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Negative Cognitive Style, Rumination, and Negative Emotionality as Mediators of the Antidepressive Effects of Physical Activity Among Young Adults

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Antidepressive Effects of Physical Activity Among Young Adults

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Dedication

To all of those who assisted me from day one and throughout this process, I thank you.

Kara L. Pegram
335 words

Abstract

This study assessed whether three potential cognitive and affective mechanisms mediated the relationship between physical activity and depressive symptoms. Participants were 143 young adults between the ages of 18 and 29 ($M = 19.29$, $SD = 1.65$); the majority of participants were female (72%) and Caucasian (69.9%). Participants were followed across eight weeks and completed measures of trait and state rumination, negative emotionality, cognitive style, depressive symptoms, and physical activity. A mediated model was proposed hypothesizing negative association between physical activity and depression mediated by less rumination, negative emotionality, and cognitive style. Hypotheses were examined cross-sectionally and prospectively. Contrary to hypotheses, physical activity was not associated with depressive symptoms concurrently ($B = -.0010$, 95% CI $= -.007$ to $.005$), prospectively for variability in physical activity [unstandardized coefficient $= -0.0008$, $t(147) = -0.74$, $p = 0.462$], or prospectively for aggregate mean of physical activity [unstandardized coefficient $= 0.0013$, $t(144) = 0.88$, $p = 0.382$]. Physical activity was not associated with any of the proposed mediators concurrently or prospectively meaning the first criterion was not met in establishing mediation. This study was a novel contribution to the literature as it explored theory-driven psychological mechanisms through which physical activity may confer protection for onset and maintenance of depressive symptomatology. Post hoc analyses highlight important aspects for future research to encompass, such as the need to prescribe physical activity for individuals experiencing depressive symptoms. Post hoc analyses were conducted and demonstrated areas for further research. Of note, physical activity was significantly correlated with depression for low CES-D individuals [$r(85) = .236$, $p = .029$] but not high CES-D individuals [$r(58) = -.054$, p

= .689]. The low CES-D participants had a higher mean level of physical activity (M = 185.79, SD = 196.38) relative to the high CES-D people (M = 121.24, SD = 146.87). Due to lack of overall understanding of the mediators explaining the antidepressive effects of physical activity, the next step must be an intervention study. Clinical implications, limitations, and directions for future research are discussed.

Keywords: physical activity, depressive symptoms, rumination, negative cognitive style, negative emotionality

CHAPTER I

Introduction and Literature Review

Purpose

"If we wish to conquer undesirable emotional tendencies in ourselves, we must assiduously and in the first instance cold-bloodedly, go through the *outward motions* of those contrary dispositions we prefer to cultivate" (James, 1884, p. 198).

Depressive symptoms are a major mental health concern for adolescents and young adults. The National Institute of Mental Health reports that the college years are a period where many will experience first onset of depressive symptoms (2012). Depressive symptoms in late adolescence and early adulthood are associated with increased risk for later depressive disorders; in fact, up to 25% of adults will experience a major depressive episode in their lifetime (Hankin et al., 1998; Kessler et al., 1994). Depressive symptoms also increase risk for suicidality, self-injury, and substance use, as well as academic and job impairment (Klein, Torpey, & Bufferd, 2008). Thus, understanding the risk and protective factors for depressive symptoms among adolescents and young adults is important.

Physical activity has been shown to be a protective factor for the development of depressive symptoms (Bhui & Fletcher, 2000; Camacho et al., 1991; Strawbridge, Deleger, Roberts, & Kaplan, 2002). For example, Taliaferro and colleagues found that in a college population, those who engaged in physical activity were at a lower risk of developing hopelessness, depression, and suicidal behavior than those who were inactive (2008). While the protective effect of physical activity on depressive symptoms is well-

established, less research has examined the mechanisms by which physical activity exerts its antidepressive effect. The purpose of this study was to examine theory-driven mechanisms for the antidepressive effects of physical activity on depressive symptoms among older adolescents and young adults.

Cognitive-affective vulnerability-stress models of depression suggest that individuals who respond to stress with negative interpretations about the causes and consequences of the stressor (e.g. negative cognitive style), perseverative attention to the stressor and associated mood (e.g. rumination), and high negative emotions (e.g. negative emotionality) are more likely to develop depressive symptoms. Individuals who engage in physical activity may be protected from depression because they are less likely to deploy these maladaptive cognitive and emotional responses to stress. Therefore I hypothesized that physical activity will be associated with lower depressive symptoms, that physical activity will be associated with less negative cognitive style for, less rumination about, and less negative emotionality to stressful events. Additionally, I hypothesized that lower cognitive style, rumination, and negative emotionality will mediate the relationship between physical activity and depressive symptoms.

Determining the cognitive and affective mediators for the antidepressive effect of physical activity can serve to better elucidate and strengthen the rationale for prescribing physical activity as a method of treatment for individuals and may be a viable alternative for those not responding to traditional psychotherapy or pharmacotherapy. A comprehensive understanding of the psychological mechanisms at play in the relationship between physical activity and depression has the potential to be enlightening for both preventing and treating depression.

Conceptualization of Depression

Depressive symptoms are a major mental health concern for adolescents and adults. Depressive symptoms are characterized as a constellation of experiences such as depressed mood, loss of interest, significant weight loss or gain, sleeping disturbance, fatigue, feelings of worthlessness and guilt, concentration difficulties, and thoughts of death (DSM-5, American Psychiatric Association, 2013). When such symptoms persist over time and cause significant life impairment, they may indicate the presence of a diagnosable depressive disorder. Hankin and colleagues (2005) suggest that depressive symptoms are distributed continuously, such that depressive symptoms and disorders exist on the same continuum and are differentiated only by number, frequency, or severity. Mulye and colleagues report that 8.9% of individuals 18-25 years old experience a diagnosed Major Depressive Episode every year (2009). In 2009, the American College Health Association reported that 30% of individuals at 2- and 4-year institutions endorsed an incapacitating depressed mood within the past year. Even subclinical depressive symptoms are a problem and are associated with greater experiences of family problems, school failure, absenteeism, suicide, and substance use in undergraduate students (Hysenbegasi, Hass, & Rowland, 2005). Depressive symptoms are also an indicator of risk for clinically significant depressive disorders. According to Blazer, Kessler, McGonagle, and Swartz (1994) individuals between the ages of 15 and 24 are most susceptible to developing depressive symptoms or Major Depressive Disorder (MDD), and up to 20% of adolescents and young adults will experience a diagnosable major depressive episode (Hankin & Abramson, 1999). Thus, untreated depressive symptoms in adolescence and young adulthood have the potential to result in

serious consequences for these individuals in adulthood. Knowing this potential trajectory in adolescents and young adults experiencing depressive symptoms, it is beneficial to explore ways to reduce or prevent depressive symptoms among this population.

Theoretical Framework

   Vulnerability-stress models of depression. Cognitive and affective models of depression developed out of the vulnerability-stress models of depression. The vulnerability-stress models of depression emphasize that the effect of stress exposure on depression outcomes is moderated by individual differences, for example vulnerabilities, which may exacerbate the effects of stress on depression outcomes. Early theoretical models of depression hypothesized that both stress and individual vulnerability factors act together to predict depressive outcomes. Stress is defined as demands on the individual, which disrupt both the physiological and psychological homeostatic balance (Selye, 1936). Selye (1974) identified two different types of stress, both of which carry with them their own psychophysiological consequences. Distress is conceptualized as stress that is damaging to an individual's health and is experienced as anger and aggression. Eustress is a more constructive and healthful experience of stress. Eustress is experienced by individuals as empathy for others and striving towards behavior that will achieve a better community. Lazarus (1966) followed suit to delineate different types of stress and defined three types of psychological stress appraisal: (a) harm and loss, (b) threat and (c) challenge. Along with separating the three types of stresses, Lazarus further hypothesized that an individual's appraisals associated with each of the types of stresses are also different.

Lazarus differentiated the *stress stimulus*, or in Seyle's terms the *stressor*, from the stress response. A stress stimulus is operationalized as an environmental condition or event that places a demand or load on the individual, which is deemed "normatively stressful but does not allow for individual differences in the evaluation of events" (Lazarus & Folkman, 1984, pp. 21). The stress response refers to an individual's response to the situation that will include cognitive components, how the person appraises or construes the meaning, the impact of the situation, and the individual's emotional response to the stressor. Lazarus opines, however, that it is somewhat futile to discuss stress as separate stimulus and response, because it is a relational process informed by the individual's appraisal (or response), that confers the severity and type of stress experienced. Currently, stress is most commonly conceptualized as environmental demands on the individual in the form of minor and major life events. It is important to note that there is nothing inherently positive or negative about life events to make them "stressful;" rather, stress is conceptualized as the total impact of the event on the individual. There is a well-established relationship between heightened levels of stress and exacerbations in onset and duration of depression (Kessler, 1997; Mazure, 1998; Monroe & Hadjiyannakis, 2002; Paykel, 2003; Tennant, 2002).

However, individual differences in the onset and course of depression even in the context of comparable stress exposure suggest that some individuals may be particularly vulnerable to developing depression when faced with stress. In the 1960s, several researchers hypothesized that some individuals may be particularly vulnerable to developing mental health disorders when faced with stress. The first "diathesis-stress" models hypothesized, for example, that individuals with a genetic predisposition to

schizophrenia who faced acute life stressors would be most at risk for developing the disorder (Bleuler, 1963; Meehl, 1962; Rosenthal, 1963). Beard defined vulnerability in 1881 as "a predispositional factor, or set of factors, that makes possible a disordered state" (Ingram & Luxton, 2005, p. 34); thus, vulnerability is further conceptualized as the factors that contribute to an individual being more susceptible to developing psychopathology in the face of stress. Vulnerabilities are the genetic, psychological, and social resources individuals possess for addressing environmental demands. Vulnerabilities are unique for individuals in the sense that deficits are impairing to varying degrees depending upon what matters to the person (Lazarus & Folkman, 1984). The common features among all individual vulnerabilities is that they are conceptualized as latent, endogenous, relatively stable, and trait-like. While separately, stress and vulnerability are able to partially explain the development and maintenance of psychopathology, when considered jointly, vulnerability-stress interactions may lend a deeper description of the processes leading to lack of wellbeing, and more importantly exacerbate the effects of stress on mental health outcomes.

Contemporary models of depression similarly emphasize the joint contributions of both stress and vulnerability in predicting depression. Cognitive models of depression hypothesize that how individuals deploy cognitive resources to attend to and interpret stressful events will explain individual differences in depressive outcomes. Similarly, affective models of depression hypothesize that how individuals respond emotionally to stressful events will explain individual differences in depressive outcomes. Below I summarize these contemporary theories of depression and how they may shed light on the potential antidepressive effects of physical activity.

Cognitive vulnerability-stress models of depression. Cognitive vulnerability-stress models of depression hypothesize that how individuals cognitively attend to, appraise, and interpret stressful events will affect their vulnerability to depression. Beck (1967) offered an early conceptualization of the cognitive vulnerability-stress model of depression. Beck hypothesized that a cognitive vulnerability for depression was conferred based upon negative cognitive schemas and dysfunctional attitudes (Abramson & Alloy, 1990; Beck, 1987; Haaga, Dyck, & Ernst, 1991). Negative cognitive schemas develop from early negative experiences, which facilitate the growth of negative attitudes about the self and with time are integrated into "the cognitive organization in the form of schemas; the schemas become activated by later adverse events impinging on the specific cognitive vulnerability and lead to the systemic negative bias at the core of depression" (Beck, 2008, pp. 970). These schemas are known as the negative cognitive triad and encompass negative views about the self, the environment, and the future (Beck, Rush, Shaw, & Emery, 1979).

