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Physical Activity and Cognitive Functioning in Older Adults: The Mediating Effect of Symptoms of Depression and Insomnia

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Physical Activity and Cognitive Functioning in Older Adults:

The Mediating Effect of Symptoms of Depression and Insomnia

By

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Kathleen T. Rhyner

Submitted to the graduate degree program in Clinical Psychology and the Graduate Faculty of the University of Kansas in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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Physical Activity and Cognitive Functioning in Older Adults:

The Mediating Effect of Symptoms of Depression and Insomnia

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Abstract

In the existing literature there is strong evidence that physical activity has a beneficial effect on cognitive maintenance in older individuals. Physical activity has been shown through brief intervention studies as well as long-term epidemiological studies to improve or maintain cognitive functioning across the lifespan. However, a key unresolved question is the mechanism by which physical activity affects cognitive performance. The present study simultaneously examined two hypotheses using longitudinal structural equation modeling (SEM). We hypothesized that higher levels of physical activity would lead to a decrease in mental health symptoms of depression and insomnia, which would in turn lead to better cognitive functioning. Community-dwelling older adults from the Health, Aging, and Body Composition (HABC) study completed a variety of measures of physical activity, depressive symptoms, insomnia symptoms, and cognitive functioning. The model of physical activity, depressive symptoms, insomnia symptoms, and cognitive functioning evaluated in the present study was found to fit the data relatively well. However, none of the hypothesized relationships between variables, beyond the autoregressive paths, were significant. The strongest predictor of cognitive functioning was an individual's prior level of cognitive functioning. This study shows if an individual has a high initial level of cognitive functioning they are likely to retain a higher level of cognitive functioning later in life.

Key Words: physical activity; cognitive functioning; depression; insomnia

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Introduction

Older adults are the most rapidly growing segment of the United States

population. The Bureau of the Census, Economics and Statistics Administration (2010)

reported that there are approximately 40.3 million people aged 65 or older living in the

United States. The previously projected rates for increases in this population continue to

be validated; the number of adults aged 65 and over increased by 15.1% from 2000 to

2010, while the overall U.S. population only increased 9.7%. One of the most common

aging-associated concerns is decline in cognitive functioning. The prevalence of

cognitive impairment generally increases with age, making it one of the most common

psychiatric disorders in older adults (Olivera et al., 2008). Older age is specifically

associated with decreased processing speed as well as impairment on other cognitive

abilities (Finkel, Reynolds, McArdle, & Pedersen, 2007; Albinet, Boucard, Bouquet, &

Audiffren, 2012; Borghesani et al., 2013). While some decline in cognitive functioning

is considered normative, individuals who meet criteria for mild cognitive impairment

transition to dementia at a higher rate than members of the general population (Petersen

et al., 2001). The development of dementia and associated problems with activities of

daily living result in a high emotional and financial cost for affected individuals, their

families, and society (Herbert, Scherr, Bienias, Bennet & Evans, 2004).

Understandably, many aging individuals are concerned about the potential

development of cognitive impairment, particularly Alzheimer's disease. A qualitative

study by Corner and Bond (2004) identified anxiety about memory loss and dementia as a

key theme, with some participants stating that Alzheimer's disease would be the most terrifying disease to die from and that individuals with the disease should "just die." The burgeoning population of older adults and increasing concern from individuals and society about cognitive decline have prompted investigations into the role of various risk or preventative factors that can influence the trajectory of cognitive functioning over time (Vemuri et al., 2012; Yaffe et al., 2009). Previous studies have examined the role of cognitive training (Rebok et al., 2014) and education (Meng & D'Arcy, 2012) on cognitive functioning. Recently, research has focused on the impact of modifiable lifestyle factors that could prevent or delay cognitive decline, such as diet and physical activity. Physical activity in particular has repeatedly been identified as an important predictor of successful cognitive maintenance during the aging process (Yaffe et al., 2009; Buchman et al., 2012).

Physical activity has a wide variety of benefits for older adults. It has been shown to decrease incidence of serious illnesses, including heart disease (Blumenthal et al., 2005), diabetes mellitus (Herriott, Colberg, Parson, Nunnold & Vinik, 2004), obesity (Blair & Nichaman, 2002), and osteoporosis and degenerative arthritis (Minor, Hewett, Webel, Anderson & Kay, 1989). Exercise can also improve physical functioning through increased cardiovascular fitness, bone density, and muscle mass (DiPietro, 2001). Other benefits include reduced falls, reduced general health care costs, increased strength, and increased self-confidence and social skills (Cox, 2007). Physical activity also causes changes at the biological level, many of which are hypothesized to influence brain activity. It alters endocrine activity, lowers inflammatory cytokines, and prevents certain alterations of gene expression, which are likely to influence cognitive functioning and

depressive symptoms (Kohut et al., 2006; Bronikowski et al., 2002). Finally, exercise

has been shown to improve overall quality of life for older individuals (Rejeski &

Mihalko, 2001).

Physical Activity and Cognitive Functioning

In addition to these benefits, physical activity has been identified as a modifiable

lifestyle factor that is beneficial for maintaining cognitive functioning over time (Yaffe et

al., 2009; Buchman et al., 2012). The association between physical activity and cognition

in older individuals has been explored with different populations around the world using

both epidemiological and intervention methods. Intervention studies provide information

about the effects of time-limited exercise interventions on cognitive functioning, while

epidemiological studies provide information about the correlation between living a

physically active lifestyle and later cognitive functioning. Cognitive functioning or

decline can be assessed in different ways depending on the research hypothesis being

tested. Some studies focus on the effect of physical activity on global cognitive

functioning, generally assessed using the Mini-Mental State Examination (MMSE) or

equivalent measure, while others focus on specific domains of cognitive functioning such

as memory, attention, or processing speed. While the MMSE is useful as a screening tool

for dementia, it actually lacks tasks targeting a wider range of cognitive domains

necessary to make claims about overall cognition (Stewart, O'Riley, Edelstein & Gould,

2012).

Intervention studies.

Intervention studies with healthy older adults have provided a large body of

literature supporting the beneficial effect of exercise on cognitive functioning. This

effect has been shown in studies using aerobic activities as well as resistance and mind/body activities. Moderate intensity aerobic activity interventions have been shown to improve measures of reaction time (Kamijo et al., 2009) as well as scores on the MMSE (Muscari et al., 2010). In contrast, a study by Ijuin et al. (2013) did not find any improvement in a variety of cognitive domains after a 20-week walking intervention, with the exception of improved scores on a measure of processing speed. However, even a single bout of moderate intensity exercise has been shown to improve logical memory and MMSE scores (Molloy, Beerschoten, Borrie, Crilly, & Cape, 1988). One session of aerobic activity has also been shown to improve verbal fluency for COPD patients (Emery, Honn, Frid, Lebowitz, & Diaz, 2001) and tests of working memory in multiple age groups, including older individuals (Hogan, Mata, & Carstensen, 2013). In addition to studies demonstrating the beneficial effects of aerobic exercise, studies have also shown that resistance exercise improves both general cognitive functioning, as well as executive functioning in particular, for older adults (Chang, Tsai, Huang, Wang & Chu, 2014). Commonly, exercise intervention studies for older adults are conducted with medically healthy individuals; however, physically frail adults also showed improved cognition, including executive functioning, memory, and processing speed, after 12 weeks of endurance exercise (Langlois et al., 2013). Since many older adults are physically frail or have chronic medical conditions this study provided valuable information about the ability to generalize the results of these intervention studies to physically frail older adults.

These studies demonstrate that structured exercise is beneficial for older adults who do not have significant problems with cognitive functioning, but studies have also

shown that exercise is beneficial for those who are at high risk for the development of dementia or have already begun to experience cognitive problems. A study by Lautenschlager and colleagues (2008) found that a physical activity intervention prevented further cognitive decline in individuals with pre-existing memory problems. Individuals with a high genetic risk of developing Alzheimer's disease also experienced improved executive functioning skills after an exercise intervention (Baker et al., 2010). Intervention studies with individuals who have been diagnosed with dementia also show cognitive benefits from physical activity, including aerobic (Venturelli, Scarsini & Shena, 2011), non-aerobic (Yaguez, Shaw, Morris, & Matthews, 2011), and combined (Rolland et al., 2007) activities.

The positive effect of physical activity on cognitive functioning is not restricted to the results of neuropsychological tests. Similar results have also been shown through brain imaging studies. A study by Colcombe et al. (2006) found that sedentary older adults who participated in an exercise intervention for six months showed an increase in gray and white-matter brain regions compared to control participants on an MRI scan. Brain volume changes of this type have been associated with improved task-switching cognitive skills. Another 16-week multimodal physical exercise program showed reduction in low-grade inflammation and improvement in Brain-Derived Neurotrophic Factor (BDNF), as well as associated cognitive function improvement, for older adults with mild cognitive impairment (Crispim et al., 2015).

Epidemiological studies.

In addition to the encouraging results of intervention studies, epidemiological studies evaluating the effect of physical activity as part of a lifestyle over a long period of

time have produced similar results. A study by van Gelder et al. (2004) found that individuals who decreased their physical activity as they aged also experienced a decline in cognitive functioning while individuals who maintained their levels of physical activity over time were more successful at maintaining cognitive functioning. Participation in moderate to vigorous activity over time has been identified as one of the primary predictors of cognitive maintenance along with younger age, white race, high school education level or greater, ninth grade literacy level or greater, and not smoking (Yaffe et al., 2009). Studies have particularly found a positive correlation between higher levels of physical activity and faster processing speed in older adults (Hillman et al., 2006; Marmeleira, Ferreira, Melo & Godinho, 2012; Chang et al., 2010).

However, there is not yet a consensus on whether physical activity needs to fall in the "moderate to vigorous physical activity" range in order to provide a cognitive benefit. A study by Weuve et al. (2004) demonstrated that participating in a walking program over approximately ten years was associated with improved cognitive scores. Even leisure related physical activity and work related physical activity have been associated with higher levels of cognitive functioning in older individuals, demonstrating that leading a generally active lifestyle is similarly beneficial to structured exercise programs (Ku, Fox, Chen & Chou, 2012).

