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The Role of Self-Efficacy in Mediating the Effect of Physical Activity on Adolescent Depression

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THE ROLE OF SELF-EFFICACY IN MEDIATING THE EFFECT OF PHYSICAL
ACTIVITY ON ADOLESCENT DEPRESSION

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DEDICATION

Dedicated, with love and gratitude, to Kevin, Will, and Béla.

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With deepest gratitude, I would like to acknowledge several individuals, without whom this work would not have been possible: my parents, who always valued and prioritized my education; my family, Kevin, Will, and Béla, who have sacrificed time, travel, and togetherness; my extended family, the Noblits and the Trumpeters, who have helped me balance all the hard work with a lot of fun; my friends, especially, Sara, Sara, Sandra, Kassy, and Hannah – grad school would have been impossible without you by my side; my former teachers and mentors, Mr. Wells, Ms. Mines, Mr. Smith, Ms. Neal, Ms. Waddell, Dr. Metzger and Dr. Watson, who taught me how to inquire, think, and write; my statistical support, Dr. Van Horn and Dr. Siceloff, who graciously gave their time and expertise; my committee, Dr. Weist, Dr. Zarrett, and Dr. Kitzman-Ulrich, who gave their time, support, and guidance on this document and throughout my training; my clinical mentor, Dr. Burnette, who encouraged me weekly to keep at it; and, most of all, my mentor, Dawn, who gave me a chance, accepted me into her life as an employee, mentee, daughter, and friend, and lived up to her promise that she would both demand of me and support me relentlessly. Thank you, all.

ABSTRACT

Depression is a common psychiatric problem experienced in adolescence with nearly a quarter of all adolescents experiencing a major depressive episode before adulthood. Previous evidence indicates that physical activity (PA) is a known protective factor for depressive symptoms and major depressive disorder in adolescents and that PA self-efficacy (i.e., self-confidence) improves self-esteem, which in turn reduces depressive symptoms. Furthermore, PA self-efficacy may be more protective against depression for individuals who value or consider PA to be highly important. The proposed study aimed to determine the cross-sectional and longitudinal PA-depressive symptoms relation in adolescents. The study was designed specifically to test a mediation model, wherein PA self-efficacy mediated the cross-sectional and longitudinal PA-depressive symptoms relations, and to test a moderation model, wherein PA valuation moderated the PA self-efficacy-depressive symptoms relation. The participants were a subsample of youth enrolled in the Active by Choice Today trial, a randomized controlled trial that tested the efficacy of an afterschool theory-based PA intervention. Students were recruited for the larger trial through flyers and events at their middle schools. These 409 sixth graders were enrolled in the first two years of the trial. The mean age was 11.4 (SD=0.65), most (72%) were African American, and about half (55%) were females. Participants completed the Children's Depression Inventory (CDI) and self-report measures of PA self-efficacy and PA valuation. Participants wore accelerometers and seven-day moderate-to-vigorous PA (MVPA) estimates were calculated from the raw PA

counts. In both the cross-sectional and longitudinal models, there was no direct effect of MVPA and depressive symptoms ($p>0.05$). In the cross-sectional model, there was a significant relation between MVPA and PA self-efficacy (a path; $B=0.006$, $SE=0.002$, $p=0.009$) and a significant relation between PA self-efficacy and depressive symptoms (b path; $B=-0.262$, $SE=0.069$, $p<0.001$), partially supporting the proposed mediation model. The hypothesized moderating effect of PA valuation was not supported in the cross-sectional or longitudinal models. A secondary finding of interest was that African Americans reported significantly fewer depressive symptoms than their peers at T1 ($t=-2.32$, $p=0.021$) and at T2 ($t=-1.99$, $p=0.047$). Overall, these findings suggest that, when objectively measured, MVPA may not be correlated with depressive symptoms in young adolescents. However, the findings supported the notion that domain-specific PA self-efficacy may be related to depressive symptoms. Finally, from a developmental perspective the data suggested that, in early adolescence, African American youth may experience fewer depressive symptoms than their peers. The theoretical and clinical implications of these findings are discussed.

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CHAPTER 1

INTRODUCTION

Background

Depression is projected to be the second leading cause of global disease burden by 2030 (Mathers & Loncar, 2006). Thus, the rising rates of adolescent depression are a growing public health concern (Ali, Fang, & Rizzo, 2010; McElroy et al., 2004).

Depression is the most common psychiatric problem experienced by adolescents with a quarter of adolescents experiencing at least one major depressive episode (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Lewinsohn, Rohde, & Seeley, 1998; Stice, Shaw, Bohon, Marti, & Rohde, 2009) and a third experiencing elevated depressive symptoms (Kubik, Lytle, Birnbaum, Murray, & Perry, 2003; Saluja et al., 2004).

Depressed mood is not only a predictor of major depression (Garber, 2006; Van Voorhees et al., 2008), it is associated with both concurrent and subsequent medical, social, and emotional problems, including educational and occupational problems, impaired social functioning, substance use, disordered eating, sexual risk taking, physical inactivity, obesity and suicide (Dockray, Susman, & Dorn, 2009; Franko et al., 2005; Fulkerson, Sherwood, Perry, Neumark-Sztainer, & Story, 2004; Gillham, Shatté, & Reivich, 2001; Goodman & Whitaker, 2002; Jerstad, Boutelle, Ness, & Stice, 2010; Johnson, Rohan, & Kirk, 2002; Katon et al., 2010; Merten, Wickrama, & Williams, 2008; Motl, Birnbaum, Kubik, & Dishman, 2004; Puder & Munsch, 2010; Tanofsky-Kraff et al., 2008).

Although the literature is mixed, ethnic minority adolescents may be at relatively higher risk for developing major depressive disorder than their peers (Alloy et al., 2012; Anderson & Mayes, 2010; Doi, Roberts, Takeuchi, & Suzuki, 2001; Flay, 2002; Kennard, Stewart, Hughes, Patel, & Emslie, 2006; Van Voorhees et al., 2008). For example, studies using a large, nationally representative sample of adolescents in grades 7-12 showed that African American youth consistently reported more depressive symptoms across three time points over seven years (Halfon, Larson, & Slusser, 2013) and that African American youth were twice as likely to develop major depression (Van Voorhees et al., 2008). In contrast, there is some evidence suggesting that the rates of depressive symptoms are similar or lower in African American adolescents as compared to Caucasians (grades 5-12; Costello et al., 1996; Kennard et al., 2006; Latzman et al., 2011; McLaughlin, Hilt, & Nolen-Hoeksema, 2007; Saluja et al., 2004; Schraedley, Gotlib, & Hayward, 1999). Overall, the results of studies that include younger adolescents tend to suggest that African American youth have lower or similar rates of depression than their peers, but this trend changes when older adolescents are studied. Nonetheless, with depressive symptoms in adolescence often predicting depression in adulthood (Franko et al., 2005; Garber, 2006; Halfon et al., 2013; Kessler et al., 1994; Lewinsohn, Rohde, Klein, & Seeley, 1999; Pine, Cohen, Gurley, Brook, & Ma, 1998), it is important to understand these relations starting in early adolescence to inform prevention and early intervention, and ongoing research is needed in underserved populations such as African American youth.

Physical activity (PA) may be protective against depressive symptoms in adolescence (Janssen & Leblanc, 2010) and an important point of intervention for

adolescent depression. Recent reviews highlight longitudinal and intervention effects of PA on decreased adolescent depressive symptoms (Biddle & Asare, 2011; Brown, Pearson, Braithwaite, Brown, & Biddle, 2013; Camero, Hobbs, Stringer, Branscum, & Taylor, 2013; Dunn & Weintraub, 2008; Hoare, Skouteris, Fuller-Tyszkiewicz, Millar, & Allender, 2013). However, the current literature is limited in two significant ways. First, the preponderance of evidence comes from observational studies using self-reported PA data, which may bias results (Bassett, Mahar, Rowe, & Morrow, 2008; Kohl, Fulton, & Caspersen, 2000; Sallis, Taylor, Dowda, Freedson, & Pate, 2002). Although the existing intervention studies, in which PA was manipulated to improve depressive symptoms, provide stronger support for the effect of PA on adolescent depressive symptoms, the trials to date are few and lack scientific rigor (Biddle & Asare, 2011; Dunn & Weintraub, 2008). Second, little research has investigated the mechanisms by which PA reduces adolescent depressive symptoms. Although biological (e.g., endorphins) and social factors (e.g., peer social support) have been the primary focus for explaining these relations, key psychological factors are also likely to contribute to these effects (MacMahon, 1990; Phillips, Kiernan, & King, 2001). Theory and empirical data point to self-efficacy as a key mediating construct (MacMahon, 1990; Ryan, 2008; Sonstroem & Morgan, 1989; Trumpeter & Wilson, 2013; Wilson, 2015). Self-efficacy is defined as one's sense of competence for engaging in a goal-directed behavior (Bandura, 1977) and is conceptualized as domain-specific - in reference to the specific behavior or life domain, for example physical activity - rather than as a global construct (Bandura, 1997; Bandura, 1978). There is data to suggest that PA self-efficacy may mediate the effect of PA on depressive symptoms in adolescents (Annesi, 2004; Dishman et al., 2006).