During the time that Beck was developing his cognitive theory of depression, Seligman was also developing his learned helplessness theory of depression (1975), which was shortly thereafter critiqued and reformulated (Abramson, Seligman, & Teasdale, 1978). Learned helplessness theory of depression posits that individuals learn that their responses and outcomes are separate and independent of one another when they experience uncontrollable events (Lazarus, 1975). Learned helplessness can be specific to a particular situation but can also generalize across situations. This dichotomy is explained by two factors: (a) the causal attribution the individual makes regarding the uncontrollable situation experienced and (b) the similarity between the original situation

and the novel situation (Alloy, Peterson, Abramson, & Seligman, 1984). The degree to which the individual makes global attributions about the experience in the original uncontrollable situation, the more likely they will generalize the experience.

Abramson, Metalsky, and Alloy (1989) highlighted that it is the cognitive expectation of an inability to control outcomes that elicits and maintains depression. This reformulated hopelessness theory of depression, here forward called hopelessness theory, suggests that the response of hopelessness depression is elicited by an individual's perception of a negative life event. Thus, similar to other vulnerability-stress models, the hopelessness theory posits that negative life events are not sufficient for the development of depression. Abramson and colleagues outlined three cognitive factors that may moderate the relationship between negative life events and depression: (a) causal inference regarding event, (b) inferences regarding the outcome, and (c) inferences about the self (1989). Along with these three types of inferences, an individual's perception of the importance these negative events hold contributes to the development and maintenance of hopelessness depression. The hopelessness theory of depression suggests that when stable and global causes are attached to a negative life event, an individual is at a greater risk of developing generalized hopelessness and eventual depression. This triumvirate of cognitive responses (negative inferences about cause, consequences, and self) is termed "negative cognitive style" and is the key cognitive vulnerability identified by the hopelessness theory of depression.

An additional cognitive model of depression is the response styles theory, proposed by Nolen-Hoeksema and Morrow (1991). The response styles theory of depression is similar to the aforementioned models of depression in that the

vulnerabilities conceptualized are cognitive. However, this model offers something different from hopelessness theory, in that the focus is on cognitive processes rather than content. The response styles theory of depression posits that the duration of negative affect is predicted in part by the individual's response style to the negative emotions. Individuals who spend more time contemplating etiology and consequences of symptoms tend to remain symptomatic longer. The counter-response to the ruminative style is the distracting style. Distracting responses incorporate an individual "engaging in pleasurable events that can give one a sense of efficacy and take one's mind off of a negative mood" (Nolen-Hoeksema & Morrow, 1991, p. 120). Distracting responses in individuals with depression have been demonstrated to shorten experiences of depressed mood (Nolen-Hoeksema & Morrow, 1993). An individual's response style, ruminative versus distracting, tends to be consistent and thus more of a characterological quality. Those who engage in more ruminative responses to negative emotions report greater periods of depressed mood, as well as exacerbation and maintenance of preexisting depressed mood (Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema, Morrow, & Fredrickson, 1993).

Affective models of depression. Affective models of psychopathology theorize affective vulnerabilities as trait and state negative emotionality to stressful events. Trait affective components are conceptualized as temperament, which is understood as "biologically rooted individual differences in behavior tendencies" (Bates, 1987, p. 1101). These noted differences are relatively stable and enduring, meaning present from infancy, and believed to be determined by the interplay between an individual's genetics and environment. Temperament influences how one responds to environmental events,

in general and in times of stress. Temperament is a crucial component of whether an individual will go on to develop adaptive or maladaptive responses as an adult (Muris & Ollendick, 2005).

Extant research has demonstrated that one component of temperament associated with elicitation and maintenance of depressive symptoms is trait-negative affectivity (Anthony, Lonigan, Hooe, & Phillips, 2002; Kendler, Gatz, Gardner, & Pedersen, 2006; Klein, Durbin, & Shankman, 2009; Ormel, Oldehinkel, & Vollebergh, 2004). Trait-NA is characterized by a high frequency and intensity of negative emotions such as fear, distress, sadness, anger, disgust, and/or worry (Watson & Clark, 1984). Individuals high in trait-NA experience negative emotions more frequently, more intensely, and for longer than individuals low in trait-NA. Given that depressive disorders are characterized by negative affective states, it is not surprising that extensive research has linked trait-NA with depression (Clark & Watson, 1991; Eisenberg et al., 2005; Simonson, Sánchez, Arger, Mezulis, 2011).

State-NA represents the immediate experience of negative affect occurring in response to stress. This *in-the-moment* experience of negative affect or distress is associated with feelings such as sadness, fear, and/or shame (Watson, Clark, & Tellegen, 1988). When individuals high in trait-NA encounter stress, they are more likely to experience high levels of immediate negative affect, for example, state-NA (Simonson, Sanchez, Arger, & Mezulis, 2011). Both trait- and state-NA have been well established as risk factors for the development of various psychological problems and maladaptive behaviors, including depression (Tellegen, 1985). My dissertation will focus on state-NA as the proximal affective risk factor for depression.

Physical Activity is Associated with Lower Depressive Symptoms

Extant research has demonstrated that physical activity, whether aerobic or strength training has been shown to have both physical and psychological benefits (Galper, Trivedi, Barlow, Dunn, & Kampert, 2006; Häkkinen, Häkkinen, Hannonen, & Alen, 2001; Harris, Cronkite, & Moos, 2006). Regular physical activity has been shown to reduce the risk of overall mortality, heart disease, cancer, stroke, diabetes, and other common diseases (Bauman, 2004; Brosse, Sheets, Lett, & Blumenthal, 2002; Dahn et al., 2005). Psychologically, physical activity has a demonstrated effect on mood states, general well-being, anxiety, and the focus of this study, depression (Dimeo, Bauer, Varahram, Proest, & Halter, 2001; Galper et al., 2006; Stephens, 1988). One of the hallmark studies demonstrating the psychological benefits conferred by engaging in physical activity is the Alameda County Study (Camacho et al., 1991). The Alameda County Study was a longitudinal study that followed non-depressed individuals from 1965 through 1983. Individuals who reported low activity level at baseline, in contrast to those who reported high activity levels, were found to have a significantly greater risk for experiencing depression at follow-up. A finding to highlight was that inactive individuals in 1965, who had increased activity level by 1974, were not at any greater risk for depression in 1983 than those who had consistently been active. Those who had reported being active in 1965 but had decreased their activity level by 1974 were "more than one and half times as likely to become depressed by 1983 as were those who maintained high levels of activity" (Camacho et al., 1991, p. 229). Thus, one finding of the Alameda County Study is that physical activity protective effects against development of depression.

Physical activity has been shown to mitigate both the severity and duration of depression following stressful life events (Harris et al., 2006; Roth & Holmes, 1987), as well as reduce the risk for developing depression in high-risk individuals (Jerstad, Boutelle, Ness, & Stice, 2010). In individuals diagnosed with MDD, physical activity has demonstrated potential as a therapeutic option (Babyak et al., 2000). While the antidepressive effects of physical activity have been well elucidated, the psychological mechanisms remain unclear.

Potential Mediators for Antidepressive Effect of Physical Activity

Negative cognitive style. Negative cognitive style is a cognitive vulnerability factor that was elucidated by Abramson and colleagues in the hopelessness theory (1989). Negative cognitive style refers to an individual's propensity to make event-specific inferences in three domains: (a) stable, global causes (b) negative self-inferences and (c) negative consequences. The hopelessness theory conceptualizes that in light of a negative event, an individual may respond with negative cognitive style by those three inferences. From those negative evaluations, the individual is likely to become hopeless and subsequently experience depressive symptomatology. Negative cognitive style confers a heightened risk for development and maintenance of depression in the context of stress exposure. Joiner and colleagues found that negative cognitive style exacerbates an individual's hopelessness, which will subsequently increase risk for depressive symptomatology (2005). Alloy and colleagues found that in a sample of college students, those with greater negative cognitive styles experienced onset of depressive symptomatology at significantly greater rates than those students at low risk in regards to cognitive style (2006).

Individuals with negative cognitive styles fall into a negatively reinforcing cycle, wherein negative attributions increase experiences of negative life events (Joiner et al., 2005). Meaning, those at a heightened risk for development and maintenance of depressive symptoms experience less positive reinforcement in daily life (Jacobson, Martell, & Dimidjian, 2001). Physical activity has the potential to offer an individual positive reinforcement. Physical activity may offer protection for an individual with negative cognitive style by providing an alternative, positively reinforcing option to making and acting on negative cognitive inferences about a stressful event. Therefore, reductions in negative cognitive style for stressful events may serve as one of the mechanisms for physical activity's ability to function as an antidepressant.

Rumination. Ruminative response style describes a repetitive and passive thought process focused on negative emotions stemming from negative events (Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1993). Trait rumination is a proposed predictor of depressive symptoms, as is stress-reactive, rumination. Rumination not only predicts depressive disorders and subclinical depressive symptoms, it also predicts new depressive episodes and chronicity of these episodes (Nolen-Hoeksema, 2000). Anxiety symptoms are also predicted by rumination, a finding that Nolen-Hoeksema argues may be at the crux of the high comorbidity between depression and anxiety (2000). Nolen-Hoeksema and Morrow (1991) differentiated between distracting and suppressive responses. Suppressive responses entail an individual attempting to simply not think of the negative event, without changing the behavior or cognitions. Distracting responses involve "actively engaging in pleasurable events that can give one a sense of efficacy and take one's mind off a negative mood" (Nolen-Hoeksema & Morrow, 1991, p. 120).

Nolen-Hoeksema and Morrow found that distracting responses are associated with emotional well-being (1991). It is with this rationale that I hypothesize physical activity will decrease rumination in times of stress and subsequently reduce experiences of depressive symptomatology.

Negative emotionality. Negative emotionality describes the individual differences in emotional response to a negative event, namely the level of intensity and degree of negativity (Charbonneua, Mezulis, & Hyde, 2009). A component of negative emotionality is the regulatory process regarding the experience of emotions. Gross defines emotion regulation as "the processes by which individuals influence which emotions they have, when they have them, and how the experience and express these emotions" (1998, p. 275). These processes can either be automatic or controlled and purposeful. Negative emotionality is generally divided into two processes: (a) antecedent focused, which encompasses attempts to alter emotion production prior to the generation of said emotions and (b) response focused, meaning the emotional response has already begun and the individual endeavors to change said response (Gross, 2002). Depression has been shown to support an ability to alter negative emotionality and thus emotion regulation dysfunction by conferring difficulty in ability to inhibit "processing of negative material" (Joorman & Gotlib, 2010, p. 281). For example, Campbell-Sills and colleagues (2004) found that presence of depression reduced emotional responding to positive stimuli.

Gross and Muñoz (1995) discussed how emotion regulatory processes have implications in functioning, specifically in the area of work, relationships, and inner life. Thus, the inability to adequately and constructively modulate emotions leads to

impairment in function and subsequent issues with mental health. Specifically, in discussion of depression, Gross and Muñoz reported that for vulnerable individuals "increases in depressed mood are not met by successful regulatory measures. When this happens, the person may cross the diagnostic threshold into an episode of major depressive disorder" (1995, p. 156).

The literature has discussed numerous ways that negative emotionality can be regulated. These regulatory processes have been organized into the following five areas: (a) situation selection, (b) situation modification, (c) attention deployment, (d) cognitive change, and (e) response modulation (see Gross, 1998, for a more detailed discussion). It is within these regulatory processes that interventions, such as behavioral activation, have shown efficacy. Thus, if scheduling pleasant activities is known to function as an emotional regulatory process, it follows that physical activity may work in much the same way. Therefore, negative emotionality may serve as a mediator between physical activity and depression, such that physical activity will allow for emotion regulatory processes to occur that will decrease negative affect and thus reduce reports of depressive symptoms.

