ABSTRACT

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This study sought to determine what kind of causal relationship, if any, exists between exercise and depression. A university student population (N = 178) was given the Godin Leisure Time Exercise Questionnaire and the Beck Depression Inventory-II at two time points separated by approximately one month. Cross-lagged panel correlation was used to make causal inferences based on the strength of the temporal relationships. After meeting the assumptions of synchronicity and stationarity, there was no significant difference between the cross-lagged correlations (ZPF = -0.4599, p = 0.65). Thus, no single causal pathway was dominant. While equal cross-lagged correlations can indicate spuriousness, it can also signify reciprocal causation. Exercise was not clearly the cause of reductions in depression, but neither was depression clearly the cause of physical inactivity. More complex causal pathways, such as reciprocal causation, warrant further investigation.

EXERCISE AND DEPRESSION: CAUSAL SEQUENCE USING CROSS-
LAGGED PANEL CORRELATION ANALYSIS

By

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Chapter 1: Introduction

Depression is one of the most common mental disorders in America, affecting an estimated 19 million adults and costing upwards of $44 billion in medical care and lost work time (Johnson, 2003). According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (as cited in Brosse, Sheets, Lett, & Blumenthal, 2002), a person with clinical depression shows either a depressed mood or a loss of interest or pleasure (known as anhedonia) for at least two weeks. Other symptoms include changes in weight, appetite, or sleep patterns, feeling "slowed down" or restless, fatigue, feelings of guilt or worthlessness, an inability to concentrate, and recurring thoughts of death or suicide. These symptoms cannot be the result of a physical illness or medication, nor can they be a response to substance abuse or bereavement. In addition, these symptoms must considerably impair the individual's functioning on a daily basis. Fortunately, there are many ways of reducing the symptoms of depression. While psychotherapy and antidepressant medication are the most common treatment modalities, many alternative treatments are available.

Exercise, widely known for its benefits to physical health, has also been studied for its benefits to mental health. Epidemiological studies have shown that exercise is inversely associated with depression (Kritz-Silverstein, Barrett-Connor, & Corbeau, 2001; see also Strawbridge, Deleger, Roberts, & Kaplan, 2002) using cross-sectional data. While a statistical association is necessary to infer a causal relationship, statistical association alone is not sufficient to conclude that exercise reduces depression; it may be that depression reduces exercise and physical activity. Some studies (Strawbridge et al., 2002), but not all (Kritz-Silverstein et al., 2001) have demonstrated that exercise is

associated with a reduced risk of future depression using prospective designs. Noting this temporal sequence, numerous exercise intervention studies have examined the role of exercise in reducing depressive symptoms, both in clinical (Blumenthal, Babyak, Moore, Craighead, Herman, Khatri, et al., 1999; see also Babyak, Blumenthal, Herman, Khatri, Doraiswamy, Moore, et al.. 2000 and Dunn, Trivedi, Kampert, Clark, & Chambliss 2005) and nonclinical populations (Hassmen, Koivula, & Uutela, 2000). By default, these interventions assume that exercise is the cause of reduced depression. This alterability of depression levels with exercise gives further evidence towards a causal relationship where exercise reduces depression. In fact, exercise intervention in clinical patients showed exercise to be as effective as a common antidepressant (Blumenthal et al., 1999), and may even be superior to antidepressants with respect to long-term relapse (Babyak et al., 2000). While older studies merely compared exercise to a standard treatment, more recent investigations have additionally included a placebo group for better comparison (Blumenthal, Babyak, Doraiswamy, Watkins, Hoffman, Barbour, et al., 2007). In addition, the potential dose-response relationship between exercise and depression has begun to be examined. One group of researchers examined this potential dose-response relationship with an intervention in a clinical population (Dunn et al., 2005). Those who received the largest dose of exercise, approximately 30 minutes of moderate intensity aerobic activity on most days, experienced more relief from depressive symptoms than those who performed less aerobic activity or stretched. The group receiving less exercise did not differ from the stretching placebo group when depression was measured at follow-up. This potential dose-response relationship gives more evidence for a causal relationship where exercise reduces depression.

However, it may be the case that the presence of depressive symptoms is incompatible with beginning or maintaining habitual exercise. It should be noted that the intervention studies mentioned previously used a self-selected population of clinical patients, which may not be representative of the all individuals with depression. Studies measuring physical activity as an outcome have noted that depression is associated with physical inactivity. This relationship was noted for several age groups, including older adults (Kaplan, Newsom, McFarland, & Lu, 2001) and adolescents (Sallis, Prochaska, & Taylor, 2000). This is not surprising, given that the symptoms of depression include fatigue and feeling slowed down. Exercise requires a certain amount of energy and motivation to get started, and many depressed patients lack these qualities due to their illness. It is possible that people with depression may not feel worthy of the benefits of exercise. If a previously active person becomes depressed, he or she may not find the same amounts of pleasure in the activity, and may drop out of exercise altogether.

However, the notion that depression reduces physical activity remains a largely untested possibility in the existing literature. Prospective epidemiological studies linking depression and exercise have typically excluded depressed subjects from follow-up analysis (Strawbridge et al., 2002; see also Kritz-Silverstein et al., 2001). Additionally, because depressive symptoms are attributed to subjects and cannot be experimentally manipulated, this idea cannot be examined in an experiment. Consequently, it has not received as much support in the literature, although many interventions note the difficulties in patient adherence to the exercise program. One study that did examine this causal role of depression in a sample of cardiac patients found that depression during hospitalization predicted physical activity one year later (Allan, Johnston, Johnston, &

Mant, 2007). Furthermore, even subclinical levels of depression were associated with reduced physical activity, suggesting that even minor depressive symptoms may inhibit exercise behavior.

A third possibility is that there may be a reciprocal relationship between exercise and depression. One intervention follow-up noted that subjects who began an exercise program, regardless of original treatment group, felt less depressed when contacted six months after the intervention ended (Babyak et al., 2000). This same group of researchers also noted that subjects who dropped out of the study tended to be more depressed initially. In this case, feeling less depressed increased the chances of a person continuing or adopting an exercise program, which decreased the odds of a depressive relapse. Conversely, severe depression may prevent one from successfully adopting an exercise program.

To date, many studies have examined the nature of the relationship between depression and exercise. However, few studies have done so in a way that allows either variable to cause the other. Most studies have examined exercise as a presumed cause of reductions in depression, but others have used depression to predict exercise habits. Although experiments often test whether exercise plays a causal role in reducing depressive symptoms, they cannot test for reverse causation. While a reciprocal relationship between these two variables cannot be excluded, this study seeks to address the following research question: What is the predominant causal relationship between exercise and depression?

Chapter 2: Literature Review

Statistical Association Between Depression and Exercise

Several large epidemiological studies have noted a statistical association between mental health and exercise. A statistical association is necessary, but not sufficient by itself, to infer causal relationships. While cross sectional studies are able to provide one type of evidence towards causation, cross sectional studies by themselves are insufficient to infer causality. Several of these cross sectional studies have examined exercise in relation to overall mental health, both its positive and negative aspects. The ones included here specifically look at depression as one of many negative mental health outcomes. One of these studies examined the association between exercise frequency and a host of mental and emotional outcomes, including depressive symptoms, using a general population (Hassmen, Koivula, & Uutela 2000). Exercise was operationally defined as the response to "How often do you exercise physically in your spare time for at least 20-30 minutes to the extent that you at least slightly lose your breath and perspire?" Six response categories indicated the frequency of exercising to that extent, ranging from "daily" to "cannot perform exercise." Depression was measured using the Beck Depression Inventory (BDI). Results showed that the more physically active participants experienced less depression and other negative mental health outcomes, such as anger and perceived stress, when compared to those who exercised less frequently. In addition, subjects who exercised frequently experienced more positive mental health outcomes, such as a sense of coherence and stronger social integration, than those who

were less active. This gives evidence towards an inverse association between exercise

and negative mental health outcomes such as depression. The main limitation to this

study is that self reports were the sole method of assessing exercise and mental health

outcomes. There is also the possibility that confounding variables explain the

association, as there may be something fundamentally different about those who exercise

versus those who do not. This is a likely scenario, as social factors such as

socioeconomic status and satisfaction with marital status may play a role in both exercise

behavior and the development of depressive symptoms.

 While the previous study was conducted in Finland, the relationship between

physical activity and mental health has also been studied in the United States (Goodwin,

2003). However, rather than examining mental both the positive and negative aspects of

mental health, as Hassmen and colleagues did, Goodwin looked at diagnosable mental

disorders in relation to regular physical activity. Additionally, the impact of potential

confounders such as physical health problems and demographic characteristics was

statistically controlled. Exercise behavior was operationally defined as the response to

"How often do you get physical exercise- either on your job or in a recreational activity?"

While five response categories were originally used, these were later grouped into

dichotomous categories, where regular exercisers were compared with those in all other

categories. Psychiatric diagnoses were obtained from a modified version of the World

Health Organization Composite International Diagnostic Interview, which is a structured

interview for trained non-clinician professionals. The major categories of mental

disorders examined include anxiety disorders, mood disorders, and substance

dependence. After statistically adjusting for socioeconomic status, self-reported physical

illnesses, and comorbid mental disorders, regular physical activity was significantly associated with a reduced likelihood of having major depression, agoraphobia, social phobia, specific phobias, and panic attacks. Similar to the findings of Hassmen et al. (2000), a negative association between exercise and adverse mental health outcomes, specifically depression, was demonstrated. However, it must be noted that this negative association was noted for major depression and several anxiety disorders, but not for bipolar disorder or substance dependence. Goodwin (2003) gives several explanations for the findings, but notes that the issue of causality cannot be resolved due to the cross sectional nature of the study. While exercise may reduce depression and other mental health symptoms, the notion that depression is a barrier to physical activity could also explain the association. The main limitation of the study was the lack of a precise definition of regular exercise. The actual frequency of "regular" exercise may vary widely within subjects, and the type of exercise was not specified. Also, the impact of existing fitness could not be examined because it was not measured.

Although Goodwin (2003) did not measure fitness, other studies have used fitness in conjunction with measures of exercise behavior when examining mental health outcomes. As part of a larger, ongoing epidemiological study, approximately 6450 subjects underwent maximal aerobic testing, in addition to wearing accelerometers and completing a self-reported physical activity index (Galper, Trivedi, Barlow, Dunn, & Campert, 2006). From these measures, subjects were classified into three groups based on aerobic capacity (low, moderate, and highly fit), and four groups based on the physical activity index (inactive, insufficiently active, sufficiently active, and highly active). For both cardiovascular fitness and exercise behavior, there was an inverse graded dose-

response relationship with depression, as measured by the Center for Epidemiological Studies Scale for Depression (CES-D). In addition, subjects in the lowest fitness category had significantly more symptoms of depression than those in more fit categories. Interestingly, there seemed to be a plateau in mental health benefits with the attainment of "sufficient" exercise, where being highly active did not confer additional reductions in depressive symptoms. This gives some evidence towards a nonlinear dose-response relationship. The biggest strength of this particular study was its use of objective measures of fitness in addition to self-reported exercise behavior, although similar results were seen for each. However, like all cross-sectional studies, it could not infer cause-effect relationships between exercise, fitness, and depression. Galper et al. (2006) noted that "physical inactivity is likely to be both cause and consequence of depressive symptoms" (p. 177).

In addition to studies exclusively examining the cross-sectional association between exercise and depression, many longitudinal studies of these variables also contain a cross-sectional component. One such investigation, the Upper Bavarian Field Study, found that the association between exercise and depression remained when statistically controlling for physical illness, age, sex, and social class (Weyerer, 1992). When regular exercisers were compared with occasional and nonexercisers, the nonexercisers had three times the risk of depression when compared to regular exercisers, while the occasional exercisers did not significantly differ from regular exercisers. The measurement of depression in this sample was sound, using depression diagnosed by research psychiatrists, but the interpretation of "regular" exercise was less specific. This imprecise definition was likely interpreted differently by respondents.

Longitudinal studies involving exercise and depression have also been conducted in the United States. These studies have taken differing views on the impact of physical disability as a potential confounder in the relationship between depression and exercise. Individuals who are physically limited are both more likely to be depressed and less likely to exercise, but it is unclear if or where physical disability lies in the causal chain of events. In an epidemiological study involving exercise behavior and depressed mood, Kritz-Silverstein et al. (2001) excluded subjects with physical limitations from the statistical analysis. In contrast, a similar study conducted statistical analyses with and without subjects with physical limitations (Strawbridge et al., 2002). Both of these investigations found the same trend in cross-sectional analysis; namely, that exercise is inversely related to depression. Interestingly, Strawbridge et al. (2002) found that the inclusion or exclusion of subjects with physical limitations did not alter the results. Unfortunately, both studies were part of larger projects that were not intended to measure physical activity or exercise, and neither used rigorously tested or widely used indices of physical activity. The measures of depression were better, using the BDI (Kritz-Silverstein et al., 2001) and an adaptation of the DSM-IV criteria (Strawbridge et al., 2002).

Longitudinal Associations Between Depression and Exercise

In addition to statistical association, the temporal sequence between variables must be established in order to infer causal relationships. While evidence from cross-sectional studies shows a strong inverse association between exercise and depression, the temporal sequence between the two is better demonstrated with longitudinal studies. As

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a whole, the evidence from prospective, longitudinal studies is less conclusive than the evidence from cross sectional studies. Also, many longitudinal studies have looked at the relationship between exercise and depression in only one direction. Specifically, they examined whether past exercise behavior predicted depression at a later time point, usually failing to examine the opposite temporal sequence.

In the Upper Bavarian Field Study (Weyerer, 1992), low physical activity at baseline was not a risk factor for developing depression five years later. This apparently contrasts the association found from cross-sectional data from the same study. However, physical activity was not assessed at follow-up, so it was unclear whether exercise habits changed over time. In addition, the possibility of depression preceding physical inactivity cannot be ruled out.

