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SEDENTARY BEHAVIOR-INDUCING RANDOMIZED CONTROL INTERVENTION ON VARIOUS HEALTH-RELATED OUTCOMES IN ACTIVE, YOUNG ADULTS

A thesis
presented in partial fulfillment of requirements
for the degree of Master of Science
in the Department of Health, Exercise Science
and Recreation Management
The University of Mississippi

by

Meghan K. Edwards

May 2016

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ABSTRACT

An expansive body of literature has established the positive effects of physical activity on a number of health-related outcomes, including chronic disease prevention (both physiological and psychological). In addition to growing information regarding the benefits of physical activity, recent research has suggested the need to focus future studies on the effects of sedentary behavior (independent of physical activity) on one's wellbeing. The majority of existing research on sedentary behavior, however, has used cross-sectional study designs. The few experimental studies on sedentary behavior have primarily focused on minimizing prolonged sedentary behavior among inactive individuals, rather than inducing sedentary behavior.

We hypothesized that if indeed there is an independent causal relationship between prolonged sedentary behavior and worse health-related parameters, increasing sedentary behavior among 'active' individuals should similarly induce negative changes in these parameters. The purpose of this study was to build upon the existing body of sedentary behavior literature by examining the effects of a one-week sedentary behavior intervention (where sedentary behavior was increased) on cognitive function, sleep, and mental health conditions (anxiety, depression, mood and quality of life).

Participants confirmed to be active (i.e., acquiring 150 min/week of physical activity) via self-report and accelerometry were randomly assigned into a sedentary behavior intervention group (n = 26) or a control group (n = 13). For one week, the intervention group eliminated exercise and minimized steps to ≤5000 steps/day whereas the control group continued normal

physical activity levels. Both groups completed various cognitive tests, as well as a comprehensive survey assessing sleep, life satisfaction, anxiety, depression, and mood, both pre and post-intervention. The intervention group resumed normal physical activity levels for one week post-intervention and then completed the survey once more. Significant group x time interaction effects were observed in all health outcomes with the exception of cognitive function.

In conclusion, a one-week sedentary behavior-inducing intervention has deleterious effects on sleep, life satisfaction, anxiety, depression, and mood in an active, young adult population. To promote and maintain desirable levels of these health outcomes among active individuals, consistent regular physical activity may be necessary.

LIST OF ABREVVIATIONS

CVD = cardiovascular disease
DSM-5 = Diagnostic & Statistical Manual of Mental Disorders, 5th Edition
GAD = Generalized Anxiety Disorder
HDL = high-density lipoprotein
LPL = lipoprotein lipase
MVPA = moderate-to-vigorous physical activity
OASIS = Overall Anxiety Severity Impairment Scale
PA = physical activity
PHQ-9 = Patient Health Questionnaire-9
POMS = Profile of Mood States
PSQI = Pittsburgh Quality Sleep Index
RCT = randomized control trial
SD = standard deviation
SWB = subjective well-being
SWLS = Satisfaction with Life Scale

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CHAPTER 1: INTRODUCTION

COGNITIVE FUNCTION

Chronic disease is pervasive in our society;[1] for example, 25% of adults are considered to be multimorbid (having 2 or more coexisting chronic diseases), and 65% of older adults are multimorbid with three or more conditions.[2,3] Many of the chronic conditions individuals suffer from are preventable with the right health-enhancing behaviors and habits.[1] For instance, physical activity (any bodily movement produced by skeletal muscles that results in energy expenditure)[4] has many known health benefits and has been suggested as an optimal method for preventing and treating morbidity and mortality.[5] Of interest to this study, recent research suggests that there is a bi-directional relationship between physical activity and cognitive function (i.e., individuals with higher level executive functioning may choose to participate more in physical activity or individuals who are more physically active may in turn observe improved cognitive function).[6] Recent research also suggests that physical activity (PA) may serve as a moderator between executive functioning and smoking.[7] It is conceivable that PA may also moderate the relationship between executive function (complex cognitive processing requiring the coordination of several processes to achieve a particular goal[8]) and other health behaviors, such as participating in physical activity. Notably, a recent study demonstrated that multimorbidity and cognitive function are inversely associated, with physical activity moderating this relationship.[9] Collectively, this previous research has raised questions and ultimately led to

our current experimental investigation of the association between sedentary behavior and cognitive function.

Cognitive function is a broad construct that includes several sub-components such as memory, attention, executive functioning, psychomotor speed, language, and visuospatial ability.[10]Adequate cognitive function is crucial for optimal functioning, including having an accurate awareness of one's situation, setting goals, and meeting the challenges of daily life.[10] Mechanisms proposed to mediate the relationship between PA and cognitive function include physical activity's effects on the brain's neural systems (such as memory encoding and information processing);[11] molecular mediators for PA and cognition (such as brain-derived neurotrophic factor); and cellular environments that enhance cognition through neurogenesis and vascular function.[12]

Various interventions have evaluated and demonstrated favorable effects of both acute bouts of exercise and chronic exercise patterns on brain functions such as executive processing, short-term memory and long-term memory.[13-16] While research regarding youth and older adults is prevalent, fewer studies have evaluated the effects of PA on cognitive function in younger adults.[17] It is important to examine the relationship between exercise and cognitive function in this younger population because there is some evidence that suggests various neurocognitive parameters may begin to decline in young adult years (ie, 20s).[18]

Furthermore, recent research suggests it is imperative that in addition to evaluating physical activity levels, sedentary behavior also be taken into consideration. Independent of physical activity, sedentary behavior (activity that requires minimal body movement resulting in low energy expenditure similar to resting levels)[19] has been found to be a negative indicator of health in adult populations.[20] A number of studies have evaluated the relationship between

sedentary behavior time and cardiovascular disease, among other cardiometabolic diseases.[21-25] One such study compared the effects of sitting time amongst individuals with different classifications of time-spent-sitting and found that the risk for cardiovascular disease was 2.7 fold greater in high sitters when compared to low sitters.[24] Another study demonstrated that prolonged sitting predicted an increased risk of CVD independent of age or recreational energy expenditure.[25] Research investigating the underlying physiological mechanisms of sedentary behavior proposes that a short-lived inhibitor protein for post-translational regulation of lipoprotein lipase (LPL), the essential enzyme for the breakdown of triglycerides in circulating lipoproteins,[26] is induced by physical inactivity stimuli.[27] Another study has suggested that there is a regulatory factor up-regulated during inactivity that suppresses the amount of capillary LPL in muscle tissue.[28] LPL levels in skeletal muscle knowingly increase after short-term exercise training[29] and have been shown to decrease as a result of physical inactivity.[30] Low LPL levels have been associated with two risk factors often associated with CVD, reduced plasma HDL and lowered plasma triglyceride uptake.[31,32] This is important, because there is abundant evidence of various CVD risk factors being associated negatively with components of cognitive function.[33,34] For instance, associations between high blood pressure and poor performance on neuropsychological tests have been documented, with high blood pressure negatively affecting cognitive processes including reasoning and planning.[35] It has been suggested that CVD may result in pathological alterations in brain structure, including grey matter atrophy, increases in white matter lesions, and damage to white subcortical matter pathways, all of which have been negatively correlated with scores on various neuropsychological tests.[36] A recent literature review of sedentary behavior research suggested that future studies of sedentary behavior should focus on the physiological and neurological effects of a “lack of human movement” in

contradistinction to the effects of physical movement, including studies that evaluate more than just time spent sitting (a common parameter used to evaluate sedentary behavior).[37] Only when this distinction is made and studied appropriately can we begin to truly understand the underlying mechanisms for chronic disease development and various health outcomes in sedentary individuals.

The primary purpose of this study was to address this gap in literature by examining the effects of a 1-week, experimentally controlled, free-living sedentary behavior intervention. Specifically, this study examined cognitive function in young, active adults in efforts to better understand the relationship between brain function and sedentary behavior. This design is unique in that, unlike other observational studies that have examined the association between sedentary behavior and health outcomes among participants with variability in their activity levels, we experimentally manipulated movement-based behaviors among active individuals.

We hypothesized that cognitive function in active individuals would decrease from baseline after a 1-week sedentary behavior intervention and would resemble baseline scores again after normal activity levels had been resumed. This hypothesis was plausible as previous research has suggested that if you increase PA, cognitive benefits may arise.[13] Thus, it was reasonable for us to suggest that cognitive function may decline if sedentary behavior is increased. We believe this approach may provide the strongest evidence of a potential cause-and-effect relationship between sedentary behavior and cognitive function.

SLEEP QUALITY

Sleep deprivation (failure to obtain adequate amounts of sleep, with adult recommendations being 7-8 hours/night)[38] and sleep deficiency (the presence of sleep

deprivation, disrupted sleep cycles, or a sleeping disorder)[39] have been associated with a number of modifiable negative health outcomes, such as obesity, depression, diabetes, cardiovascular disease, and all-cause mortality.[40] There are over 100 recognized sleep disorders, which can be classified into the following categories: insomnia disorders, sleep-related breathing disorders, central disorders for hypersomnolence, circadian rhythm sleep-wake disorders, sleep-related movement disorders, and parasomnias.[41] Insomnia disorders (i.e., trouble falling asleep or staying asleep) affect approximately 10 percent of adults.[42] Obstructive sleep apnea, a sleep-related breathing disorder has been estimated to affect between 2 and 7 percent of the adult population.[43] Existing treatments for sleep insomnia include sedatives and hypnotics, cognitive behavioral therapy and lifestyle interventions.[42] Prescription medications utilized as sleep aids can be costly, do not cure the condition, and may induce maladaptive side effects, central nervous system toxicities, dependency, or potential rebound sleep impairment after discontinuation.[42] With regards to sleep apnea, weight status is a significant risk factor, with approximately 50 percent of sleep apnea patients being classified as overweight or obese.[44] Treatments for sleep apnea typically involve lifestyle adjustments (i.e., increasing exercise and modifying diet to promote weight loss), wearing a mouth guard to assist with maintaining an open airway passage at night, or use of a continuous positive airway pressure machine to assist with keeping airways open during sleep.[44]

Poor sleep quality can also extend to individuals with sub-clinical poor sleep symptomology (lacking a diagnosed sleep disorder). For instance, troubles with falling asleep or daytime sleepiness affect approximately 35 to 40% of the U.S. adult population.[45] While excessive daytime sleepiness is a common side effect of numerous sleep disorders, it is also associated with sleep deprivation and is often observed in patients with psychiatric disorders

(e.g., depression, seasonal affective disorder).[46] It is evident that sleep quality may be impeded by both intrinsic factors (i.e., an existing sleep disorder) and extrinsic factors (e.g., chronic alcohol or drug use, excessive caffeine consumption, poor sleep hygiene).[47] Regardless of the cause, failure to obtain optimal sleep quality can negatively impact individuals in a myriad of ways, including cognitive impairment (e.g., decreased attention, working memory, long-term memory, and decision-making abilities).[48] Additionally, as previously mentioned, inadequate sleep may also have a negative influence on one's mental health[46] and also puts an individual at an increased risk for a number of physical, chronic diseases.[40]

As a result of the comorbidities[49] often associated with poor sleep quality or sleep disorders, recent research has examined alternative methods of promoting better quality sleep. Physical activity, which has many well-established global health benefits (decreased risk for cardiovascular disease, heart attack, stroke, certain cancers, Type II diabetes and obesity as well as an increased overall quality of life),[50-55] has been evaluated as a possible method for improving various sleep-related parameters. Epidemiological work has demonstrated a positive relationship between physical activity and sleep.[56-59] Research utilizing objective measurements of physical activity to evaluate its effects on sleep quality is sparse, however a recent study using accelerometry to measure physical activity found that physical activity significantly negatively influenced perceived daytime sleepiness as well as the ability to focus when tired.[60] One proposed mechanism to explain physical activity's positive influences on sleep is the temperature down-regulation theory, which postulates that sleep onset is associated with a decline in body temperature experienced post-exercise (via peripheral heat dissipation through vasodilatation).[61]

In addition to the demonstrated positive effects of physical activity on sleep, most health scientists would likely agree that physical activity is imperative for good overall health and

wellbeing. Emerging research suggests that regardless of time spent being physically active, sedentary behavior is linked with a number of negative health outcomes.[20,62] For instance, recent epidemiological work has demonstrated a negative association between sedentary behavior and daytime sleepiness, independent of physical activity levels.[60] However, few studies have utilized experimentally designed interventions and an objective measurement of sedentary behavior to draw conclusions upon.[37]

The purpose of this study was to build upon current understandings of the sedentary behavior-sleep relationship via a randomized, controlled sedentary behavior intervention. Among an "active" sample, we assessed several sleep-related parameters (detailed later herein) to determine if a sedentary behavior intervention (i.e., minimizing physical activity and increasing sedentary behavior) had a significant effect on any of these parameters. We hypothesized active individuals whose sedentary behavior was increased for one week would have worse post-intervention sleep. In addition, we hypothesized that sleep would again improve after normal activity was resumed (i.e. returned to baseline levels). This hypothesis is plausible because, as mentioned previously, research has demonstrated a positive relationship between physical activity and sleep quality.[61] Thus, it is reasonable to suggest that sleep quality may worsen if sedentary behavior is increased. This approach may provide the strongest evidence of a potential cause-and-effect relationship between sedentary behavior and sleep quality.

LIFE SATISFACTION

Quality of life is a term used to describe the overall human experience.[63] Improving quality of life is a common goal that most individuals, communities, and societies share.[63] There are considered to be two major components of quality of life. The first component involves the

examination of various social and economic indicators that ultimately aim to demonstrate *how well an individual's needs are being met*.[68] These needs may include things such as subsistence, security, affection, understanding, creativity, leisure, identity, and freedom.[63] The measurement of needs being met has been described as relatively (when compared to the second quality of life component) objective in nature (i.e., the needs are either being met or they are not).[63] Health-related quality of life measurement tools, for example, tend to focus on monitoring health status and comparing the burdens of different diseases.[69] These tools may measure the physical or mental health status of an individual (e.g. weight, body mass index, prevalence of chronic conditions, the affect to which a chronic condition interferes with activities of daily life, etc.), but alone they are not believed to adequately assess overall quality of life.[63]

The second type of quality of life assessment is a general construct referred to as *subjective well-being* (SWB), which evaluates outcomes that are subjective in nature (e.g., self-reported happiness, pleasure, and fulfillment).[70,71] The last decade has yielded a significant increase in the research on subjective well-being, as the study of subjective well-being can contribute valuable knowledge and insights to important psychology topics, such as the root causes of unhappiness and diseases related to unhappiness (e.g., depression).[72] Previous work on the topic has identified two components of subjective well-being. The first component of subjective well-being is affective in nature (i.e. having to do with mood states), typically further distinguished as either pleasant affect or unpleasant affect.[73] The second type of subjective well-being is cognitive in nature and is referred to as life satisfaction.[74] Previous work regarding subjective well-being has focused mainly on the affect-related parameters, with less emphasis on the life satisfaction component of subjective well-being.[75,76]

Life satisfaction, the outcome variable of interest in our study, is said to involve a conscious judgmental process in which individuals employ a unique set of criteria to assess the quality of their own lives.[77] Global judgment of life satisfaction is predicted to depend upon the comparison of one's life circumstances to their unique standards.[75] Much research regarding life satisfaction supports the opinion that factors influencing this outcome may have either a top-down or a bottom-up effect.[72] Top-down influences are time-invariant, trait-like (e.g., mental health, body mass index, overall physical activity, personality, self-esteem, sex) and allow for the evaluation of between-person differences and similarities.[78] Bottom-up influences vary with time, are state-like (e.g. fatigue, self-esteem, mental health, daily physical activity) and allow for the evaluation of within-person differences and similarities.[78] Some factors, such as physical activity, have been demonstrated to have both top-down and bottom-up positive associations with life satisfaction and have thus received considerable recent attention in research aiming to distinguish which effects (i.e., top-down or bottom-up) may have the most significant influences.[78,79] Also contributing to our knowledge of the physical activity-life satisfaction relationship are several prospective studies on elderly populations, which have demonstrated the positive effects on life satisfaction as related to regular physical activity.[80-82] Physical activity is believed to indirectly enhance one's life satisfaction via influences on affect, physical self-worth, self-efficacy, and mental health.[80-82]

While it is important to understand how physical activity may influence one's life satisfaction, emerging research suggests that regardless of time spent being physically active, sedentary behavior is associated with a number of negative health outcomes (i.e., individuals who are more sedentary may have an increased risk of deleterious health consequences, including those who achieve the recommended levels of physical activity).[20] Of particular

relevance to our present study, recent epidemiological work has demonstrated a negative association between objectively measured sedentary behavior and life satisfaction, independent of (objectively measured) physical activity levels.[79] However, no studies (to our knowledge) have utilized an experimentally designed sedentary behavior intervention to draw conclusions upon.