My Study

Past research has clearly established a link between physical activity and reduction in onset and maintenance of depressive symptoms. Previous research has also been able to support that individuals presenting with vulnerabilities such as negative cognitive style, rumination, and negative emotionality are at heightened risk of experiencing depression when faced with stressful events. However, the psychological mechanisms through which physical activity confers protection for development and

maintenance depressive symptomatology in vulnerable individuals has yet to be fully

described. Historically, studies examining physical activity and depression have simply

explored the relation between those two variables and have not attempted to gain insight

into why the relationship exists. Stemming from the empirical support of the cognitive

vulnerability-stress models of depression, through my study I aimed to investigate the

relationship between physical activity and depression, by identifying potential theory-

driven mediators of this relationship. This effort represents a key step in validating

physical activity as a potential alternative treatment for individuals with depressive

symptomatology.

My study was a short-term prospective diary study among undergraduate

university students, which took place over a period of eight weeks. My study examined

the following hypotheses:

(1) Physical activity would be associated with lower depressive symptoms (H1).

(2) Physical activity would be associated with less negative cognitive style for

stressful events (H2a); less rumination about stressful events (H2b); and less

negative emotionality (ER) to stressful events (H2c).

(3) Cognitive style, rumination, and ER would mediate the relationship between

physical activity and depressive symptoms (H3).
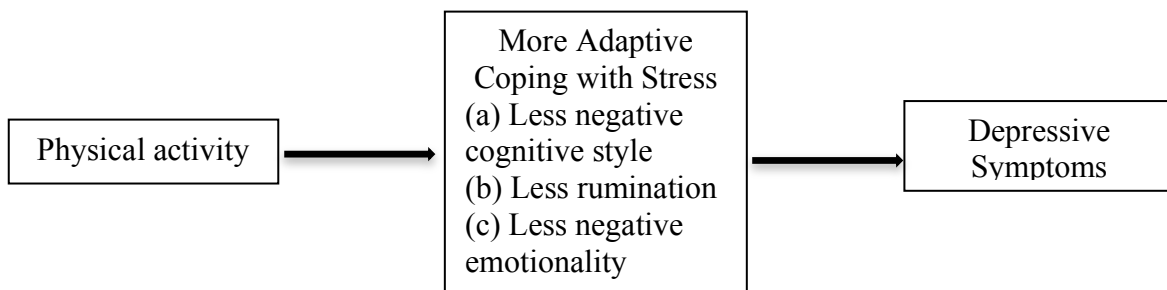
Figure 1. Mediated Model for Predicting Depressive Symptoms (H3)

CHAPTER II

Method

Sample and Participant Selection

Participants. I recruited undergraduate college students from a private, Pacific Northwestern university. A power analysis was conducted for the PROCESS style analyses for a multiple regression analysis with 4 predictors, using G*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007), which estimated that 85 participants would be required for a medium effect size (.15) with a power of .80. Power analysis for HLM remains both controversial and complicated, meaning for this study I utilized the same standards proposed for the PROCESS analyses.

Participant demographics are presented in Table 1. Participants were 143 (103 female; 72%) undergraduate students located in the Pacific Northwest. Participants ranged in age from 18 to 29 years old ($M = 19.29$, $SD = 1.65$). Approximately 70% of the sample identified as Caucasian, 17% Asian, 4% African American, and 4% as other. Approximately 5% of the sample identified as Hispanic or Latino. For the 2013 Autumn Quarter, the most recent statistics available, Seattle Pacific University (SPU) reported a total population of 3,366 undergraduate students. SPU reported that the average age of these students is 21; females represent 67% of the undergraduate population, and 32% of the undergraduate students fall under the broad category of "ethnic minority." As such, the recruited sample is comparable to the characteristics of the whole undergraduate population sampled.

Table 1

Participant Demographics

	N	%
Sex		
Male	40	28
Female	103	72
Race/Ethnicity		
Caucasian	100	69.9
Asian	24	16.8
African American	6	4.2
Hispanic/Latino	7	4.9
Other	6	4.2
Year In College		
Freshman	66	46.2
Sophomore	41	28.7
Junior	22	15.4
Senior	11	7.7

Recruitment. All study procedures and materials were approved by the SPU Institutional Review Board (see Appendix A). Participants were recruited via in-person classroom presentations as well as through posted flyers around the SPU campus. During the classroom presentations, students interested in participating in the current study were asked to provide email addresses. The posted flyers offered tear-off slips, which listed the online survey link for Part 1. Both the in-person classroom presentations and the

posted flyers gave a brief description of the study, along with the potential risks and benefits.

Procedure

The current study followed participants across eight weeks, and involved participants answering weekly questionnaires. All questionnaires were completed electronically on an online survey tool, Qualtrics. The baseline (T1) questionnaire included demographic questionnaires and self-report measures assessing physical activity over the past year, baseline depressive symptoms, trait rumination, trait cognitive style, and trait negative emotionality. T_2 through T_8 included identical questionnaires that participants completed weekly, assessing physical activity, cognitive style, rumination, and state negative emotionality for ideographically identified most stressful event, and depressive symptoms. All data was transferred by members of the research team to a secure online database, which was accessible to the faculty sponsor and myself. The data was downloaded by members of the research team to a secure password-protected computer.

Measures

Physical activity. Physical activity literature most commonly gauges frequency, duration, and intensity of physical activity (Kriska & Caspersen, 1997). Kriska and Caspersen reported that it is best practice to obtain physical activity levels over both a short and long time period (1997). Short-term weekly questionnaires are advantageous as they are influenced less by recall bias. However, historical questionnaires regarding engagement in physical activity are more predictive of typical activity patterns. Thus, in

adhering to the best practice for collecting self-report data on physical activity, both weekly and past-year reports were collected in the current study.

Past-year physical activity was measured with the CARDIA Physical Activity Questionnaire at T_1 (CARDIA; Jacobs, Hahn, Haskell, Pirie, & Sidney, 1989). This measure is comprised of nine-items, which assess participation in moderate intensity activities (e.g., volleyball, leisurely jogging) and vigorous activities (e.g., heart rate increased while engaged in activity) over the past 12 months. Activity scores are derived from summing reported participation both in frequency (per month) and total number of months (Buckworth & Nigg, 2004). Researchers have found support for utilization of CARDIA activity scores due to the measurements significant relationship with physiological markers and objective measures of exercise (Jacobs et al., 1989). In an undergraduate population, Wallace and colleagues found acceptable one-week test-retest reliabilities for moderate (.81), vigorous (.89), and total (.88) activity levels (2003). In my study, the overall CARDIA index, which was the sum of duration (frequency and months) spent engaged in moderate and vigorous activity over the past year, was used to yield one score for the past year. The Cronbach's alpha for the CARDIA within the current study was .78.

Weekly physical activity, T_2 through T_8, was measured by having participant's track intensity, duration (min), and frequency (days per week) of daily physical activity. Total scores will be derived from summing time weighted by an estimate of the intensity of the activity reported (Kriska & Caspersen, 1997). Intensity of physical activity is categorized based upon the National College Health Risk Behavior Survey (NCHRBS; Douglas et al., 1997). The NCHRBS divides physical activity intensity into the following

four categories: vigorous (e.g., basketball, swimming laps, or similar aerobic activities), stretch (e.g., toe touching or leg stretching), strength (e.g., push-ups or sit-ups), and moderate (walking or biking to class). Taylor and colleagues compared self-report physical activity logs and directly measured physical activity over seven days and reported acceptable accuracy in the self-report measures (1984). Wallace and colleagues administered an analogous format of this questionnaire to 56 undergraduate students and found adequate test-retest reliabilities for frequency and duration (.90 and 0.93, respectively; 2003). Total scores for physical activity were calculated by summing time engaged in physical activity weighted by the reported intensity of that activity, yielding 7 scores accounting for weekly reporting of physical activity. The Cronbach's alpha for the weekly physical activity questionnaire for the current study ranged from .59 to .82.

Depressive symptoms. Baseline depressive symptoms were measured at T_1 using the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), which is a 20-item self-report inventory designed to measure current depressive symptoms in community samples. Weekly depressive symptoms, T_2 through T_8, were measured using the Center for Epidemiological Studies Depression Scale Short Form (CES-D SF; Martens et al. 2006), which is a 9-item self-report inventory. Higher scores indicate more severe depressive symptoms for both forms. Scores for the full form CES-D range from 0 to 60, with a clinical cutoff of 16 (Radloff, 1977). Martens and colleagues suggest a clinical cutoff of 5 for the CES-D SF (2006). The CES-D is theoretically based on the affective and depressed mood symptoms consistent with a diagnosis of clinical depression along with other diagnoses as opposed to having a specific underlying theory.

The elements that are represented by the CES-D include the following: depressed mood, feelings of guilt and worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, loss of appetite, and sleep disturbance (Radloff, 1977). The CES-D measures a full range of depressive symptoms, including affect, somatic, and interpersonal. Items are worded in a positive direction so positive affect can be assessed and also to reduce response bias, 4 items in the CES-D and 2 in the CES-D SF. The CES-D measures current mood states, hence the directions ask for individuals to report depressive symptoms based on the past week.

The CES-D measure has been found to have high internal consistency with the clinical sample having a higher coefficient alpha (.90) than the general population (.85). Inter-item and item-scale correlations are, as predicted, higher in clinical samples than in the general population. CES-D scores are able to differentiate clinical samples from the general population, supporting the measure as a valid assessment of depressive symptoms. Convergent validity is supported with other measures of depression (Radloff, 1977). Martens and colleagues opined that use of the CES-D SF is equally capable of identifying individuals meeting criteria for diagnosis of Major Depressive Disorder with a sensitivity of .96 (2006).

The CES-D has been used cross-culturally with mental health professionals, in outpatient and inpatient settings, with ages ranging from adolescent through older adults. The CES-D has been shown to be a better predictor of depressive symptoms than the BDI-II in an undergraduate sample (Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). The Cronbach's alpha for the CES-D within the current study was .87 at baseline and ranged from .55 to .86 for the weekly CES-D SF.

Negative cognitive style.

Trait cognitive style. Trait cognitive style to depression was measured with the Cognitive Style Questionnaire (CSQ; Abramson, Metalsky, & Alloy, 2000). The CSQ is comprised of hypothetical positive and negative events, however only the negative events will be used for this study. There are 12 negative scenarios, such as "Imagine that you take a test and get a bad grade" with a follow-up question of "Did you get a bad grade because of something about you or because of something else?" Participants were then asked to respond along the range from 1 (e.g., *totally caused by something else*) to 7 (e.g., *totally caused by something about me*). The composite negative event score has a reliability of $\alpha = .88$ with a test-retest reliability of $r = .80$ (Alloy & Abramson, 1999). Alloy and colleagues demonstrated that the CSQ has predictive validity for episodes of depression (1997). The Cronbach's alpha for the CSQ within the current study was .81.

Weekly cognitive style. The Event-Anchored Cognitive Style Questionnaire (EA-CSQ; Haeffel et al., 2008) is a 5-item self-report measure looking at coping strategies as related to the worst weekly event for the individual. While the initial prompt differs from the aforementioned CSQ, the remainder of the questionnaire is intact. Participants were asked to first report the worst event experienced in the past 7 days and to reflect upon said event when responding to the scenario-based items. Participants were then asked to rate the 5-items on a 7-point Likert scale, ranging from 1 (e.g., *totally caused by other people or circumstances*) to 7 (e.g., *totally caused by me*). The Cronbach's alpha for weekly CSQ within the current study ranged from .73 to .82.

Rumination.