Similarly, the Rancho Bernardo study (Kritz-Silverstein et al., 2001) also found that physical activity at baseline did not predict depressive symptoms when measured eight years later, both before and after adjusting for potential confounders. Again, this is a direct contrast to the cross-sectional findings of the study. Unlike the Upper Bavarian Field Study (Weyerer, 1992), physical activity was measured at both baseline and follow-up. Those who reported no exercise at baseline, but became more physically active by the follow-up measure had fewer symptoms of depression at follow-up than those who remained inactive. However, the Rancho Bernardo study also has its limitations when determining the temporal relationship between depression and physical inactivity. Subjects who initially presented with high levels of depressive symptoms (scores >13 on the BDI) were excluded from follow-up analysis, so the researchers were unable to determine whether depression preceded inactivity. Another limitation was the use of an

older, high socioeconomic status population. Kritz-Silverstein et al. (2001) noted that a prospective association between exercise and depression might have been shown in a younger cohort with a wider range of exercise and depression scores. Finally, the measure of exercise took only vigorous activity into account, which may not accurately reflect the physical activity habits of the older population used in this study.

While some longitudinal studies have not found a prospective relationship between exercise and depression, others have found an inverse relationship. In the Alameda County study (Strawbridge et al., 2002), physical activity habits at baseline were associated with clinically diagnosed depression at a five year follow-up. In comparison to the Rancho Bernardo investigation (Kritz-Silverstein et al., 2001), the measure of exercise used took a wider variety of activities into account.   Perhaps the inclusion of moderate intensity activity, such as walking, explains why a relationship was shown in this study. Interestingly, the prospective association between physical activity and depression was shown when physically disabled subjects were both included and excluded from the analysis.

Additionally, data from the Australian Longitudinal Study on Women's Health showed a prospective relationship between exercise and depression (Brown et al., 2005). Instead of focusing on older individuals, this particular investigation used middle-aged women as the population of interest. Depression was measured using the CES-D, and scores >10 were indicative of depressive symptoms. The measure of physical activity took both vigorous and less vigorous activity into account, combining them to create a physical activity score. As such, the volume of physical activity was the variable of interest. The authors assessed subjects at three time points over a five year time span, in

order to determine current, previous, and habitual levels of physical activity. All assessments of physical activity were associated with reductions in depression, indicating both a cross-sectional relationship and a longitudinal relationship. Furthermore, women in the lowest physical activity category who increased their exercise to recommended levels (150 minutes per week) were less likely to report depressive symptoms after five years than women who remained inactive. The protective effects of exercise remained after adjusting for potential confounders, including education, occupation, smoking, and the presence of chronic health conditions. In this particular study, even low levels of physical activity (60 to 150 minutes of moderate intensity activity per week) were associated with a reduction of risk when compared with no physical activity.

Some of the studies mentioned did not measure changes in physical activity over time, while others took note of changes in physical activity but analyzed these changes tangentially. Lampinen, Heikkinen, and Ruoppila (2000) explicitly focused on changes in the intensity of exercise as a predictive factor for depression, using change scores (increase, decrease, or remained the same) as the primary variable of interest. The authors used an older, community-dwelling Finnish population, and followed them for eight years. The intensity of physical activity was measured on a seven-point scale, with the lowest category consisting of necessary chores only and the highest category consisting of competitive sports. These categories were later combined into three levels for statistical analysis: necessary chores only, regular walking, and strenuous physical exercise. Depressive symptoms were measured with a modified BDI. Results showed that in general, the proportion of subjects who did necessary chores only increased over the eight year follow-up, while the proportion of those participating in strenuous exercise

decreased. This trend might be expected in an older population, although it should be noted that chronic health conditions and difficulties in activities of daily living were controlled for in the statistical analysis. As a whole, those who reduced the intensity of exercise experienced more depressive symptoms at follow-up than individuals who increased the intensity of exercise or whose intensity did not change. For all levels of exercise intensity, depressive symptoms at baseline significantly predicted depressive symptoms at follow-up.

In summary, the data from longitudinal studies is mixed. While some studies show a prospective association between exercise and subsequent depression, others do not show this association. There are several potential explanations for these mixed findings. First, the measurement of physical activity may have been inappropriate for the populations used. While younger populations are likely to engage in vigorous exercise, older cohorts are more likely to participate in light or moderate activity. In addition, it is unknown which aspects of physical activity are important when it comes to reducing depression. A majority of studies examined volume or frequency of physical activity, but the intensity or duration may be important factors as well.   Additionally, it is unknown whether current physical activity or cumulative exercise over many years is more useful when predicting depression. Other explanations involve the episodic nature of depression. All of the longitudinal studies mentioned follow subjects over a course of at least five years. Because depression was measured at time points so far apart, researchers may not capture all cases of depression that occur during that time frame. For example, a person may become depressed and recover within five years, and this would not be captured in most of these studies. Use of a shorter time frame would reduce or eliminate

this problem. The effects of exercise on depression may be more immediate than these longitudinal studies can capture, and depression may inhibit exercise only in the short-term. Finally, none of the longitudinal studies took lifetime prevalence of depression into account. Even though most studies took baseline depressive symptoms into account (by excluding these subjects from further analysis or using statistical methods), it is unknown how many subjects had experienced a prior episode of depression in their lifetime. This puts them at risk for developing subsequent episodes of depression, regardless of physical activity.

Temporal Sequence: Changes in Exercise Precede Changes in Depression

Building on the cross sectional association, data from some longitudinal studies demonstrate that changes in exercise predict future changes in depression. Generally, longitudinal studies have been designed to indicate that changes in exercise predict later changes in depression. Noting the possible temporal sequence indicated by longitudinal studies, many exercise interventions have been conducted. However, changing exercise habits in either direction would change later depression levels, if this temporal sequence is correct. Subjects in exercise intervention studies greatly increase their participation in physical activity, resulting in mental health benefits. Conversely, reducing or eliminating a physical activity program for someone who is regularly active would be expected to be detrimental to mental health.

Exercise Withdrawal

Berlin, Kop, and Deuster (2006) examined the hypothesis that exercise withdrawal would result in increasing symptoms of depression. Additionally, they suspected that somatic depressive symptoms would develop before the cognitive-affective symptoms of depression, and that reductions in fitness would be related to the magnitude of emerging depressive symptoms. The subjects consisted of forty regular exercisers, aged 18-45. Before participating in the study, all subjects participated in at least 30 minutes of continuous aerobic exercise, three or more days per week for over six months. Subjects were initially healthy. They had no history of cardiovascular disease, did not suffer from hypertension or obesity, and were not receiving treatment for any psychological disorder.

After recruitment into the study, subjects were randomized to either continue their exercise routine as usual, or to withdraw from exercise for two weeks. Accelerometers were used as a manipulation check, to ensure compliance for both groups. Outcome measures included the Profile of Mood States (POMS), BDI-II, and the Multidimensional Fatigue Inventory (MFI) to reflect changes in mood, depression, and fatigue respectively. Submaximal exercise testing was also completed to measure changes in fitness. These outcomes were measured at baseline, 1 week, and 2 weeks.

Results from the accelerometers indicated that subjects in the exercise-withdrawal group did reduce their high-intensity physical activity compared to those who continued their exercise program. Withdrawal from exercise resulted in higher scores on the POMS and BDI-II at follow-up, indicating more a negative mood and increases in depressive symptoms. Specific analysis of depressive symptoms indicated that changes in somatic

symptoms were evident after only one week, and these changes in somatic symptoms predicted cognitive-affective changes at two weeks. Although overall reductions in fitness in the exercise-withdrawal group were not significant, there are likely individual differences. Individuals whose aerobic capacity decreased were more likely to develop depressive symptoms when compared to those whose fitness did not decrease. Although Berlin et al. (2006) showed that reductions in exercise preceded an increase in depressive symptoms, there are several limitations. Notably, the voluntary exercise withdrawal used may not reflect actual circumstances that involve a reduction or withdrawal from exercise. Injuries and illness are two main reasons why a previously active person would become less active, and both of these are known to impact depression levels. In addition, the two week time interval used here does not necessarily simulate real-world conditions. Although some causes of exercise withdrawal result in brief periods of inactivity, it takes much longer to recover from some illnesses and injuries. The full extent of changes in depression may not become apparent in two weeks, and there was no follow-up assessment to examine the long-term impact of exercise withdrawal.

Exercise Interventions

If exercise withdrawal results in depressive symptoms, then beginning an exercise program may relieve existing symptoms of depression. Even though psychotherapy and antidepressant medication are the traditional treatments for depression, they have some limitations. For example, medication can relieve symptoms, but does not protect against relapse if discontinued. The side effects of treatment may also be problematic. In addition, approximately 30% of patients do not respond adequately to drug treatment (Baghai, Moller, & Rupprecht, 2006). Noting the problems with standard treatments for

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depression, many types of exercise interventions have been conducted. By nature, these interventions assume that exercise plays a causal role in the reduction of depression. Some of these exercise interventions examine various components of exercise, comparing two or more exercise programs, while others compare exercise to more conventional treatments for depression. Exercise has been examined as a monotherapy for clinical patients and also as an adjunct treatment in combination with psychotherapy or medication. Some of the exercise intervention studies focus on the short-term mental health benefits of exercise, while others look at longer lasting reductions in depression. In addition, fitness has been investigated as a possible mediating factor when looking at the relationship between exercise and depression.

While the best evidence for the notion that exercise causes reductions in depression comes from randomized controlled trials, this relationship has also been noted tangentially in the use of exercise interventions for weight control and overall health. In one intervention, hypertensive men and women were randomly assigned into one of three groups: aerobic exercise, exercise and weight loss, or a wait-list control (Smith, Blumenthal, Babyak, Georgiades, Hinderliter, & Sherwood, 2007). Despite the low prevalence of depression at baseline, as measured by the BDI, there was an interaction between treatment group and baseline depression. Individuals in the exercise groups with higher BDI scores at baseline showed a reduction in their BDI scores at the end of the intervention, when compared with wait-list controls. While weight loss and reductions in blood pressure did not mediate the relationship between exercise and depression, subjects with greater improvements in aerobic capacity showed greater reductions in depression. Smith et al. (2007) note the lack of clarity in the direction of the relationship between

exercise, depression, and fitness, stating "improved aerobic capacity could result in reduced depression, or reduced depression may contribute to improved aerobic capacity" (p. 467). However, this study was not originally designed to examine the effects of exercise on depression, but rather on physical health indicators.

Although Smith et al. (2007) noted reductions in depressive symptoms when using exercise programs for physical health benefits, others have overtly set out to measure the effects of exercise on depression. Some studies have simply compared exercise training to a placebo or wait-list control group, while others have made multiple comparisons (for example, comparing two different exercise programs with a placebo or comparing exercise, medication, and combination therapy). The nature of the exercise programs varied widely. Some of the exercise programs used only aerobic exercise, some compared aerobic activity with weight training, and still others used a multimodal exercise intervention. One of these multimodal exercise interventions was conducted using a worksite population over 24 weeks (Atlantis, Chow, Kirby, & Singh, 2004). Like most exercise intervention programs, subjects were physically healthy but minimally active prior to entering the study. In addition, none of the subjects had a diagnosable mental disorder. Prior to randomization, subjects were stratified by gender and initial scores on the Depression, Anxiety, and Stress Scales (DASS). Initially, 16% of subjects scored above normal on the depression scale when compared to Australian norms, and a similar percentage was above normal for anxiety and stress. Participants were then randomized into the treatment group or the wait-list control. Those in the treatment group participated in both aerobic exercise and strength training for 24 weeks. The subjects performed a minimum of 20 minutes aerobic exercise, three days per week,

using a variety of cardiovascular machines such as a treadmill or bicycle. The subjects performed the aerobic exercise at a prescribed percentage of their age-estimated maximal heart rate, and the intensity increased throughout the duration of the study. The subjects also participated in prescribed strength training exercises. Initially, these were done in machines, but free weight exercises were later introduced. Over the course of the intervention, the load steadily increased while the number of repetitions decreased. Most subjects were advised to perform a whole-body strength training routine a minimum of two times per week, but a few used a split-body workout due to their frequent attendance at the fitness center. In addition to the aerobic and strength training exercise, behavior modification strategies were also employed through the use of several health education seminars. Subjects in the wait-list control did not receive any of the previously mentioned interventions, and minimal contact was made to collect questionnaires. They were instructed to continue life as normal, including any existing physical activity. Subject compliance was problematic with this study, as 40% dropped out before the completion of the 24-week program. Prior to the intervention, subjects in the treatment and control groups did not differ significantly on any outcome. After the intervention, subjects in the intervention group significantly improved depression and stress scores, compared to those in the control group. Among mental health outcomes, an interaction effect was seen, where those with worse scores at baseline improved the most. However, due to the multimodal nature of this intervention, the authors were unable to determine which components were most beneficial for their effects on depressive symptoms and overall mental health (Atlantis et al., 2004).

While Atlantis and colleagues (2004) studied an exercise training program over the course of several months, the short-term benefits of exercise may also be important. A single exercise bout may temporarily enhance mood, but these effects may only become clinically significant and enduring with exercise training. Bartholomew, Morrison, and Ciccolo (2005) studied the effects of a single exercise session on mood and well being, using a sample of newly diagnosed patients with Major Depressive Disorder (MDD). Mood was assessed using the Profile of Mood States (POMS) and the Subjective Exercise Experiences Scale (SEES) at several time points before and after the exercise session. Participants were assigned to the control group, which consisted of 30 minutes of quiet rest, or the exercise group, which consisted of a 30 minute brisk walk on a treadmill. The results indicated that a single exercise session showed benefits over quiet rest for some, but not all of the subscales assessed. Specifically, the exercise bout increased psychological well-being and vigor, as measured by the SEES and POMS respectively. It appears that exercise enhances these positively valenced states for a brief time period following an exercise session. While Bartholomew et al. (2005) stress that these transient changes in mood are unlikely to impact the underlying depression, they also note that exercise may be useful for short-term mood regulation. Thus, exercise sessions may benefit patients in its role as a coping mechanism, rather than being viewed as treatment in a traditional sense. This may be especially important given the length of time before antidepressants exhibit therapeutic effects.