These results are consistent with the broader adolescent depression literature which suggests that positive self-schemas, including high self-efficacy, are protective against depressive symptoms in adolescence (Bandura, Pastorelli, Barbaranelli, & Caprara, 1999; Garber, 2006; Hyun, Chung, & Lee, 2005; Orth, Robins, & Roberts, 2008). However, several theorists suggest that there are individual differences in the behavioral domains that are most relevant to sense of self, self-worth, and wellbeing (Bandura, 1978; Eccles et al., 1989; Harter & Whitesell, 2001; Marsh, 1986, 1993); therefore, PA self-efficacy, for example, may mediate the effect of PA on depressive symptoms only in the subset of adolescents who value PA. To the author's knowledge, no studies have investigated whether PA valuation (PAV), or the extent to which PA is important to youths' definition of self, may moderate the effect of PA self-efficacy on depressive symptoms in youth.

The purpose of the proposed study is to fill the gap in the literature by investigating the relation between accelerometry measured PA and depressive symptoms in a sample of underserved adolescents. PA measured with accelerometers reduces measurement error due to participant bias (Bassett et al., 2008) and reduces common-method variance typical for self-report data (Sallis et al., 2002). Moreover, this study will test PA self-efficacy as a mediator of the relation between PA and depressive symptoms and include PAV as a moderator of the PA self-efficacy-depressive symptoms relation in underserved youth in early adolescence.

Integrated Theoretical Framework

The theoretical model for this study (see Figure 1.1) incorporates cognitive explanations of the impact of PA on depression, which generally point to PA self-efficacy

as an important mediator (Annesi, 2005; MacMahon, 1990; Phillips et al., 2001). These explanations are built on social cognitive theory (SCT), which emphasizes the role of self-efficacy, or beliefs about one's ability to engage in desired behaviors, as influencing all aspects of human agency (Bandura, 1977; Bandura, 1991). Bandura, like other theorists, emphasizes that self-representations and related constructs (e.g., self-efficacy) are best conceptualized in reference to the specific behavior or life domain, for example physical activity, rather than as a global construct (Bandura, 1978; Bracken & Lamprecht, 2003; Marsh, 1990). This may be especially true in adolescence when self-representations become more differentiated with exposure to increasingly varied situations and experiences (Bracken & Lamprecht, 2003; Hankin & Abela, 2005; Marsh, 1990). SCT asserts that self-efficacy influences not only behavior (i.e., choices, motivation, perseverance), but also cognitive processing and affective reactions to the environment, and vulnerability to depression (Bandura, 1991). More specifically, self-efficacy influences emotional wellbeing by increasing positive evaluative self-reactions, or feelings of self-satisfaction, self-pride or self-worth (Bandura et al., 1999; Bandura, 1978; Bandura, 1991). Self-efficacy, for example, impacts causal attributions of successes and failure, with highly efficacious individuals ascribing successes internally and failures to low effort (Bandura, 1991), which is protective against depression (Alloy et al., 2012; Hankin & Abramson, 2001). Self-efficacy is developed in part through personal experiences of mastery, in which the person successfully performs a desired behavior (Bandura, 1977). Mastery experiences in PA, for instance, build PA self-efficacy. The effect of PA on PA self-efficacy is, in fact, quite robust in correlational (Sallis et al., 2002), longitudinal (Neissaar & Raudsepp, 2011; Stein, Fisher, Berkey, &

Colditz, 2007), and intervention studies (e.g., Annesi, 2004; Dishman et al., 2005; Goldfield et al., 2007; Haerens et al., 2008; Lubans, Foster, & Biddle, 2008; Lytle et al., 2009; Taymoori & Lubans, 2008; van Stralen et al., 2011).

The predominate psychological model explaining the influence of PA on depressive symptoms, the so called “mastery hypothesis”, suggests that PA can afford a sense of accomplishment, thereby increasing PA self-efficacy and alleviating or protecting against depressive symptoms (Annesi, 2005; MacMahon, 1990; Martinsen, 2008; Rethon et al., 2010; Ryan, 2008; Sonstroem & Morgan, 1989). In other words, heightened PA self-efficacy mediates the relation between physical activity and depression (Annesi, 2005). Provisional support for a mediational influence of cognitive factors, such as PA self-efficacy, was recently reported in review of the effects of physical activity interventions on improving self-perceptions and depressive symptoms in adolescents (Trumpeter & Wilson, 2013). One study showed that changes in exercise self-efficacy were related to changes in depression, but did not formally test a mediation model (Annesi, 2004). Dishman et al. (2006) found that physical self-esteem mediated the cross-sectional relation between PA and depressive symptoms in a sample of 12th grade girls (54% African American). However, this cross-sectional study does not establish temporal ordering of the variables and provides only weak support for the mediation model. Two studies have shown global self-perception constructs, such as self-esteem and global self-worth, to mediate the PA-depression relation longitudinally and PA intervention effects on depressive symptoms (McPhie & Rawana, 2012; Petty, Davis, Tkacz, Young-Hyman, & Waller, 2009), but none have directly demonstrated the mediating effect of PA self-efficacy. The current study aims to inform intervention

development and, therefore, expands on the existing literature by specifically examining the mediating role of domain-specific PA self-efficacy both through cross-sectional and longitudinal analyses.

The mastery hypothesis is consistent with cognitive theories of depression that purport negative cognitions about the self (e.g. perceived inefficacy) as increasing vulnerability for depression in youth (Beck, Rush, Shaw, & Emery, 1979; Garber, 2006; Hankin & Abramson, 2001; Weersing & Brent, 2006). Broadly, negative cognitive tendencies, including dysfunctional attitudes, negative inferential style, negative self-perceptions, and ruminative response style, interact with negative life events to activate negative attention and interpretation biases (e.g., global, stable, internal interpretations of negative events) to increase the likelihood of depressive symptoms (Alloy et al., 2012; Hankin & Abramson, 2001; Jacobs, Reinecke, Gollan, & Kane, 2008). Dysfunctional attitudes refer to deep-seeded, negative self-schemata, or cognitive representations of the self, and drive negative automatic thoughts, sometimes called cognitive distortions (Beck et al., 1979). Negative inferential style, sometimes called negative attributional style, negative explanatory style, or pessimism (Contrada & Goyal, 2004), refers to a tendency to attribute the causes and consequences of negative events to internal, stable, and global factors (Hankin & Abramson, 2001). Negative self-perceptions include low self-esteem and perceptions of incompetence and are thought to be caused by negative competency evaluations by important significant others (Jacobs et al., 2008). Finally, ruminative response style refers to a coping style characterized by directing attention towards negative feelings and thoughts (Nolen-Hoeksema, 1991). Rumination increases access and recall of negative events and focuses attention on negative self-referent cognitive

content provided by dysfunctional attitudes and negative inferential style (Hankin & Abramson, 2001). In other words, ruminative response style refers to a cognitive process whereas dysfunctional attitudes and negative inferential style refer to cognitive content. Unlike dysfunctional attitudes and negative inferential style, ruminative response style has not been shown to be depression-specific (Alloy et al., 2012; Hankin & Abramson, 2001). Therefore, negative cognitions about the self, rather than cognitive processes, may be more relevant to the development of adolescent depressive symptoms. The research supports that negative self-perceptions in general are risk factors for depression in adolescence (Bandura et al., 1999; Caprara, Gerbino, Paciello, Di Giunta, & Pastorelli, 2010; Carter & Garber, 2011; Garber, 2006; Hyun et al., 2005; Orth et al., 2008; Pössel, Baldus, Horn, Groen, & Hautzinger, 2005) and that this cognitive model extends to ethnic minority adolescents (Kennard et al., 2006; Stewart et al., 2004; Van Voorhees et al., 2008). Kennard and colleagues (2006), for example, showed that low self-efficacy was correlated with depressive symptoms across all ethnic groups in their sample of 450 adolescents.

Cognitive depression theorists, like self-concept theorists, further propose that cognitive vulnerabilities exist in specific life domains (Hankin & Abela, 2005; Hankin & Abramson, 2001). From a developmental perspective, life domains, and therefore cognitive vulnerabilities, influencing depression likely change over the lifespan (Eccles et al., 1989; Hankin & Abramson, 2001; Harter & Whitesell, 2001). For example, peer-oriented domains, such as physical attractiveness and athletics, may be most relevant for adolescents (Hankin & Abramson, 2001; Harter, Marold, & Whitesell, 1992; Harter & Whitesell, 1996). Several studies have also demonstrated that domain-specific self-

perceptions, i.e, perceived athletic competence (a construct closely related to PA self-efficacy) are related to depressive symptoms (Cole, Martin, & Powers, 1997; Cole, Maxwell, et al., 2001; Dozois, Eichstedt, Collins, Phoenix, & Harris, 2012; Kim-Spoon, Ollendick, & Seligman, 2012). In a longitudinal study of 617 (66% White, 30% African American) elementary school students, perceived competence predicted change in depressive symptoms over time. Athletic competence was negatively correlated with depressive symptoms in both 3rd and 6th grade, for both boys and girls (Cole et al., 1997). These relations held in the longitudinal analyses after controlling for baseline depression and self-evaluations. Dozois et al. (2012) compared adolescents, aged 13-17, with major depressive disorder to nonpsychiatric controls and found that the clinically depressed adolescents demonstrated poorer self-concept than controls in a number of domains, including athletic competence.