Trait rumination. The Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991) is a 22-item self-report inventory designed to measure the tendency to ruminate on negative affect. Participants were asked to rate items on a 4-point Likert scale ranging from 1 (*almost never*) to 4 (*almost always*), with higher scores indicating greater tendency to ruminate. Some examples of ruminative responses are the following: "I think about how hard it is to concentrate" and "I go away by myself and think about why I feel this way." Scores for the RRS range from 22 to 88 and are assessed in severity along a continuum. Previous research has provided internal consistency ranging from .88 to .92 (Bagby, Rector, & Bacchiochi, & McBride, 2004; Moberly & Watkins, 2008; Nolen-Hoeksema & Morrow, 1991). Regarding specific undergraduate populations, studies have found good internal consistency (α = .89) and test-retest reliability of .80 (Roelofs, Muris, Huibers, Peeters & Arntz, 2006; Sakamoto, Kambara, & Tanno, 2001). The Cronbach's alpha for the RRS within the current study was .91.

Weekly rumination. The Event-Anchored Ruminative Response Scale (EA-RRS; Nolen-Hoeksmea & Morrow, 1991) is comprised of the 5-item *brooding* subscale from the full RRS. Item examples include "think about the situation, wishing it had gone better" and "think, why can't I handle things better?" The EA-RRS is designed to measure preoccupation with moodiness upon reflecting on the worst event experienced in the past week. Participants were asked to rate items according to how frequently they engaged in the brooding response on a 4-point Likert scale ranging from 1 (*almost never*) to 4 (*almost always*), with higher scores indicating greater moody preoccupation. The *brooding* subscale of the RRS has been found to have good internal consistency (α = .89;

Mezulis, Simonson, McCauley, & Vander Stoep, 2011). The Cronbach's alpha for the weekly subscale ranged from .82 to .84.

Negative emotionality.

Trait negative emotionality. Trait negative emotionality was assessed at baseline using the Negative Affect superscale of the Adult Temperament Questionnaire (NA-ATQ; Evans & Rothbart, 2007). The NA-trait ATQ consists of 26 items in total based on 4 subscales: "I become easily frightened" is an example of a Fear item; "I seldom become sad when I hear an unhappy event" is an example of a Sadness item; "Colorful flashing lights bother me" is an example of a Discomfort item; and "I am rarely a patient person" is an example of a Frustration item. Participants rated each statement on a 7-point Likert scale with scores ranging from 1 (*extremely untrue of you*) to 7 (*extremely true of you*). The NA-ATQ measure has been found to have adequate reliability ($\alpha = .72$; Evans & Rothbart, 2007). Authors have also found support for construct validity of the NA-ATQ ($r = .74$; McCrae & John, 1992). Cronbach's alpha for this measure was .88.

Weekly negative emotionality. State negative emotionality was measured at baseline using the Negative Affect NA subscale of the Positive Affect Negative Affect Schedule (NA-PANAS; Watson, Clark, & Tellegen, 1988). The NA-PANAS is a 10-item measure describing different negative emotions and feelings. Participants were asked to rate items on a 5-point Likert scale ranging from 1 (*slightly or not at all*) to 5 (*very much*), with higher scores indicating greater state negative emotionality. Watson et al. (1988) reported that the NA-PANAS demonstrates strong internal consistency ($\alpha = .84$-.87) and adequate test-retest reliability ($r = .39$-.71). The Cronbach's alpha for NA-PANAS within the current study ranged weekly from .79 to .84.

Data Analytic Plan

Due to the nature of the study design, this allowed me to test my hypotheses both cross-sectionally and prospectively.

Cross-sectionally. The data used in the multiple mediation analyses were the baseline measures collected at T1. Multiple mediation was conducted using the PROCESS macro for SPSS 22.0 developed by Preacher and Hayes (2008), which is able to test via the product of coefficients test. In addition to this macro, bootstrapping was utilized to garner a reliable measure of the strength of the indirect pathway (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). As opposed to running all three mediators as separate models, a multiple mediation model was tested as the ability to examine "several mediators in the same model is one way to pit competing theories against one another within a single model" (Preacher & Hayes, 2008, p. 881). Preacher and Hayes outline that investigation of the total indirect effect and "investigating the specific indirect effect associated with each putative mediator" encompass the two essential components in multiple mediation (2008, p. 882).

Prospectively. The data used in the prospective data analysis contains all of the measures from T_2-T_8 and controlled for depressive symptoms at T_1. Prospective data analyses were conducted using the multilevel modeling in Hierarchical Linear Modeling (HLM) 6.04 (Raudenbusch & Bryk, 2002). HLM allows for the examination of within subject fluctuations in the outcome variable (depressive symptoms) as a function of within subject fluctuations in predictor variables (here, physical activity and cognitive and affective mediators; Bolger, Davis, & Rafaeli, 2003; Curran & Willoughby, 2003). Level 1 regression equations model variation in the repeated-measures outcome variable

(e.g., depressive symptoms) as a function of the within-subject predictors (e.g., weekly physical activity and weekly negative cognitive style, rumination, and negative emotionality) while also taking into account the extent to which these predictors are fluctuation around an individual's mean level (e.g. within context) by entering that mean level in Level 2. This allows me to examine the effects of both between-subject differences in predictors and within-subjects variability in predictors. Thus, I examined whether weekly within-person increases in physical activity were associated with weekly decreases in depressive symptoms, and whether this effect (if any) was mediated by weekly fluctuations in cognitive style, rumination, and/or negative emotionality to weekly stressful events.

Multilevel modeling does not offer a direct test of mediation, however, Nezlek (2007) provides a recommended methodology in establishing mediation in a multilevel model stemming from the Baron and Kenny (1986) procedure. The following four criteria needed to be met in order to demonstrate mediation in this study: (a) physical activity predicting depressive symptoms; (b) physical activity predicting hypothesized mediators; (c) the relationship between the mediators and depressive symptoms needed to be maintained when both physical activity and the mediator were entered into the model; (d) entering physical activity and the mediators into the model needed to reduce or eliminate the relationship between physical activity and depressive symptoms. The simple model and the full model then was compared to determine if there was evidence of mediation.

CHAPTER III

Results

Data Screening and Analysis

Data preparation. Prior to analysis, I inspected data for missing values, accuracy, multicollinearity, and normality of distribution. No variables were significantly skewed or kurtotic, indicating the data meets the basic assumptions of normality needed for analyses. Following Parent (2013) recommendations, I used person-mean imputation to impute scale scores on an item basis for participants who completed at least 80% of given scale.

Participants. A total of 151 participants initiated the baseline questionnaire for the present study. Of these, eight participants failed to complete any weekly data. Utilizing simple t-tests, I compared these two groups and found no significant differences regarding gender, age, and all baseline measures. Thus the final N is 143.

Descriptive. Bivariate correlations, means, and standard deviations for baseline study variables are presented in Table 2, correlations for weekly study variables are described below, and descriptive data for weekly study variables are presented in Table 3.

Baseline. Depressive symptoms were positively correlated with negative cognitive style, rumination, and negative emotionality. Physical activity was negatively correlated with negative emotionality, but not correlated with negative cognitive style or rumination. In addition, negative cognitive style, rumination, and negative emotionality were positively correlated. Contrary to hypotheses, depressive symptoms and physical activity were not significantly correlated.

Table 2

Bivariate Correlations and Descriptives among Variables at Baseline

N	Variable	1	2	3	4	5	M (SD)
143	1. Gender						1.72 (.45)
143	2. CES-D	.136					14.25 (8.76)
143	3. CARDIA	-.147	-.090				159.61 (180.23)
143	4. ATQ	.422**	.548**	-.171*			3.97 (0.66)
143	5. RRS	.154	.518**	-.059	.548**		53.47 (13.17)
143	6. CSQ	.018	.367**	.003	.303**	.344**	3.92 (0.93)

Notes: CES-D = Center for Epidemiological Studies Depression Scale; CARDIA = Physical Activity Questionnaire; ATQ = Adult

Temperament Questionnaire Negative Affect superscale; RRS = Ruminative Response Scale; CSQ = Cognitive Style Questionnaire.

*$p < .05$ ** $p < .01$

Weekly. Gender correlated with cognitive style from $r = -.033$ to .177, rumination

from $r = .009$ to .272, negative emotionality from $r = .056$ to .214, and depressive

symptoms from $r = .017$ to .175. Gender correlated with physical activity from $r = -.185$

to .023. Negative cognitive style correlated with rumination from $r = .101$ to .488 and

negative emotionality from $r = .012$ to .585. Physical activity correlated with negative

cognitive style from $r = -.127$ to .135, rumination from $r = -.116$ to .197, and negative

emotionality from $r = -.172$ to .175. Depressive symptoms correlated with negative

cognitive style from $r = -.224$ to 334, rumination from $r = -.166$ to .560, and negative

emotionality from $r = -.105$ to .509. Depressive symptoms correlated with physical

activity weekly from $r = -.079$ to .189. Most of the correlations within the weekly

variables did not reach significance.

Table 3

Descriptives for Weekly Study Variables

		CES-D SF	PA	EA-CSQ	EA-RRS	NA-PANAS
Week	*N*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
2	139	6.60 (4.99)	221.38 (261.97)	3.85 (1.24)	9.80 (3.71)	12.14 (5.08)
3	136	6.57 (4.93)	211.49 (248.19)	3.98 (1.21)	9.91 (4.00)	12.24 (4.45)
4	132	6.14 (5.06)	169.41 (187.38)	3.91 (1.36)	9.73 (3.98)	11.51 (4.65)
5	126	5.76 (4.43)	220.54 (296.04)	3.79 (1.31)	8.93 (3.54)	11.60 (4.48)
6	130	6.19 (4.90)	190.17 (232.40)	3.93 (1.25)	8.81 (3.62)	10.99 (4.33)
7	129	6.32 (5.14)	193.89 (260.66)	3.83 (1.33)	8.90 (3.60)	11.67 (4.60)
8	105	10. 11 (2.17)	187.27 (261.95)	3.78 (1.36)	8.79 (3.68)	11.11 (4.55)

Notes: CES-D SF = Center for Epidemiological Studies Depression Scale Short Form; PA = Weekly Physical Activity Questionnaire;

EA-CSQ = Event-Anchored Cognitive Style Questionnaire; EA-RRS = Event-Anchored Ruminative Response Scale; NA-PANAS =

Negative Affect NA subscale of the Positive Affect Negative Affect Schedule.

Cross-Sectional Analyses

Data analytic plan. My study examined a multiple mediation model. The independent variable was physical activity, further defined as summation of past year frequency (per month) multiplied by total number of months of reported participation across moderate and vigorous activities. The dependent variable was depressive symptoms reported over the past two weeks. The proposed mediators were trait negative cognitive style, trait rumination, and trait negative emotionality. Gender was entered as a covariate in the multiple mediation model.

Analysis. I assessed my mediation hypotheses with the script version of the PROCESS macro for SPSS v.2.12.1 developed by Preacher and Hayes (2004). The PROCESS macro allows for serial multiple mediation, model 4, which is recommended over separate simple mediation models by Preacher and Hayes (2008). I analyzed the strength and significance specific indirect effects, the total indirect effects, the direct effects, and the total effects, as outlined by Hayes (2013). In addition, I had the macro generate 1000 bootstrapped resamples, a nonparametric sampling procedure, from my data to avoid issues with skewness produced by the multiplication of path coefficients and to test the significance of the indirect effects. Indirect effects are considered significant when the confidence intervals do not include 0 whereas confidence intervals including 0 are considered non-significant. Gender was controlled for in the model.

Descriptive analysis. Results suggest that 39.68% of the variance in depressive symptoms was accounted for by the four variables in the model. The specific indirect effects, total effect, and direct effect are discussed below and reported in Table 4. There was not a significant direct effect of baseline physical activity on baseline depressive

symptoms ($B = -.0010$, 95% CI $= -.007$ to .005). While this non-significance demonstrates there was no effect to be mediated, I continued to examine the relationships of the different variables in the model.

A path: Did physical activity predict proposed mediator?