Similar to the results of intervention studies with cognitively impaired or at-risk individuals, studies assessing the positive impacts of living a physically active lifestyle have shown a preventative effect on the development of serious cognitive problems. Buchman et al. (2012) reported that individuals with higher amounts of total physical activity experienced a decreased risk for developing Alzheimer's disease. Another study

found cognitive benefits for all older adults with higher levels of physical activity, but they found the benefits were particularly high for individuals with a high genetic risk for Alzheimer's disease (Pizzie et al., 2014). A study of individuals with early signs of Alzheimer's disease found those who engaged in walking activities over the course of a year maintained their levels of cognitive functioning, while sedentary older adults showed a decline in scores on the MMSE. Additionally, individuals who walked more than 2 hours per week actually showed improvement in their MMSE scores (Winchester et al., 2013). Although it is not possible to determine causal influence from epidemiological studies, the results of these studies consistently show a strong relationship between living a physically active lifestyle and better cognitive functioning over time.

Reviews on physical activity and cognitive functioning.

The large volume and diversity of studies addressing the relationship between physical activity and cognitive functioning have led to several reviews of the topic. A review by Colcombe and Kramer (2003) evaluated the effect of aerobic exercise interventions on a variety of cognitive skills and found a combined effect size of 0.48 for exercise on cognitive functioning, with the largest benefits occurring for executive control tasks. A review by Busse, Gil, Santarem, and Filho (2009) found support for the effect of aerobic and resistance exercises on improved cognitive functioning for healthy and cognitively impaired participants. The use of resistance training for improving cognitive functioning was also supported by the results of a review by Chang, Pan, Chen, Tsai, and Huang (2012). Alternative exercises, including Tai Chi, have also been found to provide beneficial cognitive effects over time (Chang, Nien, Tsai, & Etnier, 2010).

However, not all reviews have shown overwhelming support for the positive effect of physical activity on cognition in older individuals. A review of 30 studies by Snowden et al. (2011) concluded that there was insufficient evidence to support the effect of most types of exercise on cognitive functioning. However, this review divided the included studies into many different groups based on the type of exercise used and the specific cognitive function evaluated. This led to an extremely small number of studies in any given group, which resulted in the authors' decision to deem the results inconclusive. A review by Gates, Singh, Sachdev, and Valenzuela (2013) that dealt exclusively with individuals with mild cognitive impairment found only limited support for improved reaction times in cognitive processing after physical activity interventions. Overall, most intervention studies assessing physical activity and cognitive functioning are positive at best and limited at worst.

Although it can be difficult to synthesize the literature on physical activity and cognition due to the wide range of possible cognitive abilities and lack of consistency in tasks used to measure those abilities, both intervention studies and large-scale epidemiological studies show support for the benefits of a physical activity for the maintenance of cognitive functioning and prevention of severe decline due to dementia processes. Sharing the information that lifestyle factors can have a large influence on the trajectory of cognitive functioning over the lifespan, and even the expression of genetic predispositions to cognitive impairment, could provide valuable hope and motivation for individuals who are concerned about cognitive decline during aging.

Physical Activity and Depressive Symptoms

Although evidence suggests that physical activity and cognitive function may be related, the mechanism by which they are related has not been clearly established. One potential mechanism is through decreasing depressive symptoms. Depression is one of the most common mental disorders in older adults, and depressive symptoms can significantly decrease a person's quality of life even if they do not meet the threshold for a diagnosis of major depressive disorder (Blazer, 2003). Studies in the United States estimate that 4% of older adults are affected by major depression, while 10% have depressive symptoms that do not meet criteria for a diagnosis (Blazer & Williams, 1980; Hybels, Blazer & Pieper, 2001). While older adults are less likely than their younger counterparts to be diagnosed with major depression, they are more likely to be diagnosed with dysthymia, minor depression, and other less severe depressive disorders (Blazer, 2003).

Antidepressant medications and psychotherapy are the most commonly used methods to treat depression; however, the potential barriers and drawbacks of those treatments for older individuals have led exercise to emerge as a possible alternative treatment for depressive symptoms. Although studies on exercise as a treatment for depression have been overwhelmingly positive in younger adults, studies involving older adult populations are less common, and the results of such studies have been mixed (Sjösten & Kivelä, 2006). This may be due in part to the different types of exercise used with older adults. Exercise interventions for older adult populations must include activities that are appropriate for their fitness level and physical ability. Therefore, these exercise interventions vary widely across studies and include activities such as resistance training, walking, yoga, and tai chi (Blake, Mo, Malik & Thomas, 2009). Previous

research with younger adults, which suggested that aerobic exercise was necessary in order to derive a mood benefit, often focused on activities like running or biking that are unsuitable for many older adults (Singh et al., 2005). In contrast, studies with older adults have primarily utilized walking as the method for aerobic exercise. Anaerobic exercise, often referred to as resistance training or strength training, has also been found to be effective for decreasing depressive symptoms in younger and older adults (Doyne et al., 1987; Singh, Clements & Fiatarone, 1997).

Reviews on physical activity and depressive symptoms.

There have been several systematic reviews and meta-analyses of the effect of exercise on depressive symptoms in older adults in recent years. A review by Arent, Landers, and Etnier (2000) evaluated 32 studies involving the impact of exercise on mood and found a mean effect size of 0.38. A review by Netz, Wu, Becker, and Tenebaum (2005) evaluated 36 studies involving physical activity and psychological well-being. They found a weighted mean effect size for treatment of 0.24 compared to 0.09 for the control groups. Both of these articles included studies using a variety of methods including non-randomized trials and correlational studies. A systematic review by Sjösten and Kivelä (2006) reviewed 13 randomized controlled trials (RCTs) of exercise for depressed older adults and reported a beneficial effect for exercise. A systematic review by Blake et al. (2009) detailed 11 RCTs of exercise for the treatment of older individuals with diagnosed depression and reported mixed results for the included studies. However, neither of these studies included statistical analyses to assess the overall effect size of the intervention.

Three recent reviews have addressed the question of the effect of exercise on depressive symptoms in older adults from a meta-analytic perspective. A dissertation by Cox (2007) reviewed 9 RCTs for the effect of exercise on depressive symptoms in adults over age 50. He found an overall mean change effect size of -0.46, indicating a greater decrease in depressive symptoms with exercise interventions compared to control groups. A meta-analysis by Bridle, Spanjers, Patel, Atherton, and Lamb (2012) investigated the effect of exercise interventions for older individuals who had been diagnosed with depression. Seven articles were meta-analyzed, resulting in a small but significant effect for the exercise interventions decreasing depressive symptoms (SMD = -0.34). This meta-analysis included only published RCTs of exercise interventions for depression in older adults. Finally, a recent meta-analysis of RCTs of exercise interventions compared to non-exercise control conditions for individuals with a range of depressive symptoms found a moderate effect size for exercise interventions decreasing depressive symptoms (SMD = -0.57). This effect was not significantly different for different ages of participants, types of control groups, or types of exercise interventions (Rhyner & Watts, unpublished manuscript). While many of these studies compared exercise interventions to some type of social contact control group, the social component of physical activity has also been shown to contribute to the effect of physical activity on mood (Teychenne, Ball, & Salmon, 2008; Wolinsky, Stump, & Clark, 1995). Overall, exercise has been shown to be a promising tool for decreasing depressive symptoms. In addition to the benefits of improved mood, decreasing depressive symptoms can also affect cognitive functioning.

Depressive Symptoms and Cognitive Functioning

Individuals who experience a decline, or perceived decline, in cognitive functioning are often at greater risk for depressive symptoms (Perrino, Mason, Brown, Spokane & Szapocznik, 2008). However, the reverse is also true; the prevention and treatment of depressive symptoms in older adults is crucial for helping them maintain cognitive functioning as they age. In fact, a study by Brommelhoff et al. (2009) hypothesized that depression may be a prodrome rather than a risk factor for dementia due to the fact that depression earlier in life was not associated with increased dementia risk while individuals who had experienced depression within the past 10 years were almost 4 times as likely to have dementia than those without an episode of depression. Similar to studies evaluating the direct effect of physical activity on cognitive functioning, studies evaluating the effect of depressive symptoms on cognitive functioning often use the MMSE to assess cognitive functioning. Multiple studies related lower MMSE scores with increased depressive symptoms (Baune et al., 2006; van den Kommer et al., 2013; Shimada, Park, Makizako, Doi, Lee, & Suzuki, 2014). One study with a large representative sample found depressive symptoms were associated with poorer general cognitive status on the MMSE and a steeper decline in these scores over time (Dotson, Resnick & Zonderman, 2008). Individuals with depression also show lower scores on the MMSE after two years and exhibit a greater reduction in hippocampal volume, which has been related to cognitive decline, compared to non-depressed individuals (Steffens, McQuoid, Payne & Potter, 2011).

These studies show depressive symptoms can affect general cognitive status on the MMSE. However, the results of studies evaluating specific cognitive abilities differ in their conclusions of exactly which cognitive functions are affected. In many studies

depressed mood has been related to poor executive functioning skills (Dotson et al., 2008; Reppermund et al., 2011; McBride & Abeles, 2000; Avila et al., 2009; Shimada et al., 2014). Depression has also consistently been shown to negatively impact processing speed (Baune et al., 2006; Avila et al., 2009; van den Kommer et al., 2013; Shimada et al., 2014). Particularly relevant to the current study, this effect has also been found in a population of older non-demented African Americans (Hamilton et al., 2014). Additionally, depression has been associated with lower scores on measures of memory, attention, and language (Avila et al., 2009; Shimada et al., 2014; Dotson et al., 2008; Reppermund et al., 2011). However, some studies have found that there is no connection between depression and memory (Baune et al., 2006; Comijs, Jonker, Beekman, & Deeg, 2001), attention (Baune et al., 2006), or visuo-spatial skills (Reppermund et al., 2001).

Epidemiological studies.