Similar to these studies demonstrating domain specific cognitions are related to depressive symptoms generally, other researchers have looked at the relation at the individual level. Bandura recognized that different behavioral domains are regarded differently by each individual (Bandura, 1978; Bandura, 1991). He further suggested that individuals attend more to valued activities, that is, activities affecting one's sense of welfare and self-esteem (Bandura, 1978). Therefore, performance and, in turn, self-efficacy is most relevant to one's self-worth in domains which are valued by or significant for the individual. Moreover, Bandura and colleagues have shown that low self-efficacy for valued activities can give rise to depression (Bandura, Pastorelli, Barbaranelli, & Caprara, 1999). These ideas are consistent with arguments made by Harter, Eccles, and their colleagues. Harter's research has supported the Jamesian

perspective on self-worth which suggests that the salience or “importance” of a domain affects the extent to which that domain impacts self-worth and depression (Harter, Marold, & Whitesell, 1992; Harter & Whitesell, 1996, 2001; Harter, Whitesell, & Junkin, 1998). Similarly, Eccles purports that self-esteem is grounded in the context of values, or valued life domains (e.g., Eccles et al., 1989). Although different statistical approaches have been used, research has shown that the importance of a life domain interacts with domain-specific self-perceptions to contribute to global self-esteem (Harter & Whitesell, 2001; Harter, Whitesell, & Junkin, 1998; Rodriguez, Wigfield, & Eccles, 2003). Therefore, the current theoretical model suggests that PAV may moderate the PA self-efficacy-depression relation such that the relation is stronger for people who value PA more highly.

Developmental Perspective

Beliefs about the self, and specifically self-efficacy beliefs, are especially relevant to depression in early adolescence, which is a transitional developmental period, when capacity for self-awareness and self-evaluation increase (Cole, Maxwell, et al., 2001; Harter & Whitesell, 2001; Steinberg et al., 2006; Wilson, Zarrett, & Kitzman-Ulrich, 2011) and identity formation, including a sense of one’s competencies, becomes a major developmental objective (Cole et al., 1997; Eccles et al., 1989). Cole, Martin, and Powers (1997) further note that negative competence self-perceptions are more common in depressed youth than adults and suggest that the association of self-competence and depression may be specific to this developmental period. In early adolescence, sense of competence, or self-efficacy, is influenced not only by cognitive changes, but also coinciding biological changes, namely puberty, and social changes, such as the transition

to middle school. Simmons and colleagues have demonstrated that the compounding effects of pubertal changes and school transition put students at the greatest risk for negative outcomes (e.g., Blythe, Simmons, & Carlton-Ford, 1983; Simmons, Burgeson, Carlton-Ford, & Blyth, 1987). Furthermore, theory suggests that changes in the social environment (more performance oriented, competitive school environments) as well as changes in cognitive capacities and processes (abstract thinking abilities and being more self-focused) lead to declines in self-efficacy for two reasons. Adolescents, as compared to children, tend to have a more realistic view of their own competencies as they compare themselves to a larger peer group leading to relative declines in competence ratings. Adolescents genuinely demonstrate declines in their self-evaluations due to the nature of the school environment and increasing self-consciousness (Cole, Jacquez, & Maschman, 2001; Harter & Whitesell, 2001; Wigfield & Eccles, 2002; Zanobini & Usai, 2002). The increased strength and salience of affective responses to self-perceptions, due in part to hormonal and neurological changes, increases risk for negative emotional outcomes, including depression (Steinberg et al., 2006). Indeed, perceptions of competencies, including PA competence and PA self-efficacy, have been shown to decline and be less stable at the transition to middle school (Cole, Maxwell, et al., 2001; Garcia, Pender, Antonakos, & Ronis, 1998; Wigfield & Eccles, 1994; Wigfield, Eccles, Mac Iver, Reuman, & Midgley, 1991). Depressive symptoms also tend to increase and be less stable at the middle school transition (Roeser, Eccles, & Sameroff, 1998; Tram & Cole, 2006).

In addition to significant changes in self-evaluations and affective reactions to those evaluations, valued life domains are also changing in early adolescence as a result of changing social and biological factors (Eccles et al., 1989; Harter & Whitesell, 2001;

Steinberg et al., 2006). Eccles et al. (1989) highlighted the importance of understanding one's competencies and formulating self-evaluations in the context of valued life domains in early adolescence. In other words, understanding how perceived competencies and valuation within a domain develop together provides a richer understanding of self-worth and related emotional outcomes such as depressive symptoms (Eccles et al., 1989; Harter & Whitesell, 2001; Harter et al., 1998). For some, changes in perceived competence in a domain may lead to a change in valuation for that domain (Rodriguez, Wigfield, & Eccles, 2003; Wigfield & Eccles, 2002). That is, some adolescents are able to "discount" behavioral domains for which perceived competence is low, thereby protecting themselves from depressive symptoms such as low self-worth (Harter et al., 1998). Others may continue to engage in a valued activity even when they do not have high self-efficacy for the activity (Schunk & Meece, 2005). It is these adolescents, those who value an activity, for example PA, but who have low self-efficacy for the activity, who are at the greatest risk for depressive symptoms (Bandura, 1978; Harter & Whitesell, 2001).

Although the initial transition to middle school disrupts stability of self-perceptions, values, and depressive symptoms, during the transition year (e.g., sixth grade), self-perceptions, values, and depressive symptoms tend to stabilize. For example, research has shown that constructs related to PA self-efficacy, such as perceived sports competence, are relatively stable from fall to spring of the transition year (Cole, Maxwell, et al., 2001; Eccles et al., 1989). The same pattern is observed for PAV (Eccles et al., 1989) and depressive symptoms (Hoffman, Cole, Martin, Tram, & Seroczynski, 2000; Tram & Cole, 2006). These studies are consistent with other research that has shown the

ability and tendency to make stable, internal attributions for events and behavior increase with development and that cognitive processes may become more firmly established with age (Gibb & Coles, 2005). Similarly, over the course of adolescence, cognitive vulnerabilities to depression become fully consolidated and functioning (Hankin & Abramson, 2001), and the relation between negative cognitions and depressed mood strengthens (Garber, 2006). Examining the cognitive processes linking PA and depressive symptoms over the course of sixth grade may inform interventions aiming to change developmental trajectories that begin in this transition year.

Physical Activity and Depressive Symptoms

Increasing evidence from cross-sectional, longitudinal, and intervention studies suggests that physical activity decreases adolescent depressive symptoms (Fulkerson et al., 2004; Hoare et al., 2013; Janssen & Leblanc, 2010; Jerstad et al., 2010; Motl et al., 2004; Norris, Carroll, & Cochrane, 1992; Penedo & Dahn, 2005; Sund, Larsson, & Wichstrom, 2011; Wyatt, Winters, & Dubbert, 2006). Observational data indicate that there is not only a significant negative association between PA and depressive symptoms in adolescents (age 11-18) but that there may be a dose-response effect of intensity of PA on depressive symptoms (Fulkerson et al., 2004; Goldfield et al., 2011; Jerstad et al., 2010; Neissaar & Raudsepp, 2011; Rethon et al., 2010; Sallis, Prochaska, & Taylor, 2000; Stavrakakis, de Jonge, Ormel, & Oldehinkel, 2012; Strong et al., 2005; Tao et al., 2007). For example, Jerstad et al. (2010) followed 496 girls (average age at baseline =13) through six years of adolescence and found that self-reported PA reduced risk of future depressive symptoms and onset of depression (and vice versa). Goldfield and colleagues (2011) demonstrated that depressive symptoms were more strongly related to vigorous

PA than mild-moderate PA, measured by self-report. Likewise, a recent review of twelve studies using longitudinal data from large, representative adolescent samples (ages 11-19; four out of 12 of the studies included 11 year olds) shows that self-reported PA predicts depressive symptoms (Hoare et al., 2013). Others have shown that change in PA over time predicts change in depressive symptoms (Neissaar & Raudsepp, 2011). However, a major limitation of these observational studies is the use of self-report PA data. In fact, only three observational studies to date have investigated the relation between depressive symptoms and objectively measured PA estimates, and, although Wiles et al. (2012) showed a negative relation between total PA and depressive symptoms, none of them showed a relation between MVPA and depressive symptoms (Johnson et al., 2008; Toseeb et al., 2014; Wiles, Haase, Lawlor, Ness, & Lewis, 2012).