Negative cognitive style as a proposed mediator. The overall path with baseline physical activity predicting negative cognitive style was non-significant when controlling for gender [$R^2 = .0003$, $F(2, 140) = .024$, $p = .9759$]. Physical activity at baseline was not a significant predictor of negative emotionality, meaning there was not a true relationship between the predictor and the mediator ($B = .0000$, 95% CI $= -.0008$ to .0009). Additionally, gender was a non-significant predictor of negative cognitive style ($B = .0384$, 95% CI $= -.3107$ to .3874).

Rumination as a proposed mediator. The overall path with baseline physical activity predicting rumination was non-significant when controlling for gender [$R^2 = .0251$, $F(2, 140) = 1.80$, $p = .1688$]. Physical activity at baseline was not a significant predictor of rumination, meaning there was not a true relationship between the predictor and the mediator ($B = -.0027$, 95% CI $= -.0149$ to .0095). Additionally, gender was a non-significant predictor of rumination ($B = 4.3447$, 95% CI $= -.5311$ to 9.2204).

Negative emotionality as a proposed mediator. The overall path with baseline physical activity predicting negative emotionality was significant when controlling for gender, accounting for 19% of the variance in baseline depressive symptoms [$R^2 = .1899$, $F(2, 140) = 16.41$, $p = .000$]. Physical activity at baseline was not a significant predictor of negative emotionality, meaning there was not a true relationship between the predictor and the mediator and that a majority of the effect found was due to the gender effect ($B =$

-.0004, 95% CI = -.0010 to .0001). Additionally, gender was a significant predictor of negative emotionality (B = .5945, 95% CI = .3714 to .8176).

B path: Did proposed mediator predict depressive symptoms?

Negative cognitive style as a proposed mediator. Negative cognitive style was a significant predictor of baseline depressive symptoms (B = 1. 5290, 95% CI = .1912 to 2.8668).

Rumination as a proposed mediator. Rumination was a significant predictor of baseline depressive symptoms (B = .1750, 95% CI = .0677 to .2824).

Negative emotionality as a proposed mediator. Negative emotionality was a significant predictor of baseline depressive symptoms (B = 5. 0495, 95% CI = 2.7188 to 7.3801).

Total Indirect effects.

Negative cognitive style as a mediator. The specific indirect effect (physical activity through negative cognitive style) was not statistically significant (B = .0000, 95% CI = -.0014 to .0016).

Rumination as a mediator. The specific indirect effect (physical activity through rumination) was not statistically significant (B = -.0005, 95% CI = -.0031 to .0013).

Negative emotionality as a mediator. The specific indirect effect (physical activity through negative emotionality) was not statistically significant (B = -.0021, 95% CI = -.0056 to .0005).

Table 4

Direct, Indirect, and Total Effects of Physical Activity on Depressive Symptoms through Emotional Reactivity(M_1), Rumination (M_2), and Negative Cognitive Style (M_3)

Effect			B	*SE*	*p*	95% CI	
						LLCI	ULCI
PA → ATQ → CES-D			-.0021	.0015		-.0056	.0005
PA → RRS → CES-D			-.0005	.0011		-.0031	.0013
PA → CSQ → CES-D			.0000	.0007		-.0014	.0016
Total indirect effect			-.0025	.0024		-.0074	.0022
Total effect of X on Y (c)			-.0035	.0041	.3689	-.0116	.0046
					.3967		
Direct effect of X on Y (c')			-.0010	.0033	.7624	-.0075	.0055

Notes: CES-D = Center for Epidemiological Studies Depression Scale; CARDIA = Physical Activity Questionnaire; ATQ = Adult Temperament Questionnaire Negative Affect superscale; RRS = Ruminative Response Scale; CSQ = Cognitive Style Questionnaire.

*p < .05. ** p < .01.

Prospective Analyses

Step one: Does physical activity predict depressive symptoms? I first examined the hypothesis that increases in physical activity would predict decreases in depressive symptoms across the study period. Weekly fluctuations in physical activity was entered as the predictor variable in Level 1 with depression at T_1 as the Level 2 outcome variable (equation shown below). Aggregate physical activity (each individual's mean physical activity across the 7 week period), baseline depression, and

gender were controlled in analyses by adding the aggregate as a level two predictor. Results did not support my hypothesis and indicated non-significance for the effect of variability in physical activity on depressive symptoms [unstandardized coefficient = -0.0008, $t(147) = -0.74$, $p = 0.462$]. Aggregated mean physical activity also did not predict depressive symptoms [unstandardized coefficient = 0.0013, $t(144) = 0.88$, $p = 0.382$]. The only significant predictor of depression symptoms was baseline depression [unstandardized coefficient = 0.2826, $t(144) = 11.41$, $p < 0.001$].

$$DEP_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*CESD@W1_j + \gamma_{03}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$$

I considered the model without controlling for baseline depression as well. However, results indicated non-significance for the effect of variability in physical activity on depressive symptoms [unstandardized coefficient = -0.000848, $t(147) = -0.745$, $p = 0.458$]. Aggregated mean physical activity also did not predict depressive symptoms [unstandardized coefficient = 0.0008, $t(145) = 0.48$, $p = 0.631$].

$$DEP_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$$

Thus, in trying to predict fluctuations in depressive symptoms, neither an individual's average level of physical activity nor fluctuations in his/her physical activity explained changes in depressive symptoms. Baseline depressive symptoms were the only significant predictor of weekly depressive symptoms in this model. Even without controlling for baseline depressive symptoms, physical activity was not a significant predictor.

Table 5

Final estimation of Fixed Effects for Physical Activity to Depression with Baseline

Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. $d.f.$	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	2.0490	0.9315	2.20	144	0.029
GENDER, γ_{01}	0.3141	0.4734	0.66	144	0.508
CESD@W1, γ_{02}	0.2826	0.0248	11.41	144	<0.001
PA_MEAN, γ_{03}	0.0013	0.0015	0.88	144	0.382
For PA slope, β_1					
INTRCPT2, γ_{10}	-0.0008	0.0011	-0.74	147	0.462
Random Effect	Variance Component				
INTRCPT1, u_0	5.50611			96	<0.001
PA slope, u_1	0.00001			99	0.281
level-1, r	12.07188				

Table 6

Final Estimation of Fixed Effects for Physical Activity to Depression without Baseline

Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. d.f.	p-value
For INTRCPT1, $\beta 0$					
INTRCPT2, $\gamma 00$	5.1291	1.2114	4.23	145	<0.001
GENDER, $\gamma 01$	0.9279	0.6442	1.44	145	0.152
PA_MEAN, $\gamma 02$	0.0008	0.0018	0.48	145	0.631
For PA slope, $\beta 1$					
INTRCPT2, $\gamma 10$	-0.0008	0.0011	-0.74	147	0.458
Random Effect	Variance Component				
INTRCPT1, u_0	10.83635			97	<0.001
PA slope, u_1	0.00002			99	0.279
level-1, r	12.06246				

Step two: Does physical activity predict the hypothesized mediators? While

step one criteria was not significant and thus mediation in this prospective model cannot

be met, I examined the relationship of physical activity to each of the mediators

individually. I examined each model first with baseline depression entered as a control variable. In separate equations, each mediator was entered as the outcome variable with physical activity as the predictor in Level 1. Level 2 contained aggregate physical activity, baseline depression, and gender. I examined subsequent models without controlling for baseline depression. Each mediator was entered as the outcome variable with physical activity as the predictor in Level 1. Level 2 contained aggregate physical activity and gender. Equations are shown below.

Negative cognitive style. For the model with baseline depression, the effect of fluctuations in physical activity on negative cognitive style was non-significant [unstandardized coefficient = -0.0002, $t(147)$ = -0.59, $p = 0.557$]. The aggregate of physical activity for each individual on weekly negative cognitive style was non-significant as well [unstandardized coefficient = 0.0003, $t(144)$ = 0.74, $p = 0.461$]. The only significant effect was baseline depression as a predictor for negative cognitive style [unstandardized coefficient = 0.0273, $t(144)$ = 3.52, $p < 0.001$].

$CS_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*CESD@W1_j + \gamma_{03}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$

The effect of variability in physical activity on weekly negative cognitive style was non-significant [unstandardized coefficient = -0.000178, $t(147)$ = -0.549, $p = 0.584$]. Additionally, the effect of mean, or aggregate, physical activity for each individual on weekly negative cognitive style was non-significant [unstandardized coefficient = 0.0003, $t(145)$ = 0.58, $p = 0.562$].

$CS_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$

Table 7

Final Estimation of Fixed Effects for Physical Activity to Cognitive Style with Baseline

Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. d.f.	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	3.1849	0.2896	10.99	144	<0.001
GENDER, γ_{01}	0.1389	0.1493	0.93	144	0.354
CESD@W1, γ_{02}	0.0273	0.0077	3.52	144	<0.001
PA_MEAN, γ_{03}	0.0003	0.0004	0.74	144	0.461
For PA slope, β_1					
INTRCPT2, γ_{10}	-0.0002	0.0003	-0.59	147	0.557
Random Effect	Variance Component				
INTRCPT1, u0	0.52926			96	<0.001
PA slope, u1	0.00000			99	>0.500
level-1, r	1.12838				

Table 8

Final Estimation of Fixed Effects for Physical Activity to Cognitive Style without Baseline

Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. d.f.	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	3.4899	0.2891	12.07	145	<0.001
GENDER, γ_{01}	0.1933	0.1538	1.26	145	0.211
PA_MEAN, γ_{02}	0.0003	0.0004	0.58	145	0.562
For PA slope, β_1					
INTRCPT2, γ_{10}	-0.0002	0.0003	-0.55	147	0.584
Random Effect	Variance Component				
INTRCPT1, u0	0.60328			97	<0.001
PA slope, u1	0.00000			99	>0.500
level-1, r	1.12388				

    Rumination. For the model with baseline depression, physical activity

fluctuations on weekly rumination was non-significant [unstandardized coefficient = -

0.0005, $t(147) = -0.60$, $p = 0.552$]. The effect of aggregate physical activity for each

individual on weekly rumination was also non-significant [unstandardized coefficient = 0.0007, $t(144) = 0.52$, $p = 0.604$]. Baseline depression was a significant predictor for rumination [unstandardized coefficient = .0.0897, $t(144) = .52$, $p < 0.001$].

$$RUM_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$$

For the model without baseline depression, the effect of variability in physical activity on weekly rumination was non-significant [unstandardized coefficient = -0.0004, $t(147) = -0.53$, $p = 0.595$]. In addition, the effect of mean, or aggregate, physical activity for each individual on weekly rumination was also non-significant [unstandardized coefficient = 0.000494, $t(145) = 0.373$, $p = 0.710$].

$$RUM_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$$

Table 9

Final Estimation of Fixed Effects for Physical Activity to Rumination with Baseline Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. d.f.	p-value
For INTRCPT1, β0					
INTRCPT2, γ00	6.3098	0.9453	6.67	144	<0.001
GENDER, γ01	0.9916	0.4901	2.02	144	0.045
CESD@W1, γ02	0.0897	0.0253	3.54	144	<0.001
PA_MEAN, γ03	0.0007	0.0013	0.52	144	0.604
For PA slope, β1					
INTRCPT2, γ10	-0.0005	0.0008	-0.60	147	0.552
Random Effect	Variance Component				
INTRCPT1, u0	5.90939			96	<0.001
PA slope, u1	0.00000			99	>0.500
level-1, r	6.85319				

Table 10

Final Estimation of Fixed Effects for Physical Activity to Rumination without Baseline

Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	7.2343	0.9364	7.72	145	<0.001
GENDER, γ_{01}	1.2144	0.4997	2.43	145	0.016
PA_MEAN, γ_{02}	0.0005	0.0013	0.37	145	0.710
For PA slope, β_1					
INTRCPT2, γ_{10}	-0.0004	0.0008	-0.53	147	0.595
Random Effect	Variance Component				
INTRCPT1, u_0	6.66570			97	<0.001
PA slope, u_1	0.00001			99	>0.500
level-1, r	6.83420				