The purpose of this study was to build upon current understandings of the sedentary behavior and life satisfaction relationship via a randomized, controlled sedentary behavior intervention. Among an "active" sample, we assessed life satisfaction to determine if a sedentary behavior intervention (i.e., minimizing physical activity and increasing sedentary behavior) had a significant effect on this outcome. We hypothesized active individuals whose sedentary behavior was increased for one week would report a lower post-intervention life satisfaction. In addition, we hypothesized that life satisfaction scores would improve after normal activity was resumed (i.e. returned to baseline levels). This hypothesis is plausible because, as mentioned previously, research has demonstrated a positive relationship between physical activity and life satisfaction.[78-83] Thus, it is reasonable to suggest that life satisfaction may worsen if sedentary behavior is increased. This approach may provide the strongest evidence of a potential cause-and-effect relationship between sedentary behavior and life satisfaction.

ANXIETY

Anxiety disorders (including panic disorders, generalized anxiety disorder, post traumatic stress disorder, phobias, and separation anxiety disorder) are the most common class of mental disorders present in the general population.[84] Anxiety disorders have lifetime prevalence rates ranging between 13.6 and 28.8 percent and 12-month prevalence rates between 5.6 and 19.3

percent (in an American adult population),[85] reflecting original observations by Cattell and Scheier (1961) that anxiety may be state-like (transient; reflecting a current emotional state) or trait-like (chronic; anxiety differences resulting from differing personality traits).[86] Generalized anxiety disorder (GAD) is the most common anxiety disorder seen in primary care. 6.8 million (3.1%) of United States adults suffer from generalized anxiety disorder, which is characterized by chronic, excessive, uncontrollable worry about a number of events or activities.[87] Generalized anxiety disorder has been associated with a number of somatic complaints, including chest pain and irritable bowel syndrome, and is believed to reduce effective problem solving confidence, lower senses of personal control, and inhibit ambiguous task performance.[87] Additionally, GAD has been associated with lower levels of daily life satisfaction and may detrimentally influence one's social, family, and occupational functioning.[88]

Further emphasizing the importance of employing effective treatments to alleviate and prevent anxiety symptomology is the knowledge that comorbidity is more common among those with anxiety disorders. Approximately 75 percent of individuals with a lifetime anxiety disorder have at least one other mental disorder.[84] For instance, according to reports from the National Comorbidity Study, GAD has a 91% rate of comorbidity.[89] Specifically, GAD has been shown to have strong associations with a number of affective disorders (e.g., major depressive disorder & bipolar disorder).[90] Also of notable importance, subthreshold levels of anxiety disorders (e.g. subthreshold GAD or major depressive disorder) have been reported to be more prevalent than the threshold presentation of the same disorders and may elicit similar psychological distress and lower perceptions of health (compared to healthy individuals) as clinically diagnosed anxiety disorders.[91]

In addition to the significant personal burdens and barriers that may present themselves with presence of an anxiety disorder, anxiety disorders result in a high economic burden, costing the United States over 42 billion dollars a year (as reported in the 1990s).[92] Included within these costs are emergency room visits, specialist referrals, diagnostic tests, and prescribed medications (among others).[87] Traditional treatments for anxiety disorders include various pharmacotherapy medications (e.g. antidepressants), as well as psychosocial treatment (e.g., cognitive behavioral therapy).[87] Integrative acceptance and mindfulness-based models as well as physical activity programs have also been evaluated as alternative treatment options.[87,93,94] The multitude of global health-related benefits of physical activity (e.g., decreased risk for cardiovascular disease, heart attack, certain cancers, Type II diabetes and obesity as well as an increased overall quality of life)[50,53-55,95,96] coupled with the lack of negative physical side-effects that may result from pharmacotherapy options (e.g., withdrawal symptoms and dependency issues)[97] make physical activity a particularly appealing option for anxiety treatment and prevention. In addition to considerable epidemiological evidence of an inverse relationship between physical activity and anxiety, review papers and meta-analyses have highlighted numerous physical activity intervention studies that collectively demonstrate the anxiolytic effects of physical activity.[98-100]

While it is important to understand how physical activity may influence one's anxiety levels, emerging research suggests that regardless of time spent being physically active, sedentary behavior is associated with a number of negative health outcomes (i.e., individuals who are more sedentary may have an increased risk of deleterious health consequences, including those who achieve the recommended levels of physical activity).[20,62] Of particular relevance to our present study, a recent systematic review demonstrated a positive association between sedentary behavior and anxiety.[101] This review summarized results from nine

observational study designs (7 cross-sectional and 2 longitudinal studies) and found a moderately strong association between increased sitting time and increased risk for anxiety. The authors declared the need for future longitudinal and intervention study designs to further explore the topic.[101] No studies (to our knowledge) have utilized an experimentally designed sedentary behavior intervention (where sedentary behavior is increased) to draw conclusions upon (with regards to the health outcome of anxiety).

The purpose of this study was to build upon current understandings of the sedentary behavior and anxiety relationship via a randomized, controlled sedentary behavior intervention. Among an "active" sample, we assessed anxiety to determine if a sedentary behavior intervention (i.e., minimizing physical activity and increasing sedentary behavior) had a significant effect on this outcome. We hypothesized active individuals whose sedentary behavior was increased for one week would report higher post-intervention anxiety levels. In addition, we hypothesized that anxiety levels would decline after normal activity was resumed (i.e. returned to baseline levels). This hypothesis is plausible because, as mentioned previously, observational-based research has demonstrated an inverse relationship between physical activity and anxiety.[99,100] Thus, it is reasonable to suggest that anxiety may worsen if sedentary behavior is increased. This approach may provide the strongest evidence of a potential cause-and-effect relationship between sedentary behavior and anxiety. Evaluating this within the younger adult population is of particular importance as this is a critical time period where physical activity declines[102] and anxiety has been shown to be prevalent.[103] For instance, a recent paper demonstrated that the prevalence of anxiety disorder in a national sample of US adults ages 20-39 years is 16 percent.[104] It has been suggested that the amount of physical, cognitive, and

psychosocial changes young adults undergo increases their susceptibility to the development of anxiety disorders.[105]

DEPRESSION/MOOD

Mental illness affects nearly 25% of the U.S. adult population,[106] with mood disorders affecting nearly 10% of the adult population.[90] Depression and mood are interrelated, with depression classified as a mood disorder.[107] The depression mood disorders included in the Diagnostic & Statistical Manual of Mental Disorders, 5th Edition (DSM-5), include major depressive disorder, disruptive mood disorder, persistent depressive disorder (dysthmia), and premenstrual dysphonic disorder.[108]

Mood disorders can have a profound effect on various aspects of one's life including personal relationships as well as academic and work-related performance,[109] which has led to abundant research examining various treatment options. Individuals suffering from a mood disorder may experience feelings of hopelessness, low-self esteem, excessive guilt, difficulty concentrating, sensitivity to failure, decreased decision-making abilities, irritability, and aggression, among others.[110] While extensive previous research has evaluated the effectiveness of psychotherapy and pharmacotherapy for treatment of mental disorders, physical activity has also been implemented as an alternative treatment for mental disorders due to its many global health benefits.[55,111,112] For instance, physical activity has been attributed to decreased risk for cardiovascular disease, heart attack, stroke, certain cancers, type II diabetes and obesity as well as an increased overall quality of life.[50-55,95,96,113] Previous reviews of exercise and mental health research have highlighted that the majority of experimental research regarding exercise and mental disorders supports the anti-depressive properties of exercise as well as its ability to increase positive mood-related attributes (e.g., cheerfulness).[100] Both chronic exercise

interventions and acute bouts of exercise have been shown to improve depression and mood states.[100,114] Epidemiological work also supports the inverse association between physical activity and depression symptomology.[115-119]

There are a number of proposed explanations for the relationship between exercise and improved mood and reduced depression symptoms, including both psychological and physiological mechanisms. Among such psychological mechanisms are the *distraction hypothesis* (diversion from unpleasant stimuli of daily life can lead to improved mood state following exercise),[120,121] *self-efficacy theory*[122] (increased confidence in one's ability to exercise and maintain a schedule of regular exercise may translate to improved self-confidence in the ability to handle events that challenge one's mental health),[123,124] *mastery hypothesis* (mastering exercise techniques and completing workouts can induce feelings of independence and success, which can translate over into other areas of one's life),[125] and *social interaction hypothesis* (social relationships and mutual support from others during exercise can have a positive effect on one's mental health).[126] Among the proposed physiological mechanisms are the *monoamine hypothesis* (exercise improves brain aminergic synaptic transmission, affecting monoamines such as noradrenaline, dopamine, and serotonin, all of which have been implicated in depressive disorders),[127,128] *endorphin hypothesis* (endorphins are produced as a result of exercise, which help to reduce pain and induce a state of euphoria),[124,129] and *thermogenic hypothesis* (body temperature elevations resulting from exercise may elicit improved mood).[130] Another proposed explanation is that exercise increases resistance against stress-related psychiatric disorders such as depression, through variables such as perceived control.[131] While there are numerous possible explanations, there is no overwhelming evidence supported by randomized, controlled interventions to confidently isolate one as the prominent mechanism.[120] Considering only 48% of

adults meet recommended daily physical activity guidelines,[132] it is also imperative that we consider how sedentary behavior may affect mental health.

Recent suggestions that sedentary behavior has detrimental effects on one's health independent of one's physical activity levels has inspired a new line of research on the effects of sedentary behavior.[20] Associations between sedentary behaviors and various mental health outcomes such as depression, anxiety, and self-esteem have been demonstrated.[20,37,101] Additionally, sedentary behavior has been associated with negative physical health outcomes including increased risk of obesity and type II diabetes, both of which have been linked with poor mental health,[133,134] which may be ameliorated with physical activity.[135] Notably, the majority of research regarding the consequences of sedentary behavior has come from cross-sectional observation studies, which preclude any ability to ascertain temporality, and thus, cause-and-effect. In order to better understand the relationship between sedentary behavior and mental health outcomes as well as the mechanisms that influence these relationships (which may differ from physical activity/mental health mechanisms), future high-quality longitudinal and interventional research is needed.[37]

The purpose of this study was to build upon current understandings of these relationships via a randomized, controlled sedentary behavior intervention. Among an "active" sample, we assessed depression and mood both pre- and post-intervention to determine if a sedentary behavior-inducing intervention (i.e., minimizing physical activity and thus increasing sedentary behavior) had a detrimental effect on either of the outcomes (mood and depression). We hypothesized that active individuals whose sedentary behavior was increased for one week would have higher post-intervention levels of depression as well as worsened post-intervention mood. In addition, we hypothesized that depression levels would decrease after normal activity

was resumed whereas mood state would improve (i.e., return to their normal/baseline levels). This hypothesis is plausible because previous research has suggested that increasing physical activity may elicit mental health benefits.[100] Thus, we believe it was reasonable to suggest that mental health outcomes (i.e., depression and mood) may worsen if sedentary behavior is increased. This approach may provide the strongest evidence of a potential cause-and-effect relationship between sedentary behavior and mental health outcomes. Although recent epidemiological work has reported a positive association between sedentary behavior and depression,[136,137] no randomized controlled interventions have been conducted on this topic.

CHAPTER 2: METHODS

RECRUITMENT

Participants were eligible for the study if they were between 18 and 35 years old, were confirmed to be active, spoke English, and provided informed consent. Participants were excluded from the study if they failed to obtain adequate levels of physical activity (described below) in the week of accelerometer data collection prior to the intervention. All participants provided informed consent and the study procedures were approved by the authors' institutional review board.

The recruitment goal was 30-40 participants with at least n=22 in the intervention group; this was based off of our pilot data (prospective, non-RCT study design)[138] showing that, among a sample of 29 participants who had similar demographic characteristics to the participants in this study, prospective changes in sedentary behavior were statistically significantly associated with increased changes in depression symptomology. A student researcher at the authors' institution recruited participants using a non-probability convenience sampling approach (specifically, word of mouth was used to recruit co-workers as well as students from undergraduate classes within the authors' department). The final recruited sample size was N= 39, and using a 2:1 sample size ratio for intervention and control participants,[139] 26 participants were randomly assigned into the sedentary behavior intervention group and 13 participants were randomly assigned into the control group. A 2:1 sample size ratio was used because of considerations related to study resources (e.g., equipment and research personnel). Notably,

experimental-to-control ratios of 1:1 generate the most statistical power; however, ratios of 2:1 do not substantively reduce statistical power (e.g., ~5% difference), and unequal allocation, if performed randomly, still results in equivalent groups in terms of equal distribution of confounding parameters.

VISIT DETAILS

Each participant completed either two (control group) or three (intervention group) visits, in addition to a pre-visit meeting. Hereafter, these visits are referred to as Baseline Visit (both groups), Visit 1 (both groups), Visit 2 (both groups), and Visit 3 (intervention group only), respectively. Additionally, the week between the Baseline Visit and Visit 1 will be referred to as Baseline Week, the week between Visit 1 and Visit 2 will be referred to as Week 1, and the week between Visit 1 and Visit 2 will be referred to as Week 2. The visits were scheduled approximately one week apart and at roughly the same time of day. Figure 1[Ψ] contains a visual overview of the intervention design, with details explained in the narrative that follows.