While the previous two studies mentioned only compared exercise to a control group, other exercise interventions have made multiple comparisons (Blumenthal et al., 2007; see also Dunn et al., 2005; see also DiLorenzo et al., 1999; see also Osei-Tutu &

Campagna 2004; see also King et al., 1993; see also Legrand & Heuze, 2007). In all cases, at least one of the exercise interventions used was superior to placebo or wait-list control. There were generally no significant differences between exercise programs in regards to reductions in depression, although Dunn et al. (2005) showed that one particular exercise program was superior to another. In most cases, the specific nature of the exercise program was not important; however the participation in some form of exercise was crucial for gains in mental health.

While exercise is clearly better than no treatment for depressive symptoms, other investigations have compared exercise programs to more traditional treatments for depression. One of the earlier investigations of this type compared aerobic exercise, cognitive therapy, and combination treatment (Fremont & Craighead, 1987). Individuals were recruited using public service announcements targeted towards those who were interested in treatment for negative moods. To be included in the study, subjects were sedentary and had an elevated score on the BDI, indicating depressed mood. In addition, subjects were not receiving any kind of treatment for depression at the time of entry into the study. The subjects were randomly assigned to treatment groups, and 49 completed the ten weeks of treatment. Those assigned to the exercise group did 20 minute bouts of walking and running in a small group setting three times a week. Experienced runners served as coaches, instructing the subjects on the ideal intensity, proper running form, and how to measure their heart rate. Subjects assigned to the cognitive therapy group received individual one hour sessions with a therapist once a week. The goal of these sessions was to change negative thought patterns and attitudes. Those assigned to the combination treatment received the weekly counseling session in addition to participating

in small-group exercise. Measures included the BDI, POMS, and the State-Trait Anxiety

Inventory (STAI) given at various time points before, during, and after the ten weeks of

treatment. Initially, all the subjects were mildly to moderately depressed, according to

BDI scores. After treatment, mean BDI scores indicated that they were no longer

depressed, and this improvement was maintained at 2 and 4 months of follow up. All

three treatment groups had similar impact on depression, with all groups experiencing

benefits. Improvement in depression occurred quickly, in the first 3 to 5 weeks of

treatment. It did not appear that changes in fitness were necessary for mental health

benefits, although Fremont and Craighead (1987) noted that better physiological

indicators were needed to demonstrate this. Combination treatment did not give further

benefits over treatment with cognitive therapy or exercise alone, and exercise was seen as

the most cost-effective method used (Fremont & Craighead, 1987).

At the time of Fremont and Craighead's (1987) publication, cognitive therapy was

the dominant treatment for depression. However, due to advancements in pharmacology,

treatment with medication has become more popular. More recent studies have

compared exercise to some of these newer, widely used medications for depression. A

series of studies (Blumenthal, Babyak, Moore, Craighead, Herman, Khatri, et al. 1999;

see also Babyak, Blumenthal, Herman, Khatri, Doraiswamy, Moore, et al. 2000)

compared the effects of a 16 week aerobic exercise program, antidepressant medication,

and combination treatment in a sample of older individuals with MDD. The presence of

depression was initially established in all subjects by using both a diagnostic interview

and the Hamilton Rating Scale for Depression (HAM-D). Subjects were then randomly

assigned into one of the three groups- exercise only, medication only, or combination

treatment. The exercise intervention consisted of approximately 45 minutes of walking

or jogging at a prescribed heart rate three times a week for the 16 week study. The

particular medication used in the study was sertraline (Zoloft), and medication

management was provided by one of the study psychiatrists. Specific dosage was titrated

according to the subject's response to treatment, both the therapeutic effects and the side

effects. Medication management in this group was similar to that in standard practice,

with the exception that subjects could not be switched to a different antidepressant during

the study. Subjects in the combination treatment received both the exercise intervention

and medication management concurrently.

At the end of the 16 week intervention, all of the groups showed significant

decreases in depression; however, no treatment was more effective than the others

(Blumenthal et al., 1999). Thus, it was concluded that all three treatments were equally

effective at reducing depression, and there was no additional benefit from combining

treatments. However, a 10 month follow-up of these individuals revealed differences in

recovery and relapse rates (Babyak et al., 2000). At this follow-up, subjects were

considered recovered if they no longer met the criteria for MDD and had an HAM-D

score below 8. Subjects were considered partially recovered if they no longer met the

diagnostic criteria for MDD, but still showed some depressive symptoms, with an HAM-

D score between 8 and 14. Subjects were considered relapsed if they were in remission

at the end of the intervention, but met the DSM-IV criteria for MDD or had an HAM-D

score above 14 at the 10 month follow-up. While all treatments were equally effective at

the end of the four months of treatment, participants in the exercise group were more

likely to be partially or fully recovered at the follow-up visit, when compared to

participants in the medication or combination group. In addition, relapse rates were much lower for the exercise group (8%) than for the medication group (38%) and combination groups (31%). This finding for the combination group was unexpected, as Babyak et al. (2000) assumed that this group would do as well or better than the exercise-only group. The authors explained this by noting the anti-medication sentiment among some study participants. The researchers also mentioned that medication might interfere with the beneficial effects of exercise, although they were unable to describe physiological mechanisms underlying this result.

While the exercise group had the best outcome at follow-up in regards to recovery, there were several reasons why it cannot be concluded that exercise caused depressive symptoms to be reduced at that time. First, there was substantial crossover between the groups after the intervention ended. For example, some subjects in the medication group began to exercise, while others quit medication altogether. More importantly, Babyak and colleagues (2000) acknowledge the possibility of reverse causation. In other words, patients who began or continued to exercise after the intervention did so because they were less depressed at the end of the treatment period. This was highly probable, as an inverse association was shown between post-treatment HAM-D scores and self-reported exercise during the follow-up period. However, when post-treatment HAM-D scores were controlled, exercise during the follow-up period still predicted depression status. This raises the possibility of a reciprocal relationship between exercise and depression, where feeling less depressed may increase the likelihood of physical activity, and continuing to exercise reduces the possibility of relapse.

Two limitations of the series of studies conducted by Blumenthal et al. (1999) and Babyak et al. (2000) are the lack of a true placebo group and the inability to account for nonspecific effects of exercise (staff attention, social support, etc). These limitations were addressed in a subsequent study by the same group of researchers (Blumenthal, Babyak, Doraiswamy, Watkins, Hoffman, Barbour, et. al, 2007). In this more recent study, subjects diagnosed with MDD were randomly assigned to supervised exercise in a group setting, home-based exercise, antidepressant medication, or a pill placebo treatment for 16 weeks. Because of the possibility of being assigned to a placebo treatment, none of the subjects were classified as severely depressed, as measured by the HAM-D. As with the prior investigation, both of the exercise treatments consisted of roughly 45 minutes of walking or jogging at a prescribed heart rate three times per week. While one group attended supervised sessions, the home-based group received minimal assistance from staff after an initial home visit. To ensure adherence to the exercise program, home-based exercisers completed weekly exercise logs. Subjects in the sertraline or pill placebo conditions received medication management by the staff psychiatrist, who was blinded to treatment condition. After four months of treatment, all of the active treatment conditions significantly reduced depressive symptoms when compared to placebo, with no significant differences between the active treatments. The setting of exercise, whether group or home-based, did not impact remission rates.

As a whole, studies comparing exercise to standard treatments show that exercise is just as effective as cognitive therapy or antidepressant medication when it comes to initial reductions in depression in a clinical population. In fact, exercise may be superior to medication in terms of relapse. Because of this, aerobic exercise has been

recommended by some as a monotherapy for the treatment of mild to moderate

depression. While some have used exercise as a sole treatment for depression, others

have used it in addition to existing treatments. The use of exercise in combination with

medication was one part of the investigation by Blumenthal et al. (1999); however, other

studies have specifically focused on the use of exercise as an adjunct treatment in those

who have not responded to traditional treatment.

The use of exercise as an adjunct treatment was recently examined using subjects

who had not responded adequately to medication, over the course of an eight month

exercise intervention (Pilu, Sorba, Hardoy, Floris, Mannu, Seruis, et al., 2007). Subjects

consisted of 40 to 60 year old females who had been diagnosed with MDD. In addition,

these subjects had not responded adequately to at least one antidepressant, defined as a

HAM-D score above 13 after at least two months of treatment. After deciding to enter

the study, subjects were randomized into two groups. One group would simply continue

pharmacological treatment, while the other group would add physical activity to the

existing treatment. It should be mentioned that no particular medication was used in all

patients. Instead, multiple classes of antidepressant medication were used in both the

exercise and the control group. The exercise program consisted of two 60 minute bouts

per week, combining aerobic and weight training activities in a group setting. At the end

of the eight month study, those in the exercise group experienced greater improvements

in depression and global functioning in comparison to the control group. While subjects

who merely continued antidepressant medication failed to show improvement in HAM-D

scores, those who added exercise to their existing medication did show improvements in

depressive symptoms. However, it is possible that the social interaction involved with

the exercise sessions played a role in this improvement (Pilu et al., 2007). Taken together

with previous research, combination treatment seems like an effective next step in

treatment-resistant depression, although it may not be necessary as an initial course of

action.

In addition to comparing aerobic exercise to traditional treatments, different

exercise programs have also been compared for their effects on depression. These

exercise programs may vary in the frequency, intensity, social nature of the program, or

in the type of exercise performed. Because of the widely varying types of exercise

programs used, it is difficult to draw firm conclusions about the most beneficial exercise

program for depression, in terms of the frequency or intensity of exercise. Because the

participation in an exercise program seems to be the most important factor, rather than

the specific qualities of the exercise program, many researchers concur with current

ACSM recommendations of 30 minutes of moderate activity on most, if not all days of

the week to improve or maintain physical and mental health.

One factor that has been examined is the setting or group nature of exercise

programs. Because depression is such an isolating illness, it is thought that encouraging

social connections and social support may play a beneficial role for recovery. However,

studies examining the social nature of exercise programs have not demonstrated this.

When comparing home and group-based exercise in a sample of depressed patients, both

groups experienced similar reductions in depression (Blumenthal et al., 2007).

The social nature of exercise has also been examined by King, Taylor, & Haskell

(1993). Before assignment into groups, participants were evaluated on multiple

psychological and physiological assessments. Physiological measures included body

weight, smoking status, and a symptom-limited treadmill test using a modified Balke protocol. Psychological measures included the BDI, the Taylor Manifest Anxiety Scale, the Perceived Stress Scale, and ratings of perceived change. These assessments were repeated at 6 months and at the end of the 12 month intervention. Subjects consisted of 357 healthy, but sedentary individuals between the ages of 50 and 65. They were assigned to one of four groups: high intensity group exercise, high intensity home exercise, lower intensity home exercise, and assessment-only control. Group-based exercise sessions were conducted at a local senior center and community college, where subjects participated in walking and jogging three times a week. Subjects in the home exercise groups were given instruction on measuring heart rate and given exercise logs and written instructions about the exercise program. Subjects using the higher intensity program were instructed to maintain a heart rate at 73-88% of their peak heart rate, while subjects in the lower intensity group maintained their heart rate at 60-73% of their peak heart rate. To ensure that caloric expenditure was similar between the low and high intensity groups, the lower intensity group was advised to exercise 5 days per week, while the high intensity group was advised to exercise 3 days per week.

Using the exercise logs and attendance at group exercise sessions as measures of adherence, it was found that those in the home-based exercise programs reported significantly greater adherence to the exercise program compared to the group-based exercisers. All exercise groups experienced a small, but significant increase in aerobic capacity when compared to the control group (King et al., 1993). Unlike the physiological changes, the exercise groups did not show significant differences from the control group with respect to BDI scores. However, the ratings of perceived change did

show that subjects in the exercise groups perceived an improvement in depression over the course of the 12 month program (King et al., 1993). This perceived improvement in depression was not reflected in the BDI scores, perhaps because this was not a clinical population and BDI scores were fairly low initially. Importantly, there were no differences between the three exercise programs in regards to any of the outcome measures. Thus, it was the participation in exercise, rather than the specific qualities of that exercise, which seemed to produce any health benefits.

The social nature of exercise has also been studied by Legrand and Heuze (2007). While the frequency of exercise was a key factor in their intervention, they also showed that there were no significant differences between an individual exercise program and one that utilized a group format. Twenty-three adult participants were recruited from a local sport and fitness facility, and an elevated BDI score (above 16) was a criterion for inclusion in the study. Initially, subjects exercised fewer than three times per week, and less than 20 minutes per exercise bout. Subjects were then randomized into one of three exercise programs: low frequency exercise, high frequency exercise, or high frequency exercise in a group format. The low frequency group, participating in one exercise session per week, served as a control group because these individuals performed the least amount of exercise. The two high frequency groups participated in three to five exercise bouts per week. The intensity and duration of the exercise bouts was similar for all groups, roughly 30 minutes of aerobic activity performed at 60-80% of age-predicted maximal heart rate. For those in the group-based intervention, several strategies were used to promote group cohesion. The BDI was used as a measure of depression, and

assessments were taken at baseline, 4 weeks, and at the completion of the 8 week exercise program.