Negative emotionality. When controlling for baseline, depression, the effect for

fluctuations in physical activity on weekly negative emotionality was non-significant

[unstandardized coefficient = -0.0012, $t(147)$ = -1.22, p = 0.226]. The aggregate of

physical activity for each individual on weekly negative emotionality was also non-

significant [unstandardized coefficient = 0.0016, $t(144)$ = 1.06, p = 0.290]. Baseline

depression was a significant predictor for negative emotionality [unstandardized

coefficient = 0.1315, $t(144)$ = 4.43, p < 0.001].

$$EMRE_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*CESD@W1_j + \gamma_{03}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$$

In the model without baseline depression, the effect of variability in physical

activity on weekly negative emotionality was non-significant [unstandardized coefficient

= -0.0011, $t(147)$ = -1.10, p = 0.274]. The effect of mean, or aggregate, physical activity

for each individual on weekly negative emotionality was non-significant [unstandardized coefficient = 0.0015, $t(145) = 0.96$, $p = 0.337$].

$$EMRE_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*PA\_MEAN_j + \gamma_{10}*PA_{ij} + u_{0j} + u_{1j}*PA_{ij} + r_{ij}$$

Table 11

Final Estimation of Fixed Effects for Physical Activity to Negative Emotionality with

Baseline Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	7.9359	1.1071	7.17	144	<0.001
GENDER, γ_{01}	1.0357	0.5788	1.79	144	0.076
CESD@W1, γ_{02}	0.1315	0.0297	4.43	144	<0.001
PA_MEAN, γ_{03}	0.0016	0.0015	1.06	144	0.290
For PA slope, β_1					
INTRCPT2, γ_{10}	-0.0012	0.0010	-1.22	147	0.226
Random Effect	Variance Component				
INTRCPT1, u_0	7.95146			96	<0.001
PA slope, u_1	0.00000			99	0.358
level-1, r	11.48065				

Table 12

Final Estimation of Fixed Effects for Physical Activity to Negative Emotionality without

Baseline Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	9.2661	1.1327	8.18	145	<0.001
GENDER, γ_{01}	1.3602	0.6074	2.24	145	0.027
PA_MEAN, γ_{02}	0.0015	0.0016	0.96	145	0.337
For PA slope, β_1					
INTRCPT2, γ_{10}	-0.0011	0.0010	-1.10	147	0.274
Random Effect	Variance Component				
INTRCPT1, u_0	9.43228			97	<0.001
PA slope, u_1	0.00001			99	0.348
level-1, r	11.44019				

Step three: Was the relationship between physical activity and depressive

symptoms mediated by cognitive style, rumination, or negative emotionality? I

continued to examine the beta path even though the main effect and alpha paths were

non-significant. I examined each model with baseline depression and without controlling

for baseline depression. Each mediator was examined separately and entered as the

predictor variable in Level 1. Aggregate mediator and gender were entered in Level 2.

When controlling for baseline depression, the effect for fluctuations in negative cognitive

style [unstandardized coefficient = 0.3149, $t(147) = 2.55$, $p = 0.011$], rumination

[unstandardized coefficient = 0.3562, $t(147) = 6.99$, $p < 0.001$] and negative emotionality

[unstandardized coefficient = 0.2281, $t(147) = 5.52$, $p < 0.001$] on depression were

significant. None of the aggregate means were significant predictors. Additionally,

depression was a significant predictor in the models with mediator as predictor and

depressive symptoms as the outcome with negative cognitive style [unstandardized

coefficient = 0.2639, $t(144) = 10.45$, $p < 0.001$], rumination [unstandardized coefficient =

0.2268, $t(144) = 10.94$, $p < 0.001$], and negative emotionality [unstandardized coefficient

= 0.2172, $t(144) = 10.03$, $p < 0.001$] were significant and predicted depression

prospectively.

Regarding the model without controlling for baseline depression, the effect for

fluctuation in negative cognitive style [unstandardized coefficient = 0.3366, $t(147) =$

2.67, $p = .008$], rumination [unstandardized coefficient = 0.3469, $t(147) = 6.87$, $p <$

0.001], and negative emotionality [unstandardized coefficient = 0.2233, $t(147) = 5.39$, p

< 0.001] were significant. Additionally, the aggregate mean of negative cognitive style

for each individual [unstandardized coefficient = 1.0771, $t(145) = 3.16$, $p = 0.002$],

rumination [unstandardized coefficient = .3037, t(145) = 3.02, p = .003], and negative

emotionality [unstandardized coefficient = 0.3266, t(145) = 4.19, p < 0.001] for each

individual on weekly mediator to depression was significant. These results support the

well-established relationship between these cognitive and affective constructs and

depressive symptoms.

Table 13

Final Estimation of Fixed Effects for Negative Cognitive Style to Depression with

Baseline Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	-0.4693	1.2320	-0.38	144	0.704
GENDER, γ_{01}	0.1904	0.4748	0.40	144	0.689
CESD@W1, γ_{02}	0.2639	0.0252	10.45	144	<0.001
CS_MEAN, γ_{03}	0.4815	0.2933	1.64	144	0.103
For CS slope, β_1					
INTRCPT2, γ_{10}	0.3149	0.1233	2.55	695	0.011
Random Effect	Variance Component				
INTRCPT1, u_0	4.20970			144	<0.001
level-1, r	12.20207				

Table 14

Final Estimation of Fixed Effects for Negative Cognitive Style to Depression without

Baseline Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	0.0665	1.5242	0.04	145	0.965
GENDER, γ_{01}	0.7220	0.5937	1.22	145	0.226
CS_MEAN, γ_{02}	1.0771	0.3409	3.16	145	0.002
For CS slope, β_1					
INTRCPT2, γ_{10}	0.3366	0.1262	2.67	147	0.008
Random Effect	Variance Component				
INTRCPT1, u_0	3.61692			139	0.100
CS slope, u_1	0.15193			141	0.349
level-1, r	12.07439				

Table 15

Final Estimation of Fixed Effects for Rumination to Depression with Baseline Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. d.f.	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	-0.5081	0.8311	-0.61	144	0.542
GENDER, γ_{01}	-0.2233	0.3837	-0.58	144	0.562
CESD@W1, γ_{02}	0.2268	0.0207	10.94	144	<0.001
RUM_MEAN, γ_{03}	0.1060	0.0837	1.27	144	0.207
For RUM slope, β_1					
INTRCPT2, γ_{10}	0.3562	0.0509	6.99	147	<0.001
Random Effect	Variance Component				
INTRCPT1, u_0	0.44066			137	>0.500
RUM slope, u_1	0.02538			140	>0.500
level-1, r	11.35977				

Table 16

Final Estimation of Fixed Effects for Rumination to Depression without Baseline

Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. d.f.	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	0.3733	1.1015	0.34	145	0.735
GENDER, γ_{01}	0.1701	0.5164	0.323	145	0.742
RUM_MEAN, γ_{02}	0.3037	0.1006	3.02	145	0.003
For RUM slope, β_1					
INTRCPT2, γ_{10}	0.3469	0.0505	6.87	147	<0.001
Random Effect	Variance Component				
INTRCPT1, u_0	1.58186			138	>0.500
RUM slope, u_1	0.02043			140	>0.500
level-1, r	11.43452				

Table 17

Final Estimation of Fixed Effects for Emotional Reactivity to Depression with

Baseline Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	-0.3914	0.8391	-0.47	144	0.642
GENDER, γ_{01}	-0.0963	0.3795	-0.25	144	0.800
CESD@W1, γ_{02}	0.2172	0.0216	10.03	144	<0.001
EMRE_MEA, γ_{03}	0.1248	0.0694	1.80	144	0.074
For EMRE slope, β_1					
INTRCPT2, γ_{10}	0.2281	0.0413	5.52	147	<0.001
Random Effect	Variance Component				
INTRCPT1, u_0	0.37529			139	>0.500
EMRE slope, u_1	0.03253			142	0.478
level-1, r	11.44069				

Table 18

Final Estimation of Fixed Effects for Emotional Reactivity to Depression without

Baseline Depression

Fixed Effect	Coefficient	Standard error	*t*-ratio	Approx. *d.f.*	*p*-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	-0.0854	1.0503	-0.08	145	0.935
GENDER, γ_{01}	0.2311	0.4821	0.48	145	0.632
EMRE_MEA, γ_{02}	0.3266	0.0780	4.19	145	<0.001
For EMRE slope, β_1					
INTRCPT2, γ_{10}	0.2233	0.0414	5.39	147	<0.001
Random Effect	Variance Component				
INTRCPT1, u_0	0.28214			140	>0.500
EMRE slope, u_1	0.03269			142	>0.500
level-1, r	11.61775				

Step four: Was the relationship between physical activity and depressive

symptoms reduced or eliminated when entering physical activity and the mediators into

the model? Due to the main effect being non-significant, no further analyses were

necessary. The alpha and beta path were analyzed in order to assess underlying relationships between the study variables to inform post hoc analyses.

Post Hoc Analyses

I had originally hypothesized that I would simply enter gender as a covariate given its significant correlation with depressive symptoms. However, some studies have suggested that effects of physical activity on depressive symptoms may vary for males and females (Bhui & Fletcher, 2000; Cerin, Leslie, Sugiyama, & Owen, 2009). I wondered if as opposed to controlling for gender in my model, it might moderate the effects of physical activity on outcomes. Thus, the first post hoc I completed was to look at gender as a moderator both in the main effect of physical activity on depression and as a moderator of the full mediation models cross-sectionally. Using process model 1, I examined if the main effect of past year physical activity on baseline depressive symptoms was moderated by gender. I entered physical activity as the predictor, baseline depression as outcome variable, and gender as the moderator. This model was nonsignificant (R^2 = .0253, $F(3, 139)$ = 1.20, p = .3119). Each model was looked at individually using process model 7 for moderated mediation with physical activity entered as the predictor, baseline depression as the outcome variable, each mediator entered individually (negative cognitive style, rumination, and negative emotionality) and gender as the moderator. The model was nonsignificant for negative cognitive style (B = -.0044, 95% CI = -.0120, .0031), rumination (B = -.0029, 95% CI = -.0099, .0040), and negative emotionality (B = .0002, 95% CI = -.0067, .0071).

In my second post hoc analysis I considered data that suggests physical activity is a useful clinical intervention for individuals with clinically significant levels of

depression (Dimeo et al., 2001; Galper et al., 2006; Stephens, 1988). I examined whether the effects of physical activity on depression might vary depending on people's level of depression. I divided baseline CES-D scores into high and low based on the clinical cutoff of 15 and then reran the cross-sectional analyses for people above and below clinical cutoffs for CES-D at baseline. First I examined the correlation between physical activity and depression at high and low baseline depression. Interestingly, physical activity was significantly correlated with depression for low CES-D individuals [$r(85) =$ .236, $p = .029$] but not high CES-D individuals [$r(58) = -.054$, $p = .689$]. I further went on to examine what the mean levels of physical activity were for those groups and interestingly, the low CES-D people had a higher mean level of physical activity ($M =$ 185.79, $SD = 196.38$) relative to the high CES-D people ($M = 121.24$, $SD = 146.87$). The implications of these patterns of findings will be discussed at length in the discussion.