At the Baseline Visit, the participant came in to our laboratory to confirm they met inclusion criteria for the study and, if deemed eligible to participate, to pick up their accelerometer. At this visit, the participant provided written informed consent of all of the study procedures. Next, they completed the International Physical Activity Questionnaire short form (IPAQ-SF) as a screening method to ensure that adequate levels of physical activity to participate in our study had been obtained (discussed in the "Physical Activity" section herein to follow). Out of the 47 individuals who were originally recruited, four were not eligible due to self-reporting inadequate levels of physical activity. These four individuals therefore did not continue

[Ψ] Figure 1 is included within the Appendix, along with all other figures and all tables.

with the study and were ultimately not included in the final sample size (N=39). The 43 individuals whose IPAQ-SF scores indicated that they met MVPA guidelines were given an accelerometer to wear for a 1-week period during which they were asked to continue with their regular physical activity and exercise patterns. Each participant was educated on correct accelerometer wear, including where to place the accelerometer on the body and for how long to wear it (described below). The purpose of having participants wear an accelerometer for a week *prior to the intervention* was to objectively confirm their self-reported physical activity levels. This is important because previous research has shown only a modest association (r = 20-.46) between self-reported and objectively measured physical activity.[140]

During the first visit containing assessments of cognitive function, sleep, life satisfaction, anxiety, depression, and mood (Visit 1), participants returned the accelerometer at which point its data was analyzed using the customized macro embedded in the ActiGraph software. The researcher first checked that the participant had adequate amounts of physical activity (defined in "Physical Activity" section to follow) and then verified that the accelerometer was worn for a minimum of 600 minutes/day for at least four out of the seven days.[141] If the participant met both of these criteria, they were then randomly assigned to either the intervention or control group. Prior to recruiting any participants, a random list was generated containing 1s and 2s on Microsoft Excel using the RAND function, where a 1 signified placement into the intervention group and a 2 signified placement into the control group. Allocation concealment was used so the recruiter remained blinded to which group the participant was in until the end of Visit 1 when the participants needed to know whether to restrict steps and eliminate exercise or not. Two recruited participants subjectively reported adequate physical activity levels, however, their accelerometry data did not support these self-reports. These individuals were therefore not

eligible to continue in the study and were not included in the final sample size (N=39). Additionally, six recruited participants did not meet accelerometer wear-time criteria. Accelerometer instructions for wearing were explained again and these six participants were asked to continue wearing the accelerometer for several days. Four of these six did meet wear-time criteria after coming back several days later and were therefore included in the final sample size (N=39), but two of the six were not included in our sample size due to not meeting wear-time requirements for a second time. These participants were not eligible for continuation of the experiment.

The intervention group was asked to be sedentary for seven days (where sedentary was defined as not participating in structured exercise and accumulating no more than 5000 steps/day).[142] Participants who exceeded the 5000-limit step count were not excluded from the study. In fact, 24 out of the 26 participants in the intervention group did exceed the 5000-steps/day limit on at least one out of the 7 days they wore the pedometer. However, we did not consider this to be problematic considering our analysis of step counts across time periods demonstrated a significant reduction in the average daily steps from Baseline Week to Week 1 in the intervention group (see Results section). Because we used accelerometer-derived step-counts for the baseline week and pedometer-derived step counts for the week of the sedentary behavior intervention, we applied a correction factor to adjust for the over-estimation of accelerometer-derived steps.[143] Notably, pedometers were used following the baseline week because (unlike accelerometers) they allow for participants to monitor their steps, which was necessary for those in the intervention group needing to keep steps at or below 5000/day.

The control group was asked to continue with their normal exercise and physical activity habits for the following seven days. Both groups were given a pedometer (DigiWalk SW-200) to

wear for the following seven days as well as a pedometer log sheet where they were asked to record the total number of steps at the end of each day. Also during Visit 1, participants completed the cognitive tests and took a survey that assessed sleep, life satisfaction, anxiety, depression, and mood (testing protocol and survey details will be elaborated on in future text). Herein later to follow, the outcomes of our study (cognition, sleep, life satisfaction, anxiety, depression, and mood) may be referred to collectively as "health outcomes" to avoid redundancy that would result from repeatedly listing throughout the document.

At the second visit containing the health outcomes assessment (Visit 2), both groups were asked to recomplete the same assessments as during Visit 1. The control group returned their pedometers and log sheets and were thanked for their participation in the study. After completing both the cognitive tests and the survey, the intervention group was asked to continue wearing the pedometer for one more week but to resume to their normal physical activity levels.

At the third assessment visit (Visit 3), the intervention group returned their pedometers and log sheets. They then performed the same cognitive tests and completed the same survey as during Visit 1 and Visit 2. The total time required to complete the cognitive tests and the survey at all visits was approximately 30 minutes. All data collection occurred between September 1, 2015 and December 1, 2015.

ASSESSMENT TOOLS

PHYSICAL ACTIVITY

The initial assessment of physical activity levels was gathered via the IPAQ-SF.[144] Reliability of this survey has been established among many differing populations, including young adults demographically similar to our sample.[145] The IPAQ-SF has also been

demonstrated to have adequate levels of criterion validity (r=.35-.43, *P*<.05) and concurrent validity when scores have been compared to objective treadmill tests and accelerometers.[144,146,147] Additionally, adequate test-retest reliability (ICC > 0.75, *P*<.05) of the IPAQ-SF with regards to the vigorous and moderate intensity physical activity questions has been supported in previous research.[148] To be classified as active, participants were required to obtain a minimum of 150 minutes/week of moderate to vigorous physical activity (as recommended by the USDHHS)[149] as reported by the IPAQ-SF.

Physical activity was objectively measured using the ActiGraph GT9X accelerometer, which has demonstrated evidence of reliability and validity.[150,151] Participants wore the accelerometer on the mid-axillary line of the right hip at the level of the iliac crest. Accelerometers were initialized to a 1-minute epoch length using a raw data sampling frequency of 30 Hz. Moderate-to-vigorous physical activity was defined as at least 1952 counts/min.[152] To monitor device wear time, nonwear was defined as a minimum of 60 consecutive minutes of zero activity counts, with the allowance of 1-2 minutes of activity counts between 0 and 100.[102] Only participants with at least four valid days of monitoring were included in the analyses, with a valid day defined as 10+ hrs of wear time.[141] As with the IPAQ-SF, participants needed to demonstrate a minimum of 150 minutes/week of MVPA to be eligible to continue in the study. In addition to the GT9X accelerometers, participants wore the Digi-Walk SW-200 pedometer, which is ranked highly for its accuracy and reliability when compared to other pedometers.[153]

COGNITIVE TESTS

Participants each completed eight cognitive tests of varying complexity, all designed to assess different types of brain function. Evidence suggests that task complexity may moderate

the relationship between exercise and cognition and exercise may affect separate areas of the brain (i.e. frontal versus temporal lobe) in different ways.[154] The eight tests were administered electronically through the Cambridge Brain Sciences website:[155] Spatial Span[156] and Paired Associates[157,158] (to assess working memory), Grammatical Reasoning and Odd One Out[159] (to assess reasoning), Feature Match[160,161] and Polygon[162] (to assess concentration), and Spatial Search[163] and Spatial Slider[164] (to assess planning). All ten tests were administered in a completely random order. Previous research has demonstrated convergent validity of these cognitive tests as they have associated with acute exercise bouts.[17,165] To provide evidence of the reliability of these measures, the researcher completed the cognitive tests the exact same way multiple times (e.g., purposefully answered each question incorrectly each time), and received the same scores. Notably, an intraclass correlation analysis was not performed among the control group as a measure of reliability, as this would assess within-subject cognitive variability, as opposed to reliability of the specific instrument used to assess cognitive function.

SLEEP

To assess the parameter of sleep quality we utilized the Pittsburgh Sleep Quality Index (PSQI). This survey contains four initial short-answer questions (i.e., What time do you normally go to bed?) and 19 four-point, likert scale multiple-choice questions. The survey evaluates seven components of sleep, including general sleep quality, sleep latency, sleep duration, habitual sleep efficiency (measured by frequency of waking up in the middle of the night or waking up earlier than intended), sleep disturbance, use of sleep medication, and daytime dysfunction (measured by difficulty staying awake during the day or lacking enthusiasm for tasks).[166] Each PSQI item asks the participant to recall something about their sleep within the past month (i.e., “In the past

month, how often have you had trouble sleeping because you cannot get to sleep within 30 minutes?"), however due to the timeline of our study having one week in between visits, we had participants answer each question as it pertained to the *previous week*. The scoring algorithm for the PSQI is explained elsewhere[166]. Briefly, the 19 items are used to calculate an overall sleep quality score, with higher scores indicating worse overall sleep quality. The PSQI has demonstrated adequate test-retest reliability (ICC = 0.87),[167] internal consistency (α = 0.80, $p<.05$) and construct validity.[168]

LIFE SATISFACTION

To assess life satisfaction, we utilized the Satisfaction with Life Scale (SWLS).[169] This survey includes five statements (e.g., "The conditions of my life are excellent."), to which respondents must rate how much they agree with, on a 7-point likert scale (1 = strongly disagree and 7= strongly agree).[169] The SWLS items are global in nature, which gives respondents the opportunity to weigh the various domains of their lives in terms of their own values. It is thus considered to provide a global judgment of life satisfaction.[75] In a study of college-aged students (with similar ages as individuals in our current study), the SWLS was found to have good test-retest reliability (.82). The SWLS has also been demonstrated to have adequate levels of internal consistency (α=.61-.81) and convergent validity when compared with the Life Styles Inventory (r=.46).[169]

In the present study, internal consistency, as measured by Cronbach's alpha, was .80 during Visit 1 within the intervention group. Internal consistency was .93 during Visit 1 within the control group. During Visit 2 within the intervention group, internal consistency was .93.

Internal consistency for the control group during Visit 2 was .92. Internal consistency for the intervention group during Visit 3 was .88.

ANXIETY

To assess anxiety levels we utilized the Overall Anxiety Severity and Impairment Scale (OASIS). This five-question evaluation employs a continuous measure of anxiety and therefore can be used to assess those with single or multiple anxiety disorders as well as those with subthreshold anxiety symptoms.[170] The OASIS has a high internal consistency (α = .80) and adequate levels of convergent validity with the Brief Symptom Inventory-18 (r =.58), the Fear Questionnaire (r=.41), the Spielberger Trait Anxiety Questionnaire (r=.62), and the Beck Depression Index (r=.51).[170] Additionally, one-month test-retest reliability among a population of college-aged students (similar to our population of interest) was high (ICC =.82).[170]

In the present study, internal consistency, as measured by Cronbach's alpha, was .83 during Visit 1 within the intervention group. Internal consistency was .98 during Visit 1 within the control group. During Visit 2 within the intervention group, internal consistency was .87. Internal consistency for the control group during Visit 2 was .96. Internal consistency for the intervention group during Visit 3 was .80.

DEPRESSION

To assess depression levels we used the Patient Health Questionnaire (PHQ-9).[171] This self-report questionnaire consists of nine questions asking about depression symptoms. The questionnaire instructs participants to answer the question, "Over the last two weeks, how often have you been bothered by any of the following problems?" In this study, and based on our 1-week study time periods between visits, participants were instructed to answer the questions for

the past one-week as opposed to the past two weeks. Response options ranged from "not at all" to "nearly every day" with corresponding numeric scores ranging from 0-3. The total possible score on the PHQ-9, therefore, ranged from 0-27. Due to a limited sample size, we did not apply a specific PHQ-9 cut-point, but instead, treated the PHQ-9 score as a continuous variable. Notably, all participants provided complete data on the PHQ-9 assessment (i.e., no missing data). The PHQ-9 has been used in previous research as a screen for depression and has been found to be a reliable and valid measure of depression severity.[171] Strong associations between PHQ-9 depression severity and convergent variables have also been found with the Beck Depression Inventory (r=.73) and the General Health Questionnaire-12 (r=.59). [172] The PHQ-9 has also been found to have an acceptable internal consistency (α = .76).[173] In the present study, internal consistency across all three time points, as measured by Cronbach's alpha, was .87 (intervention group) and .94 (control group) for Visit 1, .90 (intervention group) and .97 (control group) for Visit 2, and .89 (intervention group) for Visit 3.

MOOD

Identical to a previous study,[17] the Profile of Moods States (POMS) questionnaire was used to assess affect, with the *depression-dejection* (13 items), *anger-hostility* (11 items), and *fatigue-inertia* (7 items) subscales used for the present study. In addition to evaluating each of these subscales individually, an overall mood score was created by summing the responses from the *depression-dejection*, *anger-hostility* and *fatigue-inertia* subscales, with higher values indicating worse affect. Notably, all participants provided complete data on the POMS assessment (i.e., no missing data).

The POMS survey has demonstrated adequate levels of internal consistency (α = .779-.926)[174] as well as criterion validity[175] and construct validity.[176] In the present study, internal

consistency, as measured by Cronbach's alpha, was .87, .79, and .77, respectively, for *depression-dejection, anger-hostility,* and *fatigue-inertia* during Visit 1 within the intervention group. Internal consistency was .94, .97 and .80 during Visit 1 within the control group. During Visit 2 and within the intervention group, internal consistency was .90, .89, and .83. Internal consistency for the control group during Visit 2 was .97, .96, and .94. Internal consistency for the intervention group during Visit 3 was .89, .86, and .70.

STATISTICAL ANALYSIS

Analysis was computed using SPSS software (version 22.0) and Stata software (version 12.0). Demographic differences between the two groups at baseline were compared via independent t tests for any continuous data (age, BMI, and mean MVPA) and via chi-square tests for any nominal data (education status, race/ethnicity and gender). To examine the effects of the sedentary behavior intervention each health outcome individually, split-plot 2 x 2 ANOVAs were computed in SPSS, running one with each of the 8 cognitive tests (as well as a combined overall cognition score), PSQI, SWLS, OASIS, PHQ-9, and POMS (including overall mood profile as well as the three sub-categories of the POMS survey) as the outcome variables. For each split-plot analysis, condition (sedentary intervention and control) served as the between-subject variable, and time (Visit 1 or Visit 2) served as the within-subject variable. We employed 2 x 2 ANOVAs due to the fact that the control group met for one less visit than the intervention group. All assumptions for this repeated measures ANOVA were checked and confirmed to not be violated. Following the split-plot ANOVAs, paired t-tests were conducted in Stata (version 12.0) to examine differences in the health outcomes between the second and third visit among those randomized into the intervention group. Notably, given the relatively small sample size, additional sensitivity analyses used the non-parametric t-test (Wilcoxon signed rank sum test) for

these analyses, but results were similar to the parametric paired-samples t-test (data not shown). Statistical significance was set at a two-tailed nominal α of .05.

CHAPTER 3: RESULTS

DESCRIPTIVE CHARACTERISTICS

Descriptive characteristics of the study sample are shown in Table 1. The included sample sizes of the two groups were n=26 in the intervention and n=13 in the control. The mean (SD) age of the intervention group was 21.69 (2.71) years and 38% of the participants were male compared to the control group, which had a mean age of 22.08 (2.75) years and 46% males. Demographic comparisons between the intervention and control groups revealed that there were no statistically significant differences among the groups with regards to age, gender, education status, BMI, race/ethnicity, accelerometer-derived baseline physical activity levels, or self-reported baseline physical activity levels (Table 1). Thus, we did not include these parameters as covariates in our analytic models.

STEP COUNTS

Table 2 reports the mean ± SD step counts for each group across the time periods. Step counts were significantly reduced ($P<.0001$) after the 1-week sedentary behavior-inducing intervention (steps/day decreased from 8475.13 ± 1902.96 to 5648.60 ± 1646.37). Then in the intervention group, after resuming normal physical activity (Week 2), steps (as measured via pedometry) were significantly higher than in Week 1 (9508.35 ± 2172.80 vs. 5648.60 ± 1646.37, $P<.0001$), but were not significantly different than Baseline steps (9508.35 ± 2172.80 vs. 8475.13 ± 1902.96, $P=.06$). These findings are expected and demonstrate that the intervention

group significantly reduced their steps from Baseline to Week 1, with physical activity returning to near baseline levels in Week 2. In the control group we also saw a significant increase in the number of steps from Baseline Week to Week 1 (8983.60 ± 3679.83 vs. 11,165.73 ± 3654.08, *P*=.03). Importantly, adequate pedometry wear time was reported for all participants with 14.25 hours/day of wear time during Week 1, the initial intervention week (N=39) and 14.93 hours/day during Week 2, the one-week of resumed physical activity following the intervention (n=26). Figure 2 contains a graphical display of the mean daily steps over time within each group.