A series of longitudinal studies have also provided evidence of the directional nature of the relationship between depressive symptoms and cognitive functioning over time. In individuals with no pre-existing symptoms of dementia, greater baseline symptoms of depression predict poorer cognitive performance and cognitive decline after four years (Yaffe et al., 1999; Paterniti, Verdier-Taillefer, Dufouil & Alperovitch, 2002). The connection between baseline depressive symptoms and the development of mild cognitive impairment has been shown even after controlling for other sociodemographic factors, vascular risk factors, and antidepressant use (Ravaglia et al., 2008). Kohler et al. (2010) observed cognitively healthy individuals over time and found that those who had persistent depressive symptoms at baseline showed significant declines in processing speed and global functioning but not attention. There was also a dose effect noted, the

greater the number of depressive symptoms the greater the amount of cognitive decline. Another study also found a similar dose response for depressive symptoms on recall test scores (Chodosh, Miller-Martinez, Aneshensel, Wight & Karlamangla, 2010). Overall, the results of these studies suggest that the presence of depressive symptoms has a negative effect on cognitive functioning over time, although there are still inconsistencies in exactly which cognitive abilities are predominantly affected.

In addition to the direct effect of depressive symptoms on cognitive functioning there is also an interaction between these variables, which can contribute to the development of severe cognitive problems. Milwain and Nagy (2004) emphasized the potential harm that could be caused through the interaction of depressive symptoms and cognitive impairment. Their study found no relationship between depressive symptoms and cognitive decline in the beginning and late stages of Alzheimer's disease. However, the study showed a significant decline in functioning during intermediate stages of the disease caused by depressive symptoms (Milwain & Nagy, 2004). Similarly, depressive symptoms have a stronger effect on increasing cognitive impairment for individuals with the APOE-4 genotype, a genotype that has been identified as a risk factor for development of dementia (Corsentino, Sawyer, Sachs-Ericsson & Blazer, 2009). Unfortunately, the effect of depression on cognition may continue even without the current presence of depressive symptoms. A study by Yeh et al. (2011) compared individuals with no history of depression to individuals with remitted depressive symptoms. Individuals who had previously experienced depression showed greater declines in processing speed and memory compared to those who had never been depressed. While the interaction between depressive symptoms and cognitive

functioning is undeniably complex, these studies show that depressive symptoms can be a contributing factor for later cognitive decline.

Physical Activity and Sleep Problems

Another potential mechanism through which physical activity could affect cognitive functioning is through decreasing symptoms of insomnia. Like symptoms of depression, symptoms of insomnia are common in older adults. Older adults are more likely than younger adults to have problems initiating or maintaining sleep, with over 50% reporting problems (Wennberg, Canham, Smith & Spira, 2013). Contrary to popular belief, older adults do not require less sleep than younger adults, and poor sleep has harmful effects in several areas including functional impairments, increased risk of falls, and cognitive impairments (Bruce & Aloia, 2006). While the literature on the effect of physical activity on sleep problems in older individuals is not as extensive as the literature related to depressive symptoms, several epidemiological and intervention studies have demonstrated the positive effect of physical activity on decreasing sleep problems.

Epidemiological Studies.

Similar to the diversity of measurements used to assess cognition, sleep can also be measured in a variety of ways. Common assessments of sleep include global sleep quality, time spent asleep, sleep efficiency, sleep onset latency, nighttime awakenings, and daytime fatigue. In a large epidemiological study conducted in Japan, habitual physical activity over time was associated with lower rates of insomnia (Inoue et al., 2013). A ten-year follow-up study of Taiwanese older adults found that individuals who maintained high levels of leisure physical activity were significantly less likely to

experience symptoms of insomnia compared to individuals who were low or variable in their physical activity (Chen, Fox, Sun, Lo, Ku, 2014). A similar effect of more physical activity associated with fewer symptoms of late-life insomnia was found over a 9-year period after accounting for age and sex of participants (Morgan, 2003). Among African American women, higher levels of depressive symptoms and lower levels of exercise predicted greater symptoms of insomnia (Bazargan, 1996). This is particularly relevant to the current study since African Americans are typically underrepresented in research samples and it is important to be able to provide greater generalizations of results. Therefore, although insomnia can be considered one symptom of depression, physical activity appears to have some level of independent effect on sleep problems apart from its effect on depressive symptoms.

Intervention Studies.

Several intervention studies involving physical exercise have also targeted sleep quality improvement and decreased daytime fatigue as a goal. These interventions have been shown to decrease sleep onset latency, nighttime awakenings, and daytime dysfunction and increase sleep duration, efficiency, and global sleep improvement when compared to an educational sleep hygiene program (Reid et al., 2010; Burman, Hekler, Bliwise & King, 2011a; King et al., 2008). Physical activity has also been shown to alter objective assessments of sleep architecture, including the pattern of REM and NREM sleep stages. Participating in physical activity has been shown to improve slow wave sleep and performance on a memory task compared to a control group (Naylor et al., 2000). Physical activity has also been shown to affect stage 1 sleep and number of

nighttime awakenings, but this connection was mediated by change in depressive symptoms (Burman, Hekler, Bliwise & King, 2011b).

Exercise interventions for sleep problems have not been limited to aerobic activity. A variety of mind/body exercises have also become popular for promoting healthy sleep. Qigong, a traditional Chinese physical exercise, involves deep breathing, meditation, and slow structured movements (Chen & Turner, 2004). Tai Chi and Baduanjin are types of Qigong practice that focus on slow stylized movements and holding static postures respectively (Yan & Downing, 1998). These types of exercises may be more appropriate for the functional capabilities of older adults and may be considered more culturally appropriate in particular groups of the aging population around the world (Tsang, Mok, Au Yeung, & Chan, 2003). Yoga is also frequently used with older adult since it allows for different adaptations, including the use of belts and chairs, to make it appropriate for older adults' abilities (DiBenedetto et al., 2005). The effect of Baduanjin exercise, one of the most common forms of Qigong, has been investigated in a sample of older adult Taiwanese individuals and was shown to improve almost all measured areas of sleep functioning (Chen, Liu, Huang & Chiou, 2012). A study by Chen et al. (2009) reported beneficial changes in sleep quality for seniors participating in a silver yoga class intervention

Sleep and Cognitive Functioning

Investigating the effect of physical activity on sleep is valuable because poor sleep can have a negative effect on cognitive functioning over time. The relationship between sleep and cognitive functioning can at first appear contradictory because of the different methods for measuring sleep. For example, in one study objective measures of

good sleep were associated with better cognitive scores for healthy individuals and individuals with insomnia who were not taking sleep medication (Bastien et al., 2003). However, the subjective impression of having slept well was associated with better cognitive scores for healthy individuals and individuals with insomnia who took benzodiazepine medications to help them sleep. Another study reported that deviating from one's average sleep time has a more prominent effect on impairing cognitive performance than a summary score of one's sleep functioning (Gamaldo, Allaire & Whitfield, 2010).

Despite the variations in methodology among studies, a relatively consistent pattern emerges implicating poor sleep in lower cognitive functioning for older individuals. Individuals with chronic insomnia show lower functioning than individuals without sleep problems on measures of memory, attention, time estimation, executive functioning, and integration of two dimensions (Haimov, Hanuka & Horowitz, 2008). Poor sleep efficiency and shorter sleep time have also been associated with lower cognitive scores (Miyata et al., 2013), and longer sleep onset latency has been linked to lower scores on measures of verbal knowledge, long-term memory, fund of information, and visuospatial reasoning (Schmutte et al., 2007) for individuals without a diagnosis of insomnia. Nebes, Buysse, Halligan, Houch & Monk (2009) found that poor sleep quality in medically healthy individuals was associated with lower scores on tests of working memory, attention, set shifting, and abstract problem solving, but not processing speed, inhibitory function, or episodic memory. This association was not accounted for by symptoms of depression. Other studies have also found that poor sleep quality is associated with greater cognitive impairment even after controlling for levels of

depression and that the connection between insomnia symptoms and cognitive functioning is not accounted for by depressive symptoms (Kociuba et al., 2010; Naismith, Norrie, Lewis, Rogers, Scott, & Hickie, 2009). However, Saint Martin and colleagues (2012) did not find any effect of sleep on cognition in healthy older individuals with the exception of slower processing speed.

Both cross-sectional and longitudinal studies conducted on large samples have shown that having less sleep time at night is associated with greater cognitive decline in older adults (Ohayon & Vecchierini, 2005; Keage et al., 2012). Objective sleep monitoring has produced similar results to the studies based on subjective reports. Lim, Kowgier, Yu, Buchman, and Bennett (2013) conducted a prospective 6-year study evaluating sleep fragmentation using actigraph technology. They found that increased sleep fragmentation over time leads to an increased risk of developing Alzheimer's disease after six years. A study by Osorio et al. (2011) also found that the presence of insomnia was significantly associated with development of dementia over a 7-year period.   This association may be due to the removal of neurotoxic proteins during sleep that are known to contribute to the pathology of Alzheimer's disease, such as β-amyloids (Xie et al., 2013; Mendelsohn & Larrick, 2013).

Demographic variables have been shown to moderate the complex relationship between sleep and cognition. While women have a higher incidence of insomnia, men show greater cognitive impairment from symptoms of insomnia than women (Cricco, Simonsick & Foley, 2001; Potvin et al., 2012). Additionally, Zimmerman, Bigal, Katz, Brickman, and Lipton (2012) conducted a study to evaluate the impact of education level on cognitive decline. The study showed that individuals with low levels of education

who had problems with sleep onset or maintenance showed impaired performance on a test of category fluency, while individuals with higher levels of education who also had similar sleep problems showed no evidence of cognitive decline. Therefore, gender and education may have an important role in explaining the variations seen in study results about the association between sleep and cognition.

