ALL DATA, THEN READ INELIGIBILITY SCRIPT BEFORE ITEM 15.

10a. How much difficulty do you have reading ordinary print in the newspaper, [wearing glasses or contact lenses]? Would you say...

no difficulty.....1 (11)

a little or some difficulty.....2 (11)

extreme difficulty .....3 = **INELIGIBLE** (11)

you stopped reading because of your eyesight .....4 = **INELIGIBLE** (11)

you stopped reading for other reasons or you are not interested in reading.....5 (11)

10b. How much difficulty do you have hearing conversation partners, when in small groups and there is background noise? (multiple conversations, music, white noise) What about with your hearing aid(s) on? Would you say...

no difficulty.....1 (11)

a little or some difficulty.....2 (11)

extreme difficulty .....3 = **INELIGIBLE** (11)

you stopped participating in small group/ social settings because of your hearing.....4 = **INELIGIBLE** (11)

you stopped participating in small group/social settings for other reasons or you are not interested in reading.....5 (11)

The next few questions are about medical conditions you might have.

11. Has a doctor or a nurse ever told you that you have:

	YES	**NO**	**DK**	**N/A**
a. Alzheimer's Disease, or dementia	1 = **INELIGIBLE**	2	8	
b. Huntington's disease, with dementia symptoms	1 = **INELIGIBLE**	2	8	
c. Parkinson's disease with dementia symptoms	1 = **INELIGIBLE**	2	8	
d. Recurring epilepsy?	1 = **INELIGIBLE**	2	8	
e. Stroke?	1 = ASK NEXT QUESTION	2	8	
f. [Was it in the past year?]	1**= INELIGIBLE**	2	8	- 7
g. [Do you have limb weakness or paralysis as a result?]	1= **INELIGIBLE**	2	8	-7
h. heart attack or myocardial infarction?	1 = ASK NEXT QUESTION	2	8	
i. [Was it in the past year?]	1**= INELIGIBLE**	2	8	- 7
j. A head injury requiring hospitalization any time in your lifetime?	1= **INELIGIBLE**	2	8	
k. been hospitalized for psychiatric illness at any point in your lifetime, or do you currently have a psychiatric illness?	1= **INELIGIBLE**	2	8	
l. cancer, other than skin cancer, within the past 5 years?	1= ASK NEXT QUESTION	2 **(14)**	8 **(14)**	
m. [Are you <u>currently</u> receiving chemotherapy or radiation treatment for this cancer?]	1= **INELIGIBLE**	2	8	-7
n. Did you ever receive radiation treatment for a cancer above the chest?	1= **INELIGIBLE**	2	8	-7

o. Do you have a pacemaker or internal defibrillator?	1= **INELIGIBLE**	2	8	
p. Do you use portable oxygen?	1= **INELIGIBLE**	2	8	
q. Do you take steroids or cortisone?	1= **INELIGIBLE**	2	8	Meds for Asthma,OK
r. Do you use a cane or walker?	1= **INELIGIBLE**	2	8	
s. Did you ever have medical problems as a consequence of alcohol or drug use?	1= **INELIGIBLE**	2	8	
t. Did you ever have legal problems as a consequence of alcohol or drug use?	1= **INELIGIBLE**	2	8	
u. Did you ever have withdrawal symptoms related to alcohol or drug use?	1= **INELIGIBLE**	2	8	
v. Are you currently on any medication?	1 = ASK NEXT QUESTION	2	8	

w. [If known, are you currently on any calcium channel blockers or beta blockers?] List Meds: (Name and purpose) _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____ _____	1= **INELIGIBLE** **Acebutolol (Monitan, Secral)** **Atenolol (Apo- Atenolol, Novo-Atenol, Ternormin)** **Betaxolol (Kerlone)** **Bisprolol (Zebeta)** **Carteolol (Cartrol)** **Labetalol (Normodyne)** **Oxprenolol (Trasicor, Slow-Trasicor)** **Bepridi (Vascor)** **Diltazem (Cardizem, Cardizem CD, Cardizem LA, Cardizem SR, Dilacor-XR)** **Betaxolo (Kerlone)** **Toprol** **Norvasc** If still unsure, look in book or Rxlist.com (clinical pharmacology)	2	8	

TELEPHONE INTERVIEW FOR COGNITIVE STATUS (TICS)

For the next few questions, it is very important that you turn off your television or radio, so that you can concentrate and hear me clearly.