In my final post hoc analysis, I followed up on the prospective analyses, which found that although there was no prospective effect of weekly fluctuations of PA on depression, there were unique effects of two of the three mediators on weekly fluctuations on depression. I examined all three mediators simultaneously in a model where weekly cognitive style, rumination, and negative emotionality were entered into Level 1. Aggregate cognitive style, aggregate rumination, and aggregate negative emotionality, depression at baseline, and gender were entered into Level 2 to see if the effects held when all three mediators were considered simultaneously. Additionally, the model was examined without depression at baseline in the equation. For the model with depression at baseline there were significant effects for both rumination [unstandardized coefficient = 0.3009, $t(147) = 5.06$, $p = <0.001$] and negative emotionality

[unstandardized coefficient = 0.1256, $t(147) = 2.79$, $p = 0.006$]. Additionally, baseline depression was a significant predictor within the model [unstandardized coefficient = 0.2166, $t(142) = 10.59$, $p < 0.001$] (see Table 19).

$$DEP_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*CESD@W1_j + \gamma_{03}*CS\_MEAN_j + \gamma_{04}*RUM\_MEAN_j + \gamma_{05}*EMRE\_MEA_j + \gamma_{10}*CS_{ij} + \gamma_{20}*RUM_{ij} + \gamma_{30}*EMRE_{ij} + u_{0j} + u_{1j}*CS_{ij} + u_{2j}*RUM_{ij} + u_{3j}*EMRE_{ij} + r_{ij}$$

When depression at baseline was removed from the model, there were significant effects for fluctuations for rumination [unstandardized coefficient = 0.2886, $t(147) = 4.83$, $p = <0.001$] and negative emotionality [unstandardized coefficient = 0.1211, $t(147) = 2.69$, $p = 0.008$]. However, there were no main effects of any of the aggregated means (see Table 20).

$$DEP_{ij} = \gamma_{00} + \gamma_{01}*GENDER_j + \gamma_{02}*CS\_MEAN_j + \gamma_{03}*RUM\_MEAN_j + \gamma_{04}*EMRE\_MEA_j + \gamma_{10}*CS_{ij} + \gamma_{20}*RUM_{ij} + \gamma_{30}*EMRE_{ij} + u_{0j} + u_{1j}*CS_{ij} + u_{2j}*RUM_{ij} + u_{3j}*EMRE_{ij} + r_{ij}$$

Table 19

Final Estimation of Fixed Effects for Simultaneous Beta Path with Baseline Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. $d.f.$	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	-0.6262	0.9725	-0.64	142	0.521
GENDER, γ_{01}	-0.2843	0.3591	-0.79	142	0.430
CESD@W1, γ_{02}	0.2166	0.0204	10.59	142	<0.001
CS_MEAN, γ_{03}	-0.0219	0.2716	-0.08	142	0.936
RUM_MEAN, γ_{04}	0.0457	0.1137	0.40	142	0.688
EMRE_MEA, γ_{05}	0.0376	0.0904	0.42	142	0.678
For CS slope, β_1					
INTRCPT2, γ_{10}	-0.0999	0.1317	-0.76	147	0.449
For RUM slope, β_2					
INTRCPT2, γ_{20}	0.3009	0.0594	5.06	147	<0.001
For EMRE slope, β_3					
INTRCPT2, γ_{30}	0.1256	0.0451	2.79	147	0.006
Random Effect	Variance Component				
INTRCPT1, u_0	1.06807			119	>0.500
CS slope, u_1	0.05906			124	>0.500
RUM slope, u_2	0.00923			124	>0.500
EMRE slope, u_3	0.02541			124	0.271
level-1, r	10.95203				

Table 20

Final Estimation of Fixed Effects for Simultaneous Beta Path without Baseline

Depression

Fixed Effect	Coefficient	Standard error	t-ratio	Approx. $d.f.$	p-value
For INTRCPT1, β_0					
INTRCPT2, γ_{00}	-1.1311	1.2399	-0.91	143	0.363
GENDER, γ_{01}	0.0986	0.4659	0.21	143	0.833
CS_MEAN, γ_{02}	0.3470	0.3339	1.04	143	0.300
RUM_MEAN, γ_{03}	-0.0202	0.1392	-0.14	143	0.885
EMRE_MEA, γ_{04}	0.2274	0.108866	2.09	143	0.038
For CS slope, β_1					
INTRCPT2, γ_{10}	-0.0617	0.1322	-0.47	147	0.641
For RUM slope, β_2					
INTRCPT2, γ_{20}	0.2886	0.059756	4.83	147	<0.001
For EMRE slope, β_3					
INTRCPT2, γ_{30}	0.1211	0.0450	2.69	147	0.008
Random Effect	Variance Component				
INTRCPT1, u_0	0.59635			120	>0.500
CS slope, u_1	0.05570			124	>0.500
RUM slope, u_2	0.01051			124	>0.500
EMRE slope, u_3	0.02286			124	0.303
level-1, r	11.09049				

CHAPTER IV

Discussion

The purpose of my study was to assess three theory-driven mediators, as outlined in the integrated affective-cognitive vulnerability-stress model of depression, as potential mechanisms accounting for the antidepressive effects of physical activity on depressive symptoms. I examined whether negative cognitive style, rumination, and negative emotionality accounted for the relationship between physical activity and depressive symptoms. Due to the nature of the study design, I proposed to examine my hypotheses both cross-sectionally and prospectively. My goal for cross-sectional analyses was to test my overall hypothesis that people who exercise tend to report less negative cognitive style, less rumination, and less negative emotionality and this in turn is why they are less depressed at a trait level. My goal for prospective analyses was to answer the overarching hypothesis that when individuals are physically active, that makes an independent contribution to how they cope with weekly events on a week-to-week basis which would in turn explain weekly fluctuations in depressive symptoms. The current study was a novel contribution to the literature, as it attempted to delineate the specific cognitive and affective factors, which may interact to explain why physical activity is often associated with lower depressive symptoms. Also, unique to this study was the separate evaluation of both the cross-sectional and prospective data. While past research demonstrates the inverse relationship between physical activity and negative mood states, minimal research has been conducted in assessing the psychological mechanisms by which physical activity exerts its antidepressive effects (Dimea, Bauer, Varahram, Proest,

& Halter, 2001; Galper et al., 2006; Stephens, 1988). Therefore, my study provided exploration of potential novel links between the aforementioned variables.

I will first review what I found when analyzing my data cross-sectionally. Baseline physical activity was not a significant predictor for any of the proposed mediators. The overall model for the three mediators predicting depressive symptoms at baseline when controlling for gender was significant. This finding will be discussed later. The direct effect of physical activity on depressive symptoms was non-significant. Examinations of the indirect effects, physical activity via each of the individual three proposed mediators, were all non-significant. Thus, none of my hypotheses were supported in the cross-sectional analyses of my multiple mediation model.

For my second set of analyses, I looked at the data prospectively. Results did not support significance of the main effect, which indicated that the amount of weekly physical activity an individual reported did not impact weekly depressive symptoms. Results showed that my first hypothesis, physical activity will be associated with lower depressive symptoms, was not supported. My second hypothesis also was not significant; I did not find support for weekly physical activity being associated with less weekly negative cognitive style, less weekly rumination, or less weekly negative emotionality (A path). Of note, baseline depression was a significant predictor for both negative cognitive style and rumination within the alpha path. My third hypothesis, predicting a pathway between the three proposed mediators and depressive symptoms, was significant for the effect of fluctuations in rumination and negative emotionality on depressive symptoms in the model with baseline depression and the model without baseline depression. The aggregate mean of negative emotionality on depression was significant

in the model controlling for baseline depression. Additionally, baseline depression was significant in the model with negative cognitive style as the predictor and depressive symptoms as the outcome (B path).

Was Physical Activity Associated with Lower Depressive Symptoms?

I did not find a direct relationship between physical activity and lower depressive symptoms. Therefore, my hypothesis that physical activity will be associated with lower depressive symptoms (H_1) was not supported. These findings did not support previous research that physical activity is negatively correlated with experience of depressive symptoms (Babyak et al., 2000; Camacho et al., 1991; Dimeo et al., 2001; Galper et al., 2006; Häkkinen et al., 2001; Harris et al., 2006; Roth & Holmes, 1987; Stephens, 1988; Jerstad et al., 2010). Therefore, I cannot make conclusions regarding the effectiveness of physical activity in reducing onset or maintenance of depressive symptoms. The current results do not demonstrate, among a college population, if the antidepressive effects yoked with physical activity generalize to non-prescribed physical activity in a population with predominantly subclinical depressive symptoms. Speculations for this lack of finding will be discussed below.

My study did not take into account many of the variables that have potential to explain the lack of expected relationship between physical activity and depression. One such potential explanation found in the literature is that the function or goal of physical activity is associated with changes in mood (Abele & Brehn, 1993; Berger & Motl, 2000). Meaning that we should discern if the emphasis is the end-product of winning, such as interpersonal competition, or the process of engaging in physical activity for enjoyment or associated health benefits. Past research demonstrates that losing

performance, which leads to sense of failure or internalizing idea of lack of ability, is associated with negative mood (Abele & Brehn, 1993). Losing performance may extend towards injuries or having a poor workout. Thus, if an individual historically engages in physical activity as a stress reliever or mood elevator, if they incur injury during the workout or are injured and attempted similar workouts without success, the physical activity may have the opposite of the intended effect. Extending from this important differential in function of exercise, competition with others or self versus enjoyment and attainment of health goals, physical activity has a greater likelihood of functioning as an antidepressant when it is "externally paced" and "fosters a sense of achievement" (Lane & Lovejoy, pp. 544, 2001). Thus, it is important to assess and control for interpersonal competition, injury status, and overall intent of physical activity (Berger & Motl, 2000). Stemming from this data, it would also be advantageous to assess the individual's perceived experience of physical activity in order to qualify if the experience was rewarding, frustrating, or neutral as this may explain lack of variability.

A second possibility is presence of mood symptoms in participants. A majority of studies available, which demonstrate the inverse relationship between physical activity and depression, select participants who meet diagnostic criteria for depression. For example, Lane and Lovejoy (2001) found individuals in their depressed mood group "reported significantly greater mood changes" (pp. 544). Researchers found that physical activity interventions are efficacious for treatment of individuals experiencing mild to moderate depression (Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005; Harris et al., 2006). Additionally, Lennox, Bedell, and Stone (1990) reported that in a non-clinical population, physical activity failed to imbue long-term beneficial effect. However, for

my study, participants were not selected based on diagnostic criteria for depression. In conjunction with the positive depressive symptoms, many studies focus on prescribed physical activity (Babyak, et al., 2000; Blumenthal et al., 2007; Dunn et al., 2005; Pischke, Frenda, Ornish, & Weidner, 2010). My study looked at naturally occurring physical activity and did not encourage a set amount of physical activity. This may be problematic in individuals experiencing difficulty with motivation, particularly in those with depressed mood. Past research has demonstrated that low physical activity may be a behavioral correlate of depression, consistent with the premise that depression is associated with fatigue and loss of motivation (Jerstad et al., 2010).

Was Physical Activity Associated with Less Negative Cognitive Style For, Less Rumination About, or Less Emotional Reactivity to Stressful Events?

Negative cognitive style. Results did not support my hypothesis that physical activity would be associated with less negative cognitive style for stressful events (H_{2a}). Based on the hypothesis that physical activity potentially may offer positive reinforcement, which would disrupt the negatively reinforcing style found in individuals with negative cognitive style, I believed reductions in negative cognitive style would be one function of engaging in physical activity (Joiner et al., 2005). An explanation for the findings standing in contrast to this hypothesis may be the characterological nature of negative cognitive style makes the time-limited engagement of physical activity not potent enough to sustain long-term changes in thinking styles. Another potential explanation is that the act of engaging in physical activity is not potent enough to change the trajectory of an individual who is currently engaged in the negatively reinforcing cycle experienced by those with negative cognitive style (Jacobson et al., 2001).

Rumination.   Results did not support my hypothesis that physical activity will be associated with less rumination about stressful events (H_{2b}). Rumination involves passively engaging with negative material as opposed to actively engaging in pleasurable events. I hypothesized that physical activity would act as a distracting response, which stands in contrast to rumination. Physical activity was proposed as a form of distracting response due to both of these constructs association with emotional well-being. However, an explanation for this unsupported hypothesis may be that individuals prone to ruminate may engage in physical activity in order to further engage in the repetitive and passive thought process focused on negative emotions stemming from negative events (Nolen-Hoeksema, 2000; Nolen-Hoeksema & Marrow, 1993). Further, engagement in physical activity does not imbue a sense of efficacy and emotional well-being. This idea will be further discussed in limitations and future directions.