The results showed that participants in the two high-frequency exercise groups, regardless of social setting, experienced greater reductions in depression when compared to the low-frequency control group. Although the frequency of exercise was an important factor in reducing depressive symptoms, social setting was less important. Although group cohesion was enhanced for those in the group intervention, this did not result in further reductions in depression.   While most participants in the high-frequency groups were no longer classified as depressed (BDI below 13) at the end of the study, regardless of social setting, this was not true for those in the control group. In addition, reductions in depression happened relatively quickly, within the first 4 weeks of the exercise program (Legrand & Heuze, 2007). The authors noted that while social relationships did not have any short term effects on depression, there may be unmeasured long-term effects. The main limitations of the study included a small sample size and the method of recruiting subjects. Because all subjects were recruited from one fitness facility, the results may not generalize to the broader population. In addition, no clinical diagnosis was used to assess participants for the presence of depression, merely self-reported symptoms. Finally, it should be noted that the control group was not completely sedentary, which could minimize true differences between groups.

The social setting of exercise seems to be relatively unimportant for reductions in depression. However, as found by Legrand and Heuze (2007), the frequency of exercise may be an influential factor. While that study used exercise programs of widely varying frequency, comparing those who exercised one day per week with those who exercised

three to five times per week, it did not keep the volume of exercise constant between the programs. Thus, while the frequency of exercise varied, the total amount of exercise also varied. However, other studies have examined the frequency of exercise programs while keeping the total volume of exercise constant. One of these studies compared short and long-bout exercise (Osei-Tutu & Campagna, 2004). Because the ACSM currently recommends that adults should accumulate 30 minutes of activity on most days of the week, this can be accomplished in frequent short bursts of activity or a longer continuous bout of exercise. In this particular study, both exercise programs consisted of 30 minutes of walking, 5 days per week for eight weeks. However, one group accomplished this in one exercise bout, while the other group completed three 10 minute sessions to accumulate 30 minutes of activity. Subjects in the two exercise groups were compared to a control group, which did not make any lifestyle changes during the study. Outcome measures included the POMS to assess mental health, and VO_2 max and percent body fat to assess physical health. The results showed that long-bout exercise had a significant impact on mood compared to those who remained sedentary, and short-bout exercise showed a nonsignificant trend in this direction. Osei-Tutu and Campagna (2004) speculated that the 8 week duration of the exercise program was not long enough to see significant changes in the short-bout group. Unlike most exercise intervention studies, these exercise programs did not have a specific impact on depression, as measured in a subscale of the POMS. While the mental health benefits of short and long-bout exercise differed, the two exercise programs showed comparable changes in aerobic fitness, as both groups improved their VO_2 max relative to the control group. Thus, it is unlikely that psychological improvements were due to improved aerobic fitness. In contrast to

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aerobic fitness, only long-bout exercise was found to reduce body fat. Interestingly, the more body fat was lost by those in the long-bout exercise program, the more their mood improved (Osei-Tutu & Campagna, 2004). Although the overall amount of time spent exercising was the same for both exercise groups, the mental health benefits were not equal. More frequent exercise bouts were not more beneficial; however this may be due to the extremely short duration of the exercise sessions. It is possible that there is a threshold for mental health benefits to occur, and the 10 minute sessions were not sufficient to result in mental health benefits.

While Osei-Tutu and Campagna (2004) manipulated frequency and duration to keep the total volume of exercise constant, others have looked at frequency and volume simultaneously. In one such comparison, four aerobic exercise programs were compared to an exercise placebo to determine the impact of exercise frequency and total energy expenditure (or exercise dose) on depression (Dunn et al., 2005). Subjects initially were mildly to moderately depressed, as assessed by HAM-D scores, and this was confirmed with a clinical interview. The study utilized a 2 X 2 factorial design, emphasizing the frequency (3 or 5 days per week) and energy expenditure (public health dose or low dose) of aerobic exercise. Additionally, an exercise placebo control group was used, and the subjects in this group performed flexibility exercise three times per week. The public health dose of exercise roughly corresponds to current ACSM recommendations for physical activity, while the low dose corresponds to less than half that amount of exercise. To ensure that social support did not confound the results, participants exercised individually in rooms by themselves for the duration of the 12-week study. The results indicated that the public health dose of exercise effectively

treated depression, reducing the HAM-D scores below clinical levels. The low dose groups did not respond better than the exercise placebo, although both groups experienced some reduction in symptoms. Because the frequency of exercise by itself did not impact depression scores, it was determined that total energy expenditure was the important variable of interest for reducing symptoms of depression.

Taken together, the combined results of these exercise interventions show that the frequency of an exercise program by itself has small effects on depression, if any. Rather than the frequency, it is the total volume of activity that is important. Aside from increasing the frequency of exercise, another way to increase the volume of activity is to increase the intensity or duration of individual exercise sessions. As mentioned earlier, King et al. (1993) found no differences between low and high intensity exercise programs when the total volume of activity was kept constant. However, it is possible that no differences were shown because of the population used. Since there were low BDI scores initially, there was not room for much improvement. None of the exercise programs were more beneficial than the control in reducing BDI scores; however all of the exercise programs resulted in perceived benefits from the participants. These perceived benefits did not vary by the intensity of the exercise program.

The intensity of exercise has also been examined in conjunction with the duration of exercise (DiLorenzo, Bargman, Stucky-Ropp, Brassington, Frensch, & LaFontaine, 1999). Similar to King et al. (1993), a community sample was used, and small mental health benefits were expected. The fitness program consisted of 12 weeks of bicycle ergometry, with outcome measures taken at the end of the intervention and at a 1 year follow-up. Subjects were initially randomly assigned into a 24 minute variable intensity

exercise group, a 48 minute continuous intensity exercise group, or a wait-list control group. Those in the exercise groups trained four days per week for the duration of the 12-week study. Psychological constructs, including depression and anxiety, were measured with STAI, the BDI, and some subscales of the POMS. Performance on both maximal and submaximal bicycle tests was used to indicate changes in aerobic fitness. As a manipulation check, both of the exercise groups showed increases in aerobic fitness when compared to the control group, indicating the exercise to be of sufficient intensity. Because both of the exercise groups experienced similar improvements in fitness, they were combined for further analysis. Because of this, one cannot determine which intensity or duration was more beneficial. However, at the end of the aerobic training program, exercisers showed significant improvements in all psychological measures used, including a reduction in depression. In addition, these improvements were maintained over a 1-year follow-up period. DiLorenzo et al. (1999) listed social support and enhanced body image as some of the reasons why the participants experienced mental health benefits, even in the absence of clinical problems.

Thus, preliminary evidence indicates that intensity of physical activity is not a major factor in the antidepressant effects of exercise. While the intensity, frequency, and social nature of exercise do not appear to be important, the total amount of physical activity is a relevant factor. However, there are many different types of exercise, and each may have different effects on depression. In most of the exercise interventions mentioned above, only cardiovascular exercise was used as the mode of activity. The mode of exercise, aerobic or non-aerobic, is another avenue for exploration. Although differences between aerobic exercise programs have been compared, the differences

between aerobic exercise and strength training have also been examined (Motl, Konopack, McAuley, Elavsky, Jerome, & Marquez, 2005; see also Doyne, Ossip-Klein, Bowman, Osborn, McDougall-Wilson, & Neimeyer, 1987).

Motl et al. (2005) compared cardiovascular exercise and strength training in a sample of nondepressed elderly. For the aerobic exercise condition, subjects met at a local shopping mall and walked three times a week. The intensity and duration of the exercise increased throughout the duration of the six month program. Subjects who were randomized into the resistance and flexibility group completed one set of 8-12 repetitions per major muscle group, and flexibility exercises for the large muscle groups. The frequency and duration was the same used for the aerobic group, and increased gradually during the program. Because an older population was used in the study, the Geriatric Depression Scale (GDS) was used to assess depression immediately after the six month intervention and again at one and five-year follow-ups. The primary finding concerned the long lasting reductions in depression following the exercise program. Despite the fact that subjects were not clinically depressed, the reduction in depressive symptoms was maintained at all follow-up assessments. These changes in depression did not differ between the two exercise groups, as both were effective in reducing depression. Because the subjects were not clinically depressed, this supports exercise for the prevention of depressive symptoms in the general population of older adults.

Doyne et al. (1987) also compared aerobic exercise to weight training, although using a much shorter follow-up time period. Instead of using a nondepressed older population, subjects were limited to 18-35 year old women diagnosed with major or minor depressive disorder. Participants were advised to attend four exercise sessions per

week over the eight-week program. Subjects randomized to the aerobic exercise

condition walked or ran around an indoor track, while subjects in the weight-lifting

condition completed a 10-station program using a Universal Exercise Machine. In

addition, a wait-list control was used, and subjects in this condition were told the exercise

program would be delayed for eight weeks. Like the study done by Motl et al. (2005),

both exercise groups showed reductions in depression symptoms when compared to the

wait-list control group, and these improvements were maintained throughout the one-year

follow-up period. Despite the vastly different nature of the two exercise programs, they

were equally effective in reducing depression (Doyne et al., 1987).

As a whole, the literature on exercise interventions shows that participation in an

exercise program reduces symptoms of depression. While some studies have looked at

specific features of exercise programs, these have all shown large differences between

exercise and control groups, and few if any differences between different exercise groups.

While the frequency, intensity, mode, and social nature of exercise programs do not

appear to be important, the total amount of activity appears affect symptoms of

depression. The temporal relationship between exercise and depression in exercise

intervention studies gives evidence towards the notion that exercise plays a causal role in

the reduction in depression. In addition, experiments rule out the possibility of

spuriousness through randomization and other methods in their design.

Review Articles and Meta-Analyses

Due to the sheer number of exercise intervention studies, it is no surprise that

there have been several attempts to summarize this literature in the form of narrative

reviews and meta-analyses. These reviews often note the differences in the measurement and classification of depression, as well as the variety of populations studied. For example, some reviews allow only studies using a clinical population, while others include both clinical and nonclinical populations. In addition, they examine different types of exercise programs, and consider possible mechanisms that explain the relationship between exercise and depression.

One of the earliest reviews sought to examine the use of exercise in a clinically depressed population (Martinsen, 1990). At the time, diagnostic systems such as the Research Diagnostic Criteria (RDC) and the DSM-III were the best tools available to determine the presence of clinical depression. Martinsen (1990) noted that while the BDI was often used, this type of self-report was not adequate for obtaining an official diagnosis. Each of the 10 studies included used at least a quasi-experimental design, and 8 of these were true experimental studies using random assignment to treatment conditions. Six of the 10 included studies used strict diagnostic criteria to determine the presence of clinical depression, rather than simply using self-reported symptoms. All the included studies showed that exercise programs resulted in significant improvements in depression. When exercise was compared to standard treatments for depression such as psychotherapy, exercise was comparable or even superior to counseling. However, it is unclear if the counseling offered was effective, as at least one study did not report significant reductions in depression for the group receiving psychotherapy.

Two studies included in Martinsen's (1990) review compared aerobic and non-aerobic exercise, and found that they had similar benefits for depressive symptoms. Cardiovascular exercise and strength training were both superior to a control group.

Despite the small number of studies included and methodological limitations such as small sample sizes, no study showed that exercise had harmful effects, and all pointed towards the benefits of exercise on depression. Therefore, Martinsen (1990) concluded that "aerobic exercise is more effective than no treatment, and not significantly different from other forms of treatment" (p. 384). In addition, changes in aerobic fitness did not appear to be necessary for mental health benefits to occur. Because similar benefits resulted from aerobic and strength training exercise, Martinsen (1990) argued that psychological mechanisms were likely responsible for the improvement in depression, since the two forms of exercise engage different biological pathways.

A later review examined exercise therapy for patients with a variety of psychiatric disorders, including but not limited to depression (Tkachuk & Martin, 1999). In their investigation, they separated studies by the primary diagnosed psychiatric disorder, and exercise was used as a primary or adjunct treatment for that psychiatric disorder. Exercise was found to have the most beneficial impact on depression when compared to other psychiatric disorders. In the 14 studies used to evaluate the effectiveness of exercise in alleviating depression, all found exercise to be beneficial. Similar to Martinsen's (1990) review, Tkachuk and Martin (1999) included quasi-experimental and experimental studies using a clinical population of mild to moderately depressed patients. Many of the included studies used diagnostic criteria to determine the presence of clinical depression, and all were clear about the specific nature of the exercise program used. A minimum of three 20-60 minute exercise sessions per week resulted in significant improvements in depression after only five weeks. Importantly, exercise was found to be more cost-effective than standard treatments. For diagnoses other than depression, such

as schizophrenia or substance abuse disorders, evidence was mixed or there were too few studies to draw sound conclusions.

While the effect of exercise on a variety of psychological disorders was studied by Tkachuk and Martin (1999), Dunn, Trivedi, and O'Neal (2001) examined a very limited set of psychological disorders. The *International Classification of Disease* (ICD) 9, ICD-10, and DSM-IV diagnostic criteria were used to determine the presence of primary depressive and anxiety disorders of interest. Hence, only studies utilizing clinical populations were used in the analysis. In total, 37 studies met inclusion criteria for the review. Analysis of cross-sectional studies showed a clear inverse relationship between physical activity and depression, and the prospective studies included indicated that changes in physical activity predicted later depressive symptoms. Despite the clear inverse relationship, only one prospective study examined the potential dose-response gradient between physical activity and depression. Dunn et al. (2001) also looked at clinical relevance, and they defined a treatment response as a 50% reduction in symptoms. Although smaller reductions may be statistically significant, reductions less than 50% were not considered clinically meaningful. Of the 18 exercise training studies included, 8 showed a treatment response which was maintained during follow-up periods. While exercise was shown to be beneficial, the studies were limited by small sample sizes, lack of control groups, and combining exercise with other treatment modalities. The three studies that examined resistance training showed it to be as effective in reducing depression as the more commonly used aerobic activity. In short, while both moderate and vigorous exercise were shown to reduce symptoms of depression, it is

unclear whether this occurs as a direct result of manipulating the intensity, frequency, or duration of exercise in a dose-response manner.