Please look around you, and move all papers, newspapers, pens and pencils away from where you are. I will be asking you some questions that require you to use your memory and it's important that you don't write anything down for this part.

[**NOTE TO SCREENERS**: SINGLE REPETITIONS ARE PERMITTED ON ALL ITEMS EXCEPT FOR T5 AND T8.

T1. "Please tell me your full name." 1 point for first name, 1 point for last name ____ / 2

_____

T2. "What is today's date?" 1 point for month ____ / 5

_____

1 point for date
1 point for year

Prompt for missing parts (month, date, year, day of week, season)

1 point for day of week
1 point for season

T3. "Please tell me your age and phone number." _____	1 point for age 1 point for phone number	____ / 2
T4. "Count backwards from 20 to 1." _____	2 points if completely correct on first trial; 1 point if completely correct on second trial; 0 points for anything else	____ / 2
T5. "I'm going to read you a list of ten words. Please listen carefully and try to remember them. When I am done, tell me as many words as you can, in any order. Ready? The words are: cabin, pipe, elephant, chest, silk, theatre, watch, whip, pillow, giant. Now tell me all the words you can remember." _____ _____ _____ _____ _____	1 point for each correct response. No penalty for repetitions or intrusions. (*cabin, pipe, elephant, chest, silk, theatre, watch, whip, pillow, giant*)	____ / 10
T6. "One hundred minus 7 equals what?" _____ "And 7 from that?" _____ "Keep going"… _____ "Keep going"… _____ "Keep going"… _____	Stop at 5 serial subtractions. 1 point for each correct subtraction. (*93-86-79-72-65*) Do not inform the participant of incorrect responses, but allow subtractions to be made from his/her last response (e.g., "93-85-78-71-65" would get 3 points).	____ / 5
T7. "What do people usually use to cut paper?" _____ "How many things are in a dozen" ____ "What do you call the prickly green plant that lives in the desert?" _____ "What animal does wool come from?" _____	1 point for *scissor* or *shears* 1 point for *12* 1 point for *cactus* 1 point *sheep* or *lamb*	____ / 4

T8. "Say this: 'No ifs, ands, or buts.'" "Say this: Methodist Episcopal.'"	1 point for complete repetition on the first trial. Repeat only if poorly presented.	____ / 2
T9. "What is the full name of the President of the United States right now?" _____ "What is the full name of the Vice President? _____	1 point for correct first and last name. (*George W. Bush in 2002/2003*) 1 point for correct first and last name. (*Richard Cheney in 2002/2003*)	____ / 4
T10. "With your finger, tap 5 times on the part of the phone you speak into."	2 points if *5 taps* are heard 1 point if participant taps *more or less* than 5 times	____ / 2
T11. "I'm going to give you a word and I want you to give me its opposite. For example, the opposite of hot is cold. "What is the opposite of 'west';" _____ "What is the opposite of 'generous'?' _____	1 point for *east*. 1 point for *selfish, greedy, stingy, tight, cheap, mean, meager, skimpy*, or other good antonym.	____ / 2
T12. "Please tell me all the words you remember from the list I gave you before." _____ _____ _____ _____	1 point for each correct response. No penalty for repetitions or intrusions. (*cabin, pipe, elephant, chest, silk, theatre, watch, whip, pillow, giant*)	____ / 10
TOTAL TICS SCORE	If score is 27 or below, complete rest of screener, then confer with another AAMP staff member to determine eligibility.	_____ / 50