Present Study

In the existing literature there is strong evidence that physical activity has a beneficial effect on cognitive maintenance in older individuals. Physical activity has been shown through brief intervention studies as well as long-term epidemiological studies to improve or maintain cognitive functioning across the lifespan. However, a key unresolved question is the mechanism by which physical activity affects cognitive performance . A recent study by Wilckens (2014) investigated the possible mediational role of sleep. They found that sleep did mediate the relationship between physical activity and cognitive performance in several different domains including task switching, inhibition, working memory, and memory retrieval. Vance and colleagues (2005) used structural equation modeling (SEM) to investigate the mediating effect of depression and social stimulation on the relationship between physical activity and cognition in an attempt to answer this question. They found partial support for their hypotheses; however, they were severely limited by a small sample size and could not make a strong case for the directionality or causality of their proposed models because the data were not longitudinal. Although the relationships between the primary variables included in this study have been investigated separately, using a large longitudinal sample, like the Health

ABC study, allowed for more accurate testing of a complex model integrating

relationships among multiple variables.

In the present study, I used longitudinal structural equation modeling to

simultaneously test two hypothesized mediational mechanisms of the relationship

between physical activity and cognitive functioning:

1. Higher levels of physical activity lead to lower levels of depressive symptoms, which in turn lead to better cognitive scores.

2. Higher levels of physical activity lead to fewer symptoms of insomnia, which in turn lead to better cognitive scores.

For a visual representation of the hypothesized models see Figure 2. Using a large

representative sample, SEM provided several advantages for analysis over traditional

statistical methods, including correcting for measurement error and yielding more

accurate estimates of unbiased population parameters. The longitudinal nature of the data

allowed stronger causal inferences to be drawn, as the data came from three different

time points that were each separated by 2 years. Identification of potential mediators of

the relationship between physical activity and cognitive functioning will help determine

if the mechanism of action between these variables is related to common mental health

concerns in older adults.

Method

Participants

Participants were part of the ongoing Health, Aging and Body Composition

(Health ABC) study, a prospective investigation of the interrelationships between health

conditions, body composition, social and behavioral factors, and change in physical

function. The Health ABC study enrolled a random sample of 3,075 community-dwelling and Medicare-eligible participants living in Memphis, Tennessee or Pittsburgh, Pennsylvania. Participants were aged 70-79 years at the time of recruitment in 1997, with a mean age of 73.6 years. The population was 42% black and 52% female. Exclusion criteria for the study included reported difficulty with activities of daily living, walking a quarter of a mile, or climbing 10 steps without resting. Individuals were also excluded if they had a life-threatening cancer diagnosis, were currently enrolled in a lifestyle intervention trial, or had plans to move out of the study area within three years.

In Year 3 of the Health ABC study, the Cognitive Vitality Substudy was initiated. Participants included in the substudy represent approximately the top 20% on an endurance walk test in Year 2 from each of eight groups defined by sex, race, and study site and an equal number drawn at random from the remaining members of each group. Exclusion criteria included reported difficulty seeing large print or holding a pen. Participants included in this substudy received additional cognitive testing. All eligible participants signed a written informed consent, approved by the institutional review boards of the clinical sites (University of Pittsburgh and University of Tennessee, Memphis) and the coordinating center (University of California, San Francisco). A detailed description of the Health ABC sample can be found in Rooks et al. (2002).

The total Health ABC population included 3,075 participants. For the present analyses, we excluded individuals with any indications of dementia, including use of dementia related medications Aricept and Exelon ($N = 67$) or mental status scores two standard deviations below the mean stratified by race ($N = 125$). We also excluded respondents who died over the 7-year follow up period used for the current study ($N =$

444) or whose vital status at the 7-year follow up was unknown ($N = 7$). Finally, we removed all participants who did not have scores for the primary cognitive outcome measures because they were not included in the cognitive vitality substudy ($N = 1448$). Thus, the primary analytic cohort for this study contained 984 participants. A flow-chart depicting the criteria for participant exclusion is presented in Figure 1. The demographic characteristics for this sample are presented in Table 1.

Measures

Center for epidemiological studies depression scale-10.

The Center for Epidemiological Studies Depression Scale-10 (CESD-10; Andresen, Malmgren, Carter & Patrick, 1994) is a 10-item self-report screening measure for assessing depressive symptoms during the past week. This measure comprises a short form of the original Center for Epidemiological Studies—Depression Scale (CES-D; Radloff, 1977), a 20-item self-report measure with total scores ranging from 0-60. The CESD-10 has total scores ranging from 0-30 with higher scores indicating greater depressive symptoms. Items are scored on a 4-point Likert scale ranging from 0 (*rarely or none of the time*) to 3 (*all of the time*). The original CES-D has been shown to be reliable and valid when used with older adult populations (Radloff & Teri, 1986; Hertzog, Van Alstine, Usala, Hultsch & Dixon, 1990; Beekman, Kriesgsman, Deeg, & van Tilburg, 1995). The CESD-10 has shown comparable retest correlations ($r = 0.71$) and good predictive accuracy in older adults when compared to the CES-D. The CESD-10 has also shown an expected strong negative correlation with positive affect ($r = -0.63$) and positive correlation with self-assessed stress ($r = 0.43$). (Andresen et al., 1994) Using a cut-off score of 16 was shown to have adequate sensitivity (.67) and good

specificity (.81) for identifying individuals experiencing a current major depressive episode (Weiss, Aderka, Lee, Beard, & Bjorgvinsson, 2014).

Insomnia symptom assessment.

Insomnia symptoms were evaluated with standardized questionnaires created by Health ABC that included detailed questions about sleep and fatigue. Participants were asked how often they: "have trouble falling asleep," "wake up during the night and have difficulty getting back to sleep," "wake up too early in the morning and [are] unable to get back to sleep," "feel excessively (overly) sleepy during the day," and "take sleeping pills or other medication to help you sleep." Responses were categorized as: never (*0 times/month*), rarely (*1 time/month*), sometimes (*2-4 times/month*), often (*5-15 times/month*), or almost always (*16-30/month*).

Cognitive assessment.

The following tests were administered to participants in the Cognitive Vitality substudy and were used to evaluate cognitive functioning in the current study: The Boxes and Digit Copying (BDC) tests are timed tests of psychomotor speed (Salthouse, 1996). The participant is asked to complete as many boxes and copy as many digits as possible within 30 seconds for each test. Psychomotor speed is scored as the sum of total boxes and digits completed ($\rho = 0.77$). The Pattern and Letter Comparison (PLC) tests are timed tests of attention and perceptual speed (Salthouse, 1996). The participant is asked to determine whether pairs of patterns and letters are the same or different within 30 seconds for each test. Perceptual speed is scored as the sum of correct pattern and letter comparisons ($\rho = 0.64$). The Digit-Digit and Digit-Symbol tests are computerized tests of perceptual speed based on Salthouse's (1996) Letter-Letter and Letter-Symbol tests. In

the Digit-Digit test the participant is asked to determine if two numbers that appear in a box are the same or different as quickly and accurately as possible. In the Digit-Symbol test the participant is asked to determine if a box with a number in the top part and symbol in the bottom part matches a key of number-symbol pairs displayed at the top of the screen. Results were scored as the percent accuracy during the fastest trial for each test.

Physical activity assessment.

Physical activity measures were based on self-reports of physical activity obtained from a standardized face-to-face interview and included minutes spent walking during the past week. Participants were asked, "In the past 7 days, did you go walking?" If participants responded "yes" to this question the number of times they went walking during the past week and amount of time spent walking each time was obtained. This information was used to create a total value of minutes spent walking per week. This value included minutes spent walking at a brisk pace as well as minutes spent walking at a moderate or leisurely pace.

Demographic characteristics.

Demographic characteristics were collected during the standardized interview conducted at baseline. Participant age, gender, ethnicity, and level of education were obtained and included as covariates in the current analysis.

Data Analysis

Longitudinal structural equation modeling (SEM) was used to evaluate the study hypotheses. SEM is superior to null hypothesis testing for evaluating these hypotheses because it allows for complex models of the relationships between variables to be tested.

SEM allows for the longitudinal assessment of two potential mediators simultaneously and can provide support for stronger claims about the causal relationships between variables than variables assessed at the same time point. Importantly, SEM also corrects for measurement error. All variables were assessed during years 3, 5, and 7 of the Health ABC study, with the exception of the variables measuring insomnia symptoms, which were not collected during year 7.

All variables were screened using SPSS version 22.0 statistical software for missing values and normality of the data. We assessed normality by examining the skewness and kurtosis values and inspecting histograms for each variable. The variables used to assess symptoms of insomnia consisted of nominal values on a 5-point scale and the variables used to assess symptoms of depression consisted of nominal values on a 4-point scale. Previous research has demonstrated that nominal variables with more than four categories can be treated as continuous variables without introducing significant bias into parameter estimates (Beaujean, 2014). The variables used to assess physical activity and cognitive functioning were continuous variables. However, several variables were not normally distributed, primarily due to floor effects present in the data.

Due to the violations of normality on some variables and the use of a 4-point nominal variable a robust maximum likelihood estimator (MLR) was used for all analyses. MLR uses Huber-White robust standard errors and computes a test statistic that is asymptotically equivalent to the Yuan-Bentler test statistic (Beaujean, 2014). This process creates standard errors that are based on the expected information matrix and corrected using the Yuan-Bentler approach, and also allows for the imputation of missing data. The variable used to assess physical activity, minutes spent walking each week,

was found to have a scale for variance values that was extremely different from other variables used in the analyses. Therefore, this variable was transformed into hours spent walking each week by dividing the value of minutes by sixty in order to put this variable on a more comparable scale.

The *lavaan* package for *R* (version 0.5-17) was used to conduct all robust maximum likelihood estimations using raw data. Full information maximum likelihood (FIML) was used for imputation of missing data. The model fit was evaluated using multiple fit statistics, including the chi-square goodness-of-fit statistic, Comparative Fit Index (CFI), Tucker Lewis Index (TLI), Standardized Root Mean Square Residual (SRMR), and Root Mean Square Error of Approximation (RMSEA), as well as by assessing localized areas of strain through residuals and modification indices.