Evidence from intervention studies further strengthens the case that PA influences depressive symptoms. The majority of intervention studies to date have been prevention focused, as opposed to treatment programs, using adolescents who are healthy or at risk for developing depression (Biddle & Asare, 2011; Larun, Nordheim, Ekeland, Hagen, & Heian, 2006). Recent reviews similarly concluded that there is a small significant treatment effect of PA on depressive symptoms in these community samples (Biddle & Asare, 2011; Brown et al., 2013; Camero et al., 2013; Larun et al., 2006). Moreover, some studies indicate a dose-response effect with higher intensity PA (aerobic exercise) producing greater decreases in depressive symptoms than low-intensity comparison conditions (Crews, Lochbaum, & Landers, 2004; Petty et al., 2009). Crews et al. (2004) compared an aerobic exercise intervention to a moderate PA comparison condition and found reductions in depressive symptoms in the aerobic exercise group only. As

measured by the Beck Depression Inventory, there was a 2-point decrease in depression in the aerobic condition ($M=11.6\pm 9.1$ to $M=9.5\pm 9.5$) as compared to a 1-point increase in the control group ($M=17.5\pm 9.5$ to $M=18.7\pm 11.0$), and the between groups effect was significant ($F=6.93, p<.05$). Petty et al. (2009) demonstrated a linear dose-response effect of PA on depressive symptoms in a sample of overweight youth aged 7-11. Comparing a high-dose (40 minutes/day) aerobic exercise condition, a low-dose (20 minutes/day) aerobic exercise condition, and a control condition, these researchers showed, after adjusting for baseline depression score and body mass index, an intervention effect ($p=0.045$) on depression measured with the Reynolds Child Depression Scale, with the high-dose group reporting the lowest depression scores, followed by the low-dose group and then the control group. Pairwise comparisons revealed that there was a statistically significant difference ($p=0.02$) in depression scores between the high-dose group and the control group at post-intervention, but not between the high- and low-dose conditions or between the low-dose and control conditions. Only a few studies have examined PA as a treatment for clinically depressed adolescents (see Dunn & Weintraub, 2008; Larun et al., 2006 for reviews; Philipsson, Duberg, Moller, & Hagberg, 2013). Although preliminary, the results of these trials provide supportive data for continued treatment research and further strengthen the evidence that PA reduces depressive symptoms across a range of symptom severity (Dunn & Weintraub, 2008).

PA Self-Efficacy as a Mediator

Support for a mediational influence of cognitive factors was recently reported in a review of physical activity interventions on improving self-perceptions and depressive symptoms (Trumpeter & Wilson, 2013). In this review, the authors showed that

interventions targeting both physical health (weight and/or PA) and depression outcomes were, overall, also affecting cognitive mediators including self-efficacy for PA. However, as mentioned above, the mediational effect of PA self-efficacy on the association between PA and depressive symptoms has not been tested directly in youth. Although the majority of the studies in this review conceptualized PA self-efficacy (and related constructs) as an outcome, two specifically conceptualized and provided evidence in support of the cognitive variables as mediators. Annesi (2004) conducted a single-group, pre-post pilot study examining the effects of a PA intervention on depression with 54 young adolescents (93% African American; 9-12 years old). He showed not only that a PA intervention improved both depressive symptoms and exercise self-efficacy, but that the changes in depressive symptoms were related specifically to the changes in exercise self-efficacy. Petty et al. (2009) showed in a dose-response randomized trial of exercise on depression for overweight youth (age 7-11) that global self-worth mediated the PA intervention on depressive symptoms for white youth only and that physical appearance self-perceptions partially mediated the intervention effects for the entire sample. It is important to note that both of these studies included young adolescent samples. Furthermore, Dishman et al. (2006) demonstrated that PA self-efficacy mediates the relation between physical activity and depression in adolescents; in a cross-sectional study, they found that self-esteem for PA (and global self-esteem) mediated the relation between PA and depressive symptoms in 12th grade girls (54% African American). Lastly, two studies with adults have demonstrated that PA self-efficacy mediates cross-sectional PA-depression associations (Pickett, Yardley, & Kendrick, 2012; Ryan, 2008). Together, these studies, using samples across adolescence and adulthood, provide

preliminary support for the proposed theoretical framework that hypothesizes the impact of physical activity on depressive symptoms is mediated by PA self-efficacy. The present study expands on this past research by being the first study to test a longitudinal model wherein the specific construct, PA self-efficacy, mediates PA-depressive symptoms associations in underserved, young adolescent youth.

Valuation as a Moderator

Few studies have investigated the proposed notion that the importance (value) of a life domain may interact with self-perceptions in that domain to influence depression. To the author's knowledge, only one study has examined the domain-specificity of cognitive vulnerabilities and depression in youth (Rood, Roelofs, Bogels, & Meesters, 2012). In a sample of 10-18 year olds ($M = 12.4 \pm 1.9$ years old) Rood et al. (2012) found that negative cognitive style for appearance was more strongly related to depressive symptoms for girls, and negative cognitive style in the interpersonal domain was related to depressive symptoms for girls and boys. Assuming that appearance is more important for adolescent girls than boys, these results support a valuation by cognition interaction effect on depressive symptoms. Marsh (1986) investigated the effect of importance ratings by domain-specific self-esteem ratings in a university sample. He found that for the physical abilities domain, which demonstrated the most inter-individual variability, the effect of importance interacted with the physical self-esteem measure to contribute significantly to global self-esteem. Similarly, in a study with adolescent boys, global academic self-esteem was best predicted by academic subdomains that have been empirically shown to be generally more important, i.e. core subjects, such as English, math, and science versus noncore subjects, such as art, music, and physical education

(Marsh, 1993). These studies provide preliminary evidence supporting that those with higher valuation may demonstrate greater depressive symptoms given more negative self-perceptions (self-efficacy), but more research is needed.

Purpose of the Present Study

The current study aimed to fill a gap in the literature by testing the relations between PA and depressive symptoms in adolescents and investigating PA self-efficacy as a potential mediator of the PA-depressive symptom relation and PA valuation as a potential moderator of the PA self-efficacy-depressive symptoms association. This study used data from the Active by Choice Today (ACT) trial, which has been described in detail previously (Wilson et al., 2008; Wilson, Van Horn, et al., 2011). The purpose of the ACT trial was to test the efficacy of a motivational plus behavioral skills theory-based PA intervention for early adolescents. The ACT trial was a group-randomized cohort design in which schools were paired pre-randomization based on school size, percentage of ethnic minorities, percentage of students on free or reduced lunch, and urban or rural setting. The ACT program was implemented after school three days per week for 17 weeks. Measures were collected at baseline before randomization (T1) and 2-weeks after the 17-week intervention (T2). Schools were randomized to receive the theory-based PA program or a comprehensive health education program. Although there was an increase in PA in the intervention group as compared to the control group at mid-point, there were no significant intervention effects of PA at post-intervention (Wilson, Van Horn, et al., 2011). Therefore, for this study, the data were collapsed across intervention assignment.

A number of covariates were included in the statistical models in order to understand the proposed relation over and above variables known to influence MVPA,

depression, or both. BMI is known to be correlated with both MVPA and depressive symptoms (Dockray et al., 2009; Janssen et al., 2005). Girls tend to report more depressive symptoms and less PA than boys (Hankin & Abramson, 2001; Troiano et al., 2008). Race and sex interact to influence PA with African American girls engaging in the least amount of PA (Troiano et al., 2008). Peer social support is associated with PA and depression (Lawman, Wilson, Van Horn, & Zarrett, 2012; Stice, Rohde, Gau, & Ochner, 2011). School (dummy coded) was also included as a covariate to control for school, cohort, and intervention effects.

The first aim of the study was to test a cross-sectional simple mediation model and moderated-mediation model positing that (a) PA decreases depressive symptoms by increasing PA self-efficacy (PASE) and (b) PA valuation (PAV) moderates the relation between PASE and depressive symptoms such that individuals with higher PAV demonstrate a stronger PASE-depressive symptoms relation. Additionally, a discrepancy score analysis (discrepancy score = T1 PASE Z-score - T1 PAV Z-score), which identified individuals with higher PAV relative to their PASE, served as an additional test of the moderated-mediation model (Harter, 1990). In consideration of the primary aim, it was hypothesized that:

- (1) Controlling for covariates (BMI, sex, age, ethnicity, school, peer social support), T1 MVPA is positively associated with T1 PASE (a path) and lower levels of T1 depressive symptoms (c path).
- (2) Controlling for covariates (BMI, sex, age, ethnicity, school, peer social support), and partialling out the effects of T1 MVPA, T1 PASE is negatively associated with T1 depressive symptoms (b path).