While previous reviews have focused on clinical populations, others have included individuals with a wider range of depressive symptoms. Brosse, Sheets, Lett, and Blumenthal (2002) noted that some exercise training studies have shown reductions in depressive symptoms in a healthy population, while others have not shown these same benefits. Most likely, this is because there is not much room for improvement in healthy, nondepressed subjects. Among those with chronic medical conditions, evidence for the antidepressant effects of exercise was also mixed. Some studies in the review by Brosse et al. (2002) reported reductions in depression for individuals with chronic medical conditions, while others did not. However, it should be noted that some, but not all individuals with chronic medical conditions were also clinically depressed. This is likely to explain the mixed findings. The antidepressant effects of exercise became more apparent when a clinically depressed population was studied, because there was more room for improvement. Exercise was more effective in reducing depression than no treatment, and the antidepressant effects of exercise were comparable to that of psychotherapy and medication. Nevertheless, Brosse et al. (2002) noted that it is uncertain whether combining exercise with standard treatments produces an additive effect. The authors recognized its promise, however they concluded that "if the prescription of exercise for MDD required approval from the FDA, it probably would not pass current standards" (Brosse et al., 2002, p. 754).

While Brosse et al. (2002) specifically looked at depression, Landers and Arent (2001) took a much broader view of mental health. In addition to examining the

antidepressant effects of exercise, Landers and Arent (2001) also noted exercise's

potential benefits to cognitive functioning, anxiety, stress reactivity, and self-esteem. In

particular, exercise had beneficial effects on depression for all types of people, varying in

gender, fitness, activity level, physical health, and depressive status. Exercise had

especially large benefits to clinical patients who were moderately to severely depressed,

as well as individuals who were initially unfit. Similar to other review articles, Landers

and Arent (2001) note the equal benefits for aerobic and resistance exercise. While there

was some evidence for a dose-response relationship, there appeared to be a threshold

effect for the antidepressant effects of exercise to occur.

In addition to qualitative reviews, quantitative meta-analysis has also been used to

summarize the literature on physical activity and depression. Some meta-analyses have

included only randomized controlled trials, while others have included studies of lesser

quality. Lawlor and Hopker (2001) analyzed 14 randomized controlled trials, and found

a large effect size of 1.1. All the studies used an adult population, and many of the

studies used the BDI as their measure of depression. In addition to the large effect size,

the authors found that across studies, BDI scores decreased by approximately seven

points. Despite these strong findings, Lawlor and Hopker (2001) concluded that it was

not possible to determine the effectiveness of exercise in the management of depression

due to methodological weaknesses in the studies examined. Some of the methodological

weaknesses mentioned include a lack of follow up, adequate concealment, intention to

treat analysis, and blinding. In addition, Lawlor and Hopker (2001) recommended that a

dichotomous measure of depression be used to better assess clinical relevance, as this is

more clinically meaningful than change scores on the BDI.

A more recent meta-analysis calculated an effect size of 1.42, indicating a very large effect for exercise interventions relative to control conditions (Stathopoulou, Powers, Berry, Smits, & Otto, 2006). To be included in the meta-analysis, studies were required to use an inactive control group and focus on affective disorders as an outcome. Among the included studies, the frequency of exercise varied from twice to four times per week. The duration varied from 20 to 45 minutes, and the intensity ranged from unspecified to 70-85% of maximal heart rate. Stathopoulou et al. (2006) found that although more recently published studies were associated with higher effect sizes, it would require over 350 unpublished null findings to reduce the overall effect size to nonsignificance. When aerobic and anaerobic exercise were compared, they reduced depression equally. When exercise was examined in combination with standard treatments such as pharmacotherapy or psychotherapy, there was little additional benefit from combining treatments.

Opposite Temporal Sequence: Changes in Depression Precede Changes in Exercise

While studies examining exercise as a causal factor are plentiful, the notion that depression may influence exercise habits has been examined less frequently. Partly, this is because depression is a characteristic of an individual, and cannot be manipulated as easily as exercise behavior. Research subjects can be assigned to high or low levels of exercise, but they cannot be assigned to high or low levels of depression. Even though most longitudinal studies have looked at depression as an outcome, a few have investigated the possibility that depression could reduce or inhibit exercise. The few

studies that have done so have generally looked at psychosocial correlates of exercise behavior.

One study that did examine depression as a factor affecting physical activity was only able to do so using cross-sectional data, and was therefore unable to draw conclusions about causation (Kaplan et al., 2001). In addition to examining a wide variety of demographic, psychological, and physical factors in relation to exercise, the researchers overtly sought to determine if individuals experiencing greater levels of psychological distress were less likely to engage in physical activity. The National Population Health Survey was used to collect data from older individuals in Canada. A six-item version of the Generalized Distress Scale (GDS) was used to assess symptoms of anxiety and depression. The frequency of moderate-intensity physical activity was also measured, provided that the activity occurred in bouts lasting 15 minutes or more. Subjects who exercised at least three times per week were classified as frequent exercisers, while those who participated in less physical activity were categorized as infrequent. Results indicated that psychological distress was associated with decreased physical activity for both men and women. However, the use of cross sectional data merely confirms the known inverse association between exercise and depression; it cannot be used to infer causal relationships between them.

Other researchers have examined exercise in the context of overall health behavior, where depression can lead to an unhealthy lifestyle in general (Fulkerson, Sherwood, Perry, Neumark-Sztainer, & Story, 2004; see also van Gool, Kempen, Penninx, Deeg, Beekman, & van Eijk, 2003). Fulkerson et al. (2004) looked at depression in relation to a variety of health-compromising and health-promoting

behaviors in a sample of adolescents, focusing on eating and nutritional factors in addition to physical activity and sedentary behavior. A modified version of the Godin Leisure Time Exercise Questionnaire (GLTEQ) was used to assess physical activity, and strenuous and moderate responses were summed to give a moderate-to-vigorous activity score. Weekend and weekday television viewing were used to assess sedentary behavior. Results indicated that depressive symptoms were negatively associated with physical activity for males, but not females. Females in the moderate-depressive symptom group spent significantly more time watching television than those in the low or high depressive symptom group. Aside from physical activity, adolescents who reported more depressive symptoms were less likely to engage in health promoting behaviors, such as eating regular meals. Depressive symptoms were also linked with the use of cigarettes, marijuana, alcohol, and other drugs. Although the study provided evidence for an association between depression and an unhealthy lifestyle, its cross-sectional nature precluded conclusions about causation.

Building off the cross-sectional data linking depression and unhealthy lifestyles, longitudinal studies investigating these variables have been undertaken. As part of the Longitudinal Aging Study Amsterdam (LASA), individuals aged 55-85 were asked about a variety of health behaviors, including physical activity, over a period of six years (van Gool et al., 2003). Depression was measured by the CES-D scale, and a score of 16 was used as the cutoff for depression. Initial categorization (depressed or not depressed) and change scores were used in the analysis. Physical activity was measured in minutes per day of various types of exercise and household chores. Physical activity was then dichotomized into the lowest quartile, representing a sedentary lifestyle, and the upper

three quartiles, representing an active lifestyle. Longitudinal analysis of the data showed that those with emerging depression (individuals who were not initially depressed but who were depressed at follow-up) were significantly more likely to adopt a sedentary lifestyle. Those whose initial depression remitted tended to become more physically active. Despite these results, van Gool et al. (2003) noted that "the inference of depression being a risk factor for simultaneous physical inactivity cannot be made" (p. 85). In addition to its relationship with exercise, clinically relevant depressive symptoms were associated with other indicators of an unhealthy lifestyle, such as smoking behavior and excessive alcohol use.

The previous three studies were unable to draw causal inferences due to the use of cross-sectional data or because of concurrent changes in exercise and depression in the longitudinal analysis. Perhaps the strongest evidence towards the idea that depression influences exercise comes from a study utilizing a cardiac population (Allan et al., 2007). Depression is relatively common in the first year after a myocardial infarction or other coronary event, yet its treatment can be more complicated in this population because of underlying medical issues. Drawing from the literature linking depression and cardiac events, Allen et al. (2007) noted that adherence to medical treatment was a behavioral mechanism potentially linking the two. While adherence to diet and medication is important, the researchers focused on depression's effects on exercise adherence. One of the study's goals was to determine whether depression immediately after hospitalization predicted exercise and fitness 12 months later, and whether the predictive effects of depression and perceived behavioral control were independent of each other. Approximately 600 subjects were recruited into the study during hospitalization for a

coronary syndrome event. Prior to discharge from the hospital, depression was assessed using the Hospital Anxiety and Depression Scale (HADS). Items pertaining to anxiety were not relevant to the study and excluded from analysis. At the 12 month follow-up, exercise was measured using the GLTEQ, and subjects were asked if they were taking regular exercise. Fitness was measured using a 6 minute walk test, and the HADS was administered again. Results of logistic regression analysis showed that depression prior to hospital discharge reliably predicted fitness and physical activity level 12 months later. However, depression assessed at the 12 month follow-up was a much stronger correlate of fitness and activity level than initial depression. While this could be due to the time difference between assessments, initial depression may predict future activity because it predicts future depression. Interestingly, the predictive effects of depression remained when clinical cases were excluded, indicating that even low levels of depressive symptoms could negatively influence exercise behavior (Allan et al., 2007). The authors concluded that perceived behavioral control and depression independently predict physical activity and fitness 12 months after hospitalization from a coronary event.

In summary, the literature examining depression as a factor influencing physical activity is less plentiful and of lesser quality research than the research noting the antidepressant effects of exercise. Because depression is a variable attributed to an individual, it cannot be experimentally manipulated to observe its effects on physical activity. In the absence of true experiments, more longitudinal research needs to be conducted before ruling out this possibility. In addition, many of these studies use exercise in conjunction with other indicators of a healthy lifestyle. While this is a

worthwhile area of research, it would also be helpful to focus on the direct effects of depression on exercise, without other health behaviors.

Other Possibilities

The evidence supporting exercise as a causal factor in the reduction of depressive symptoms is plentiful, while the evidence for depression playing a causal role is sparse. However, there are some studies that do not overtly adopt either of these viewpoints in their investigation of exercise and depression. One such inquiry looked at predictors of early dropout and treatment failure in an exercise and medication intervention for depression (Herman, Blumenthal, Babyak, Khatri, Craighead, Krishnan, et al. 2002). Psychological factors such as anxiety and life satisfaction were predictive of treatment dropout and failure, regardless of initial treatment group. However, severity of depression, as measured by HAM-D score, did not predict treatment dropout. The severity of depression did, however, contribute to the prediction of treatment failure. The overall dropout rate for patients in the exercise groups was relatively low (23%), and did not differ markedly from the dropout rate for those receiving medication only. Because attrition rates for exercise are typically much higher, Herman et al. (2002) remarked that "clinicians familiar with the often profound motivational deficits and low energy exhibited by depressed patients may wonder why the dropout rates for exercise therapy were not higher" (p. 559). These types of comments give insight into the relationship between exercise and depression that statistical analysis alone cannot. Exercise

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interventions may be highly effective at reducing depression, but only if individuals with depression are willing and able to undergo an exercise program.

In addition to statistical analysis, the feasibility of exercise therapy for individuals with depression was also studied using qualitative methods. Specifically, the unique motives and barriers to exercise in a depressed population were examined (Faulkner & Biddle, 2004). Six clients with clinical depression were used as a convenience sample for the research. All of them had been referred to a local fitness center by a community mental health team, where they were then recruited into the study. All had the sole diagnosis of clinical depression, and all were taking antidepressant medication. Each individual participated in five semi-structured interviews over the course of a year. In the end, three types of profiles were shown in regards to exercise adherence: the initial enthusiast, the late starter, and the regular attendee. The initial enthusiast used the referral to the fitness center as a start towards becoming more active, citing weight control and other health reasons as motives for attending. Even after six weeks, the initial enthusiast went to the fitness center to relieve boredom and get out of the house. However, employment issues later took priority over attendance at the fitness center. Despite the lack of attendance at the center, the initial enthusiast continued to remain active in other ways. The slow starter showed almost the exact opposite pattern from the initial enthusiast. For the slow starter, initial attendance was minimal but increased throughout the year. Initially, the slow starter was hesitant to use available social support and experienced a lack of confidence in her ability to use the fitness center. Although a sense of achievement was noted for the slow starter, the lack of confidence in social situations was a pervasive barrier. The regular attendee also cited lack of confidence and

social anxiety as barriers to attending the fitness center, although it did not prohibit the regular attendee from visiting the center several times a week (Faulkner & Biddle, 2004)

As a whole, all three profiles found in qualitative research viewed exercise as a coping strategy in the recovery from depression, rather than treatment for it. This concurs with experimental work by Bartholomew et al. (2005), a single exercise session was viewed as a potential coping strategy for dealing with the daily effects of depression, rather than treatment per se. Initially, all participants in Faulkner and Biddle's (2004) study mentioned structure and distraction as motives for initiation of exercise. In addition, a sense of accomplishment was mentioned by all subjects, even though this was relatively transient. Distraction and mastery have been widely noted as potential psychological mechanisms for the antidepressant effect of exercise. Although there are many other psychological and physiological mechanisms explaining the antidepressant effects of exercise, a complete discussion of them is beyond the scope of this paper. Many of the barriers to physical activity mentioned by the participants in Faulkner and Biddle's (2004) study were not vastly different from barriers for nonclinical populations. Life events also influenced the subjects' exercise habits. Despite their awareness of the benefits of exercise, there were times when participation was difficult for all subjects. While depressive symptoms sometimes made it difficult to exercise, other times it was life events that took priority. While some exercise intervention studies show little dropout, this is likely to be markedly different from exercise undertaken by those in the community (Faulkner & Biddle, 2004).