A non-significant chi-square value ($p < 0.05$) indicates acceptable fit between the estimated covariance matrix and the sample covariance matrix. However, the chi-square test has been found to be too strict for use in larger samples (Little, 2013) and therefore was not used as the primary tool for evaluating model fit in these analyses. The CFI and TLI are relative fit indices that compare the model to an alternative model, with values greater than 0.90 indicating acceptable fit (Hu & Bentler, 1995). The SRMR and RMSEA are measures of the discrepancy between predicted and observed model values; values closer to 0 indicate better fit, with values less than 0.08 indicating acceptable fit (Little, 2013). When evaluating alternative models, including invariance testing, the chi-square difference test is considered to be too strict; therefore, reasonableness tests were used for evaluation. These tests included if the RMSEA value for the new model fell within the confidence interval for the previous model and if the change in CFI and TLI

between models was less than or equal to 0.01. The *R* syntax for all models is included in supplemental materials.

Results

Descriptive Statistics

The skewness and kurtosis values indicated that several variables were not normally distributed; therefore, a robust maximum likelihood estimator (MLR) was used to decrease bias in parameter estimates. The mean scores for individual CESD-10 items, which had a possible range from 0-3, were generally low at all time points (0.17-1.01). The mean scores for individual insomnia symptom items, which had a possible range from 0-4, were slightly higher but similarly consistent across time points (1.21-1.46). The mean number of hours walked each week was also generally low and declined slightly at each time point (Year 3 $M = 1.81$, Year 5 $M = 1.65$, Year 7 $M = 1.35$).

The percentage of missing data for the included variables ranged from 2.0% (Year 3 hours walked per week) to 33.9% (Year 7 Digit-Symbol cognitive test). The majority of variables had less than 15% missing data. The exceptions to this was CESD-10 items during year 7, as only a subset of participants were administered these items during that time periods. If participants were missing data on all variables used in a particular analysis missing data was not imputed and they were not included in that analytic sample. This resulted in samples ranging from 933 participants for invariance testing to 984 participants for calculation of measurement and structural models.

Confirmatory Factor Analysis

As recommended by Little (2013), we first used confirmatory factor analysis (CFA) to test whether the measurement model of the latent variables included in our

analysis was valid. When evaluating a longitudinal model, it is necessary to correctly specify the null model to be used for comparison, as the default null model provided by statistical programs is usually incorrect as it is only appropriate for single-time point models (Little, 2013). Since the model contained indicators that were represented at multiple time points, the null model was specified to reflect the longitudinal null model expectation that the variances and means of the like indicators did not change across time. Otherwise the null model was essentially an independence model with no associations estimated among the indicators.

An initial longitudinal CFA model was created for three latent variables (depressive symptoms, insomnia symptoms, processing speed) at three time points (years 3, 5, and 7). For the depressive symptoms latent variable, ten indicators were used reflecting the ten items from the CESD-10 scale. To set the scale for the latent variable the marker variable method was used with the questionnaire item "I felt depressed" identified as the marker variable due to its high face validity and previous factor analytic results that this item accounted for 45% of the variance of the total scale (Andresen et al., 1994). A correlated residual was estimated between the indicators "I was happy" and "I felt hopeful about the future" at each time point due to their unique measurement properties as the only positively worded items, which are reverse-scored for calculation of the total scale.

For the insomnia symptoms latent variable, four indicators were used. The fifth item "taking sleeping pills or other medications to help you sleep" was not included, as prior investigation had determined that it did not correlate significantly with the other indicators and had a non-significant loading on the insomnia symptoms latent variable.

To set the scale for the latent variable the marker variable method was used with the questionnaire item "have trouble falling asleep" identified as the marker variable due to its high face validity. Symptoms of insomnia were not assessed during year 7 of the study, so the latent factor of insomnia symptoms was only included for years 3 and 5.

For the latent variable of cognition, six indicators were used from the available cognitive test scores. All of these tests evaluated cognitive processing speed. These indicators included scores on Boxes Copying and Digit Copying tests, Digit-Digit and Digit-Symbol tests with a correlated residual between these tests at each time point reflecting similar measurement methodology for these tests, and Pattern Comparison and Letter Comparison tests with a correlated residual between these tests at each time point reflecting similar measurement methodology for these tests. To set the scale for the latent variable the marker variable method was used with the Boxes Copying score identified as the marker variable. The factor structure for the latent cognitive variable in this dataset had previously been identified (Watts, Kritchevsky, & Yaffe, unpublished data) and was confirmed for this study. Other cognitive abilities were not explored because they generally only had one test to indicate the particular ability (i.e. executive functioning) and the same tests were not given at each time point.

We were unable to create a latent factor for physical activity due to inconsistencies in the particular physical activity variables reported at each time point and lack of relationship between eligible variables. Therefore, physical activity was included in the model using a manifest variable for hours walked during the week and was added to the model along with other manifest covariates after the latent confirmatory factor structure was established and invariance for the factors over time was assessed. For the

initial CFA, the manifest variables were restricted to load onto their corresponding latent variables and the latent variables were freely allowed to covary with all other latent variables. Unlike the null model, the variance and intercepts of like indicators across time were freely estimated.

The initial longitudinal CFA model was compared to the longitudinal null model to evaluate the overall model fit for the measurement structure of the latent variables. The overall goodness-of-fit indices showed that the fit of the model to the data was acceptable. The values for the SRMR (0.060) and RMSEA (0.031; $CI = 0.030\text{-}0.033$) indicated that the model had acceptable to close fit. The TLI (0.898) and CFI (0.912) indicated that the model had mediocre to acceptable fit. Therefore, the latent variables were adequately measured by their specified manifest variables. However, an examination of the residuals and modification indices revealed specific areas of localized strain related to the CESD-10 item "my sleep was restless". The modification indices suggested that there would be a significant improvement to the model fit if this item was allowed to freely load on the latent insomnia symptoms factor in addition to loading on the depressive symptoms factor. As this made conceptual as well as empirical sense, this item was allowed to have a dual loading on the depressive symptoms and insomnia symptoms latent factors.

The revised CFA model including the dual loading was shown to have good fit compared to the null model when using the goodness-of-fit indices. The values for the SRMR (0.053) and RMSEA (0.026; $CI = 0.024\text{-}0.028$) indicated that the model had acceptable to close fit. The TLI (0.928) and CFI (0.938) also indicated that the model had acceptable fit. These valuables represented a substantial improvement in model fit

compared to the initial CFA model. An examination of the residuals and modification indices revealed no theoretically relevant areas of localized strain. Therefore, this model of the latent factors was considered to be a good fit for the data. The fit statistics for the initial CFA model and final CFA model are presented in Table 2. The parameter estimates for the factor loadings of the final CFA model are presented in Table 3.

Factorial Invariance Testing

After establishing the factor structure for the latent variables, we tested the invariance of that structure across the time points included in the study. Configural invariance was evaluated to determine if the pattern of fixed and free parameters was the same across time. The configural model is identical to the final CFA model that was evaluated, please see previous section for fit statistics. As the assumption of configural invariance was deemed tenable, weak invariance was then tested to determine if the corresponding factor loadings were equal across time. When evaluating tests of invariance the chi-square difference test is considered to be too strict; therefore, reasonableness tests were used for evaluation. These tests included if the RMSEA value fell within the confidence interval for the configural model and if the change in CFI between models was less than or equal to 0.01 (Cheung & Rensvold, 2002). The RMSEA value for the weak invariance model (0.026; CI = 0.025-0.028) fell within the RMSEA confidence interval for the configural invariance model and the change in the CFI value (0.002) was less than 0.01 from the CFI value of the configural invariance model. Therefore, the assumption of weak invariance was tenable, and the factor loadings could be considered invariant across time with no significant decrease in model fit.

Strong invariance was then tested to determine whether the intercepts of the indicators were invariant across time. The RMSEA value for the strong invariance model (0.028; CI = 0.027-0.030) fell within the RMSEA confidence interval for the weak invariance model but the change in the CFI value (0.012) was greater than 0.01 from the CFI value of the weak invariance model. Therefore, the assumption of strong invariance was not tenable, and the intercepts of the indicators were not shown to be invariant across time. Partial strong invariance was evaluated after allowing the intercepts for the CESD-10 item "my sleep was restless" to vary freely at each time point. This made empirical as well as conceptual sense since this item was allowed to dual load on the depression and sleep factors for the first two time points but only loaded on depression for the last time point due to the lack of a sleep factor at year 7. The RMSEA value for the partial strong invariance model (0.027; CI = 0.025-0.029) fell within the RMSEA confidence interval for the weak invariance model and the change in the CFI value (0.003) was less than 0.01 from the CFI value of the weak invariance model. Therefore, the assumption of partial strong invariance was tenable after allowing the aforementioned intercepts to freely vary, and this model was retained for all additional analyses. The values for invariance testing are presented in Table 4.

Structural Equation Models

After establishing weak factorial invariance for the latent factors, it was necessary to respecify the longitudinal null model to include manifest variables that would ultimately be used in the structural model. This included the addition of the variable assessing hours walked each week which was allowed to freely covary with all other latent variables, and covariates for age, race, gender, and education that were regressed

on each of the constructs at each time point to provide full control of the covariate influences.

The final CFA measurement model was then compared to the longitudinal null model. The revised model was shown to have acceptable fit compared to the null model when using the goodness-of-fit indices. The values for the SRMR (0.054) and RMSEA (0.026; CI = 0.025-0.028) indicated that the model had acceptable to close fit. The values for the TLI (0.916) and CFI (0.926) also indicated that the model had acceptable fit. The fit statistics for the final measurement model and subsequent SEM models are presented in Table 5.