15. EXERCISE STAGING ALGORITHM (ESA)

"I am now going to ask you a few questions about your recent exercise habits. To do this, I will need to read to you a definition of what we mean by "regular exercise" so that we understand each other. Are you ready to hear the definition?" [WAIT UNTIL PARTICIPANT SEEMS ATTENTIVE AND READY TO LISTEN]

"Regular exercise is any planned voluntary physical activity (such as brisk walking, aerobics, jogging, bicycling, swimming, basketball, etc.) performed to increase physical fitness. Such activity should be performed *3 to 5 times per week* for a minimum of *20 minutes per session*. Exercise does not have to be painful to be effective, but should be done at a level that increases your breathing rate and causes you to break a sweat. Is this definition clear to you?" [IF YES, CONTINUE. IF NO, CLARIFY ANY CONFUSIONS, PROBE FOR EXAMPLES OF ACTIVITIES THEY SUGGEST]

(Record Persons report of Routine)_____
_____
_____
_____
_____

15a. Based on this definition, do you *currently* exercise regularly?

YES.....1 = **INELIGIBLE**

NO.....2 GO TO QUESTION #15b

[QUESTIONS #2 AND #3 ARE CODED AS ONE ITEM]

15b. Do you intend to begin exercising regularly?

YES.....1 GO TO QUESTION #15c

NO.....2 SKIP QUESTION #15c

15c. Do you intend to begin exercising regularly in the next 30 days or the next 6 months?

Next 30 days……..1 Next 6 months…….2

Determine eligibility here, before continuing. If ineligible, skip to last page and read closeout script.

If eligible say:

"This study has several sections. First, based on our phone call today, I would like to schedule an in-person visit, during which I can assess aspects of your mental and physical fitness, your health, and your everyday functioning, as well as to begin your enrollment at our Living Well facility. I would need to schedule this meeting within the next few weeks. Depending on individual circumstances, this session can take anywhere from 1 to 3 hours."

16. Are you willing to schedule this in-person meeting?

YES 1

NO .....2 = **INELIGIBLE**

"In order to be eligible to participate in this study and to ensure that it is safe for you to be physically active, we also need a signed checklist from your doctor or nurse clearing you to exercise. We have prepared this checklist, and you should be receiving it by mail soon. It should only take your doctor a few moments to complete it. We will ask you to get it filled out in the next 1-2 weeks."

17. Will you be able to make sure that your doctor or health care provider completes this checklist?

YES 1

NO .....2 = **INELIGIBLE**

"After our first in-person session and clearance from your doctor, you may be eligible to participate in our program. At the outset, however, you should know that this is an involved study and will require a significant time commitment on your part. Over the 16-week study period, you will be asked to devote approximately 1.5 hours each week to meeting with research staff. Although we provide convenient parking and flexible scheduling, it is important for you to consider whether this is reasonable for you."

18. Are you able to participate in the study for the entire 16-week study period?

YES 1

NO .....2 = **INELIGIBLE**

"In addition to the weekly time commitment, you will be asked to wear an activity monitoring device throughout the study. These devices, either an accelerometer or pedometer, should not restrict any of your normal daily activity. However it is important that you wear this device daily

throughout the entire 16-week study period. It is important for you to consider whether this is reasonable for you."

19. Do you think you would be able to wear the accelerometer or pedometer daily throughout the study period?

YES.....1

NO.....2 = **INELIGIBLE**

"You will also be asked to complete daily questionnaires that ask questions about your sleep and exercise behaviors during the previous day. Although these questionnaires should require no more than 5 minutes per day, its important that they are complete *daily* and that few days are missed. It is important for you to consider whether this is reasonable for you."

20. Would you be willing to complete daily questionnaires consistently throughout the study period?

YES.....1

NO.....2 = **INELIGIBLE**

TAKE A PAUSE WHILE YOU BRIEFLY ASSESS THE FOLLOWING TWO ITEMS.