This null hypothesis was consistent with findings from past research. Craft (2005) found response style did not predict depression in those who engaged in physical activity.

Negative emotionality. Results did not support my hypothesis that physical activity will be association with less negative emotionality to stressful events (H_{2c}). My hypothesis was based on efficacy of behavioral activation as a supported intervention for modifying the regulatory processes involved in negative emotionality (Gross, 1998). However, whereas behavioral activation is a pre-planned, scheduled, and typically gradual increases in activity with thorough rational provided to the individual asked to engage in this treatment, I looked at self-initiated and non-prescribed physical activity. Behavioral activation is successful for many reasons, pertinent to this study it is

successful because it has folks engage in opposite-action behavior when feeling depressed as opposed to allowing mood to dictate and drive behavior. It is feasible that participants engaged in activity when their mood was elevated and in a very normal experience, decreased or did not engage in physical activity when mood was depressed or experiencing increase in stress. Additionally, Gross outlined five varying methods of increasing regulatory processes (1998). The lack of significance found in my study may be calling for a more holistic and thorough approach to recalibrating an individual who is either at risk for developing or currently experiencing depressive symptoms with regards to regulatory processes. For example, as a component of physical activity it may be crucial to have the individual also engage in cognitive change or attention deployment.

Do Lower Cognitive Style, Rumination, and Emotional Reactivity Mediate the Relationship Between Physical Activity and Depressive Symptoms?

Cognitive style, rumination, and negative emotionality did not mediate the relationship between physical activity and depressive symptoms. However, I found that the well-established relationship between two of the three mediators, rumination and negative emotionality, with depressive symptoms was replicated within my data. The effect of fluctuations in rumination and negative emotionality on depressive symptoms in the model with and without baseline depression was significant as was the aggregate mean of negative emotionality on depression. This significant finding suggests that lack of significance for my overall proposed model was not necessarily due to limitations with participants or the measures utilized.

Clinical Application

Although the current study did not identify significance within the proposed model, previous research demonstrates a significant negative relationship between physical activity and depressive symptoms (Babyak et al., 2000; Camacho et al., 1991; Dimeo et al., 2001; Galper et al., 2006; Häkkinen et al., 2001; Harris et al., 2006; Roth & Holmes, 1987; Stephens, 1988; Jerstad et al., 2010). In conjunction, theory-driven research has specified negative cognitive style, rumination, and negative emotionality as both risk and maintenance factors of depressed mood (Abramson et al., 1989; Alloy et al., 2005; Campbell-Sills et al., 2004; Gross, 1998; Gross & Muñoz, 1995; Jacobson et al., 2001; Joiner et al., 2005; Joorman & Gotlib, 2010; Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1993). Therefore, clinicians should focus their attention on holistic care of mood disorders and implement physical activity as an adjunctive tool in psychotherapy.

In implementing physical activity as a treatment, there are several key topics clinicians will need to address. Assessing current level of physical activity is needed as existing research has demonstrated those who exercise on a "regular basis believe that the benefits of exercise outweigh the effort made to complete the exercise session" (Lane & Lovejoy, pp. 540, 2001). While this impacts likelihood of adherence, it also taps into another key area clinicians need to assess for, intended function of physical activity engagement (Abele & Brehn, 1993). Advising clients that engaging in physical activity with the goal of improvement of mood and health benefits rather than competing with others or self will set the client up for greater mental health gains (Abele & Brehn, 1993; Berger & Motl, 2000). Research has also suggested that the fundamental predictor of

physical activities impact on depressive symptoms is likely the psychosocial experience and not the total energy expenditure (Pickett, Yardley, & Kendrick, 2012). Brosse and colleagues outlined several additional factors that are likely to impact adherence to physical activity for individuals with depressed mood such as, "attitudes towards the value and importance of exercise, perceived behavioural control, self-efficacy, early exercise experiences and recent involvement in physical activity, physical condition, knowledge about fitness and exercise, and perceived social support/encouragement to exercise (2002, pp.753). Prochaska and colleagues (1994) illuminated the need to assess and view physical activity along the spectrum of the five stages of change (pre-contemplation, contemplation, preparation, action, and maintenance) prior to prescribing a client a routine physical activity schedule. In order to assist clients through the stages of change the therapist must understand where the client is and utilize reinforcement, supportive relationships, and insight to facilitate client growth and change. Informing the client of the expectations regarding physical activity as a treatment component for depressed mood is always important. Meaning, therapist and client need to understand that just as pharmacotherapy's effectiveness is gauged post adequate trial, so to should be physical activity. This means, the client must adhere to prescribed plan for agreed upon duration in order to gain therapeutic benefit. Additionally, informing clients that maintenance of physical activity post successful trial may be crucial in maintaining benefits and thus preventing relapse (Babyak et al, 2000). Based on the findings of this study, the theoretical rationale for why physical activity functions as an antidepressant continue to remain unclear. While the current necessary dose, duration, type, and intensity of physical activity have yet to be determined, just like with pharmacotherapy,

the clinician can and should work with the client to track benefits of physical activity until an appropriate dose, duration, type, and intensity are found. Even with the seemingly extreme amount of uncertainty, at present the literature continues to support prescription of and support of engagement in physical activity for individuals with a mood disorder (Craft & Perna, 2004; Daley, 2008; Galper et al., 2006; Jerstad et al., 2010; Taliaferro et al., 2008).

Limitations and Future Directions

My goal for this current study was to elucidate potential theory-driven mechanisms at play in explaining why physical activity functions so well as an antidepressant. This study is one of the first to examine the relationship between physical activity, depressive symptoms, and the effect of negative cognitive style, rumination, and negative emotionality, and there are many improvements to be made when exploring these variables in the future. My present study was not without limitations, such as theory, measurement, and power and sample.

Theory. I would be remiss in not discussing that I focused solely on psychological mediators. Biochemical explanations were ignored in my hypothesis due to the nature of this study and my field of expertise. Just as the psychological mediators are unknown, so to are the physiological. Craft and Perna (2004) review the most common and probable physiological explanations. However, they conclude that methodological difficulties have caused difficulty in this area of research. Rot, Collins, and Fitterling (2009) outlined the neurobiological systems that are likely involved in the antidepressive effects of physical activity, (a) opioids and cannabinoids, (b) monoamines, (c) hypothalamic-pituitary-adrenal axis, (d) neurotrophins, and (e) neurocircuitry. Craft

and Perna (2004) emphasize that understanding the mechanisms involved in the negative relationship between physical activity and depressogenic experiences are at the intersection of specific physiological, biological, and psychological construct. Thus, future research should be interdisciplinary and begin to whittle away at these potential theory-driven mechanisms (see Craft & Perna, 2004 and Rot et al., 2009, for a more detailed discussion). Future research would also benefit from studies with more comprehensive potential mediators in order to begin explaining the mechanism behind the well-established inverse relationship between physical activity and depressive symptoms. For example, there is some support for self-efficacy as a potential mediator for the antidepressive effects of physical activity (Craft, 2005).

Measurement. My study contains multiple data points, however all of the measures are self-report data. Future studies should utilize a less-biased measure of physical activity, meaning that it is not sufficient to account for physical activity via recall and self-report alone. For example using accelerometers, a device that measures and records accelerations, to assess leisure time physical activity and heart-rate monitors during non-leisure physical activity would yield greater accuracy in breaking physical activity into various domains to assess for differences in benefit based on strenuous category.

Power and sample. With regard to PROCESS, my sample size was larger than originally mandated by my power analysis, however my analyses may have lacked sufficient power to determine significant effects due to the complex nature of my entire model. Low sample size contributed to issues with poor internal consistency and not enough variability to look at data for alternate models. As stated previously, there is

currently a lack of agreement regarding the standard for sample size regarding HLM analyses. Additionally, my sample consisted of college students from a predominantly Caucasian and female university, which likely limits the generalizability of my findings as they should not be generalized to college men or to the general public. The students also were voluntary participants, which may also have affected the representativeness and generalizability of the findings to other college students. Future research should focus on seeking individuals currently engaged in mental health counseling in addition to those not meeting criteria. In order to establish the theory-driven mechanisms for the antidepressive effect of physical activity on depressive symptoms it is likely that inclusion criteria for depression will need to be established and assessed. Previous research demonstrated smaller effects are likely to be seen in healthy non-symptomatic individuals in part due to a "floor effect where healthy subjects have less room to improve depressive symptoms than subjects with clinical depression" (Conn, 2010, p.135).

So what is the next step? The most crucial next step will need to be a clinical trial of physical activity as an intervention while still looking at the same proposed mediators. Future research should continue to examine the three hypothesized cognitive and affective mediators because fluctuations in these variables do effect depression and physical activity and its effects on depression do appear related to the severity and presence of mood disorder. One argument for an intervention study is related to the presentation of depression as an individual likely with reduced interest in activity, fatigue and loss of energy, indecisiveness, and feeling worthless. Thus, an individual experiencing depressive symptoms is likely less prone to engage in self-directed physical

activity. My study, which simple asked participants to track their current behavior is a great example of what not to do when attempting to ascertain how physical activity impacts the onset and maintenance of depressive symptoms. Similar to asking therapy clients to engage in behavioral activation when depressed, basically not allowing mood to dictate behavior, future research needs to focus on behavioral activation in prescribing the physical activity.

Physical activity is a useful intervention for those who meet criteria for clinical depression (Babyak et al., 2000; Camacho et al., 1991; Dimeo et al., 2001; Galper et al., 2006; Häkkinen et al., 2001; Harris et al., 2006; Roth & Holmes, 1987; Stephens, 1988; Jerstad et al., 2010). However, due to the presentation and experience of those with depressive symptoms, such as loss of energy, fatigue, reduced pleasure, feelings of worthlessness, physical activity likely needs to be introduced as an intervention with a structured protocol. My post-hoc analyses demonstrate support for the need for prescribed physical activity in light of the fact that depression itself may be serving as a barrier to engagement in onset or increase in physical activity. In my data the following pattern was present, (a) correlation between physical activity and depression was significant for low CES-D people not high (b) Low CES-D average physical activity was higher not lower. This trend gives support for an intervention study due to the likelihood that an individual with depressive symptoms presenting with reduced interest in activity, fatigue and loss of energy, indecisiveness, and feeling worthless is less likely to engage in positive behavior change. Of note, my study looked at currently ongoing physical activity and did not encourage any change or a set amount of physical activity. This may be problematic in individuals experiencing difficulty with motivation, particularly in

those with depressed mood. Thus, an individual experiencing depressive symptoms is likely less prone to engage in self-directed physical activity. Past research has demonstrated that low physical activity is correlated with depression (Jerstad et al., 2010). My study, which simply asked participants to track their current behavior, is a great example of what not to do when attempting to ascertain how physical activity impacts the onset and maintenance of depressive symptoms. Similar to asking therapy clients to engage in behavioral activation when depressed, basically not allowing mood to dictate behavior, future research needs to focus on behavioral activation in prescribing the physical activity. Many past studies utilize prescribed physical activity, potentially to counterbalance this pattern in individuals with depressive symptoms (Babyak, et al., 2000; Blumenthal et al., 2007; Dunn et al., 2005; Pischke, Frenda, Ornish, & Weidner, 2010).

In conclusion, this study suggests that further exploration of the mechanisms accounting for the antidepressive effects of physical activity need more rigorous and continued research. At present, the extent to which individuals can benefit from physical activity is unknown regarding mental health outcomes. Identifying those who will benefit, the specific type and duration of physical activity necessary, and longer-term behavioral change to reduce relapse remains crucial for treatment interventions. Further exploration is needed to establish physical activity as an approved treatment option for individuals at-risk of developing or currently experiencing depressive symptoms.

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