An SEM of the data was then constructed to evaluate the study hypotheses. The walking manifest variable and the three latent variables were allowed to freely covary within each time point. Auto-regressive paths were estimated so that each variable predicted later measurements of the same variable. Other regressive paths were estimated for the effect of year 3 walking on year 5 depressive symptoms and year 5 insomnia symptoms, with subsequent paths estimated for the effect of year 5 depressive symptoms and year 5 insomnia symptoms on year 7 cognitive functioning. Due to the hypothesized relationship between symptoms of depression and insomnia, cross-lagged paths between these variables were also estimated across all time points to account for their reciprocal relationship. The hypothesized structural model was shown to have acceptable fit compared to the null model when using the goodness-of-fit indices. The SRMR (0.056) and RMSEA (0.026; CI = 0.025-0.028) values indicated that the model had acceptable to close fit. The TLI (0.912) and CFI (0.922) values also indicated that the model had acceptable fit. When compared to the measurement model, the RMSEA

value for the hypothesized structural model (0.026) fell within the RMSEA confidence interval for the measurement model and the change in the CFI value (0.004) was less than 0.01 from the CFI value of the measurement model, indicating the hypothesized structural model continued to provide a good fit for the data. Although model fit was acceptable, there were no significant effects in the model beyond the autoregressive paths.

A pruned model that eliminated all non-significant paths from the hypothesized model was then evaluated and compared to the hypothesized model. The RMSEA value for the pruned structural model (0.026) fell within the RMSEA confidence interval for the hypothesized model and the change in the CFI value (0.000) was less than 0.01 from the CFI value of the hypothesized model, indicating the pruned structural model provided a similar fit for the data. As there were no other theoretically meaningful paths that were significant, this model was retained as the final structural model. The final model is depicted with unstandardized estimates in Figure 2 and standardized estimates in Figure 3.

Discussion

Measurement of Depression and Insomnia Symptoms

The model of physical activity, depressive symptoms, insomnia symptoms, and cognitive functioning evaluated in the present study was found to fit the data relatively well. However, none of the hypothesized relationships between variables, beyond the autoregressive paths, were significant. In the current study, the levels of reported depressive symptoms and sleep problems were very low. The floor effects present for these variables decreased the ability to identify possible mediation effects. The

relationship between depressive symptoms and sleep problems has been consistently identified in the literature (Pigeon et al., 2008; Sadler, McLaren & Jenkins, 2013), and the current study was no exception. The correlations between symptoms of depression and insomnia that were measured at the same time point during Year 3 and Year 5 were both significant (Year 3 $r = .385$, $p < .001$; Year 5 $r = .332$, $p < .001$). However, the reciprocal effect of these variables across time was not significant.

Since the expected effect was present within time points, it is likely that the time lag between measurements (2 years) was not ideal to capture the reciprocal effects of these variables on each other, or on other variables included in the model across time. One of the crucial elements of longitudinal research is identifying time lags between measurements that are ideal to capture the effect of the variables of interest and evaluate their hypothesized effects (Little, 2013). If the time lag is too short it may not give adequate time for the hypothesized causal effect to take effect, while if it is too long the relationship between variables may dissipate. It is likely that the 2-year time lag between each of the study periods was too long to evaluate the relationship between symptoms of depression and insomnia and between these variables and physical activity and cognitive functioning.

According to the *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*, symptoms of insomnia can vary widely in their duration and can be situational, persistent, or recurrent. Situational insomnia is often triggered by a particular event and resolves within a few days to a few weeks. However, some individuals experience symptoms of insomnia that are persistent over time or reoccur in an episodic fashion (APA, 2013). Studies by Buysse, Angst, Gamma, Ajdacic, Eich, and Rössler (2008) and Morin,

LeBlanc, Daley, Gregoire, and Merette (2009) reported chronicity rates of insomnia as 45% to 75% over 1 to 7 years. A study of older adults that specifically evaluated symptoms of insomnia over 2-year periods of time reported 38% of participants only experienced one episode of insomnia symptoms and 28% of participants experienced recurrent episodes of insomnia but did not have symptoms at all time points (Kim et al., 2013). In the current study some participants with significant symptoms of sleep problems at a particular time point in the study were possibly experiencing a brief episode of situational insomnia that did not persist or episodic symptoms that did not reoccur during the other time points in the study. Therefore, the episodic nature of sleep problems may have limited the ability to assess relationships with this variable over the 2-year time intervals included in the study.

Symptoms of depression can also vary widely in duration and time periods of remission. For individuals with symptoms of depression severe enough to warrant a diagnosis of major depressive disorder two in five individuals will begin to recover within three months of onset and four in five individuals will begin to recover within one year of onset (Coryell et al., 1994). A study by Solomon et al. (1997) identified 5.5 months as the mean time to recovery from a depressive episode and determined that this is generally stable across age and number of depressive episodes. Individuals with high levels of depressive symptoms in the current study may have received treatment to improve their depressive symptoms over the course of the study through the use of medications or other interventions, or they may have experienced spontaneous remission or reduction of symptoms after several months which decreased the ability to detect effects across time.

Measurement of Processing Speed

The current study found no significant relationships between cognitive functioning and any other variables across time points. Additionally, there were no significant relationships between cognitive functioning and the other latent variables within each time point. Measurement limitations of the cognitive functioning latent variable may have played a role in the lack of relationship of cognitive functioning to other variables in the model. Creation of the latent factor for cognitive functioning was restricted to a particular set of variables due to inconsistency in tests used across time points. The measures that were used consistently across the study periods were measures of processing speed as opposed to measures of memory, executive functioning, or a combination of cognitive abilities.

The majority of previous studies investigating the effect of physical activity on cognitive functioning used a general measure of cognitive functioning, such as a Mini-Mental Status Exam as their primary outcome variable (Kamijo et al., 2009; Muscari et al., 2010). Some studies that evaluated the effect of physical activity on processing speed did not find a significant relationship between variables (Pizzie, et al., 2014; Snowden et al., 2011). However, other studies have found connections between physical activity and improvements in processing speed (Langlois et al., 2012; Linde & Alfermann, 2014). The relationships between variables in the current study may have been limited due to the use of only processing speed measures. Similar to the findings of Yaffe et al. (2009) there was a significant relationship between younger age and higher cognitive functioning at year 3 and year 5 but not at year 7, possibly due to patterns of missing data from participant drop-out. In the current study the only significant predictor of future

cognitive functioning was level of cognitive functioning at the previous time points.

Since individuals with significantly impaired cognitive functioning were excluded from

this study this finding supports the importance of healthy cognitive functioning

throughout the lifespan as a primary predictor of higher cognitive functioning in late-life.

Measurement of Physical Activity

In this study it was not possible to create a latent variable to measure physical

activity. This was due to different physical activity measures being reported during

different years of the study and lack of relationship between the consistently reported

measures. For example, consistent with recommendations from previous studies (Watts,

Vidoni, Loskutova, Johnson & Burns, 2013) calories burned while engaged in household

chores and other unstructured physical activities were some of the measures included in

order to try to capture some of the common physical activities completed by older adults.

However, this variable was consistently negatively correlated with other measures of

physical activity, such as walking, likely due to the fact that individuals spending

significant time engaging in caregiving activities do not have time for other exercise

activities. Since it was not possible to create a latent factor for physical activity this

necessitated only using minutes walked per week to measure the impact of physical

activity in our model. Since this was not a latent factor we were not able to account for

the role of measurement error in this variable.

Rates of physical activity are typically low for older adults, and nearly all self-

report measures find a floor effect. A study by Clark (1999) found 75% of individuals

were at the floor for walking and only 13% reported engaging in other leisure activities.

The current study confirms the findings of previous studies that identified a substantial

floor effect in self-report measures of physical activity (Clark, 1999; Tudor-Locke &

Myers, 2001; Watts et al., 2013). In the current study, very few participants reported

engaging in any type of vigorous physical activity, and only approximately 50% reported

engaging in any walking behaviors during the previous week. For individuals who did

participate in walking activities, the number of hours walked in the previous week was

still quite low, ranging from 1.6 to 1.3 to 1.1 across the years used for the current study.

Although this is slightly higher than the average 48 minutes identified for this age group

in a previous study (Clark, 1999) it is still similar to the low amounts that have plagued

other studies in their attempts to accurately evaluate physical activity in more sedentary

populations.

Previous studies have demonstrated that self-reports of walking behaviors in older

adults are notoriously poor (Tudor-Locke & Myers, 2001; Kowalski, Rhodes, Naylor,

Tuokko, & MacDonald, 2012), and measurement of low-intensity activity tends to have

poor reliability and validity (Jacobs, Ainsworth, Hartman, & Leon, 1993; Shephard,

2003). Measuring intensity and distance walked from self-report is especially poor

(Durante & Ainsworth, 1996). Because of the challenges of measuring walking

behaviors through self-report, pedometer or accelerometer technologies are preferable for

measuring these behaviors (Tudor-Locke & Myers, 2001), although even these measures

are not suitable for measuring some types of physical activity such as swimming or

resistance exercise (Kowalski et al., 2012). Studies summarized by Kowalski et al.

(2012) showed high variability in the correlation between self-report and objective

measures of physical activity (-0.02 to 0.79). The challenges of ensuring accurate

assessment and recall of physical activity likely contributed to the lack of significant associations related to this variable.

Impact of Covariates

Several covariates were included in the current study to parcel out their effect from the variables of interest. The patterns of covariate results were consistent with previous research on the impact of these variables. In addition to the aforementioned effect of age on cognitive functioning, gender and race of participants also showed a significant impact on the variables of interest. The current data showed that men were significantly more likely to engage in physical activity at year 3 and 5 and women were significantly more likely to experience symptoms of depression at year 3 and 5. Finally, African American participants were less likely to engage in walking behaviors than whites at all time points. They were also less likely to experience symptoms of insomnia at year 3. In addition to the significant effect of the autoregressive paths that suggest that the best predictor of the included variables are individual's scores on those variables at earlier time points, this study also supports the continued importance of evaluating the impact of demographic variables such as age, gender, and race on all of the variables included in the model. Overall, these factors may have a greater impact on physical activity, depressive symptoms, sleep problems, and cognitive functioning.

Strengths and Limitations

The current study possessed several notable strengths. Using longitudinal data allowed for true mediation effects and direct effects across time to be evaluated. Many studies describe causal effects when they are actually evaluating variables that occur at the same time point (i.e. Vance et al., 2005). The current study also included a large

number of participants that allowed for adequate power to evaluate a complex model with multiple variables at multiple time points. The participants included in this study were also more diverse than typically found in studies of older adults allowing for greater generalization of the results to both white and African American populations. Finally, the use of SEM allowed for the evaluation of relationships between variables while accounting for measurement error.