- (3) Controlling for covariates (BMI, sex, age, ethnicity, school, peer social support), and partialling out the effects of T1 MVPA, T1 PASE is negatively associated with T1 depressive symptoms (b path) and the negative relation between T1 PASE and T1 depressive symptoms (b path) is stronger for individuals with higher PAV at T1.
- (4) Controlling for covariates (BMI, sex, age, ethnicity, school, peer social support), T1 MVPA is positively associated with a lower T1 discrepancy score (a path) and lower levels of T1 depressive symptoms (c path).
- (5) Controlling for covariates (BMI, sex, age, ethnicity, school, peer social support), and partialling out the effects of T1 MVPA, the T1 discrepancy score is positively associated with T1 depressive symptoms (b path).

The second aim of the study was to test a longitudinal moderated-mediation model. The model posited (a) that PA decreases depressive symptoms by increasing PASE and (b) that PAV moderates the relation between and depressive symptoms such that individuals with higher PAV demonstrate a stronger PASE-depressive symptoms relation. Stability analyses were conducted first in order to understand the pattern of change in MVPA, PASE, PAV, and depressive symptoms from T1 to T2. Additionally, a discrepancy score analysis (discrepancy score=T2 PASE Z-score - T2 PAV Z-score) served as an additional test of the moderated-mediation model (Harter, 1990). In consideration of the secondary aim, it was hypothesized that:

- (1) MVPA, PASE, PAV, and depressive symptoms demonstrate stability (are highly correlated) from T1 to T2.

- (2) Controlling for T1 depressive symptoms, T1 PASE, and other covariates (BMI, sex, age, ethnicity, school, peer social support), T1 MVPA is positively associated with T2 PASE (a path) and lower levels of T2 depressive symptoms (c path).
- (3) Controlling for T1 depressive symptoms, T1 PASE, and other covariates (BMI, sex, age, ethnicity, school, peer social support), and partialling out the effects of T1 MVPA, T2 PASE is negatively associated with T2 depressive symptoms (b path).
- (4) Controlling for T1 depressive symptoms, T1 PASE, and other covariates (BMI, sex, age, ethnicity, school, peer social support), and partialling out the effects of T1 MVPA, T2 PASE is negatively associated with T2 depressive symptoms (b path), and the negative relation between T2 PASE and T2 depressive symptoms (b path) is stronger for individuals with higher PAV at T2.
- (5) Controlling for T1 depressive symptoms, the T1 discrepancy score, and other covariates (BMI, sex, age, ethnicity, school, peer social support), T1 MVPA is positively associated with T2 discrepancy score (a path) and lower levels of T2 depressive symptoms (c path).
- (6) Controlling for T1 depressive symptoms, T1 discrepancy score, and other covariates (BMI, sex, age, ethnicity, school, peer social support), and partialling out the effects of T1 MVPA, the T2 discrepancy score is positively associated with T2 depressive symptoms (b path).

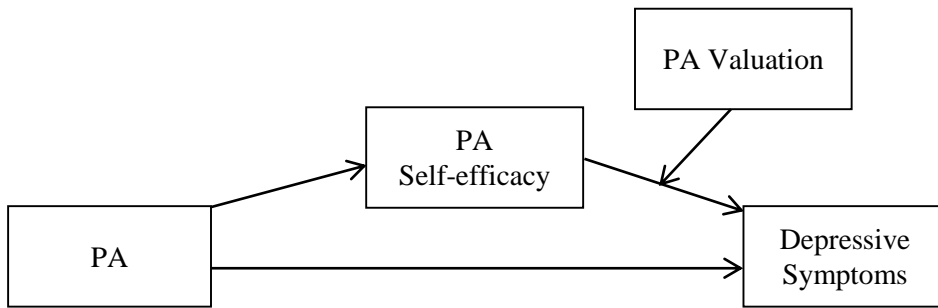


Figure 1.1. Proposed Theoretical Model

CHAPTER 2

METHOD

Study Design

This study included data from 11 of a total of 24 middle schools that were recruited to participate in the larger ACT trial. The depressive symptoms measure was collected as part of a supplemental study that collected data in the first two years of the four-year trial. Five of the six schools in the first cohort and all six schools in the second cohort agreed to participate in the additional data collection. Measures were collected at baseline before randomization (T1) and two weeks after the 17-week intervention (T2). At both time points, a trained and certified measurement team collected measures during the school day over a two-week period. Accelerometers were administered by the measurement team on the first day of data collection and collected on the last day of data collection. The depressive symptoms data were collected from participants who were present at school the day the survey was administered. This study was a cross-sectional examination of the MVPA, PASE, PAV, and depressive symptoms data collected at T1 and a longitudinal examination of the same variables collected at both T1 and T2.

Participants

Students in the 6th grade were recruited to participate. Exclusion criteria were (1) having a medical condition that interfered with PA, (2) having developmental delays such that the intervention materials were not cognitively appropriate, and (3) being in treatment for a psychiatric disorder. Students volunteered to participate in the afterschool

program at each school. The mean age was 11.4 (SD=0.65), most were African American (72%), and about half (55%) were females.

A subset of 410 (60% of 688) participants provided depressive symptoms data at T1, T2, or both. Of those data, 3.1% were missing at T1, and 38.6% were missing at T2. When comparing the groups with no depression data, T1 depression data only, T2 depression data only, and both T1 and T2 depression data, there were no significant differences in age, sex, ethnicity, intervention status, or baseline depressive symptoms scores ($ps > .05$). One subject was dropped from the analyses due to an incorrectly recorded height measurement (which caused an outlier BMI value), leaving the final sample size for this study at $N=409$. The descriptive statistics for the final sample are summarized in Table 2.1.

Measures

All measures were collected by trained staff who were blind to randomization. Staff were trained and certified on all measurement protocols and used standardized scripts when administering questionnaires and collecting anthropometric data.

Covariates. Age, sex, and ethnicity were collected via questionnaires during baseline measures. Body Mass Index (BMI) was calculated from objectively measured height and weight. Height was measured twice to the nearest 0.1 centimeter with a portable stadiometer (Shorr Productions, Olney, MD), and the average of these two measures was calculated. Weight was measured twice to the nearest 0.1 kilogram using an electronic scale (SECA, Model 880, Hamburg, Germany), and the average of the two measures was calculated. Peer social support was included as a covariate given theory suggesting social mechanisms linking PA and depressive symptoms (MacMahon, 1990;

Phillips et al., 2001). Peer social support was assessed using a well-established self-report measure, the Peer Social Support and Exercise Survey, which has previously demonstrated adequate reliability, as well as concurrent and discriminative validity (Sallis, Grossman, Pinski, Patterson, & Nader, 1987).

Moderate to vigorous physical activity (MVPA). Objective physical activity data were collected using Actical accelerometers (Mini-Mitter, Bend, OR). Acticals are omni-directional accelerometers used to estimate PA duration, intensity, and frequency. Accelerometers can provide estimates of time spent in various PA intensities when validated and calibrated in the population of interest (Puyau, Adolph, Vohra, Zakeri, & Butte, 2004; Welk, Schaben, & Morrow, 2004). The Actical device has been increasingly favored because of its improved reliability over other devices (Esliger & Tremblay, 2006). The accelerometers were initialized with a 60-second epoch length. Participants wore the accelerometers on their right hips (above the iliac crest), secured with elastic belts (Welk et al., 2004) over seven full, consecutive days. Accelerometer data were recorded as counts per minute, and these raw data were reduced to minutes of MVPA per day using cut points defined for children, MPA = 1,500 to <6,500 and VPA = \geq 6,500 (Puyau et al., 2004).

PA self-efficacy (PASE). PA self-efficacy was measured with a modified version of a scale by Saunders and colleagues (Motl et al., 2000; Saunders et al., 1997). The scale included 8 items, such as “I have the skills I need to be active in my free time on most days” and “I can be active in my free time on most days when I am busy.” Participants responded on a 3-point Likert-type scale with response options “Not at all like me”, “A little like me”, “A lot like me.” The scale demonstrated adequate reliability and construct

validity in a confirmatory factor analysis and cross-validation study with two cohorts of adolescent girls (mean age=13); the single factor model demonstrated acceptable fit ($\chi^2=39.93$; $df=20$; $RMSEA=0.031$, $RNI=.98$; $NNFI=.98$) and the factor structure was invariant across time and cohorts (Motl et al., 2000). In this sample, the internal consistency was adequate with $\alpha =0.69$ at T1 and $\alpha =0.81$ at T2.

PA valuation (PAV). PA valuation was measured using a modified version of a scale developed by Wilson and colleagues, which was originally designed to measure PA self-concept (Wilson et al., 2005; Wilson et al., 2002). This is a 10-item scale that includes items such as, “Being active is an important part of my life” and “I care a lot about being active on most days.” Participants responded on a 3-point scale with response options “Not at all like me”, “A little like me”, and “A lot like me.” In previous studies the scale demonstrated adequate reliability ($\alpha=0.71$) and construct validity, correlating ($r=0.41$, $p<.05$) with a well-validated scale of exercise habits (Wilson et al., 2005; Wilson et al., 2002). In the present study one negatively worded item (“Being active on most days is not something I care about”) was dropped, and the internal consistency was strong ($\alpha=0.85$ & 0.88 at T1 and T2, respectively).