COGNITIVE FUNCTION

Table 3 reports the mean cognitive function scores by time period (cognitive function scores from before the 1-week sedentary behavior intervention, directly following the intervention, and one week after resumption of normal physical activity levels in the intervention group). There was no significant time x group interaction effect for the overall composite cognitive function score (F value = .04, P > .84). Similarly, there were no statistically significant interaction effects for each of the individual cognitive function tests (P>0.05). Notably, however, the scores on Spatial Slider, which assesses the cognitive parameter of planning, significantly improved from Visit 2 to Visit 3 within the intervention group (38.00 vs. 47.70, P = .0063).

SLEEP

Table 4 reports the mean PSQI scores by time period (scores from before the 1-week sedentary behavior intervention, directly following the intervention, and one week after resumption of normal physical activity levels in the intervention group). The split-plot ANOVA demonstrated a statistically significant time x group interaction effect for PSQI scores (F=4.49,

P=.04). Mean ± SE PSQI scores were significantly higher (unfavorable change) after the one-week sedentary behavior-inducing intervention (17.62 ± 1.15) compared to scores from before the intervention (17.12 ± 1.15). Mean scores decreased (improved) below baseline following the one-week of resumption to normal physical activity (14.46 ± 1.06, *P*<.001). These findings suggest that, among this physically active sample, a 1-week sedentary-inducing intervention detrimentally influenced sleep, with PSQI scores returning back to baseline values after participants returned to their normal physical activity patterns. In addition to examining the effects of the sedentary behavior-inducing intervention on overall sleep quality (PSQI), sensitivity analyses examined the effects of the intervention on sleep duration and sleep latency. Although not shown in tabular format, there was no interaction effect for sleep latency (F=.09, *P*=.76) or sleep duration (F=.91, *P*=.35).

In the intervention group, there was an absolute (Visit 1 minus Baseline Visit) change of .50 for PSQI scores. The relative percentage change (Visit 1 minus Baseline Visit / Baseline Visit) was 3%. Effect size estimates were calculated to estimate strengths of associations (η^2_p; partial eta-squared). The η^2_p PSQI was 0.108, suggesting that 10.8% of the total variance for changes in sleep quality can be accounted for by group assignment. The η^2_p estimate was calculated using formula #13 in the reference by Lakens.[177] Due to the relatively small sample size of the present study, the *corrected* η^2_p (partial omega squared, ω^2_p) was calculated to be .081 denoted as formula #15 in Lankens.[177] Lastly, and although there were no differences in baseline physical activity (self-reported or accelerometer-assessed) between the intervention and control groups, it is plausible to suggest that baseline physical activity may moderate the intervention effects. For example, highly active (e.g., 300 min/week of MVPA) individuals may have a greater change in sleep quality following a sedentary intervention than minimally active (e.g.,

150 min/week) individuals. Sensitivity analysis did not, however, suggest such an effect, as determined by a visual plot (data not shown) of baseline physical activity and PSQI changes scores (Visit 2 minus Visit 1). Further, in a linear regression model, baseline accelerometer-determined steps was not associated with this PSQI change score in either the intervention (β = .040; 95% CI: -4.67-5.26; *P*=.43) or control group (β = .262; 95% CI: -10.09- 2.86; *P*=.205).

Interestingly, although the mean PSQI score significantly increased from Visit 1 to Visit 2, 38 percent of participants in the intervention group had decreased PSQI scores from Visit 1 to Visit 2. This is indicatory that the sedentary behavior inducing intervention had differing effects at the individual level. To determine if there were any moderating variables within the collected data, independent t tests were computed within the intervention group, comparing change in PSQI scores by age and by BMI. Chi squared tests were computed to compare change in PSQI scores by gender, race/ethnicity, and education status. The results of these tests indicated that age, BMI, race/ethnicity, and education status were all not significantly associated with change in PSQI scores (P >.05), however gender was significantly associated with change in PSQI scores (*P*=.007) within the intervention group. Specifically, 80 percent of males in the intervention group (n=8) had increased PSQI scores from Visit 1 to Visit 2, versus only 19 percent of females (n=3) who experienced increased PSQI scores across the same time period.

LIFE SATISFACTION

Table 5 reports the mean SWLS scores by time period (scores from before the 1-week sedentary behavior intervention, directly following the intervention, and one week after resumption of normal physical activity levels in the intervention group). The split-plot ANOVA demonstrated a statistically significant time x group interaction effect for SWLS scores

(F=32.75, *P*<.001). Mean ± SE SWLS scores were significantly lower after the one-week sedentary behavior-inducing intervention (19.04 ± 1.54) compared to scores from before the intervention (27.62 ± .92). Mean scores increased above baseline following the one-week of resumption to normal physical activity (28.16 ± 1.05, *P*<.001). These findings suggest that, among this physically active sample, a 1-week sedentary-inducing intervention detrimentally influenced quality of life, with SWLS scores returning back to baseline values after participants returned to their normal physical activity patterns. Mean results are demonstated graphically as compared with the control group in Figure 4.

In the intervention group, there was an absolute (Visit 1 minus Baseline Visit) change of -8.58 for SWLS scores. The relative percentage change (Visit 1 minus Baseline Visit / Baseline Visit) was -31.1%. Effect size estimates were calculated to estimate strengths of associations (η^2_p; partial eta-squared). The η^2_p SWLS was 0.469, suggesting that 46.9% of the total variance for changes in sleep quality can be accounted for by group assignment. The η^2_p estimate was calculated using formula #13 in the reference by Lakens.[177] Due to the relatively small sample size of the present study, the *corrected* η^2_p (partial omega squared, ω^2_p) was calculated to be .448 denoted as formula #15 in Lankens.[177] Lastly, and although there were no differences in baseline physical activity (self-reported or accelerometer-assessed) between the intervention and control groups, it is plausible to suggest that baseline physical activity may moderate the intervention effects. For example, highly active (e.g., 300 min/week of MVPA) individuals may have a greater change in sleep quality following a sedentary intervention than minimally active (e.g., 150 min/week) individuals. Sensitivity analysis did not, however, suggest such an effect, as determined by a visual plot (data not shown) of baseline physical activity and SWLS changes scores (Visit 2 minus Visit 1). Further, in a linear regression model, baseline accelerometer-

determined steps was not associated with this SWLS change score in either the intervention (β = .122; 95% CI: -21.25- -1.48; *P*=.276) or control group (β = -.317; 95% CI: -1.29- 17.21; *P*=.145).

ANXIETY

Table 6 reports the mean OASIS scores by time period (scores from before the 1-week sedentary behavior intervention, directly following the intervention, and one week after resumption of normal physical activity levels in the intervention group). The split-plot ANOVA demonstrated a statistically significant time x group interaction effect for OASIS scores (F(1,37)=11.13, *P*=.002). Mean ± SE OASIS scores were significantly higher after the one-week sedentary behavior-inducing intervention (5.35 ± .86) compared to scores from before the intervention (3.88 ± .69). Mean scores decreased below baseline following the one-week of resumption to normal physical activity (2.52 ± .56, *P*=.001). These findings suggest that, among this physically active sample, a 1-week sedentary-inducing intervention detrimentally influenced anxiety, with OASIS scores returning back to baseline values after participants returned to their normal physical activity patterns. Mean results are demonstrated graphically as compared with the control group in Figure 5.

In the intervention group, there was an absolute (Visit 1 minus Baseline Visit) change of 1.48 for OASIS scores. The relative percentage change (Visit 1 minus Baseline Visit / Baseline Visit) was 37.9%. Effect size estimates were calculated to estimate strengths of associations (η^2_p; partial eta-squared). The η^2_p OASIS was 0.231, suggesting that 23.1% of the total variance for changes in anxiety can be accounted for by group assignment. The η^2_p estimate was calculated using formula #13 in the reference by Lakens.[177] Due to the relatively small sample size of the present study, the *corrected* η^2_p (partial omega squared, ω^2_p) was calculated to be .206 denoted as

formula #15 in Lankens.[177] Lastly, and although there were no differences in baseline physical activity (self-reported or accelerometer-assessed) between the intervention and control groups, it is plausible to suggest that baseline physical activity may moderate the intervention effects. For example, highly active (e.g., 300 min/week of MVPA) individuals may have a greater change in anxiety following a sedentary intervention than minimally active (e.g., 150 min/week) individuals. Sensitivity analysis did not, however, suggest such an effect, as determined by a visual plot (data not shown) of baseline physical activity and OASIS changes scores (Visit 2 minus Visit 1). Further, in a linear regression model, baseline accelerometer-determined steps was not associated with this OASIS change score in either the intervention (β = .018; 95% CI: -3.35- -5.89; *P*=.47) or control group (β = .444; 95% CI: -8.65- -.57; *P*=.064).

Interestingly, although the mean OASIS score significantly increased from Visit 1 to Visit 2, 27 percent of participants in the intervention group had decreased OASIS scores from Visit 1 to Visit 2, and 23 percent of participants reported no change in anxiety from Visit 1 to Visit 2. This is indicatory that the sedentary behavior inducing intervention had differing effects at the individual level. To determine if there were any moderating variables within the collected data, independent t tests were computed within the intervention group, comparing change in OASIS scores by age and by BMI. Chi squared tests were computed to compare change in OASIS scores by gender, race/ethnicity, and education status. The results of these tests indicated that age, BMI, gender, race/ethnicity, and education status were all not significantly associated with change in OASIS scores (P >.05). Thus, it remains unknown as to what may be explaining these individual-level observations.

DEPRESSION/MOOD

Table 7 reports the mean PHQ-9 and POMS scores by time period (scores from before the 1-week sedentary behavior intervention, directly following the intervention, and one week after resumption of normal physical activity levels in the intervention group). With regard to the repeated measures MANOVA, there was a significant time x group interaction effect on the omnibus combined depression and mood variable (F= 6.62, *P*=.02). When examining PHQ-9 and POMS separately, there was a statistically significant time x group interaction effect for both the PHQ-9 scores (F=11.85, *P*=.001) and overall POMS scores (F= 10.03, *P*=.003). Mean ± SD PHQ-9 scores were significantly higher after the one-week sedentary behavior-inducing intervention (7.20 ± .97, *P*=.001) compared to scores from before the intervention (3.92 ± .79). Mean scores decreased below baseline following the one-week of resumption to normal physical activity (2.75 ± .55, *P*<.001). Similarly, mean ± SD POMS scores were significantly higher after the one-week sedentary behavior-inducing intervention (53.92 ± 3.65, *P*=.003) compared to scores from before the intervention (43.63 ± 2.08), with scores lowering below baseline (38.83 ± 1.87, *P*<.001) following the one-week resumption of normal physical activity. Notably, and as shown in Table 7, results for the individual subcomponents of the mood profile (depression/dejection, anger/hostility, fatigue/inertia) were similar to the results for overall mood. Collectively, these findings suggest that, among this physically active sample, a 1-week sedentary-inducing intervention detrimentally influenced depression and mood profile, with these mental health outcomes returning back to baseline values after participants returned to their normal physical activity patterns. PHQ-9 and POMS mean values by group over time are displayed graphically in Figures 6 and 7, respectively.

In the intervention group, there was a mean absolute (Visit 2 minus Visit 1) change of 3.28 for PHQ-9 scores and 10.29 for POMS scores. The relative percentage change (Baseline

Visit minus Visit 1 / Baseline Visit) was 83.67 for PHQ-9 scores and 23.58 for POMS scores. Effect size estimates were calculated to estimate strengths of associations (η^2_p; partial eta-squared). The η^2_p for PHQ-9 was 0.248, suggesting that 24.8% of the total variance for changes in depression symptomology can be accounted for by group assignment. The η^2_p for the overall POMS was .233, suggesting that 23.3% of the total variance for changes in mood symptomology can be accounted for by group assignment. The η^2_p estimate was calculated using formula #13 in the reference by Lakens.[177] Due to the relatively small sample size of the present study, the *corrected* η^2_p estimate was calculated (partial omega squared, ω^2_p), denoted as formula #15 in Lankens.[177] The ω^2_p was .217 and .188 respectively, for PHQ-9 and POMS. Lastly, and although there were no differences in baseline physical activity (self-reported or accelerometer-assessed) between the intervention and control groups, it is plausible to suggest that baseline physical activity may moderate the intervention effects. For example, highly active (e.g., 300 min/week of MVPA) individuals may have a greater change in mental health following a sedentary intervention than minimally active (e.g., 150 min/week) individuals. Sensitivity analysis did not, however, suggest such an effect, as determined by a visual plot (data not shown) of baseline physical activity and PHQ-9/POMS changes scores (Visit 2 minus Visit 1). Further, in a linear regression model, baseline accelerometer-determined steps was not associated with this PHQ-9 change score in either the intervention (β = .332; 95% CI: -6.00-5.42; *P*=.11) or control groups (β = .422; 95% CI: -9.64-.76; *P*=.17). Similarly, baseline accelerometer-determined steps was not associated with the POMS change score in either the intervention (β = .346; 95% CI: -18.72-11.07; *P*=.10) or control groups (β = .375; 95% CI: -13.39-.06; *P*=.23).

CHAPTER 4: DISCUSSION

COGNITIVE FUNCTION

Overall, despite the intervention group decreasing their steps and the control group increasing their steps over the 1-week period, we did not observe convincing statistically significant evidence of a group by time interaction effect. These findings contradict our initial hypothesis that increasing sedentary behavior would be inversely related to cognitive test scores (that test scores would decrease from Visit 1 to Visit 2 for the intervention group). These findings, however, suggest that a 1-week period of reduced physical activity does not detrimentally affect cognitive function, which may have encouraging implications for individuals going through a temporarily relapse in physical activity.

As the first study of this nature (experimentally increasing sedentary behavior while restricting physical activity), we could only speculate as to if one-week would be a sufficient amount of time to elicit unfavorable changes in cognition. While research has demonstrated that even one bout of exercise can yield improved cognitive test scores,[17] research has also suggested that there may be different physiological responses occurring molecularly within the body in response to sedentary behavior as opposed to physical activity.[27,178] Thus, increasing sedentary behavior may not necessarily result in the opposite effect of increasing physical activity.

Additionally, although all structured exercise was removed and step counts were significantly reduced for all participants in the intervention group, it is possible that there is a threshold of physical activity above which cognitive function will not be impacted significantly.

Our participants were still engaging in necessary ambulatory-type physical activity throughout the course of each day (i.e. walking to class). Perhaps the baseline daily activity levels that our participants achieved were enough to up-regulate BDNF, IGF-1, and other molecules that have been proposed to mediate the relationship between exercise and cognitive function[179-181]. Previous research has demonstrated that treadmill walking increases BDNF and its signal transductor receptor TrkB mRNA levels in rats.[182] Additionally, a recent human study evaluated the effects of a walking program on BDNF levels and observed that the walking program significantly increased BDNF via changes in the anterior hippocampal volume resulting from aerobic exercise.[183] Alternatively, it is possible that any potential changes in cognition resulting from manipulating sedentary behavior may result from a more chronic mechanism or adaptation that requires an elongated period of increased sedentary behavior greater than one week.