There were also several limitations to the current study. Physical activity was only measured through minutes spent walking per week and did not include other types of activities or objective measures, such as accelerometers or pedometers, to corroborate self-reported physical activity. Since a latent factor could not be created, it was not possible to remove the error variance inherent in this solitary measure of physical activity. The self-report of physical activity suffered from similar floor effects and possible recall bias that have affected other studies attempting to measure physical activity in older adults. The measurement of cognitive functioning may have also had an effect on the results of this study, as it was limited to the use of variables assessing processing speed. Therefore, the conclusions of this study can only be applied to effects on processing speed and not on other cognitive abilities or general cognitive functioning.

This study eliminated participants who had very low initial cognitive functioning as well as those who died during the years evaluated for the present study. These individuals may have shown a different pattern of results for cognitive functioning that was not found with the healthier participants retained for analysis. Similarly, low levels of reported symptoms of depression and insomnia limited the conclusions that could be drawn about the impact of these variables. The time lag of 2 years used in the current

study did not appear to be an appropriate amount of time to model the effects of these variables on each other across time.

Future Directions

Future research should continue to strive toward improving the self-report measurement of physical activity particularly for older adult populations. It would also be beneficial to include objective measurements of physical activity in future studies, such as accelerometers in order to provide more accurate assessments of physical activity. Improving measurement of cognitive functioning would also be beneficial. Because studies evaluating cognitive functioning often employ a wide variety of measures it is difficult to determine a consensus on the results of these studies. Many studies often use a single measure of cognitive functioning, such as a Mini-Mental Status Exam, which does not provide as much information about the complexity of cognitive functioning. The development of a standard battery of cognitive tests that are brief, readily available, and sample a range of cognitive domains would be beneficial toward standardizing the assessment of cognitive functioning across studies. Future research evaluating the impact of depression and insomnia symptoms on cognitive functioning should evaluate multiple briefer time periods to determine how far into the future the impact of these symptoms goes and whether this is similar for different levels of severity of symptoms.

Conclusions

The model of physical activity, depressive symptoms, insomnia symptoms, and cognitive functioning evaluated in the present study was found to fit the data relatively well. However, none of the hypothesized relationships between variables, beyond the

autoregressive paths, were significant. The methods used to measure the variables included in the study and the span of time between measurement occasions likely played a role in the absence of relationships between the variables over time. In the current study, the strongest predictor of cognitive functioning was an individual's prior level of cognitive functioning. If an individual has a high level of initial cognitive functioning they are likely to retain a higher level of cognitive functioning over time compared to those with initial low levels of cognitive functioning. Therefore, it is important to facilitate cognitive functioning across the lifespan since it is likely to have a strong relationship with cognitive functioning later in life.

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Table 1

Demographic characteristics of analytic sample at baseline (N = 984)

Variable	N	%
Female	513	52.1%
African American	516	52.4%
Education ≤ High School	548	55.7%
Variable	*Mean*	*SD*
Age	73.25	2.8

Table 2

Fit indices of CFA for longitudinal latent factors

Model tested	x^2	df	SRMR	RMSEA (95% CI)	CFI	TLI
Null Model	16078.636	1612				
Initial Model	2673.245	1395	.060	.031 (.030, .033)	.912	.898
Final Model	2295.588	1393	.053	.026 (.024, .028)	.938	.928

Table 3

Parameters for CFA of longitudinal latent factors

Indicator	Estimate	S. E.	Z-value	P-value	St. Estimate
Y3Depression =~					
Y3DDEP	1.000				0.726
Y3DHOPE	0.739	0.122	6.065	< 0.000**	0.302
Y3DHAPPY	1.002	0.096	10.415	< 0.000**	0.524
Y3DBOTH	0.754	0.082	9.235	< 0.000**	0.544
Y3DMIND	0.701	0.091	7.669	< 0.000**	0.467
Y3DEFFRT	0.815	0.079	10.341	< 0.000**	0.474
Y3DFEAR	0.565	0.066	8.524	< 0.000**	0.523
Y3DLONE	0.817	0.076	10.781	< 0.000**	0.586
Y3DNOGO	0.617	0.082	7.528	< 0.000**	0.459
Y3DSLEEP	0.280	0.088	3.170	0.002*	0.145
Y3Insomnia =~					
Y3SINSOM1	1.000				0.660
Y3SINSOM2	1.479	0.083	15.500	< 0.000**	0.821
Y3SINSOM3	1.330	0.086	14.167	< 0.000**	0.765
Y3SITIRED	0.413	0.063	6.577	< 0.000**	0.282

Y3DSLEEP	0.600	0.049	12.223	< 0.000**	0.564
Y3Cognitive =~					
Y3BOXT	1.000				0.826
Y3DIGITCT	0.929	0.027	34.361	< 0.000**	0.902
Y3PCSCORE	0.237	0.011	22.054	< 0.000**	0.737
Y3LCSCORE	0.184	0.009	20.395	< 0.000**	0.650
Y3ACCDST	0.352	0.048	7.367	< 0.000**	0.407
Y3ACCDDT	0.266	0.048	5.512	< 0.000**	0.307
Y5Depression =~					
Y5DDEP	1.000				0.693
Y5DHOPE	0.842	0.129	6.532	< 0.000**	0.345
Y5DHAPPY	0.960	0.094	10.224	< 0.000**	0.495
Y5DBOTH	0.802	0.078	10.305	< 0.000**	0.561
Y5DMIND	0.827	0.091	9.102	< 0.000**	0.527
Y5DEFFRT	0.978	0.095	10.314	< 0.000**	0.583
Y5DFEAR	0.567	0.071	7.971	< 0.000**	0.465
Y5DLONE	0.642	0.077	8.293	< 0.000**	0.495
Y5DNOGO	0.774	0.095	8.191	< 0.000**	0.539
Y5DSLEEP	0.232	0.082	2.818	0.005*	0.128

Y5Insomnia =~

Y5SINSOM1	1.000				0.575
Y5SINSOM2	1.479	0.113	13.091	< 0.000**	0.804
Y5SINSOM3	1.330	0.104	12.824	< 0.000**	0.704
Y5STIRED	0.569	0.081	7.037	< 0.000**	0.330
Y5DSLEEP	0.726	0.068	10.695	< 0.000**	0.587

Y5Cognitive =~

Y5BOXT	1.000				0.815
Y5DIGITCT	0.932	0.029	31.968	< 0.000**	0.884
Y5PCSCORE	0.252	0.012	20.400	< 0.000**	0.753
Y5LCSCORE	0.162	0.009	17.389	< 0.000**	0.614
Y5ACCDST	0.296	0.051	5.777	< 0.000**	0.390
Y5ACCDDT	0.253	0.053	4.820	< 0.000**	0.335

Y7Depression =~

Y7DDEP	1.000				0.745
Y7DHOPE	0.518	0.207	2.505	< 0.000**	0.206
Y7DHAPPY	1.419	0.230	6.184	< 0.000**	0.660
Y7DBOTH	0.791	0.170	4.644	< 0.000**	0.572
Y7DMIND	0.671	0.180	3.735	< 0.000**	0.402

Y7DEFFRT	0.812	0.183	4.448	< 0.000**	0.395
Y7DFEAR	0.554	0.134	4.145	< 0.000**	0.538
Y7DLONE	1.039	0.133	7.820	< 0.000**	0.704
Y7DNOGO	0.817	0.192	4.254	< 0.000**	0.502
Y7DSLEEP	0.542	0.167	3.240	0.001*	0.286
Y7Cognitive =~					
Y7BOXT	1.000				0.857
Y7DIGITCT	0.919	0.024	37.785	< 0.000**	0.913
Y7PCSCORE	0.230	0.011	21.722	< 0.000**	0.764
Y7LCSCORE	0.159	0.009	17.410	< 0.000**	0.664
Y7ACCDST	0.468	0.060	7.775	< 0.000**	0.473
Y7ACCDDT	0.340	0.047	7.240	< 0.000**	0.417

*Note. *p* < .01. **p* < .001

Table 4

Invariance for Latent Factors Across Time

Model tested	x^2	df	RMSEA (95% CI)	CFI	ΔCFI	TLI	Pass?
Null	16078.636	1612	---	---	---	---	---
Configural	2295.588	1393	.026 (.024, .028)	.938	---	.928	Yes
Weak	2355.278	1425	.026 (.025, .028)	.936	.002	.927	Yes
Strong	2557.074	1456	.028 (.027, .030)	.924	.012	.916	No
Partial Strong	2429.567	1454	.027 (.025, .029)	.933	.003	.925	Yes

Note. For the measurement model tests of invariance, a change in CFI of 0.01, or less and the RMSEA model tests are used.

Table 5

Comparison of CFA and SEM models

Model tested	x^2	df	RMSEA (95% CI)	CFI	ΔCFI	TLI	Pass?
Null	18076.331	2088	---	---	---	---	---
CFA	3019.627	1838	.026 (.024, .027)	.926	---	.916	Yes
Initial SEM	3113.585	1864	.026 (.025, .028)	.922	.004	.912	Yes
Pruned SEM	3117.296	1871	.026 (.024, .028)	.922	.000	.913	Yes

Note. For the measurement model tests of invariance, a change in CFI of 0.01, or less and the RMSEA model tests are used.

Figure 1.