Depressive symptoms. The Children’s Depression Inventory (Kovacs, 1985, 2003) is a 27-item scale designed to assess depressive symptoms in children and adolescents aged 7 to 17 years. Participants respond on a 3-point scale (0-2) on the severity of symptoms over the two past weeks. The CDI is a well-established, widely used measure and has demonstrated strong internal consistency and construct validity in both in-patient and community samples; alpha coefficients ranged from 0.80 to 0.94, and the measure was significantly negatively correlated with a measure of self-esteem, for

example (Saylor, Finch, Spirito, & Bennett, 1984). Confirmatory factor analyses with community samples of white and African American youth indicated that the overall structure and individual factor loadings were invariant across ethnicity (Steele et al., 2006). In this sample, internal consistency was good with $\alpha=0.87$ & 0.93 at T1 and T2, respectively.

Missing Data

Previous analyses with this dataset used multiple imputation to deal with missing MVPA, PA self-efficacy, and PA valuation data (Wilson, Van Horn, et al., 2011). Multiple imputation methods provide unbiased parameter estimates and standard errors (Dong & Peng, 2013; Schafer & Olsen, 1988). The PAN package (Schafer, 1997) implemented in R statistical software was used to generate 40 imputations with minutes of MVPA for each period log-transformed to correct for the skewness that is typical for count data. Missing data were estimated at the student level, and the imputation model included baseline variables for each student. For MVPA, imputations were conducted at the interval level, rather than the assessment period level, which took advantage of the information from consecutive intervals during that time period, thus improving imputed values.

Missing depressive symptoms data were imputed in a second multiple imputation phase using the MICE package (van Buuren & Groothuis-Oudshoorn, 2011). The MICE package uses predictive mean matching to predict missing items from other scale items. Therefore, it was only for individuals who provided at least partial depressive symptoms data at T1 or T2 that missing values could be imputed. In other words, we were not able to impute missing values for participants who were completely missing depressive

symptoms data at T1 and T2 because of the large percentage of missing data at the scale level and the reduced accuracy of prediction in such scenarios (Graham, 2012). In MICE, related, “auxiliary” scales are included to further increase the accuracy of the predicted value. Auxiliary variables for this imputation model were MVPA, PA self-efficacy, PA valuation, instrumental support for PA, motivation for PA, regulatory motivation for PA, social provisions for PA, social support from family, PA enjoyment, and PA relapse prevention self-efficacy from the phase one imputed dataset, as well as anxious arousal symptoms, reassurance seeking, emotional and behavioral problems, and prosocial behavior, which were collected in the same battery as the depressive symptoms scale. Covariates, including sex, ethnicity, BMI, school, and peer social support, were also included in the imputation model. The mediation models were conducted with a dataset obtained by merging the multiply-imputed MVPA, PA self-efficacy, and PA valuation data with the multiply-imputed depressive symptoms data from the subset of cases (n=410) for which depressive symptoms data and related variables were collected.

There were some limitations to this approach. First, the effects of MVPA and PA self-efficacy on depressive symptoms likely were underestimated because the imputed values for MVPA and PA self-efficacy were not calculated with depressive symptoms in the imputation model (depressive symptoms data were not included in the first phase of imputation because they were not available at the time). Second, listwise deletion may have resulted in reduced power due to the exclusion of meaningful data for some cases, and it may have resulted in potentially biased parameter estimates since the subset is not necessarily representative of the whole sample or the population (Graham, 2012).

However, a priori power analyses suggested that the sample size was adequate for the desired 0.80 power (see below).

Statistical Analyses

Statistical analyses were conducted in the statistical package R Version 2.13.1. An analysis of covariance (ANCOVA) mediation model was used to estimate the hypothesized direct and mediated effects. Prior to analyses, the depressive symptoms, PA self-efficacy, and PA valuation scales were centered and the depressive symptoms variables were square-root transformed to correct for the positive skew of these data. For both aims, the product of coefficients approach was used to test the significance of the mediated paths. The product of coefficients approach provides the most accurate Type I error rates and adequate power of the available methods for testing the mediated effect (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002), and enables testing of specific indirect effects not afforded by the difference of coefficients approach. Asymmetric confidence limits were obtained for the specific indirect effects from the distribution of the product (PRODCLIN) program (MacKinnon, Fritz, Williams, & Lockwood, 2007; Tofighi & MacKinnon, 2011). This program uses the empirically-derived distribution for the product of coefficients to estimate the standard error and confidence limits for the mediated effect; this provides accurate (asymmetric) confidence limits, which increases power for statistical tests of the mediated effect (Mackinnon, Lockwood, & Williams, 2004).

The first aim was tested through three sets of multiple linear regression equations for estimating the direct and mediated effects among measured variables. First, the simple mediation model (Figure 2.1) was tested using the following equations that regressed the

T1 PASE mediator on relevant covariates and T1 MVPA and that regressed T1 depressive symptoms on relevant covariates, T1 MVPA, and T1 PASE:

$$T1PASE = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \varepsilon$$

$$T1Depr = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \beta_3T1PASE + \varepsilon$$

In the first equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support), β_2 represents the effects of T1 MVPA on T1 PASE (a path), and ε represents variability in T1 PASE not accounted for by β_1 and β_2 . In the second equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support), β_2 represents the effects of T1 MVPA on depressive symptoms controlling for the indirect effects (c' path), β_3 represents the effects of T1 PASE on depressive symptoms controlling for T1 MVPA, and ε represents variability in T1 depressive symptoms not accounted for by $\beta_1 - \beta_3$.

Second, the moderated-mediation model (Figure 2.3) was tested using the following equations that regressed the T1 PASE mediator on relevant covariates and T1 MVPA and that regressed T1 depressive symptoms on relevant covariates, T1 MVPA, T1 PASE, T1 PAV, and the T1 PASE X T1 PAV interaction:

$$T1PASE = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \varepsilon$$

$$T1Depr = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \beta_3T1PASE + \beta_4T1PAV + \beta_5T1PASE * T1PAV + \varepsilon$$

In the first equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support), β_2 represents the effects of T1 MVPA on T1 PASE (a path), and ε represents variability in T1 PASE not accounted for by β_1 and β_2 . In the second

equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support), β_2 represents the effects of T1 MVPA on depressive symptoms controlling for the indirect effects (c' path), β_3 represents the effects of T1 PASE on depressive symptoms controlling for T1 MVPA, T1 PAV, and the interaction effects (b path), β_4 represents the effects of T1 PAV on T1 depressive symptoms controlling for the other effects in the model, β_5 represents the effects of T1 PASE at varying levels of T1 PAV, and ε represents variability in T1 depressive symptoms not accounted for by $\beta_1 - \beta_5$. Because the interaction term was not found to significantly predict T1 depressive symptoms, simple slopes for the effects of T1 PASE at varying levels of T1 PAV were not calculated.

Third, a discrepancy score model (Harter, 1990; see Figure 2.3) was tested using the following equations that regressed the T1 discrepancy score ($\text{Disc} = \text{T1 PASE} - \text{T1 PAV}$) on relevant covariates (not shown, see hypotheses) and T1 MVPA and that regressed T1 depressive symptoms on relevant covariates, T1 MVPA, and T1 PASE:

$$\text{T1Disc} = \beta_0 + \beta_1 \text{Cov} + \beta_2 \text{T1MVPA} + \varepsilon$$

$$\text{T1Depr} = \beta_0 + \beta_1 \text{Cov} + \beta_2 \text{T1MVPA} + \beta_3 \text{T1Disc} + \varepsilon$$

In the first equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support), β_2 represents the effects of T1 MVPA on the T1 discrepancy score (a path), and ε represents variability in the T1 discrepancy score not accounted for by β_1 and β_2 . In the second equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support), β_2 represents the effects of T1 MVPA on T1 depressive symptoms controlling for the indirect effect (c' path), β_3 represents the effects

of the T1 discrepancy on T1 depressive symptoms controlling for T1 MVPA (b path), and ε represents variability in T1 depressive symptoms not accounted for by $\beta_1 - \beta_3$.

The second aim was tested first through a set of multiple linear regression equations consistent with a demi-longitudinal simple mediation model (see Figure 2.4), a set of multiple linear regression equations consistent with a demi-longitudinal moderated mediation model (see Figure 2.5), and a second set of multiple linear regression equations using a discrepancy score to estimate the moderated-mediation effect in the longitudinal relations (Figure 2.6). First, the simple mediation model was tested using the following equations that regressed the T2 PASE mediator on relevant covariates and T1 MVPA and that regressed T2 depressive symptoms on relevant covariates, T1 MVPA, T2 PASE:

$$T2PASE = \beta_0 + \beta_1 Cov + \beta_2 T1MVPA + \varepsilon$$

$$T2Depr = \beta_0 + \beta_1 Cov + \beta_2 T1MVPA + \beta_3 T2PASE + \varepsilon$$

In the first equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support, T1 depressive symptoms, T1 PASE, and T1 PAV), β_2 represents the effects of T1 MVPA on T2 PASE (the a path), and ε represents variability in T2 PASE not accounted for by β_1 and β_2 . In the second equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support, T1 depressive symptoms, T1 PASE, and T1 PAV), β_2 represents the effects of T1 MVPA on T2 depressive symptoms controlling for the indirect effects (c' path), β_3 represents the effects of T2 PASE on T2 depressive symptoms controlling for T1 MVPA, and ε represents variability in T2 depressive symptoms not accounted for by $\beta_1 - \beta_3$.