SLEEP

Experimental studies have evaluated the relationship between increased physical activity and sleep-related parameters and have found increased physical activity to have positive effects on the outcome of sleep when compared to control groups.[184-186] Emerging epidemiological research suggests an independent association of sedentary behavior on sleep. While bed rest studies on astronauts have contributed important knowledge to the understanding of the relationship between prolonged sedentary behavior and sleep related parameters (i.e. biological alertness rhythms, disturbed sleep, and daytime sleepiness),[166,187,188] no randomized controlled interventions (to our knowledge) have been conducted on sedentary behavior as it relates to sleep. Experimentally evaluating the effects of sedentary behavior on sleep is important because epidemiological data does not provide the strongest evidence of a potential cause-and-effect

relationship. Our results are in accordance with our original hypothesis that a sedentary behavior inducing intervention would have detrimental effects on sleep in active, young adults. As reported within the Results section, partial eta-squared and partial omega-squared values >.06 demonstrate that the magnitudes of our observed effects are medium.[189]

The relationship between physical activity and sleep is multifarious in nature and is lacking one profound mechanism to explain the association. Similarly, the relationship between sedentary behavior and sleep is complex and not yet fully understood (especially considering the scientific community is currently working to delineate the effects of sedentary behavior versus physical inactivity on various health-related outcomes). In accordance with the temperature down-regulation theory,[61] it is plausible to believe that by inducing sedentary behavior via eliminating all bouts of exercise, the down-regulation of core body temperature may have been impeded (due to a lesser initial increase in core body temperature that would normally have been present due to exercise), ultimately leading to worsened sleep quality. Adding to the plausibility of this mechanism, gender appeared to moderate the relationship between sleep quality and sedentary behavior. Our previously detailed chi squared analysis suggests that a one-week sedentary behavior inducing intervention had a more deleterious effect among males in the intervention group when compared to females. This is not surprising when considering temperature down-regulation to be a mediating mechanism between sleep quality and sedentary behavior due to known gender differences in thermoregulation.[190] Differences in male and female thermoregulation likely result from differing body surface to body mass ratios, greater subcutaneous fat levels in females, and differing rates of sex hormone release as a result of menstruation in females.[190]

Through previous research it is evident that gender may moderate the associations between numerous observed health-outcomes as they relate to an independent variable of interest.[191-193] Another health outcome gender has been proposed to moderate is the response to psychological stress.[194] Specifically, greater hypothalamic-pituitary-adrenal (HPA) activity and autonomic responses have been observed in adult men when compared to adult women.[195,196]Additionally, hormonal differences between males and females have been shown to have marked effects on stress response, with estrogen being suggested as a buffer for HPA arousal.[197] Collectively, these differences have been identified as a "fight-or-flight" response, believed to be more naturally occurring in males, and a "tend-and-befriend" response, which is theorized to occur more often with women.[198] The fight-or-flight stress response has been associated with increased focus and alertness.[199,200] The tend-and-befriend response is believed to involve activation of the reward system under stress and ultimately the down-regulation the fight-or-flight response.[198] It is possible that our results were partially moderated by gender (with males demonstrating a more deleterious response to induced sedentary behavior) due to the psychological stress response playing a meditational role between sedentary behavior and sleep. Perhaps females were able to down-regulate their fight-or-flight response and mitigate the increased arousal and alertness, both of which may have an effect on one's sleep quality. It is likely a combination of factors are working in concert to explain the observed effects of our sedentary behavior intervention, and future research should be done in this area to better understand these underlying mechanisms.

LIFE SATISFACTION

Our results are in accordance with our original hypothesis that a sedentary behavior inducing intervention would have detrimental effects on life satisfaction in active, young adults. As reported within the Results section, partial eta-squared and partial omega-squared values >.14 demonstrate that the magnitudes of our observed effects are large.[189] The short duration of our intervention lends some support to the idea of weekly sedentary behavior having a bottom-up influence on life satisfaction and subjective well being.

The relationship between physical activity and life satisfaction lacks one profound mechanism to explain the association. Due to life satisfaction being a global health outcome (especially as assessed via the broad statements in the SWLS) where each individual consciously chooses which life aspects to weigh and how much influence they feel each aspect has on their overall satisfaction,[169] it is plausible to believe that there are numerous mechanisms that synergistically mediate the physical activity-life satisfaction relationship. As mentioned previously, affect, physical self-worth, self-efficacy, and mental health have all been suggested to mediate this relationship.[80-82] The emerging adulthood years (i.e., 18-25; ages included in our study) are characterized by increased negative self-evaluations and affective lability when compared to later life stages (e.g., midlife or older adulthood).[201,202] This may put the age group in our study at an increased risk for some of the negative mediating mechanisms (mentioned previously) that ultimately may have led to their decreased satisfaction with life scores. Given that physical activity has been positively associated with improved mental health (i.e. decreased depression and anxiety symptomology, improved mood state),[100,114,115,117] it is plausible to speculate that removing physical activity would have the opposite effect on these variables (and thus an indirect negative influence on life satisfaction).

Additional theories that may help explain the resulting decreases in life satisfaction are the activity theory and the need theory. The *activity theory* postulates that life satisfaction is determined by both the frequency of engagement in various activities as well as the degree of intimacy associated with these activities.[203] While the activity theory was developed in the context of explaining processes related to aging in the elderly population, it has also been applied to the general adult population with regards to life satisfaction.[204] Participants in our study were required to reduce the frequency of physical activity by eliminating exercise for an entire week. For any individuals in our study who potentially believe their sense of self-worth or general well-being is intimately associated with their weekly exercise routines and personal fitness levels, this reduction likely was especially difficult and detrimental to their life satisfaction. The *need theory* states that life satisfaction is mainly regulated by an individual's ability to satisfy his or her biological and psychological needs.[205] It is possible that participants in our study have needs which they use physical activity to help meet (e.g., the use of physical activity to attenuate existing symptoms of anxiety or attention deficit disorders, the participation in physical activity to maintain or hypertrophy existing muscle mass, or the participation in exercise to increase one's self-esteem). By removing physical activity, it is possible that these individual needs were not met as well, resulting in decreased life satisfaction.

ANXIETY

Physical activity is inversely associated with mental health and emerging epidemiological research suggests an independent association of sedentary behavior on mental health. However, experimentally evaluating the effects of sedentary behavior on anxiety is important because epidemiological data does not provide the strongest evidence of a potential cause-and-effect

relationship. Our results are in accordance with our original hypothesis that a sedentary behavior inducing intervention would have detrimental effects on anxiety in active, young adults. As reported within the Results section, partial eta-squared and partial omega-squared values >.14 demonstrate that the magnitudes of our observed effects are large.[189]

A number or physiological and psychological mechanisms have been proposed to explain the relationship between physical activity and anxiety,[98] several of which may help speculate about the sedentary behavior-depression relationship. Two physiological mechanisms that may have influenced the observed results are the thermogenic model and the visceral afferent feedback model. The *thermogenic model* postulates that elevation in body temperature may produce therapeutic effects. As it pertains to exercise and anxiety, core body temperature elevations, proportional to the intensity of exercise, are believed to produce the anxiolytic effects. A meta-analysis[98] by Petruzzello et al. did not demonstrate support of this model, however the authors note a common limitation of temperature-anxiety studies being that they only took indirect measurements of temperature. The authors suggested that future research regarding the influence of temperature on anxiety is needed.[98] Another physiological mechanism discussed within this meta-analysis is the *visceral-afferent feedback model*, which suggests that working muscles transmit afferent impulses to brainstem collateral neurons, resulting in increased stimulation of the ascending reticular activating system. It is believed that this cortical excitation from exercise may reach a point at which an inhibitory mechanism is triggered, ultimately reducing the somatic afferent stimulation. The decrease in cortical excitation is believed to have an effect lasting significantly longer than the original stimulus (i.e., there is a prolonged poststimulus effect). Exercise studies evaluating EEG activity have contributed some

support to this theory (an increase in alpha activity following exercise is believed to signify a relaxation response and reduction in anxiety).[206,207]

Bahrke and Morgan's (1978) *distraction hypothesis*[208] is a psychological mechanism, which suggests that distracting oneself from stressful stimuli (e.g., exercising to take time off from one's daily routine) can have anxiety-reducing effects. By removing physical activity, participants may have limited the number of distractions from their daily routine, thus resulting in higher levels of anxiety. Further, if meditation or mindfulness distraction therapies were present within the individual's exercise routine (e.g., if they practiced yoga as a part of their workout), eliminating physical activity may have had an even further deleterious effect on anxiety. It would be intriguing to evaluate the "distractive capabilities" of different modalities of exercise training (e.g., high intensity interval training, steady-state aerobic exercise, contact kickboxing, yoga, etc.) in terms of alleviating anxiety symptoms.

Though not recorded as a part of our data collection process, a prominent anecdotal theme present among the majority of participants was a fear of the negative consequences that may ensue from taking one week off of exercise. Common fears discussed by participants were muscle atrophy (expressed mainly among the men participants) and weight gain (present mainly among the female participants). Fear and anxiety are interrelated, both resulting in negative feelings and strong bodily manifestations. Fear is suggested to differ from anxiety, however, in that it typically is the result of an identifiable stimulus, whereas anxiety is often more anticipatory in nature (i.e., prestimulus).[209] Anxiety, as explained by Epstein (1972), is the result of failed coping attempts (e.g., due to uncontrollability of the situation) or unresolved fear.[210] Based off of this explanation of the fear-anxiety relationship as well as the anecdotal evidence for the presence of fear, we hypothesize that fear may have partially mediated the relationship

between sedentary behavior and the observed increases in anxiety scores. Participants with a fear of losing muscle or gaining weight (especially those who may be physically active specifically to avoid these outcomes) may have seen the one-week sedentary behavior intervention as an uncontrollable situation in which there was nothing they could do to escape or avoid (even knowing that the physical activity limitations were only temporary and exercise could be resumed at the end of one week), which ultimately may have induced anxiety.

It is also possible that exercisers with a higher exercise identity (i.e., an individual's level of identification of exercise as an integral part of the concept of self)[211] were more negatively affected by the sedentary behavior intervention (i.e., exercise identity moderated the sedentary behavior-anxiety relationship).[212] Exercise, as well as the social interactions that individuals engage in during exercise, are believed to be essential components of the exerciser's role identity (i.e., their definition of self with regards the social roles they hold).[213] Participants with a high level of exercise identity, then, may have experienced a diminished sense of role identity (via less social interactions in the gym and displacing actual exercise), which may have increased their anxiety. It would be of interest to include a fear and exercise identity assessment with future sedentary behavior-anxiety related studies in attempt to more clearly define the role of each construct in this relationship.

DEPRESSION/MOOD

Physical activity is inversely associated with mental health. Emerging epidemiological research suggests an independent association of sedentary behavior on mental health. No randomized controlled interventions have been conducted on sedentary behavior as it relates to mental health outcomes. Experimentally evaluating the effects of sedentary behavior on mental

health is important because epidemiological data does not provide the strongest evidence of a potential cause-and-effect relationship. Our results are in accordance with our original hypothesis that a sedentary behavior inducing intervention would have detrimental effects on depression and mood in active, young adults. As reported within the Results section, partial eta-squared and partial omega-squared values were greater than .14 for both depression and mood state. These effect size estimates may be viewed as relatively large. For example, Cohen[189] has provided benchmarks defined as small ($\eta^2 = 0.01$), medium ($\eta^2 = 0.06$) and large ($\eta^2 = 0.14$). However, these benchmarks were developed for comparisons between unrestricted populations and using these benchmarks for repeated measures is not consistent with the considerations in which these thresholds were based. As such, interpretation of these effect size estimates may be better considered in the light of other studies. When compared to other physical activity/sedentary behavior and depression interventions,[98,214,215] our observed effect sizes are consistent with or greater than other related interventions.

This study recruited active individuals who were accustomed to exercising multiple times each week. It is possible that by eliminating any planned physical activity and limiting steps for an entire week, these individuals had minimal perceived control over mitigating daily stress, which speculatively they may have controlled via exercise. This daily stress (possibly experienced in part as a result of removing exercise) may have induced negative mood symptomology (previous studies on the psychological impact of daily stress have demonstrated the ability to negatively alter mood).[216-218] Notably, we did not measure the degree to which the length of our intervention may have affected depression symptomology. It is conceivable that depression symptoms may have increased to a greater extent if the duration of the sedentary behavior intervention was longer (e.g., more than 1-week), or if participants were blinded to the

length of the sedentary behavior intervention (i.e., further minimizing their perceived control and ability to manage stress and any potential negatively associated mood symptomology). However, our observed statistically significant increase in depression scores as a result of the 1-week intervention suggest that in an active young adult population, one week is a sufficient length to observe large, maladaptive effects on depression (even with individuals knowing the intervention is temporary and normal physical activity may be resumed after a week).

For participants who may typically exercise with friends or in a group exercise class, it is possible that eliminating physical activity reduced the amount of positive social-supporting interactions they had throughout the week. Previous research has demonstrated strong associations between social support and mental health,[219] thus having potentially less social support or motivation in the form of an exercise partner during the intervention may have had negative implications for overall mood and depression symptoms. It would be informative, in future research, to evaluate the effects of a sedentary behavior intervention on individuals who engage in exercise alone versus those who typically participate in some form of group exercise, and to evaluate whether social support and perceived behavioral and psychological control play a mediating role. Additionally, multiple items within both the PHQ-9 and the POMS ask questions related to sleep or sleepiness. Sleep disorders such as insomnia, hypersomnia, and apnea have all been linked with depression[220-222] and symptoms of sleep disorders often overlap depression symptoms in what is typically regarded as a bi-directional relationship.[223] Knowing that sleep and depression/mood symptoms can be interrelated, it is possible that eliminating exercise could have had a negative effect on the sleep quality of participants. According to the temperature down-regulation theory, exercise has body-heating effects that can help to prime an essential down-regulation of core body temperature that is required for optimal sleep.[130] It is also possible

that some of the participants in this study utilize exercise as an outlet to help manage the stress-levels of daily life. Removing this outlet, then, may have resulted in higher levels of stress and increased depression and mood symptoms (distraction hypothesis).[121] It is also possible that the observed changes in mood mediated the relationship between the sedentary behavior intervention and decreased depression scores, acting as a mechanism through which the intervention detrimentally influenced depression symptoms.

The present findings suggest that a one-week sedentary behavior inducing intervention has a statistically significant, relatively large negative effect on mood and depression symptoms. While previous work[224] has evaluated the effects of prolonged bedrest on depression and mood state and has observed increases in depression levels, this is the first randomized controlled intervention (to our knowledge) to experimentally increase sedentary behavior and evaluate these outcome measures in a free-living setting. Coupled with known information regarding the benefits of physical activity on these outcomes, the findings from this study provide evidence for a cause-and-effect relationship between physical activity and the mental health outcomes of depression and mood, particularly among active individuals. Our observation that the intervention group (who decreased their physical activity) had unfavorable changes in depression and mood, coupled with our observation that the control group (whom inadvertently increased their physical activity) had favorable changes in depression and mood, supports this cause-and-effect relationship and highlights the powerful role of physical activity and sedentary behavior on mental health. This research may have particular implications for active athletes or general daily exercisers who sustain an injury that prevents them from obtaining their preferred method of exercise (e.g., a runner not being able to run due to an injured Achilles tendon). Hopefully, knowing the maladaptive effects of increasing sedentary behavior will encourage injured, active

individuals to consider finding another outlet of physical activity to attenuate the effects of any depression or negative mood symptoms they may already be experiencing from sustaining a serious, debilitating injury.