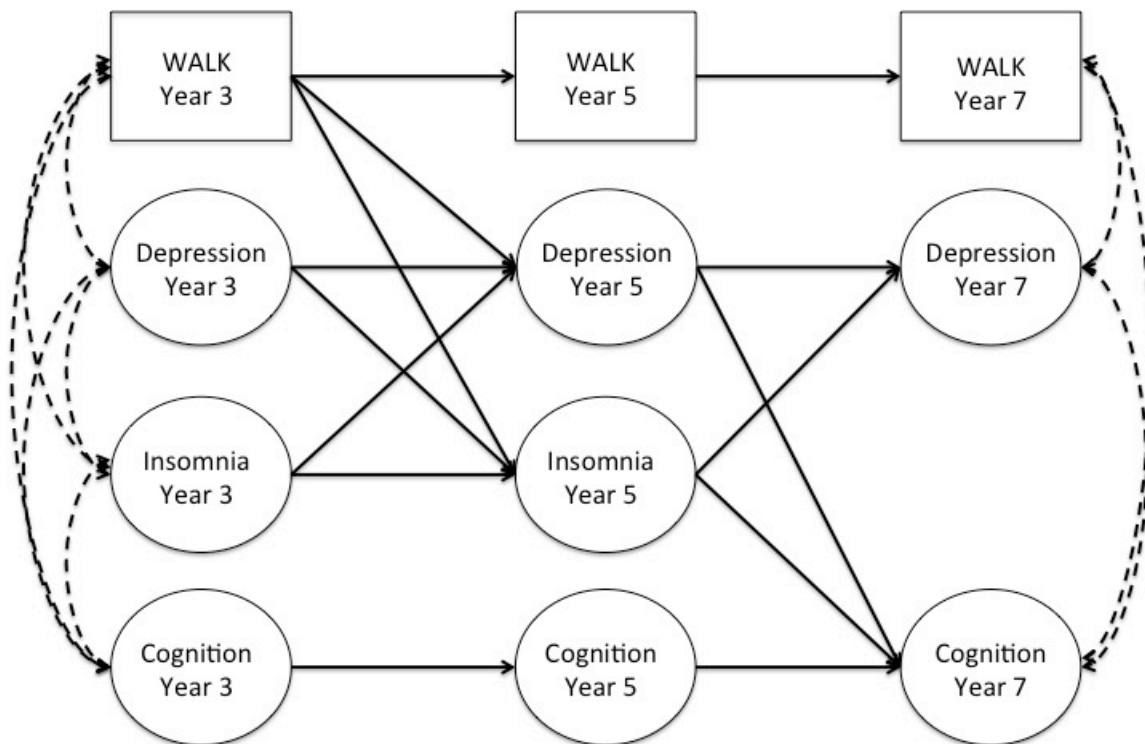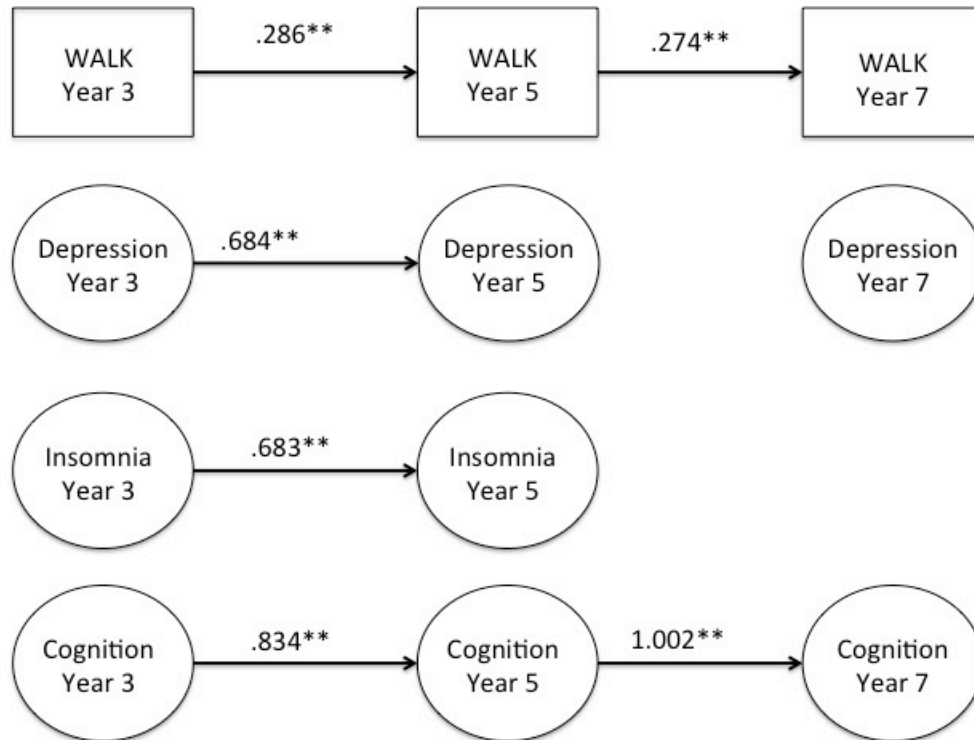Flow-chart of reasons for participant exclusion.
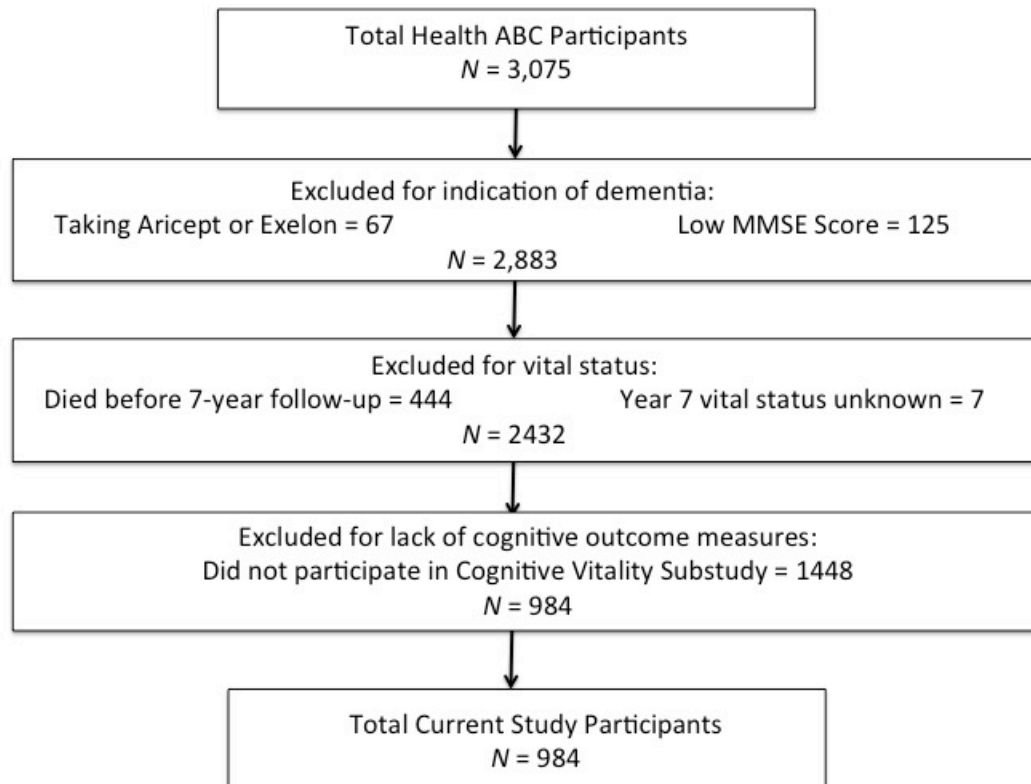
Figure 2

Longitudinal mediation model of symptoms of depression and insomnia mediating the effect of physical activity on cognition.

Note. Intercorrelations for Time 2 variables are not depicted to ensure figure clarity.

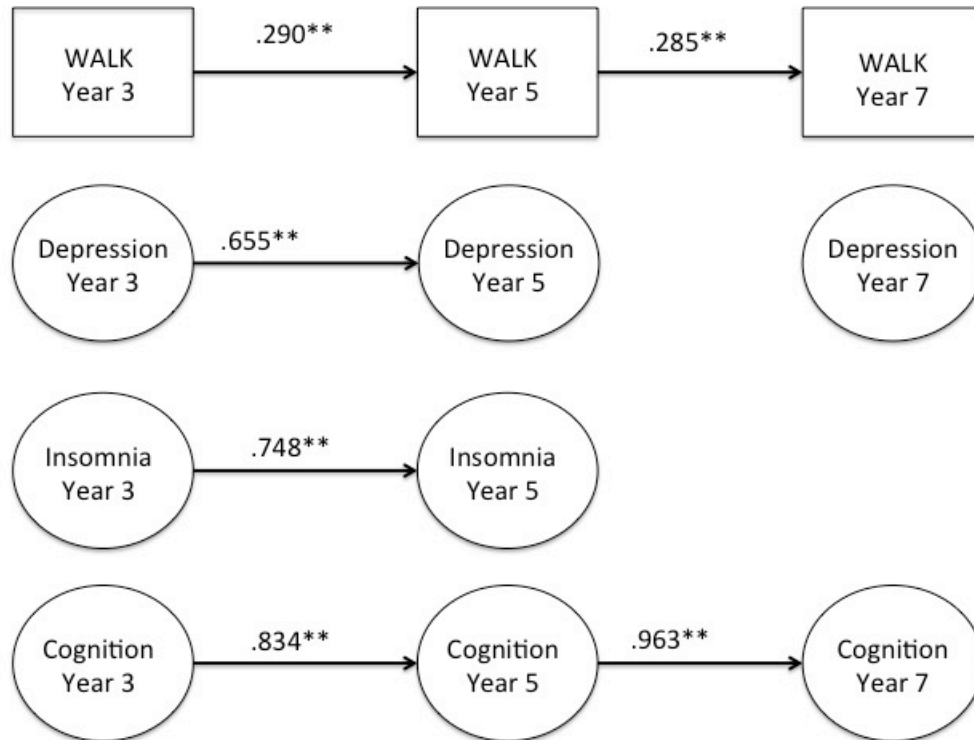Figure 3

Final model with unstandardized parameter estimates

Note. Intercorrelations within time points are not depicted to ensure figure clarity.

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

Figure 4

Final model with standardized parameter estimates

Note. Intercorrelations within time points are not depicted to ensure figure clarity.

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