INTERVIEWER ASSESSMENT OF PARTICIPANT COMMUNICATION

USING THE SCORING CRITERIA ON THE NEXT PAGE, CODE YOUR ASSESSMENT OF PARTICIPANT'S ABILITY TO MAKE SELF UNDERSTOOD AND TO UNDERSTAND OTHERS. THESE JUDGMENTS CAN BE MADE BOTH ON THE BASIS OF COGNITIVE UNDERSTANDING, AND ALSO OF ENGLISH-AS-SECOND-LANGUAGE ISSUES.

21. MAKING SELF UNDERSTOOD

UNDERSTOOD 0

USUALLY UNDERSTOOD.....1
(DIFFICULTY FINDING WORDS OR FINISHING THOUGHTS.)

SOMETIMES UNDERSTOOD .....2 = **INELIGIBLE**
(ABILITY IS LIMITED TO MAKING CONCRETE REQUESTS.)

RARELY/NEVER UNDERSTOOD.....3 = **INELIGIBLE**

22. ABILITY TO UNDERSTAND OTHERS

UNDERSTANDS 0

USUALLY UNDERSTANDS.....1
(MAY MISS SOME PART/INTENT OF MESSAGE.)

SOMETIMES UNDERSTANDS .....2 = **INELIGIBLE**
(RESPONDS ADEQUATELY ONLY TO SIMPLE, DIRECT COMMUNICATION)

RARELY/NEVER UNDERSTANDS.....3 = **INELIGIBLE**

MAKING SELF UNDERSTOOD SCORING		
0	= Understood:	The participant expresses ideas clearly.
1	= Usually Understood:	The participant has difficulty finding the right words or finishing

		thoughts, resulting in delayed responses; or requires some prompting to make self understood.
2	= Sometimes Understood:	The participant has limited ability, but is able to express concrete request regarding at least basic needs (e.g., food, drink, sleep, toilet).
3	= Rarely/Never Understood:	At best, understanding is limited to interpretation of highly individual, person-specific sounds (e.g. indicated presence of pain or need to toilet).

MAKING SELF UNDERSTOOD SCORING		
0	= Understands:	Clearly comprehends the interviewer's message(s) and demonstrates comprehension by words or questions.
1	= Usually Understands:	May miss some part of intent of the message but comprehends most of it. The participant may have periodic difficulties integrating information but generally demonstrates comprehension by responding to words or questions.
2	= Sometimes Understands:	Demonstrates frequent difficulties integrating information, and responds adequately only to simple and direct questions or directions. When the message is rephrased or simplified, the participant's comprehension is enhanced.
3	= Rarely/Never Understands:	Demonstrates very limited ability to understand communication. Or, interviewer has difficulty determining whether the participant comprehends messages, based on verbal responses. Or, the participant can hear sounds but does not understand messages.

DETERMINE PARTICIPANT ELIGIBILITY AND GO TO APPROPRIATE SCRIPT ON NEXT PAGE.

FOR ELIGIBLE PARTICPANTS

"Thank you for answering the questions. As I already implied, we would like to continue with you in the program and meet you in person and have you meet us. At this in-person meeting, we will ask you some more questions regarding your mental and physical fitness, health, and everyday functioning to determine if you are eligible for participation in the program. This meeting will take up to three hours depending on how much information is needed from you. It will be held at the Living Well Center at the University of Florida."

"Do you have any questions for me at this time?"

"You have already indicated that you would be willing to schedule this session. To help me with scheduling an appointment, could you tell me what other commitments you typically have during the week, such as work, volunteering, caring for others, or social activities?"

23. What days of the week work best for you? _____
24. Is morning or afternoon better for you? _____

"We will be getting in touch with you soon to schedule your first visit. In the meantime, you should be receiving the form for your doctor or nurse to complete to be cleared to participate in exercise, and we will need this clearance before we can enroll you in the study."

"Thank you very much for your time."

Could I schedule you for (date/time)?

25. Date ___/___/_____

26. Time:_____:_____AM / PM

27. Test Site:_____

28. Person: ___ _____ (Tester ID/Initials)

Do you know where Living Well is located? I will send you a letter and map with directions to our center.