Qualitative research, such as the study by Faulkner and Biddle (2004), provides detail about individual experiences with exercise and depression that many quantitative

studies cannot. However, the two approaches provide complementary information about the relationship between exercise and depression. Cross-lagged panel correlation is one quantitative method used to study the relationship between exercise and depression without adopting an initially strict view of cause and effect. Prior to this research project, a group of Japanese researchers used a cross-lagged panel design in conjunction with structural equation modeling to investigate age differences in the relationship between exercise and depression (Fukukawa, Nakashima, Tsuboi, Kozakai, Doyo, Niino, et al.., 2004). The subjects consisted of approximately 1100 middle-aged and older individuals, and two years elapsed between measurements. A Japanese translation of the CES-D was used to measure depressive symptoms on at both time points. To measure usual physical activity, subjects wore a pedometer for 7 days, and the number of steps was averaged. While walking steps and depressive symptoms remained relatively stable during the follow-up period, the results of the cross-lagged analysis showed that those who walked more at baseline experienced fewer depressive symptoms at follow up, particularly in the older group (age 65-79). However, this was not the case for middle aged adults (age 45-64). Fukukawa et al. (2004) reasoned that walking was more strenuous exercise for the older adults, suggesting that age and fitness should match the intensity of exercise for optimal antidepressant effects. While strengths of the study included a large sample size and the use of objective physical activity measures, it was limited by a somewhat weak (but still statistically significant) antidepressant effect of exercise. While the model predicting depression from baseline physical activity was the best fit model when structural equation modeling was applied, all models used fit the data well. By examining the strength of the temporal relationships between depression and exercise,

Fukukawa and colleagues (2004) inferred that walking has a protective effect against depression in older, but not necessarily middle-aged, individuals.

In summary, most of the research concerning exercise and depression focused on the notion that changes in exercise precede changes in depression. There are numerous exercise intervention studies and meta-analyses that adopt this viewpoint, and mechanisms for the presumed causal effect have been proposed, most of which are beyond the scope of this paper. However, a much smaller body of literature has investigated the opposite causal pathway, where depression influences exercise habits. Both viewpoints can be helpful in explaining the relationship between exercise and depression, and some qualitative and quantitative methods can be used to infer which cause-and-effect relationship plays the greater role.

Chapter 3: Methods

Subjects

The subjects consisted of students enrolled in various kinesiology classes at the University of Maryland, College Park. Most of these students were enrolled in large, lecture-style classes, but some were taking smaller, activity-based courses. The particular classes surveyed include the following: a general sport psychology course, an upper-level sport psychology course, a circuit training class, and two tennis classes. These classes contain students majoring in kinesiology, as well as non-majors. The classes surveyed had combined rosters of approximately 260 students. The sample was not chosen to reflect the characteristics of the general population in the College Park area, nor is it a representative sample of all students enrolled at the university. However, the exercise habits of these students were believed to be extremely diverse, with some getting no physical activity and others exercising frequently. The perceived range of physical activity was believed to be beneficial for analysis.

Variables and Instrumentation

Because this study aims to study the cause-and-effect relationship between exercise and depression, strict interpretations of independent and dependent variables as cause and effect cannot be used. However, due to the preponderance of literature suggesting exercise as the presumed cause of reduced depression, exercise served as the

independent variable in this study. On a conceptual level, exercise can be defined as any physical activity performed with the intent of changing the capacity of the body to do work. The broad term "exercise" includes various dimensions, such as the duration, frequency, intensity, and type of exercise.

Exercise was measured using the Godin Leisure-Time Exercise Questionnaire (GLTEQ). The GLTEQ is a self-report instrument that measures a subject's usual physical activity, and a copy of the questionnaire can be found in Appendix A. It asks about the frequency with which an individual performs various mild, moderate, and vigorous activities during a typical week, provided that each activity is performed in bouts of ≥ 15 minutes. From this information about frequency and intensity, it then produces a physical activity score. The formula for the physical activity score is as follows: Activity Score = (9 * Strenuous) + (5 * Moderate) + (3 * Mild). Higher scores are indicative of a high volume of physical activity. Reliability for the instrument is acceptable, as the total score has a test-retest reliability of .62-.81, depending on the time interval used. The validity of the instrument is fair when compared to a Caltrac activity monitor, NASA, and Baeke questionnaires (.45, .54, and .61 respectively). The portion of the questionnaire devoted to strenuous physical activity was shown to be valid at the p $< .05$ level when measured against tests of VO_2 max and body composition (Godin and Shephard, 1997). It is important to keep in mind that the Godin questionnaire is a measure of exercise behavior, not a measure of fitness. It is therefore not surprising that the portions of the questionnaire devoted to moderate and light activity were not significantly correlated to VO2max, a measure of aerobic fitness.

Depression, on a conceptual level, is the extent to which a person shows symptoms of the illness. As mentioned previously, these symptoms include a low mood, a loss of pleasure or interest in usual activities, and other cognitive, affective, and somatic symptoms. On an operational level, depression was measured using the second edition of the Beck Depression Inventory (BDI-II). Like the original BDI, the BDI-II aims to measure the presence and severity of depression in depressed and non-depressed adolescents and adults. The BDI-II is a 21-item self-report instrument where subjects are asked to report the severity of various depressive symptoms on a scale of 0-3. Scores on individual items are summed to produce a total score, with higher scores indicative of more severe symptoms. A copy of the BDI-II can be found in Appendix A.

The test-retest reliability of the BDI-II was .96 when students seeking help from a university counseling center were given the questionnaire before treatment and during their first visit one week later (Sprinkle, Lurie, Insko, Atkinson, Jones, Logan, et al. 2002). In addition, split-half reliability estimates in this study ranged from .91 to .93, indicating a high reliability for this instrument. Another study found the internal consistency of the BDI-II to be .91 (Dozois, Dobson, and Ahnberg, 1998). The validity of the BDI-II is also high. When compared to a structured interview by clinicians, students' BDI-II scores were strongly correlated (.83) to the number of symptoms shown in the clinical interview (Sprinkle et al., 2002). When compared to the original BDI, the correlation between the BDI and the BDI-II was .93 ($p < .01$), although scores on the BDI-II were significantly higher (Dozois et al., 1998).

Cross-lagged panel correlation was used to examine the strength of the temporal relationships, which could then be used to make causal inferences about the association between exercise and depression. While a true experiment would be best to rule out alternative variables, experiments require the use of random assignment and manipulation of variables. This is problematic with many psychological constructs, because a researcher cannot randomly assign subjects to varying levels of depression. Although cross-lagged panel correlation is not an experimental design, it can be used with variables that cannot be experimentally manipulated. This quasi-experimental design necessitates the use of two variables, in this case exercise and depression, measured at two points in time. From this, six correlations are calculated: two autocorrelations, two synchronous correlations, and two cross-lagged correlations (Kenny, 1975). This is graphically represented in Figure 1.

Cross-lagged panel correlation depends on the use of two assumptions to test for spuriousness: synchronicity and stationarity. If the two variables under study are measured at the same time, then the assumption of synchronicity is met. However, retrospection, or asking subjects about past behavior, can be problematic when synchronicity is assumed. This study addressed the problem of retrospection by using a measure of typical physical activity, rather than activity performed in the past week or month. Some might consider retrospection a problem with using the BDI-II, since it asks respondents to describe their feelings in the previous two weeks. However, this two week time interval is inherent to the DSM-IV definition of depression. Even though the questionnaire asks about the respondents' feelings in the previous two weeks, it measures

depression levels at the time of survey administration. Therefore, depression and

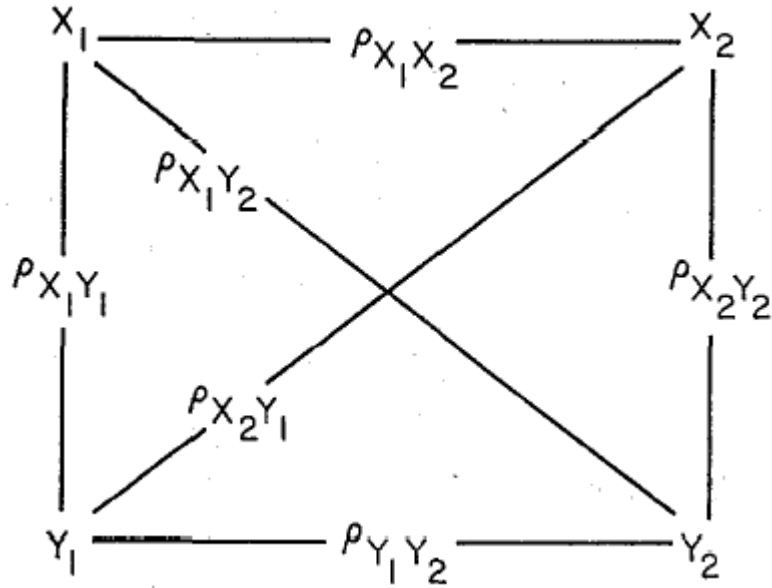exercise were assessed at the same points in time.

Figure 1: Graphical representation of the cross-lagged panel correlation. X and Y represent the variables and 1 and 2 representing the times. The autocorrelations are indicated by the top and bottom of the square, while the synchronous correlations are shown as the left and right sides. The cross-lagged correlations are found in the middle of the box. (Kenny, 1975)

The assumption of stationarity is met when the causal processes between the two

variables do not change over the course of the measurements. Stationarity is related to

the stability of the variables, measured by the autocorrelation. However, stability refers

to the lack of change of the empirical values of the variables, while stationarity refers to

the lack of change of the causes of a variable. If the synchronous correlations are equal,

this is considered evidence of stationarity. If the two assumptions of synchronicity and

stationarity are met, then the values of the cross-lagged correlations are examined to infer

the dominant causal relationship (Kenny, 1975).

Data Collection Procedures

Students completed the surveys at two time points separated by approximately one month. In no class did the time lag vary from one month by more than four days. This time frame was chosen because it allows enough time for the exercise habits and the depressive status of individuals to change yet it is short enough to minimize problems with follow-up. For each distribution, students were asked to complete the GLTEQ and the BDI-II. To reduce problems with nonresponse, surveys were distributed during regular class meeting times during the semester. In addition, consent forms were included in the first round of data collection. The subjects read and signed the consent form, then completed the two surveys. The consent form included with the first round of surveys can be found in Appendix B. In the second round of data collection, only the two surveys were included. To protect the students' identities, student identification numbers assigned by the university were used to track responses from one round of data collection to the next. The procedures used were approved by the Institutional Review Board at the University of Maryland- College Park before any data collection began.

Statistical Analysis

Descriptive information, such as the standard deviations and means, were calculated for each of the variables at each time point. Cronbach's alpha (1951) was calculated to determine the interitem reliability of the BDI-II and the Godin questionnaires. Pearson correlation coefficients were also calculated for all possible combinations of variables and time points. This includes the autocorrelations for exercise and depression, the stationary relationships between exercise and depression at baseline and follow-up, and the two cross-lagged correlations. Each correlation was tested for

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significance using a one-tailed t-test because the direction of association could be predicted from the literature.

Pairs of correlations were then compared using a modification of the Pearson-Filon test (1898). This test is designed to test for significant differences between two nonoverlapping correlations from a single sample, and it is frequently used in cross-lagged panel correlation analysis. Although the original Pearson-Filon statistic is still used, Steiger (1980) modified the statistic by transforming the original correlations into Fisher's z-scores. Raghunathan, Rosenthal, and Rubin (1996) demonstrated the superiority of this ZPF statistic, gaining accuracy without losing power. Because of this, the ZPF statistic was used to compare the two autocorrelations, the two synchronous correlations, and the two cross-lagged correlations. Since neither Microsoft Excel nor other available statistical software calculates this statistic, it was calculated by hand using the equations listed in their article (Raghunathan et. al. 1996). For ease of reference, the equations are also included here. Equation 3 gives the full formula for calculating the ZPF statistic, where k is given by Equation 2.

$$
\begin{aligned}
k = & (r_{12} - r_{24}r_{14})(r_{34} - r_{24}r_{23}) \\
& + (r_{13} - r_{12}r_{23})(r_{24} - r_{12}r_{14}) \\
& + (r_{12} - r_{13}r_{23})(r_{34} - r_{13}r_{14}) \\
& + (r_{13} - r_{14}r_{34})(r_{24} - r_{34}r_{23});
\end{aligned}
\qquad (2)
$$

$$
ZPF = \sqrt{\frac{N-3}{2}} \; \frac{(Z_{14} - Z_{23})}{\sqrt{1 - \dfrac{k}{2(1 - r_{14}^2)(1 - r_{23}^2)}}},
\qquad (3)
$$

Chapter 4: Results

Initially, 214 students from various kinesiology classes completed the first round of surveys. Of these 214 originally surveyed, 178 were available for the one month follow-up analysis, giving an adequate response rate of 83.1%. Because 36 of those originally surveyed were not available for follow-up analysis, the information obtained from those individuals was not used in any of the statistical analysis that follows. The effective sample size for this study was therefore 178. Kenny (1979) recommended using a moderate sample size, defined as 75-300 subjects, in order to obtain adequate power for the test. Since the sample size used in this study falls within that range, this study has adequate power according to Kenny's recommendations, and a more thorough power analysis will not be undertaken.

Table 1 shows the means and standard deviations for the GLTEQ and the BDI-II, at both the baseline and one-month follow-up. Table 1 also includes other descriptive information at both time points. As expected, this student population showed a wide range of physical activity, with scores varying from 0 to 178 at baseline and 0 to 495 at follow-up. While some students performed no physical activity in their leisure time, others were highly active. Although there was a large range of physical activity, depression scores were more homogenous. Depression scores varied from 0 to 28 at baseline, and from 0 to 23 at follow-up. Mean depression scores were low (approximately 5 at both time points), suggesting that a majority of students were not depressed. This was expected since a clinical sample was not used.