This research may also have important clinical implications for the general population. Our findings underscore the importance of clinicians promoting physical activity to inactive patients as well as the maintenance of physical activity among active patients. Additionally, clinicians are encouraged to assist their patients in developing relapse prevention plans or roadmaps to avoid the depression and mood symptomology associated with lapses in physical activity. As identified by Marlatt and Gordon's relapse prevention model,[225] recommended relapse prevention techniques include identifying the patient's underlying cognitive behavioral weaknesses that may influence their risk of relapse, working with the patient to develop coping strategies for challenges that may threaten their ability to remain physically active, and having the patient set several small and measurable goals that will be easy for the patient to assess. Although this model is typically applied to the addictive behaviors of smoking or alcohol consumption, techniques from the model could be modified to form a "sedentary behavior relapse prevention model."

Encouragingly, depression and mood improved post-intervention, after one-week of resumed regular physical activity. This has implications for individuals who may experience a temporary lapse in physical activity. Whether this individual has a well-established active lifestyle or has recently adopted a more active lifestyle including physical activity, it could prove beneficial to re-evaluate one's perceptions of "lapsing." Clinicians should encourage patients who do not exercise for several days in a row (or up to a week) to not view this lapse in physical activity as a failure that will inevitably lead to a complete relapse (i.e., assuming a sedentary

lifestyle and not continuing to exercise in the future). Rather, physicians can encourage patients to view a temporary physical activity lapse as a key learning opportunity that results from an interaction of coping and situational determinants (both of which may be modifiable in the future).[225] Knowing that it is possible to reverse the depression/mood symptoms (in this active sample) associated with physical inactivity could potentially serve as a motivator for individuals experiencing depression that is having a negative impact on their daily life.

Future studies should continue investigating the relationship between sedentary behavior and mental health in efforts to better understand the effects of sedentary behavior on mental health outcomes and how the underlying mechanisms mediating this relationship may be similar to or may differ from those that mediate the relationship between physical activity and mental health. Notably, others investigating the effects of sedentary behavior on depression share this opinion.[226,227] Future studies looking to build off of these observations may consider recruiting only highly active individuals (for instance, individuals who exercise 4-5 days a week and accumulate at least 300 min/week of MVPA) as compared to those who just meet MVPA guidelines, as this may help to confirm our findings which did not suggest a moderating role of physical activity on changes in mood and depression. Acquiring a larger sample size may also be a desirable goal for future studies. A larger sample may allow for greater heterogeneity in sample demographic characteristics, which thus may enable greater generalizability across other demographic characteristics (i.e., race-ethnicity, age, etc.). Having a mixed study design that employs some form of qualitative assessment of the sedentary intervention (i.e., focus groups or interviews with participants asking them how the sedentary behavior intervention affected them) may help to elaborate further on the mechanisms driving the relationship between sedentary behavior and mental health outcomes. Additionally, it would be useful for future research to

evaluate the effects of different durations of sedentary behavior interventions (e.g., greater than 1-week) as well as investigate how the participants' knowledge of the intervention duration may influence their depression and mood symptomology (e.g., would depression and mood scores be more detrimentally influenced if participants knew they had to be sedentary for a much longer period of time?).

LIMITATIONS/STRENGTHS

A limitation of this study is the utilization of a nonprobability convenience-based sampling approach likely resulting in some degree of selection and sampling biases. Sampling bias can compromise the external validity of a study by reducing the ability of the study to be generalized to the rest of the population, whereas selection bias can lead to lower levels of internal validity for any observed differences or similarities within the samples. Additionally, we employed a modified 1-week version of the PHQ-9 depression survey (which typically requests responses based on a 2-week recall) and the PSQI sleep survey (which typically requests responses based on a 1-month recall). Notably, the modified version of the both surveys used in this study demonstrated evidence of both construct validity and internal consistency, and ultimately did not influence the ability to observe meaningful changes in depression or sleep symptomology across time points within the intervention group. It is also conceivable that recall bias may have actually been less present than it would have been with use of the original assessment tools, due to a shorter period of time that participants were required to recall their depression symptoms and sleep-quality. Another limitation of our study is that we used accelerometer derived step counts as our baseline step counts and pedometer derived step counts were used for the subsequent visit(s). However, we applied a correction factor based off a well-

known comparison study by Tudor-Locke et al. to take into consideration the fact that accelerometers tend to generate higher step counts than pedometers.[143] The reduction in average steps/day from baseline to Visit 1 in the intervention group remained statistically significant with the p-value changing from $P<.001$ to $P=.0005$. Additionally, the control group actually significantly increased their average daily steps, both without and with the applied correction factor (P-value changed from $P=.03$ to $P=.001$). The step count comparisons are displayed in Table 3. The pedometer used in the 2002 Tudor-Locke et al. study[143] was the Yamax Digi-Walk 200 pedometer (i.e., the same pedometer used in our current study). The GT9X accelerometer by ActiGraph is the newest model of the 7164 ActiGraph accelerometer used for comparison in the 2002 study. In 2010, a study compared the 7164 accelerometer with three versions of the ActiGraph GT1M accelerometer, finding no statistically significant differences in their outputs.[228] In 2012, a comparison study found good agreement between the ActiGraph GT1M and the ActiGraph GT3X, which is the most recent model prior to the current ActiGraph model, the GT9X.[229] Comparison studies for the GT3X and the GT9X have not yet (to our knowledge) been published. However, our unadjusted and adjusted step count findings suggest that the different instrument (accelerometer to measure steps vs. pedometer to measure steps) used at baseline compared to the subsequent weeks did not appreciably influence our experimental findings. Major strengths of this study include the utilization of an experimental design to manipulate sedentary behavior and the use of both objective and subjective measures of physical activity to confirm study inclusion criteria.

CHAPTER 5: CONCLUSION

COGNITIVE FUNCTION

In conclusion, the results of our study raises a number of questions for future inquiry and demonstrates a need for further research to examine the effects of sedentary behavior on cognitive brain function. The present findings suggest that perhaps the effects of sedentary behavior take longer than one week to have noticeable deleterious effects on one's cognitive functioning, at least in an active population. This has implications for individuals who may suffer from an injury, illness, or exceptionally busy schedule that inhibits them from exercising for several consecutive days but still need to have optimal levels of cognitive functioning (i.e., students who skip their workouts during all of finals week to have more time to study for exams). The experimental procedure of this study may help to set the stage for future research aiming to experimentally manipulate sedentary behavior as it relates to cognition or other health outcomes (i.e., to investigate how long it takes to observe statistically significant changes in cognition as a result of chronic sedentary behavior). It is important to continue investigating sedentary behavior experimentally if we hope to ascertain a clearer picture of any potential cause-and-effect relationships between sedentary behavior cognition.

SLEEP

In conclusion, the present findings suggest that a one-week sedentary behavior inducing intervention has a statistically significant, negative effect on overall sleep quality. As mentioned

previously, this is the first randomized controlled intervention (to our knowledge) to experimentally increase sedentary behavior and evaluate these outcome measures in a free-living setting. Coupled with known information regarding the benefits of physical activity on sleep, the findings from this study provide evidence for a cause-and-effect relationship between sedentary behavior and sleep quality in active individuals. Our observation that the intervention group (who decreased their physical activity) had unfavorable changes sleep, coupled with our observation that the control group (whom inadvertently increased their physical activity) had favorable changes in sleep, supports this cause-and-effect relationship and highlights the powerful role of physical activity and sedentary behavior on this health outcome. These findings underscore the importance of maintaining a normal routine of physical activity to avoid impairments in sleep quality, especially among a young adult male population. These findings may apply to athletes or exercisers who sustain a serious injury and are unable to engage in their primary mode of exercise, offering encouragement to find alternate methods of obtaining physical activity (i.e., cross-training) to avoid prolonged periods of sedentary behavior. Additionally, clinicians may recommend that sedentary patients suffering from sleep-related issues supplement any prescribed treatment with a physical activity program to potentially better attenuate their sleep-related problems.

Future studies looking to build off of these observations may consider recruiting only highly active individuals (for instance, individuals who exercise 4-5 days a week and accumulate at least 300 min/week of MVPA) as compared to those who solely meet minimum MVPA guidelines, as this may help to confirm our findings which did not suggest a moderating role of physical activity on changes in sleep quality. Acquiring a larger sample size may also be a desirable goal for future studies. A larger sample may allow for greater heterogeneity in sample

demographic characteristics, which thus may enable greater generalizability across other demographic characteristics (i.e., race-ethnicity, age, etc.) as well as a further exploration of the potential moderating role of gender as it pertains to our observed sedentary behavior-sleep relationship. Additionally, having a mixed study design that employs some form of qualitative assessment of the sedentary intervention (i.e., focus groups or interviews with participants asking them how the sedentary behavior intervention affected them) may help to elaborate further on the mechanisms driving the relationship between sedentary behavior and sleep.

LIFE SATISFACTION

The present findings suggest that a one-week sedentary behavior inducing intervention has a statistically significant, negative effect on life satisfaction. As mentioned previously, this is the first randomized controlled intervention (to our knowledge) to experimentally increase sedentary behavior and evaluate this outcome in a free-living setting. Coupled with known information regarding the benefits of physical activity on life satisfaction, the findings from this study provide evidence for a cause-and-effect relationship between sedentary behavior and life satisfaction in active individuals. Our observation that the intervention group (who decreased their physical activity) had unfavorable changes in life satisfaction, coupled with our observation that the control group (whom inadvertently increased their physical activity) had favorable changes in life satisfaction, supports this cause-and-effect relationship and highlights the powerful role of physical activity and sedentary behavior on this health outcome. These findings underscore the importance of maintaining a normal routine of physical activity to avoid decreases in life satisfaction in the young adult population. Clinicians and counselors who work with sedentary patients suffering from low levels of subjective well being and poor life

satisfaction may recommend beginning a physical activity routine to improve these negative self-rated health outcomes.

Future studies looking to build off of these observations may consider recruiting only highly active individuals (for instance, individuals who exercise 4-5 days a week and accumulate at least 300 min/week of MVPA) as compared to those who solely meet minimum MVPA guidelines, as this may help to confirm our findings which did not suggest a moderating role of physical activity on changes in life satisfaction. Additionally, employing a mixed study design that utilizes some form of qualitative assessment of the sedentary intervention (e.g., focus groups or interviews with participants asking them how the sedentary behavior intervention affected them) may help to elaborate further on the potential mechanisms that mediate the relationship between sedentary behavior and life satisfaction. This qualitative assessment could also address individual's definitions of life satisfaction; it would have been interesting to evaluate and compare how participants in the present study would have ranked physical activity participation alongside other reported components of their overall life satisfaction. Future studies may also look to explore the influence of daily sedentary behavior in active individuals to further understand potential bottom-up influences of this health behavior by adding a daily measure of SLWL within the week-long intervention.

ANXIETY

The present findings suggest that a one-week sedentary behavior inducing intervention has a statistically significant, meaningful negative effect on anxiety. As mentioned previously, this is the first randomized controlled intervention (to our knowledge) to experimentally increase sedentary behavior and evaluate this outcome (anxiety). Coupled with known information

regarding the benefits of physical activity on anxiety, the findings from this study provide evidence for a cause-and-effect relationship between sedentary behavior and anxiety in active individuals. Our observation that the intervention group (who decreased their physical activity) had unfavorable changes in anxiety, coupled with our observation that the control group (whom inadvertently increased their physical activity) had favorable changes in anxiety, supports this cause-and-effect relationship and highlights the powerful role of physical activity and sedentary behavior on anxiety symptomology. These findings underscore the importance of maintaining a normal routine of physical activity to avoid increases in anxiety among the young adult population. Clinicians and therapists or counselors who work with sedentary patients suffering from anxiety disorders or anxiety-associated symptomology may recommend beginning a physical activity program to attenuate these symptoms.

As mentioned previously, future studies looking to build off of these observations may consider employing a more comprehensive survey that assesses additional psychological variables, such as fear and exercise identity, as well as collecting information at baseline regarding the types of physical activity that make up one's exercise routine. In addition to including more anxiety-related constructs within the survey assessment, future studies could also include a qualitative assessment (e.g. focus groups or interviews) to further explore potential mediating mechanisms.

DEPRESSION/MOOD

In conclusion, this randomized controlled intervention is novel due to manipulating free-living sedentary behavior while assessing mood state and depression levels as outcomes. A one-week sedentary behavior-inducing intervention resulted in significant, relatively large negative

changes in both depression and mood state, with depression and mood returning back to normal levels after one-week of resumed physical activity levels. Now is a critical time to be investigating the effects of sedentary behavior, as understanding any potential casual relationships between sedentary behavior and health outcomes necessitates a multitude of future longitudinal and experimental study designs where sedentary behavior is specifically manipulated. In that sense, we believe this study is an important contribution in the exploration of this topic, especially given the psychological outcome variables evaluated in contrast to the physiological, cardiometabolic risk factors often evaluated in sedentary behavior research.

COMPREHENSIVE STUDY CONCLUSION

A one-week sedentary behavior inducing intervention elicited significant, moderate-to-large, negative effects on sleep quality, life satisfaction, anxiety, depression, and mood in an active young adult population. Encouragingly, all health outcomes improved after one-week of resuming normal physical activity levels. Experimentally manipulating activity levels to specifically increase sedentary behavior while minimizing moderate-to-vigorous physical activity and light physical activity is a novel research design that has not been previously implemented (to our knowledge). We believe this study is an important response to the scientific community's call for more experimental, intervention-based sedentary behavior research to build upon existing knowledge acquired mainly through observational research. Future research should continue to explore the physical activity (including making clearer distinctions between the effects of light, moderate, and vigorous physical activity) and sedentary behavior relationship in order to better guide public health recommendations and ultimately improve the nation's health status.

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LIST OF APPENDICIES

APPENDIX I: DEMOGRAPHIC INFORMATION

Table 1. Demographic characteristics of the analyzed sample (N = 39)

Demographic Variable	Intervention Group (n=26)		Control Group (n=13)		P-Value†
	n	Mean ± SD/%	n	Mean ± SD/%	
Gender, %					*P*= .65
Male	10	38%	6	46%	
Female	16	62%	7	54%	
Race-Ethnicity, %					*P*= .65
Mexican American	1	4%	0	0%	
Non-Hispanic White	16	61.5%	9	69%	
Non-Hispanic Black	7	27%	4	31%	
Other/Multi-Race	2	7.5%	0	0%	
Education Status, %					*P*= .31
Undergraduate	20	77%	8	62%	
Graduate	6	23%	5	38%	
BMI, kg/m^2	26	25.35 (6.82)	13	26.13 (3.67)	*P*= .71
Age, years	26	21.69 (2.71)	13	22.08 (2.75)	*P*= .69
Baseline MVPA, min/week (IPAQ-SF)	26	423.85 (206.03)	13	577.50 (293.21)	*P* =.07
Baseline MVPA, min/week (Accelerometer)	26	317.65 (111.56)	13	362.29 (145.24)	*P*= .30

* BMI = body mass index; MVPA = moderate-to-vigorous physical activity; IPAQ-SF = International Physical Activity Questionnaire, Short-Form

* †An independent sample student t-test was used to calculate differences for the continuous variables across the two groups (intervention vs. control). For the categorical variables, a chi square analysis was used to calculate differences for the categorical variables across the two groups.