Thank you. If you wear glasses for distance or reading or wear a hearing aid, please bring them with you. You should have received, or will be receiving shortly, a one-paged form that needs to be signed by your physician. Please bring this signed physician's form and any medications you take with you to your appointment. We look forward to seeing you on (day/date).

INELIGIBLE SCRIPT

"This concludes your participation in this study. Thank you for answering these questions. This has been very helpful. Based on our interview today, you are not eligible to participate in the study at this time. This is typically because individuals have health conditions that have been identified as exclusion characteristics for this study. We appreciate the time you have spent answering these questions. Although you are not eligible for this study, we may want to call you in the future about your interest in some other study."

29. May we have permission to share your interest in research with our aging research colleagues here at the University of Florida, so that other researchers can call you to invite you to consider participating in future research studies?

YES.....1
NO.....2

APPENDIX B
NURSE/PHYSICIAN CHECKLIST AND PERMISSION

Project AAMP (Active Adult Mentoring Project)
College of Public Health and Health Professions
Adrienne Aiken Morgan
PO Box 100165
Gainesville, FL 32610-0165
Phone: (352) 273-5098

Please type or print clearly

Physician's Name _____ Phone # _____

Patient's Name _____ (Project AAMP participant)

Program exclusion checklist (please check any that apply to this patient):

- ☐ Terminal illness with life expectancy of < 12 months
- ☐ Myocardial infarction in the last 6 months
- ☐ Chronic heart failure (New York Classification III to IV)
- ☐ Aortic stenosis
- ☐ Cardiac arrhythmia
- ☐ Cardiac stent
- ☐ Cardiac arrest
- ☐ Implanted cardiac defibrillator
- ☐ Pacemaker
- ☐ Coronary artery bypass graft
- ☐ History of cardiac arrest
- ☐ Uncontrolled angina
- ☐ Stroke or TIA
- ☐ Peripheral vascular disease
- ☐ Pulmonary disease requiring oxygen or steroid treatment
- ☐ Receiving chemotherapy or radiation for cancers
- ☐ Ambulation with assistive devices
- ☐ Poorly controlled diabetes
- ☐ Smoked regularly (>4 cigarettes per day) in past 3 years
- ☐ Any of the following calcium channel or beta blockerS

Beta Blockers
Acebutolol (Monitan, Sectral)
Atenolol (Apo-Atenolol, Novo-Atenol, Tenormin)
Betaxolol (Kerlone)
Bisoprolol (Zebeta)
Carteolol (Cartrol)
Labetalol (Normodyne, Trandate)
Oxprenolol (Trasicor, Slow-Trasicor)

Calcium Channel Blockers
Bepridil (Vascor)
Diltiazem (Cardizem, Cardizem CD, Cardizem LA, Cardizem SR, Dilacor-XR)
Betaxolo (Kerlone)

Note: Trade Names in parenthesis

I hereby give my patient permission to:

1. Participate in an exercise program ☐ **YES** ☐ **NO**
2. Complete a health and fitness assessments* ☐ **YES** ☐ **NO**

*The **fitness assessment** includes resting heart rate and blood pressure measurements and an 85% sub-maximal cardiovascular test (heart rate only, no EKG)