Second, the moderated mediation model was tested using the following equations that regressed the T2 PASE mediator on relevant covariates and T1 MVPA and that

regressed T2 depressive symptoms on relevant covariates, T1 MVPA, T2 PASE, T2 PAV, and the interaction of T2 PASE*T2 PAV:

$$T2PASE = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \varepsilon$$

$$T2Depr = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \beta_3T2PASE + \beta_4T2PAV + \beta_5T2PASE*T2PAV + \varepsilon$$

In the first equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support, T1 depressive symptoms, T1 PASE, and T1 PAV), β_2 represents the effects of T1 MVPA on T2 PASE (the a path), and ε represents variability in T2 PASE not accounted for by β_1 and β_2 . In the second equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support, T1 depressive symptoms, T1 PASE, and T1 PAV), β_2 represents the effects of T1 MVPA on T2 depressive symptoms controlling for the indirect effects (c' path), β_3 represents the effects of T2 PASE on T2 depressive symptoms controlling for T1 MVPA, T2 PAV, and the interaction effects (b path), β_4 represents the effects of T2 PAV on T2 depressive symptoms controlling for the other effects in the model, β_5 represents the effects of T2 PASE at varying levels of T2 PAV, and ε represents variability in T2 depressive symptoms not accounted for by $\beta_1 - \beta_5$. Because the interaction term was not found to significantly predict T2 depressive symptoms, simple slopes for the effects of T2 PASE at varying levels of T2 PAV were not calculated.

Third, a discrepancy score model (Harter, 1990) was tested using the following equations that regress the T2 discrepancy score ($Disc = T2 PASE - T2 PAV$) on relevant covariates and T1 MVPA and that regress T2 depressive symptoms on relevant covariates, T1 MVPA and T2 PASE:

$$T2Disc = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \varepsilon$$

$$T2Depr = \beta_0 + \beta_1Cov + \beta_2T1MVPA + \beta_3T2Disc + \varepsilon$$

In the first equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support, T1 discrepancy score), β_2 represents the effects of T1 MVPA on T2 Disc (a path), and ε represents variability in the T2 discrepancy score not accounted for by β_1 and β_2 . In the second equation, β_1 represents the effects of covariates (BMI, sex, age, ethnicity, school, peer social support, T1 depressive symptoms, and T1 discrepancy score), β_2 represents the effects of T1 MVPA on T2 depressive symptoms controlling for the indirect effect (c' path), β_3 represents the effects of the T2 discrepancy score on T2 depressive symptoms controlling for T1 MVPA (b path), and ε represents variability in T2 depressive symptoms not accounted for by $\beta_1 - \beta_3$.

Power

A priori, effect size estimates for the proposed mediation model were drawn from previous studies (McPhie & Rawana, 2012; Ryan, 2008). Based on small-medium ($\beta=.26$) estimated effect sizes, it was estimated that 161 subjects were needed for .80 power ($\alpha =.05$) for testing whether self-efficacy mediates the effect of MVPA on depressive symptoms for the product of coefficients method with the PRODCLIN test of significance (Fritz & Mackinnon, 2007). With regards to the moderated-mediation analyses, simulation studies indicated that adequate power to detect small effect sizes were achieved with a sample size between 200-500 (Preacher, Rucker, & Hayes, 2007). Thus, it was estimated a priori that the sample size of 409 was sufficient to power the cross-sectional and longitudinal moderated mediation analyses.

Table 2.1. Participant demographics

	Full ACT sample (C1-C4)	C1 and C2 sample	Sample with CDI data	Final sample
N	1422	688	410	409
Age, M (SD)	11.34 (0.58)	11.39 (0.65)	11.4 (0.65)	11.4 (0.65)
Sex				
Girls, N (%)	770 (54%)	376 (56%)	230 (56%)	230 (56%)
Boys, N (%)	652 (46%)	304 (44%)	180 (44%)	179 (44%)
Ethnicity				
African American, N (%)	1038 (73%)	504 (73%)	293 (71%)	293 (72%)
Other, N (%)	384 (27%)	177 (26%)	117 (29%)	116 (28%)
Intervention				
PA, N (%)	729 (51%)	366 (53%)	190 (46%)	190 (46%)
GH, N (%)	693 (49%)	322 (47%)	220 (54%)	219 (54%)

Notes. C=Cohort, CDI=Children's Depression Inventory, GH=General Health, M=Mean, PA=Physical Activity, SD=Standard Deviation