Table 1: Descriptive Statistics for Exercise and Depression at Time 1 and Time 2

	N	Minimum	Maximum	Mean	Mode	Median	Std. Deviation
Ex1 Total	178	0	178	62.34	43	60	27.849
Dep1 Total	178	0	28	5.07	0	3	5.069
Ex2 Total	178	0	495	65.61	43	61	43.902
Dep2 Total	178	0	23	4.92	0	3	5.390
Valid N	178						

Table 2: Internal Consistency

	Cronbach's Alpha	N of Items
Exercise, Time 1	.491	4
Depression, Time 1	.868	21
Exercise, Time 2	.701	4
Depression, Time 2	.896	21

Cronbach's alpha was used to calculate the interitem reliability for each of the measures used, and this can be found in Table 2. The interitem reliability was above .70 for both administrations of the BDI-II, as well as the final administration of the Godin questionnaire. Table 3 shows the complete correlation matrix for the variables, and Figure 2 represents this information visually. A one-tailed t-test was used to determine statistical significance because the direction of the relationships could be predicted from existing literature. Both autocorrelations were positive and significantly different from zero, $p < .01$. The synchronous correlation at baseline was significantly different from zero, $p < .05$, while the synchronous correlation at follow-up approached significance, $p = .090$. The cross-lagged correlation linking initial exercise with later depression also approached significance, $p = .059$. The cross-lagged correlation linking initial depression

with later exercise was not significantly different from zero. The synchronous

correlations and cross-lagged correlations were all negative, indicating an inverse

relationship between exercise and depression. While the direction of these relationships

was expected, given the existing literature on the subject, the magnitude of this

relationship was smaller than anticipated. Despite the amount of literature showing an

inverse association between exercise and depression, the synchronous correlations

obtained here were small, in the range of -.10 to -.15.

Table 3: Correlation Matrix

		Ex1 Total	Dep1 Total	Ex2 Total	Dep2 Total
Ex1 Total	Pearson Correlation	1.000	-.136*	.449**	-.117
	Sig. (1-tailed)		.036	.000	.059
Dep1 Total	Pearson Correlation	-.136*	1.000	-.077	.729**
	Sig. (1-tailed)	.036		.155	.000
Ex2 Total	Pearson Correlation	.449**	-.077	1.000	-.101
	Sig. (1-tailed)	.000	.155		.090
Dep2 Total	Pearson Correlation	-.117	.729**	-.101	1.000
	Sig. (1-tailed)	.059	.000	.090	

*. Correlation is significant at the 0.05 level (1-tailed).

**. Correlation is significant at the 0.01 level (1-tailed).

Pairs of correlations were then compared using the modified Pearson-Filon

statistic suggested by Raghunathan and colleagues (1996). A two-tailed test was used

since the direction of these differences was not predicted by the existing literature.

Results of the modified Pearson-Filon test are shown in Table 4. There was a significant

difference between the two autocorrelations, $ZPF = -4.174$, $p < .01$. No significant

differences were found between the synchronous correlations, $ZPF = -0.4619$, $p = .64$. In

addition, the cross-lagged correlations were not significantly different from each other,
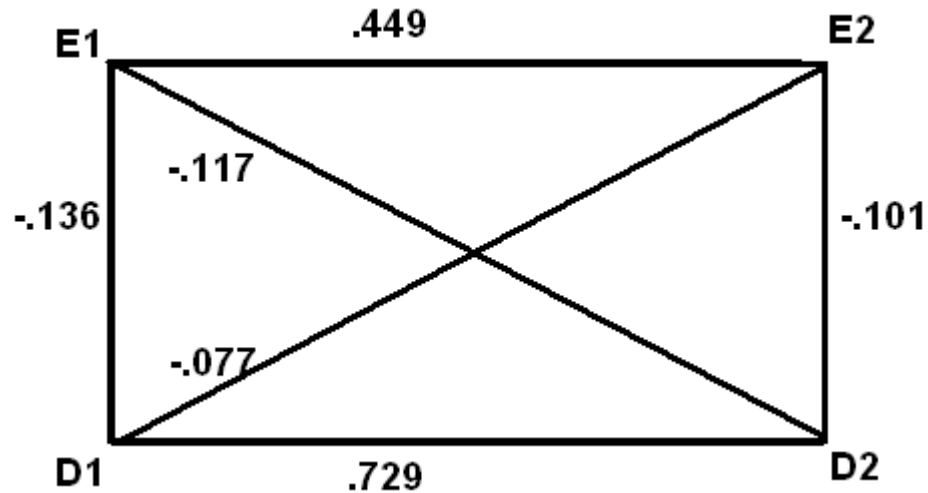
ZPF = -0.4599, p = .65.

Figure 2: Visual display of the correlation matrix. The two autocorrelations are shown on the top and bottom of the square, and are significantly different from each other. The two synchronous correlations are on the left and right, for baseline and follow-up respectively. These are not significantly different from each other. The cross-lagged correlations are shown in the middle, and are not significantly different from each other.

Table 4: Results of the Pearson Filon Test		
	ZPF	p value
Autocorrelations (E1E2 vs D1D2)	-4.174	< .01
Synchronous correlations (E1D1 vs E2D2)	-0.4619	.64
Cross-lagged correlations (E1D2 vs E2D1)	-0.4599	.65

Chapter 5: Discussion

Although the present study was not designed to test solely for the existence of a relationship between exercise and depression, a small but significant inverse association was shown at baseline between the variables. This association fell below the threshold for statistical significance at follow-up. However, results of the modified Pearson-Filon test showed no differences in the synchronous correlations from baseline to follow-up. The magnitude of the relationship between exercise and depression was small, likely because of the population under study. While many studies showing an association between exercise and depression use clinically depressed populations, this one used a nonclinical sample of college students. Depression scores were generally low, regardless of exercise habits. Because of this, a stronger association may be seen in samples with more variable depression scores.

Assumptions of Test

To interpret the results of the cross-lagged panel correlation analysis, the assumptions of the test must be considered. The assumption of synchronicity was met because exercise and depression were measured at the same points in time. This was ensured through the selection of the measures used, to avoid problems with aggregation and retrospection.

Stationarity is another assumption of the cross-lagged panel correlation design, and indicates stability in the causal processes occurring between the measured variables. A lack of change in the synchronous correlations from the first to the second time period

is considered evidence of perfect stationarity. Results of the modified Pearson-Filon test showed no significant differences between the two synchronous correlations, indicating that the assumption of stationarity was satisfied. In addition, stationarity is generally not met during period of rapid developmental growth, or when the time intervals used are long. This was not the case during this research, as a one month time interval was used on adult subjects.

Because the two assumptions of synchronicity and stationarity were met, the strength of the temporal relationships can be used to make causal inferences. If the cross-lagged correlation linking initial exercise with later depression is significantly larger than the other cross-lagged correlation, one can infer that exercise is the cause of reductions in depression due to the temporal sequence of the variables. However, results of the modified Pearson-Filon test showed no significant differences in the cross-lagged correlations. Because of this, one should not reject the null hypothesis that the cross-lagged correlations are equal. The lack of statistically significant differences can be explained by several factors, and gives rise to multiple interpretations of the findings.

Explanation of Null Findings

Despite meeting the assumption of stationarity and showing that the causal processes underlying the relationship between the variables are stable, the reliability of the measured variables can affect the causal inferences drawn from the data. Interitem reliability, measured by Cronbach's alpha, was acceptable for the depression measure at both time points, and also for the exercise measure at follow-up. However, interitem reliability was relatively low for the exercise questionnaire at baseline. There are several

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potential explanations for this finding. First, the Godin questionnaire is only a four-item instrument, and interitem reliability tends to be higher when more items assess a construct. In addition, it is a very global measure of exercise, ultimately assessing the volume of physical activity. It incorporates many different components of an exercise routine, such as frequency and intensity. It is very plausible that an individual could participate in frequent bouts of light activity, while not performing any strenuous exercise. The responses to questions on mild and strenuous exercise are likely to be very different in this scenario, yielding a low correlation between items on the instrument. However, this merely explains why the exercise questionnaire has a lower reliability in general when compared to the depression measure. It does not explain why the internal consistency of the GLTEQ was low for the first, but not the second round of data collection. It could simply be that exercise is a highly variable behavior. Perhaps students had more balance between mild, moderate, and strenuous activities at the second testing, while they predominantly performed one type of exercise at baseline.

Because of the low internal consistency of the GLTEQ at baseline, the autocorrelation of the exercise measure was significantly lower than that of the depression measure. Kenny (1975) noted that in situations where the variables had different reliabilities, the one with the lower reliability would appear to be the cause, while the variable with the higher reliability would appear to be the effect. He then suggested a method to correct for changes in reliability. However, the method he suggested cannot be used in this analysis because the calculation of reliability ratios requires the use of three or more variables. Because of the lower reliability of the exercise measure employed in this study, exercise would be seen as the cause of changes

in depression if a significant difference in the cross lags occurred. Since no differences in the cross-lagged correlations were found, it is possible that the effects of depression on future exercise habits are stronger than observed here. The failure to find a significant difference between the cross lags could be attributable to the low reliability of the exercise questionnaire.

In addition, the stationary relationships between exercise and depression are weak, in the range of -.10 to -.15. Previous research on cross-lagged panel correlation analysis suggests that moderate to large correlations, 0.3 and above, typically yield more meaningful results (Kenny and Harackiewicz, 1979). Large correlations between the variables also increase the power of the test. Therefore, the lack of a significant difference in the cross lags could also be attributed to the weak association between exercise and depression in this sample. The association between exercise and depression may be stronger in a population with clinical depression, or a population that is not as physically active as college students.

Another explanation for the findings is a lack of statistical power. Since cross-lagged panel correlation is a low power test, it is possible that the difference between the cross-lagged correlations was too small to be detected with this moderate sample size. The lack of power could be due to the low synchronous correlations between exercise and depression. The low reliability of the exercise measure used also decreases the power of the test. In addition, it may also have a more direct effect in that it could be masking the true differences in the cross lags. Although power could be increased by increasing the sample size, it is not believed that the sample size by itself ($N = 178$) was too small to ensure adequate power.

A fourth interpretation for the lack of significant differences between the cross-lagged correlations is that the one month time interval used may not be sensitive to changes in depression. While exercise was variable during this time period, as shown by the autocorrelation of .449, depression was much more stable. The time interval used may have been too short to allow for changes in depressive symptoms. However, this same time interval has been used successfully in studies using similar methodology, population, and measures (Golin, Sweeney, & Shaeffer, 1981). In addition, using such a short time interval avoids the problems of other longitudinal research, where the time lag is often too long to account for the intermittent nature of depression. The time lag used in this study did not allow for individuals to recover, only to have a new episode of depression during the time of the study. In longitudinal studies, where the time lag is often measured in years, it is much more probable that an individual could recover from a bout of depression, relapse, and recover once again.

Interpretation of Findings

Because statistically significant differences were not found between the cross-lagged correlations, the null hypothesis of equal cross lags cannot be rejected. This is generally interpreted as a spurious relationship between the variables, where no causal relationship exists. However, there are other valid interpretations for these findings. While the relationship between exercise and depression may indeed be spurious, the results of experiments by other researchers indicate other possibilities. In his critique of cross-lagged correlation, Rogosa (1980) pointed out that the difference in the stability of the variables can offset the true difference in the cross-lagged correlations. In essence,

the different reliabilities of the variables can mask differences in the cross-lagged correlations. In this situation, equal cross-lagged correlations could be consistent with unequal causal effects. This particular interpretation offered by Rogosa can certainly be applied to this study, as the reliability of the exercise measure was markedly different from the depression measure. If this is the case, then it is likely that depression has a stronger causal influence than what is demonstrated here, because the stability of the exercise measure was less than the stability of the depression measure.

In addition to spuriousness and masking, reciprocal causation is another interpretation of the data. Reciprocal causation would give rise to large causal effects in both directions, and the magnitude of these causal effects may or may not be equal. Rogosa (1980) also asserts that equal cross-lagged correlations can signify large and equal causal effects, or large and unequal causal effects. The possibility of reciprocal causation cannot be ruled out using cross-lagged panel correlation analysis, nor can it be ruled out using experimental methods. However, it remains a viable alternative, and could explain why no dominant pathway emerged. The possibility of reciprocal causation is an avenue for further exploration.

In summary, the data do not clearly indicate one dominant cause-and-effect pathway. While it cannot be concluded that exercise causes changes in depressive symptoms, neither can it be concluded that depression causes changes in physical activity. The association between exercise and depression could, in fact, be spurious and not causal in nature at all. However, the more likely scenario is one of reciprocal causation, where exercise decreases depression, which in turn increases physical activity.

This study is rather unique in that it takes a non-experimental approach. Perhaps because of this, it draws different conclusions from the experimental literature on exercise and depression. Exercise intervention studies often conclude that exercise is the cause of reductions in depression, regardless of the specific exercise program used. The results of this study are more in line with the mixed results from longitudinal studies examining exercise and depression.

One particular study that used cross-lagged panel correlation came to different primary conclusions about the nature of the exercise-depression relationship (Fukukawa et al., 2004). However, many similarities arose when the data was compared more closely. Although they used similar methodology, different measures of exercise and depression were used on a dissimilar population of middle aged and elderly Japanese individuals. While a self-reported measure of physical activity was used in this study, Fukukawa et al. (2004) measured the participants' steps per day using a pedometer, a more objective assessment of physical activity. The time lag used in their study was two years, much longer than the one month used here. They determined that exercise was the cause of reductions in depression in adults aged 65-79. However, it should be noted that all the causal models used in the Japanese study fit the data relatively well. Although the model predicting depression from prior walking behavior fit the best, other causal pathways were also a good fit for the data. The finding that multiple causal models fit their data falls in line with the research presented here, where no dominant causal relationship was found. In addition, their finding that exercise was the cause of reductions in depression was only true for the older age group, and not for adults aged

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40-64. Because of the age difference found in their study, it is possible that the students surveyed in this research were too young to show the same effect. One limitation mentioned in the study by Fukukawa et al. (2004) was that the antidepressant effect of exercise, as shown by the cross-lagged correlation linking initial exercise with later depression, was relatively weak at -0.11. However, that result is very similar to the cross-lagged correlation found in this study, at -0.117. Although the correlations obtained are very similar, the findings of Fukukawa et al. (2004) were statistically significant while the correlation found here was not. Most likely, their larger sample size (N = 1151) resulted in greater power, therefore giving them the ability to detect smaller differences. It is not known what magnitude of difference is thought to be clinically meaningful, as this is not the same as statistical significance. Although the conclusions drawn here are not an exact match to those of Fukukawa and colleagues, there are many similarities in the data.