APPENDIX II: STEP COUNTS

Table 2: Comparison of average daily step counts between groups and across time periods (N=39)

Group	Baseline (Mean ± SD)	Week 1 (Mean ± SD)	Week 2 (Mean ± SD)	P- value (Baseline → Week 1)	P-value (Week 1→2)	P-value (Baseline → Week 2)
Intervention	8475.13 (1902.96) †	5648.60 (1646.37)	9508.35 (2172.80)	$P < .0001$	$P < .0001$	$P = .06$
Control	8983.60 (3679.83) ‡	11,165.73 (3654.08)	-	$P = .03$	-	-

[*] Baseline step counts were multiplied by 0.839 to calculate the adjusted baseline values (assuming a 16% over-estimation of pedometer step counts was present in baseline accelerometer step counts) in accordance with previous research findings.[143]

[*] † The adjusted baseline steps for the intervention group = 7110.63 (1546.59), with the associated p-values of P = .0005 (Baseline → Week 1), P <.0001 (Week 1 → Week 2), and P=.001 (Baseline → Week 2)

[*] ‡ The adjusted baseline steps for the control group = 7537.25 (3087.37)

[*] Standard deviations are listed following each average daily step count

APPENDIX III: COGNITIVE FUNCTION

Table 3: Mean (SD) changes in cognitive scores across the study time period (N=39)

Group/Cognitive Test	**Visit 1 (Baseline)**	**Visit 2 (Week 1)**	**Visit 3 (Week 2)**	**F-value**	P-value (Visit 1→2)	P-value (Visit 2 →3)
Memory						
Spatial Span				.04	*P* = .85	*P* = .79
Intervention	5.61 (1.12)	5.65 (.83)	5.61 (.94)			
Control	5.70 (.82)	5.80 (1.03)	-			
Paired Associates				.01	*P* = .92	*P* = .87
Intervention	4.74 (1.10)	5.09 (1.16)	5.13 (.92)			
Control	5.10 (1.29)	5.50 (1.08)	-			
Reasoning						
Grammatical Reasoning				.87	*P* = .36	*P* = .26
Intervention	13.39 (5.30)	15.26 (5.56)	16.26 (6.63)			
Control	14.50 (4.45)	5.50 (1.08)	-			
Odd One Out				3.22	*P* = .08	*P* = .95
Intervention	9.30 (3.17)	9.96 (2.88)	10.00 (3.68)			
Control	7.60 (5.36)	11.30 (3.09)	-			
Concentration						
Feature Match				.091	*P* = .76	*P* = .67
Intervention	112.43 (24.41)	121.52 (22.73)	124.87 (29.21)			
Control	121.10 (33.39)	126.60 (27.47)	-			
Polygon				.22	*P* = .64	*P* = .82
Intervention	36.70 (16.94)	47.04 (23.89)	45.96 (19.25)			
Control	48.40 (29.36)	54.30 (20.87)	-			
Planning						
Spatial Search				2.45	*P* = .13	*P* = .76
Intervention	6.87 (2.60)	7.00 (1.98)	6.87 (1.87)			
Control	7.30 (2.63)	8.60 (2.63)	-			
Spatial Slider				.04	*P* = .84	*P* = .0063
Intervention	35.87 (17.95)	38.00 (15.47)	47.70 (19.60)			
Control	42.10 (16.40)	45.70 (23.53)	-			
Composite Score				.04	*P* = .84	*P* = .18
Intervention	224.91 (40.63)	249.52 (42.21)	262.39 (43.76)			
Control	251.80 (40.94)	272.60 (43.04)	-			

* The α value was set at .05 for all tests
* Standard deviations are reported in parentheses
* p-values for Visit 1→2 were calculated using repeated measures ANOVA and p-values for Visit 2→3 were calculated using paired t-tests
* The reported p-values and F-values from the repeated measures ANOVA are from the group*time interaction
* The Composite Score was calculated by summing the 8 individual tests

APPENDIX IV: SLEEP

Table 4: Mean changes in PSQI scores across the study time period (N=39)

PSQI Scores/Group	Visit 1 (Week 1)	Visit 2 (Week 2)	Visit 3 (Week 3)	F-value	P-value (Visit 1→2)	P-value (Visit 2 →3)
				4.49	*P*=.04	*P<.001*
Intervention	17.12 (1.15)	17.62 (1.15)	14.46 (1.06)			
Control	16.58 (1.86)	14.85 (1.68)	-			

* Standard error for each mean score are reported after the mean in parenthesis
* P-values for Visit 1→2 were calculated using repeated measures ANOVA and p-values for Visit 2→3 were calculated using paired t-tests
* The reported p-value and F-value are from the split-plot ANOVA are from the group*time interaction
* PSQI = Pittsburgh Quality Sleep Index

APPENDIX V: LIFE SATISFACTION

Table 5: Mean changes in SWLS scores across the study time period (N=39)

SWLS Scores/Group	Visit 1 (Week 1)	Visit 2 (Week 2)	Visit 3 (Week 3)	F-value	P-value (Visit 1→2)	P-value (Visit 2 →3)
				32.75		
Intervention	27.62 (.92)	19.04 (1.54)	28.16 (1.05)		*P*<.001	*P*<.001
Control	24.85 (2.18)	28.46 (1.30)	-			

* Standard error for each mean score are reported after the mean in parenthesis
* P-values for Visit 1→2 were calculated using repeated measures ANOVA and p-values for Visit 2→3 were calculated using paired t-tests
* The reported p-value and F-value are from the split-plot ANOVA are from the group*time interaction
* SWLS = Satisfaction With Life Scale

APPENDIX VI: ANXIETY

Table 6: Mean changes in OASIS scores across the study time period (N=39)

OASIS Scores/Group	Visit 1 (Week 1)	Visit 2 (Week 2)	Visit 3 (Week 3)	F-value	P-value (Visit 1→2)	P-value (Visit 2 →3)
Intervention	3.88 (.69)	5.35 (.86)	2.52 (.56)	11.13	*P*=.002	*P*=.001
Control	5.18 (1.75)	3.27 (1.40)	-			

* Standard error for each mean score are reported after the mean in parenthesis
* P-values for Visit 1→2 were calculated using repeated measures ANOVA and p-values for Visit 2→3 were calculated using paired t-tests
* The reported p-value and F-value are from the split-plot ANOVA are from the group*time interaction
* OASIS = Overall Anxiety Severity Impairment Scale

APPENDIX VII: DEPRESSION/MOOD

Table 7: Mean changes in PHQ-9 (depression) and POMS (mood) scores across the study time period (N=39)

Group/Assessment	Visit 1	Visit 2	Visit 3	F-value	P-value (Visit 1→2)	P-value (Visit 2 →3)
PHQ-9				11.85	*P*=.001	*P*<.001
Intervention	3.92 (3.97)	7.20 (4.86)	2.75 (2.67)			
Control	5.0 (5.66)	3.85 (5.18)	-			
Overall POMS				10.03	*P*= .003	*P*<.001
Intervention	43.63 (10.17)	53.92 (17.90)	38.83 (9.17)			
Control	42.18 (5.65)	38.82 (7.76)	-			
POMS Depression/Dejection				6.76	*P*=.013	*P* = .003
Intervention	16.31 (5.27)	18.77 (7.60)	14.76 (4.24)			
Control	18.31 (9.46)	16.15 (7.58)	-			
POMS Anger/Hostility				12.11	*P*=.001	*P*<.001
Intervention	13.75 (3.22)	18.88 (6.86)	13.36 (3.60)			
Control	13.73 (3.93)	13 (3.87)	-			
POMS Fatigue/Inertia				6.02	*P*=.013	*P*<.001
Intervention	13.27 (3.63)	16.23 (5.16)	10.81 (3.23)			
Control	13.83 (4.17)	12.92 (5.58)	-			

*Standard deviations for each mean score are reported after the mean in parenthesis
*P-values for Visit 1→2 were calculated using repeated measures ANOVA and p-values for Visit 2→3 were calculated using paired t-tests
*The reported p-values and F-values from the split-plot ANOVAs are from the group*time interaction

APPENDIX VIII: STUDY DESIGN

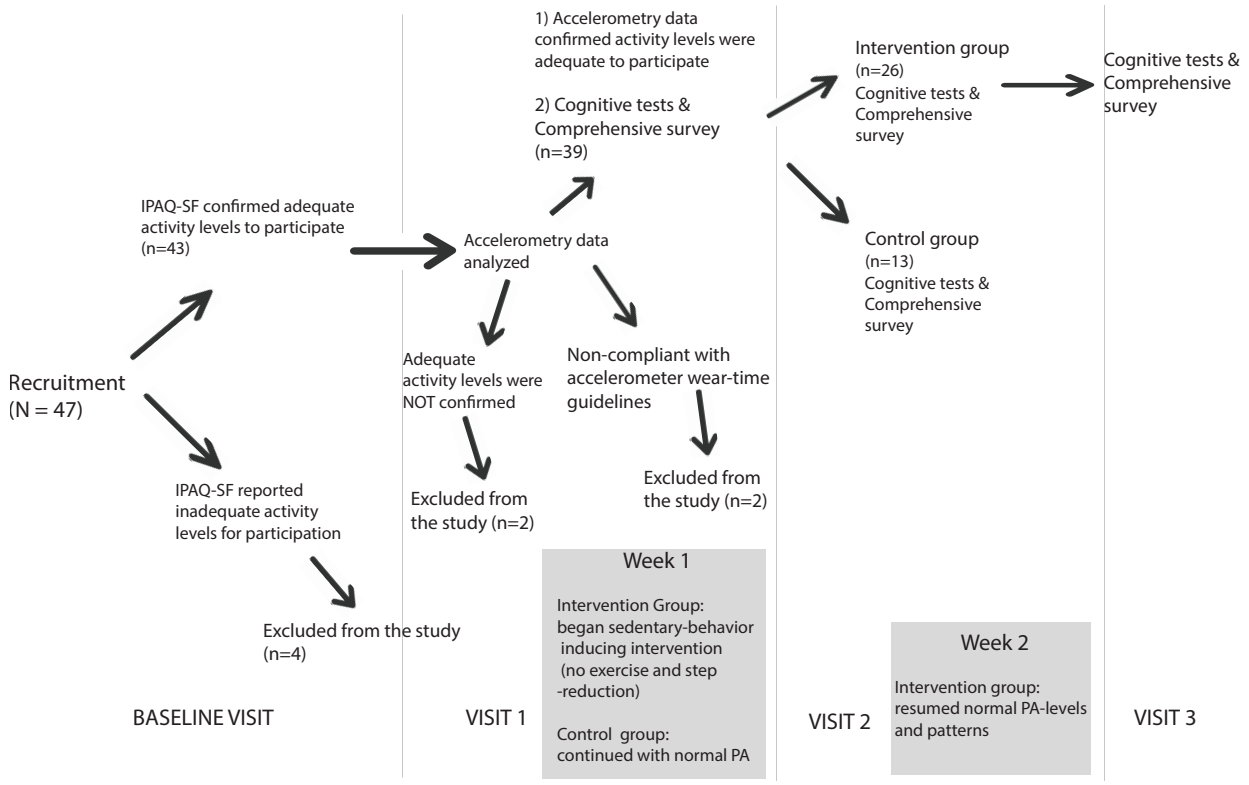

Figure 1: A schematic overview of the sedentary-behavior inducing intervention.
47 individuals were originally recruited. 43 of these individuals reported adequate physical activity (PA) levels to participate in the study. These 43 individuals wore an accelerometer for 1-week, after which 39 completed a baseline assessment all health outcomes and were randomly assigned into either the intervention or control group. 2 individuals were excluded due to accelerometry data not demonstrating adequate PA time and 2 were excluded due to non-compliance with accelerometry wear-time guidelines. The intervention group completed the health outcomes assessment at Visits 1, 2, and 3 and the control group completed the health outcomes assessment at Visits 1 and 2 only.
* IPAQ-SF= International Physical Activity Questionnaire, Short-Form

APPENDIX IX: STEP COUNTS

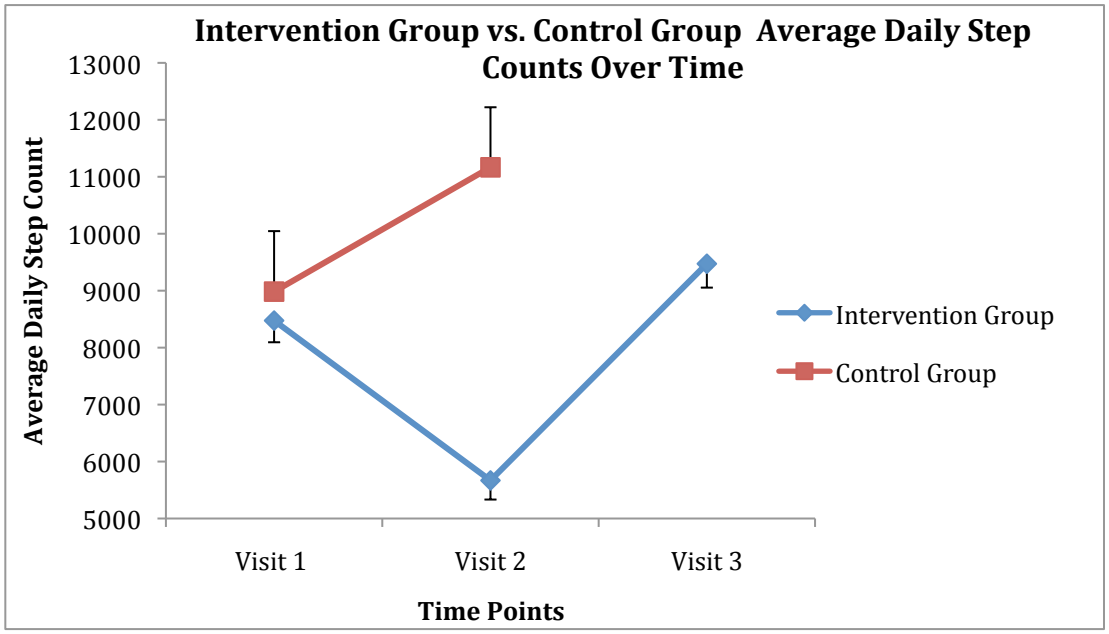

Figure 2: Intervention group vs. control group average daily step counts over time

- Standard errors are included as the error bars.

APPENDIX X: SLEEP FIGURE

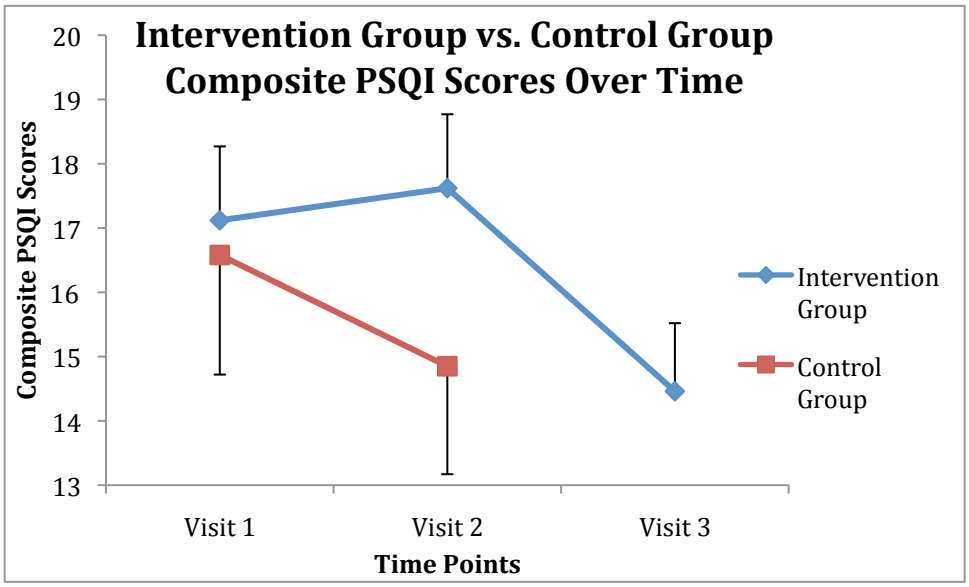

Figure 3: Intervention group vs. control group composite PSQI scores over time

- Standard errors are included as the error bars.