Special instructions or indicated activities: _____

Contraindications to any activities: _____

_____ _____
Nurse/Physicians' Signature [Required] **Date**

APPENDIX C
QUALITY CONTROL CHECKLISTS

EXERCISE PROMOTION INTERVENTION GROUP

Mentor/Coach _____ Date _____

Procedure	None	Part	Full	Score
CREATING A SUPPORTIVE ENVIRONMENT				
1. Reviews participants' exercise during previous week.	0	5	10	_____
2. Use of Open-Ended Questions ex.: "In what ways has exercise been helpful to you?"	0	5	10	_____
3. Use of Affirmations ex.: "You are a very conscientious person. That quality will help you to begin an exercise program."	0	5	10	_____
4. Use of Reflective Responses ex.: "It sounds like you are frustrated. How do you deal with that?"	0	5	10	_____
5. Use of Summary Statements	0	5	10	_____
6. Effective group management Keeps group on topic Manages time and pace of discussion Maintains leadership of discussion	0	5	10	_____
COMMUNICATION ROADBLOCKS				
7. Avoids Lecturing	0	10	20	_____
8. Avoids giving advice	0	10	20	_____
9. Avoids interpreting or analyzing	0	10	20	_____
10. Avoids questioning participant	0	10	20	_____
CONCLUSION				
11. Assures participant that all instructions are in the Workbook & reminds to bring all logs and complete any homework for next session.	0	5	10	_____
12. Makes appropriate referrals regarding questions participants may have about the study.	0	5	10	_____
13. Makes appropriate referrals regarding mental or physical health concerns observed during sessions.	0	5	10	_____

Total _____

Comments for Mentor:

Session Specific Evaluations

Procedure	None	Part	Full	Score
GOAL SETTING SESSION				
7. Use of OARS during discussions about goals	0	5	10	_____
8. Distinguishes between long- and short-term goals	0	5	10	_____
9. Clearly discusses SMART goals	0	5	10	_____
10. Encourages participants to set SMART goals in a non-judgmental manner.	0	5	10	_____
11. Gives examples of SMART goals	0	5	10	_____
	Total	_____		

Comments for Mentor:

Mental imagery SESSION				
12. Integrates mental imagery into discussions using OARS.	0	5	10	_____
13. Makes connections between fitness/health goals and mental imagery with open-ended questions.	0	5	10	_____
14. Gives examples of vivid images that evoke all five senses and feelings/emotions.	0	5	10	_____
	Total	_____		

Comments for Mentor:

CONTROL GROUP

Mentor/Coach _____ Date _____

Procedure	None	Part	Full	Score
GROUP MANAGEMENT				
1. Begin sessions with discussion about last week's topic, including quiz.	0	5	10	_____
2. Keeps group on topic	0	5	10	_____
3. Manages time and pace of discussion	0	5	10	_____
4. Maintains leadership of discussion	0	5	10	_____
PRESENTATIONAL SKILLS				
5. Use of Open-ended questions	0	5	10	_____
6. Clearly presents topic to be discussed	0	5	10	_____
7. Promotes discussion by asking questions	0	5	10	_____
8. Actively makes efforts to include all members of the group into discussion.	0	5	10	_____
COMMUNICATION ROADBLOCKS				
9. Avoids Lecturing	0	10	20	_____
10. Avoids giving advice	0	10	20	_____
11. Avoids interpreting or analyzing	0	10	20	_____
12. Avoids questioning participant	0	10	20	_____
CONCLUSION				
13. Assures participant that all instructions are in the Workbook & reminds to bring all logs and complete any homework for next session.	0	5	10	_____
14. Makes appropriate referrals regarding questions participants may have about the study.	0	5	10	_____
15. Makes appropriate referrals regarding mental or physical health concerns observed during sessions.	0	5	10	_____

Comments for Mentor:

Total _____

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BIOGRAPHICAL SKETCH

My academic and research interest in cognitive aging began when I was an undergraduate student at Florida A&M University in Tallahassee, Florida. During this time, I was a Distinguished Scholar, with a full academic scholarship. After graduating summa cum laude with a Bachelor of Arts in psychology in April 2002, I attended the University of Florida on a University of Florida Alumni Fellowship. I received a Master of Science in clinical psychology in May 2004. Throughout my graduate career, I was the recipient of various awards and grants, including a National Institute on Aging (NIA) Aging Research Dissertation Award to Increase Diversity (Grant #1R36AG029664-01) to fund the present dissertation research. In June 2008, I completed a pre-doctoral clinical internship in clinical neuropsychology at the University of Chicago. In addition, I will earn a Doctor of Philosophy degree in clinical psychology, with a specialty in neuropsychology and a Graduate Certificate in gerontology in August 2008 from the University of Florida. I will begin a post-doctoral fellowship in geropsychology and geriatric rehabilitation at Rush University Medical Center in July 2008, and I look forward to a career studying the influence of heath and disease on cognitive aging in racial/ethnic minority elders.