Limitations

There are several limitations to the current study. First, the reliability of the exercise measure was low (one month test-retest reliability of .449, Cronbach's alpha of .491 and .701 at baseline and follow-up, respectively). The GLTEQ was chosen because of its simplicity and ease-of-use. In addition, it is very short, only 4 items, and takes minimal time to complete. A more detailed assessment of physical activity, such as a 3 day diary, could provide more comprehensive information about the types of exercise performed. Since the Godin questionnaire is a subjective self report instrument, it is

therefore prone to bias. The use of objective physical activity measures, such as accelerometers or pedometers, would decrease this bias. Because is it a subjective measure, it is likely that students would classify similar activities very differently, since there are few specifications of what is considered "mild," "moderate," and "vigorous." For example, a highly fit student might classify walking 4 mph as light activity, while a less fit student might classify that same activity as "moderate" or even "vigorous." If a three day diary was used, the intensity of the exercise could be classified according to standardized MET values, and then objectively sorted into categories based on the MET value of the exercise. Furthermore, it might have been difficult for subjects to recall episodes of mild exercise, since they require minimal effort and often are not thought of as "exercise" at all. Finally, the GLTEQ only captures activity performed in an individual's leisure time. It does not include other forms of physical activity, such as transportation or employment-related activity. For instance, students who walk or bike for transportation would not include this in their total activity, as its primary purpose is not to improve the body and it doesn't occur during a person's free time.

Aside from the exercise questionnaire, another limitation of this study is the population used. The sample of college students selected was not intended to be representative of the general population or representative of all college students. Besides not being able to generalize the results, the population used was also a captive audience. Because the surveys were distributed during normal class meeting times during the semester, it is unknown how truthfully students answered the questionnaires. In addition, the population used may not be the best for showing the relationship between exercise and depression. Since Fukukawa and colleagues (2004) were able to show that exercise

caused changes in depressive symptoms for elderly, but not middle-aged adults, it is possible that the lack of significant findings in this study is due to the age of the participants. College students are typically healthy young adults, and one would not expect high levels of depression in this population. With the population used, the modal response to the BDI was zero and the median score was 3, indicating that most of the students were not depressed. Since depression scores in this population were low regardless of exercise habits, this weakened the association between exercise and depression. The use of a clinical population would give rise to more varied depression scores, and would allow for more improvement of depression scores over time. This could strengthen the association between the variables.

A third limitation of this study is the small synchronous correlations between exercise and depression. A moderate to large correlation between variables (~0.3 and above) is best for interpreting the results of cross-lagged panel correlation. However, the correlation between exercise and depression was small in this study, in the range of -0.10 to -0.15. While the synchronous correlation was statistically significant at baseline, it was not significant at follow-up. While this could be due to the sample size, it more likely reflects the weak association between exercise and depression in this population. Using a different exercise measure to improve the reliability, as well as studying a different population, would likely strengthen the association between exercise and depression. In turn, this would improve the power of the test to detect differences in the cross-lagged correlations.

Finally, the study was limited by the time constraints of the academic school year, as well as the calendar year. Students were initially surveyed at the beginning of the

semester, where the workload was relatively light. At the follow-up assessment, the workload for most of the students had increased, and many of them had major assignments and exams around the time of the follow-up. While this was unlikely to bring any individual from perfectly normal to highly depressed, it could have influenced the BDI scores by a few points. In addition, students were surveyed during September and October. At the follow-up assessment, the temperature was colder and the days were shorter than at baseline. Again, this seasonal effect could influence depression scores by a few points, but would be unlikely to have a major impact in such a short time frame.

Conclusions and Suggestions for Further Research

Further research needs to be done concerning the relationship between exercise and depression. Although many exercise intervention studies show that exercise causes changes in depression, these types of studies cannot test for the possibility that depression influences physical activity habits. In addition, experimental studies generally take place in a laboratory setting, which does not always mimic real-world conditions. Future studies should use a longitudinal approach, using real-world observations to supplement the existing experimental literature. Methods that do not make causal assumptions up front need to be used more frequently. Particularly, the use of cross-lagged panel correlation has had mixed results when applied to the relationship between exercise and depression. More studies using this methodology need to be completed, using a variety of populations and exercise measures. Additionally, the possibility of reciprocal causation needs to be studied in further detail. Qualitative research, such as the work by

Faulkner and Biddle (2004), would give more detailed insight into the possibility of reciprocal causation. More complex means of path analysis could also be undertaken.

In conclusion, the data did not indicate that exercise caused reductions in depression in a sample of college students. In fact, the data did not support one particular causal model over another. This is in contrast to the plentiful experimental studies that show exercise to be the cause of reductions in depression, but falls in line with the mixed results of longitudinal studies. More research needs to be done, especially observational studies that do not make initial causal assumptions. The notion of reciprocal causation warrants further investigation.

Appendix A: Surveys

UID #: _____

Please do NOT include your name or any other identifying information on this page OR on the attached version of the Beck Depression Inventory. Use ONLY your UID #.

Godin Leisure-Time Exercise Questionnaire

1. 1. During a typical **7-Day period** (a week), how many times on the average do you do the following kinds of exercise for **more than 15 minutes** during your free time (write on each line the appropriate number).

	Times Per Week

a) **STRENUOUS EXERCISE**

(HEART BEATS RAPIDLY)

(e.g., running, jogging, hockey, football, soccer,

squash, basketball, cross country skiing, judo,

roller skating, vigorous swimming,

 vigorous long distance bicycling)   _____

b) **MODERATE EXERCISE**

(NOT EXHAUSTING)

(e.g., fast walking, baseball, tennis, easy bicycling,

volleyball, badminton, easy swimming, alpine skiing,

popular and folk dancing)   _____

c) **MILD EXERCISE**

(MINIMAL EFFORT)

(e.g., yoga, archery, fishing from river bank, bowling,

horseshoes, golf, snow-mobiling, easy walking)   _____

2. 2. During a typical **7-Day period** (a week), in your leisure time, how often do you engage in any regular activity **long enough to work up a sweat** (heart beats rapidly)?

OFTEN	SOMETIMES	NEVER/RARELY
1. ▪	2. ▪	3. ▪

BDI-II

Date:

Name: _____   Marital Status: _____ Age: _____ Sex: _____

Occupation: _____   Education: _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today.** Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
0   I do not feel sad.
1   I feel sad much of the time.
2   I am sad all the time.
3   I am so sad or unhappy that I can't stand it.

2. Pessimism
0   I am not discouraged about my future.
1   I feel more discouraged about my future than I used to be.
2   I do not expect things to work out for me.
3   I feel my future is hopeless and will only get worse.

3. Past Failure
0   I do not feel like a failure.
1   I have failed more than I should have.
2   As I look back, I see a lot of failures.
3   I feel I am a total failure as a person.

4. Loss of Pleasure
0   I get as much pleasure as I ever did from the things I enjoy.
1   I don't enjoy things as much as I used to.
2   I get very little pleasure from the things I used to enjoy.
3   I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
0   I don't feel particularly guilty.
1   I feel guilty over many things I have done or should have done.
2   I feel quite guilty most of the time.
3   I feel guilty all of the time.

6. Punishment Feelings
0   I don't feel I am being punished.
1   I feel I may be punished.
2   I expect to be punished.
3   I feel I am being punished.

7. Self-Dislike
0   I feel the same about myself as ever.
1   I have lost confidence in myself.
2   I am disappointed in myself.
3   I dislike myself.

8. Self-Criticalness
0   I don't criticize or blame myself more than usual.
1   I am more critical of myself than I used to be.
2   I criticize myself for all of my faults.
3   I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
0   I don't have any thoughts of killing myself.
1   I have thoughts of killing myself, but I would not carry them out.
2   I would like to kill myself.
3   I would kill myself if I had the chance.

10. Crying
0   I don't cry anymore than I used to.
1   I cry more than I used to.
2   I cry over every little thing.
3   I feel like crying, but I can't.

Subtotal Page 1

Continued on Back

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11. Agitation

0 I am no more restless or wound up than usual.

1 I feel more restless or wound up than usual.

2 I am so restless or agitated that it's hard to stay still.

3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interest in other people or activities.

1 I am less interested in other people or things than before.

2 I have lost most of my interest in other people or things.

3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.

1 I find it more difficult to make decisions than usual.

2 I have much greater difficulty in making decisions than I used to.

3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.

1 I don't consider myself as worthwhile and useful as I used to.

2 I feel more worthless as compared to other people.

3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.

1 I have less energy than I used to have.

2 I don't have enough energy to do very much.

3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.

1a I sleep somewhat more than usual.

1b I sleep somewhat less than usual.

2a I sleep a lot more than usual.

2b I sleep a lot less than usual.

3a I sleep most of the day.

3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability

0 I am no more irritable than usual.

1 I am more irritable than usual.

2 I am much more irritable than usual.

3 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.

1b My appetite is somewhat greater than usual.

2a My appetite is much less than before.

2b My appetite is much greater than usual.

3a I have no appetite at all.

3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.

1 I can't concentrate as well as usual.

2 It's hard to keep my mind on anything for very long.

3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.

1 I get more tired or fatigued more easily than usual.

2 I am too tired or fatigued to do a lot of the things I used to do.

3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I am much less interested in sex now.

3 I have lost interest in sex completely.

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_____ Subtotal Page 2

Appendix B: IRB Consent Form

CONSENT FORM

Project Title	*Causal Relationship Between Exercise and Depression*
Why is this research being done?	*This is a research project being conducted by Dr. David Andrews and Ginny Scott at the University of Maryland, College Park. We are inviting you to participate in this research project because you are currently enrolled at the University of Maryland. The purpose of this research project is to clarify the relationship between exercise and depression. Specifically, does exercise reduce symptoms of depression? Or, are individuals experiencing symptoms of depression less likely to exercise? This knowledge could be used to enhance the current treatments for depression.*
What will I be asked to do?	*The procedures involve completing surveys about exercise and depression on two separate occasions. These surveys are expected to take less than 15 minutes to complete. The first survey you will complete is the Godin Leisure-Time Exercise Questionnaire. In this survey, you will be asked to describe the frequency with which you perform different types of activity during a typical week. For example, you might be asked questions such as "During a typical 7 day period (a week, in your leisure time, how often do you engage in any regular activity long enough to work up a sweat (heart beats rapidly)?" The second survey you will be asked to complete is the Beck Depression Inventory-II. You will be asked to report the severity of any current symptoms of depression, such as guilt, sadness, or changes in sleeping patterns.*
What about confidentiality?	*We will do our best to keep your personal information confidential. To help protect your confidentiality, completed surveys will be stored in a locked file cabinet, and only the researchers will have access to the locked storage. The information collected during this study will be numerically coded, and your name will not be identified at any time. In addition, the responses will be destroyed after the results are published or a period of 2 years has elapsed. If we write a report or article about this research project, your identity will be protected to the maximum extent possible. Your information may be shared with representatives of the University of Maryland, College Park or governmental authorities if you or someone else is in danger or if we are required to do so by law.*

Project Title:	*Causal Relationship Between Exercise and Depression*
What are the risks of this research?	*There may be some risks from participating in this research study. Risks to the subjects will be minimized by keeping survey responses confidential. While there are no physical or legal risks by completing the surveys, you may feel uncomfortable answering certain questions in regards to the symptoms of depression. Specifically, you may feel fearful or embarrassed when asked about sensitive information, such as feelings of sadness or thoughts of death. You might feel stigmatized if others knew your responses to survey questions, but your responses will remain confidential. In addition, there may be risks that are currently unforeseeable at this time.*
What are the benefits of this research?	*This research is not designed to help you personally, but the results may help the investigator learn more about the relationship between exercise and depression. We hope that, in the future, other people might benefit from this study through improved understanding of the causal nature of this relationship. This information will be used to enhance the current knowledge of treatments for depression.*
Is any medical treatment available if I am injured?	*The University of Maryland does not provide any medical, hospitalization, or other insurance for participants in this research study, nor will the University of Maryland provide any medical treatment or compensation for any injury sustained as a result of participation in this research study, except as required by law.*
Do I have to be in this research? May I stop participating at any time?	*Your participation in this research is completely voluntary. You may choose not to take part at all. If you decide to participate in this research, you may stop participating at any time. If you decide not to participate in this study or if you stop participating at any time, you will not be penalized or experience negative consequences from your decision. Your grade in any class will not be impacted by your decision to participate in this study.*

Project Title	*Causal Relationship Between Exercise and Depression*
What if I have questions?	*This research is being conducted by Dr. David Andrews, Department of Kinesiology, at the University of Maryland, College Park. If you have any questions about the research study itself, please contact Dr. Andrews at: 2359 SPH Building, Dept. of Kinesiology, College Park, MD 20740; office (301) 405-2474; email dla@umd.edu. You may also contact Ginny Scott at: 2132 SPH Building, Dept. of Kinesiology, College Park, MD 20740; email gscott2@gmail.com. If you have questions about your rights as a research subject, please contact:* **Institutional Review Board Office, University of Maryland, College Park, Maryland, 20742; (e-mail) irb@deans.umd.edu; (telephone) 301-405-0678** *This research has been reviewed according to the University of Maryland, College Park IRB procedures for research involving human subjects.*
Statement of Age of Subject and Consent	*Your signature indicates that:* * you are at least 18 years of age;* * the research has been explained to you;* * your questions have been fully answered; and* * you freely and voluntarily choose to participate in this research project.*
Signature and Date	**NAME OF SUBJECT**	
	SIGNATURE OF SUBJECT	
	DATE	

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