APPENDIX XI: LIFE SATISFACTION FIGURE

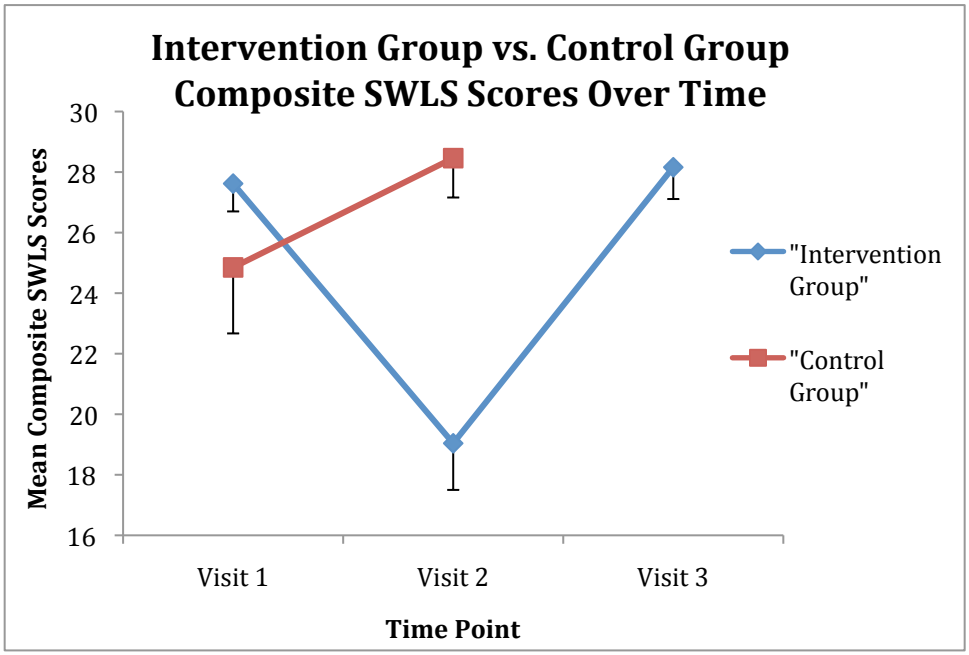

Figure 4: Intervention group vs. control group average daily step counts over time

- Standard deviations are included as the error bars.

APPENDIX XII: ANXIETY FIGURE

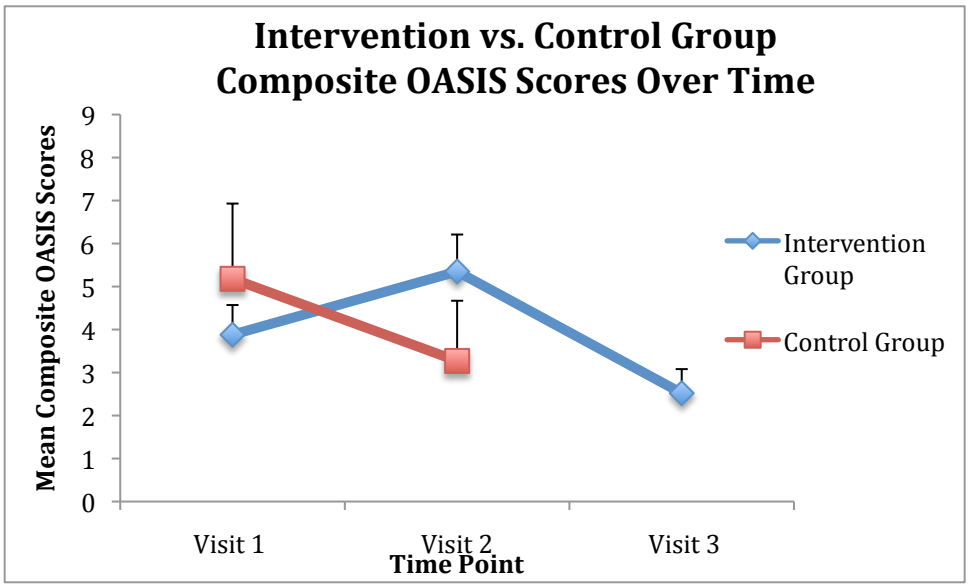

Figure 5: Intervention group vs. control group composite OASIS scores over time

- Standard deviations are included as the error bars.

APPENDIX XIII: DEPRESSION/MOOD FIGURES

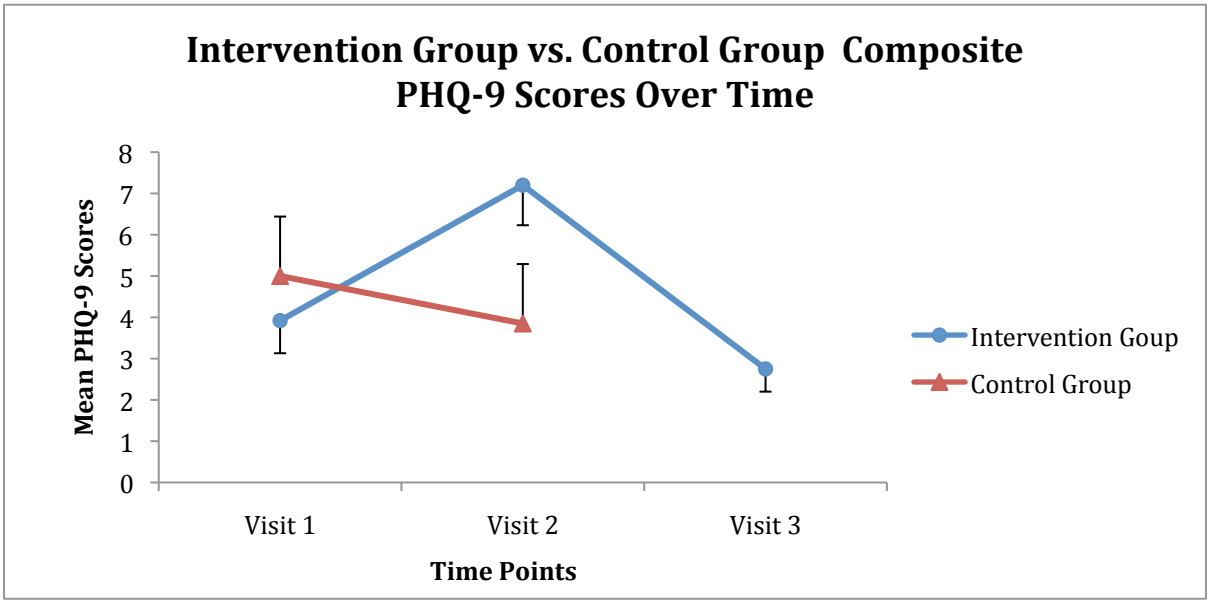

Figure 6: Intervention group vs. control group composite PHQ-9 scores over time

- Standard errors are included as the error bars.

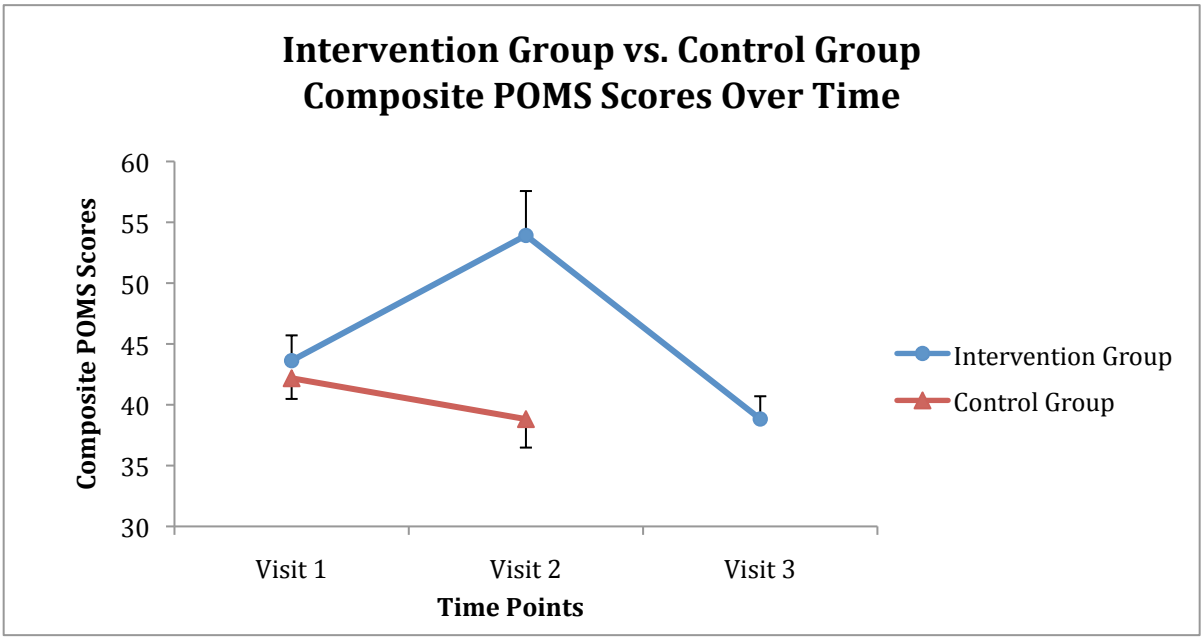

Figure 7: Intervention group vs. control group composite POMS scores over time

- Standard errors are included as the error bars.

VITA

Education

University of Minnesota, Twin Cities, MN ***September 2010 - May 2014***
Bachelor of Science in Kinesiology, Spanish Minor
Wayzata High School, Wayzata, MN ***September 2006- May 2010***

Work Experience

University of Mississippi Department of Campus Recreation **Oxford, MS**
Fitness Graduate Assistant ***August 2014- Present***

- Supervise and mentor a student staff consisting of 40-50 Fitness Center Supervisors, Personal Trainers, and Group Fitness Instructors
- Responsible for various administrative and programming tasks including conducting skill assessments and performance evaluations of fitness center employees, completing employee payroll, creating schedules, leading staff meetings and continuing education sessions, planning and implementing fitness-related special events, leading an instructional program for newly hired group fitness instructors, and teaching an 8-week personal training exam preparatory course

Group Fitness Instructor ***August 2014 - Present***

- Teach between 6-12 classes per week including yoga, cardio kickboxing, cycling, Pilates, strength, and high intensity interval classes.
- Regularly attend continuing education conferences and workshops as well as research current trends in the fitness industry to further my knowledge and skills as an instructor

Personal Trainer ***April 2012 - Present***

- Design individualized programs that assist to accomplish specific client goals, including special populations
- Maintain a thorough understanding of fitness assessment and functional movement screening procedures

Southern Star Yoga Center **Oxford, MS**
Yoga Instructor ***August 2014- Present***

- Instruct weekly yoga classes and attend various in-house continuing education workshops

OrangeTheory Fitness **Oxford, MS**
Fitness Instructor ***March 2016- Present***

- Regularly instruct fitness classes as well as volunteer at various workshops and special events

Oxford Fitness Kickboxing **Oxford, MS**
Kickboxing Instructor ***March 2015- Present***

- Instruct multiple weekly kickboxing classes

University of Minnesota Department of Recreation and Wellness **Twin Cities, MN**
Group Fitness Program Assistant ***April 2011 - May 2014***

- Led a team of 30-40 Group Fitness instructors, regularly conducted instructor performance evaluations, led staff meetings, and organized continuing education opportunities for instructors

Teaching Experience and Research Interests

EDHE 105, University of Mississippi

- Instructed a 3-credit first-year experience course designed to help students transition successfully from high school to college; examples of curriculum topics include: stress reduction/time management, race and diversity at the University, history of the University, substance abuse/misuse, sexual education, and physical activity/mindfulness

Red Cross First Aid/CPR/AED, University of Minnesota & University of Mississippi

- Regularly instructed First Aid/CPR/AED courses for student/staff RecWell employees at the University of Minnesota and currently instruct newly-hired student employees at the University of Mississippi

Research Interests

- Physical activity promotion, community health, chronic disease prevention, sedentary behavior, yoga, stress management, mindfulness-based therapy, quality of life, body image, exercise identity

Publications

- Loprinzi PD & **Edwards M**. Association between objectively measured physical activity and erectile dysfunction a nationally representative sample of American men. *J Sex Med* 2015;12(9):1862-4.

Additional Information

Professional Development

- Presented at the University of Mississippi Graduate Student Counsil Research Day Symposium *April, 2016*
- Accepted to present at the ACSM 2016 Annual Conference (Boston, MA) *April, 2016*
- Presented at the 2016 Evolve Fitness Expo (Atlanta, GA) *March, 2016*
- Presented at the 2016 University of Mississippi/UM Medical Center Research Day *March 2016*
- Attended and presented on multiple topics at the Evolve Fitness Expo (Auburn, AL) *March, 2015*
- Attended the MS/AL NIRSA State Workshop (Starkville, MS) *January, 2015*
- Attended the NIRSA Region III Student Lead-On Conference (West Lafayette, IN) *January, 2015*
- Attended the University of Mississippi Student Affairs Empower U Professional Development Conference (Oxford, MS) *January, 2015*
- Presented on multiple topics at the MS/AL Fitness Expo (Hattiesburg, MS) *November, 2014*
- Volunteered at and attended the BigTen NIRSA Conference (Minneapolis, MN) *May, 2014*
- Graduate of the University of Minnesota Student Employment Leadership Program *May, 2014*
- Attended the Annual NIRSA Conference (Nashville, TN) *April, 2014*
- Attended the Region V NIRSA Student Lead-On Conference (Omaha, NE) *October, 2013*
- Attended the 2012 & 2013 Minneapolis Empower Fitness Conference *October, 2012 & 2013*

Volunteer Work

- Volunteered at Sojourner Truth Academy during the 2013- 2014 academic year
- Volunteer instructed weekly yoga classes at the Minneapolis YMCA during the 2013-2014 academic year
- Volunteered at Comunidades Latinas Unidas En Servicio during the 2011-2013 academic years
- Volunteered as a NCKA and NASKA judge at various karate tournaments from 2009-2014
- Raised over $6000 for the Susan G. Komen Breast Cancer Foundation in 2013

Certifications

- 2014: PiYo® LIVE Certified Instructor

- 2014: 200-Hour Yoga Alliance Registered Yoga Teacher, University of Minnesota Center for Spirituality and Healing Instructor Training Program
- 2014: Schwinn® Indoor Cycling Certified Instructor
- 2013: American Council on Exercise (ACE) Certified Group Fitness Instructor
- 2013: TRX® Certified Instructor
- 2013: Indo-Row® Certified Instructor
- 2013: Shockwave® Certified Instructor
- 2013: National Academy of Sports Medicine (NASM) Certified Personal Trainer
- 2013: Red Cross Certified Instructor in First Aid and CPR/AED for adults, children and infants