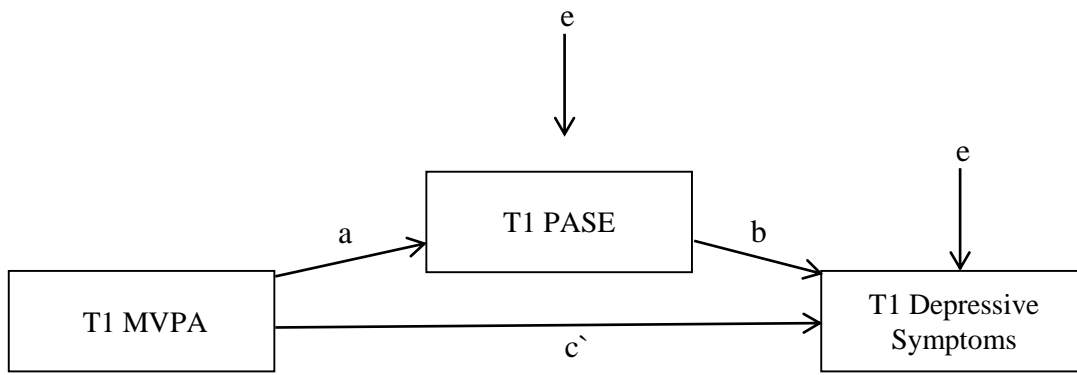


Figure 2.1. Cross-Sectional Simple Mediation Model for Aim 1

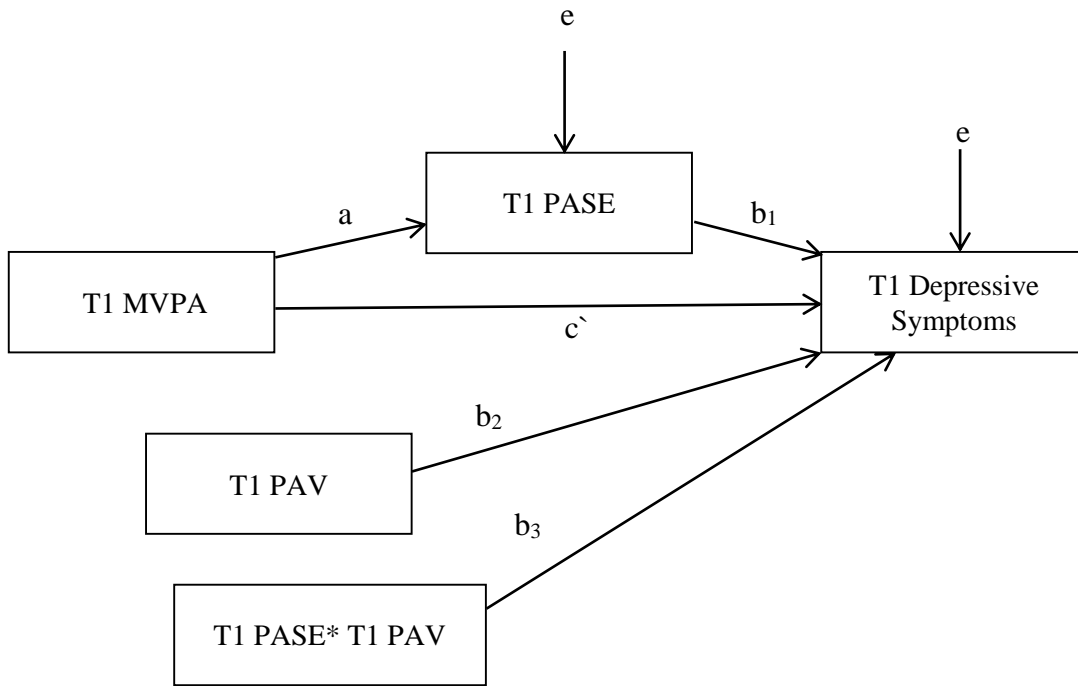


Figure 2.2. Cross-Sectional Moderated-Mediation Model for Aim 1

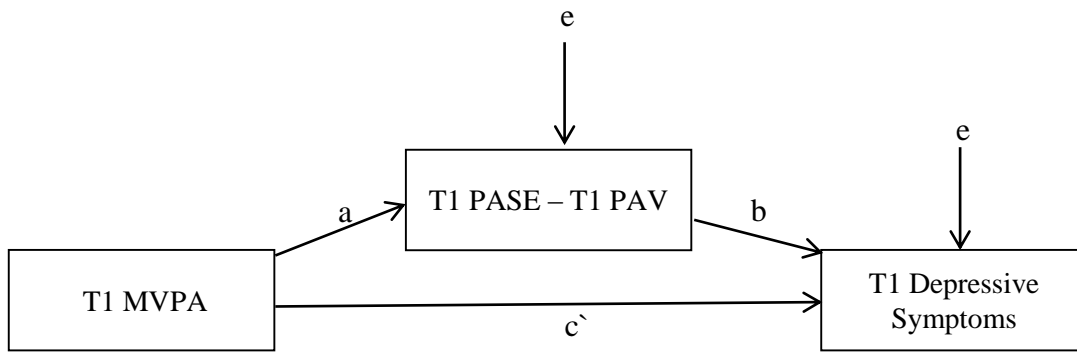


Figure 2.3. Cross-Sectional Discrepancy Mediation Model for Aim 1

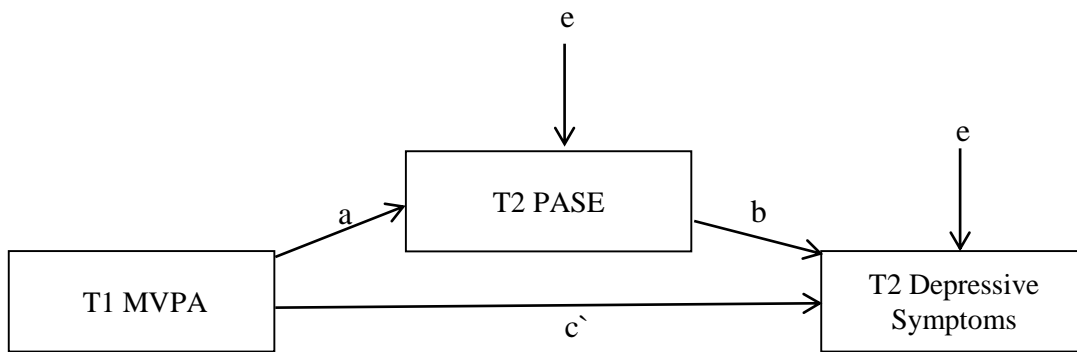


Figure 2.4. Demi-Longitudinal Simple Mediation Model for Aim 2

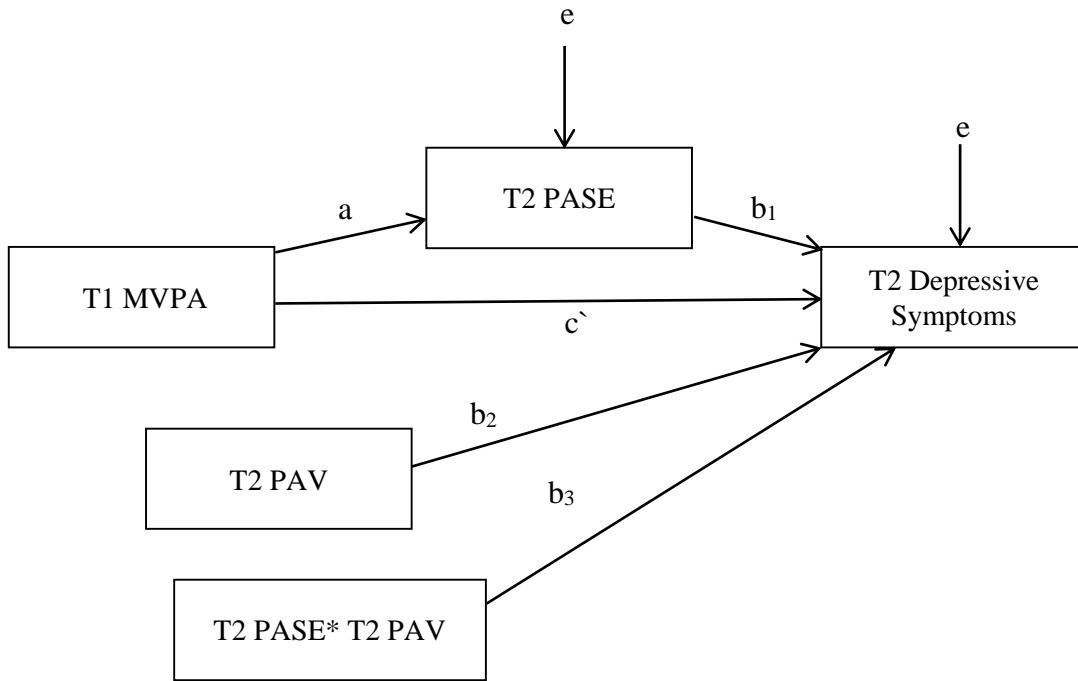


Figure 2.5. Demi-Longitudinal Moderated Mediation Statistical Model for Aim 2

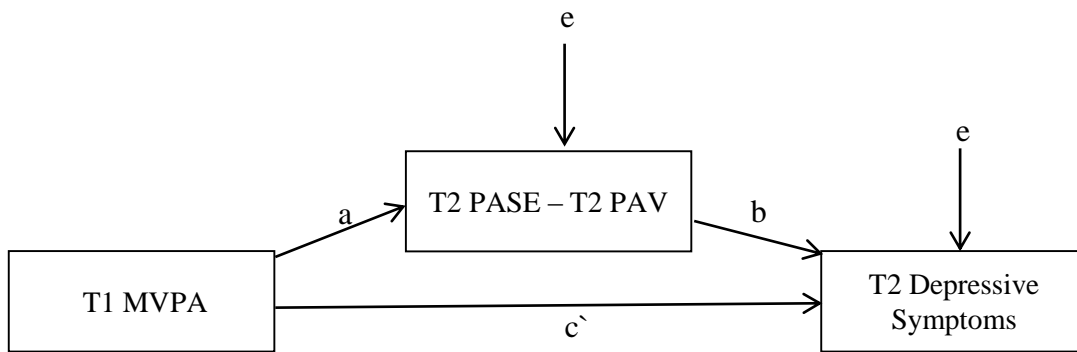


Figure 2.6. Demi-Longitudinal Discrepancy Mediation Model for Aim 2

CHAPTER 3

RESULTS

Statistical Assumptions

A series of multiple regression models was used to test mediation, and statistical assumptions of Ordinary Least Squared regression and mediation models were tested. Residual scores were normally distributed, except in the models where T1 depressive symptoms or T2 depressive symptoms was the dependent variable. Both of these variables were square root transformed to correct for the positive skew (Cohen, Cohen, West, & Aiken, 2003). Homoscedasticity and independence of the residuals were examined with residual versus fitted scatterplots and autocorrelation function plots, respectively, and the results suggested no violation of these assumptions. Multiple regressions testing for predictor-mediator interactions produced no significant results. Outliers were identified with diagnostic plots and Bonferroni-corrected outlier tests of the Studentized residuals for each model (Fox & Weisberg, 2011). Twenty-nine individuals were flagged as potential outliers. Therefore, sensitivity analyses with these participants removed from the dataset were conducted (see below). Finally, it is noted that, because there were not three time points of data, the assumption of temporal precedence (the predictor preceded the mediators which preceded the outcome) was violated in the cross-sectional mediation model and partially violated in the demi-longitudinal model. Therefore, results were interpreted as relational rather than causal when appropriate.

Descriptive Statistics

Table 3.1 summarizes the distributions of the primary variables of interest. The participants engaged in an average of 41.79 (SD=23.34) minutes of daily MVPA at T1 and 37.12 (SD=26.79) minutes at time T2. The average CDI score was 8.93 (SD=7.30) at T1 and 11.95 (SD=10.78) at T2. The depressive symptoms variables at T1 and T2 were not normally distributed, and a square root transformation was done to correct for the observed skewness for the analyses. On average across time points, PA self-efficacy ranged from 2.24-2.27 (SD=0.36-0.42), and PA valuation ranged between 2.16-2.28 (SD=0.48-0.52), each with a maximum score of 3.00.

Correlations

Zero-order correlations between all covariates and variables of interest are presented in Table 3.2. Alpha was set to 0.05 for tests of significance. T1 MVPA was significantly positively correlated with T1 PA self-efficacy ($r=0.15$), T2 PA self-efficacy ($r=0.15$), and T2 PA valuation ($r=0.12$). T1 MVPA was significantly negatively correlated with BMI ($r=-0.24$) and sex ($r=-0.26$), indicating that boys were engaging in more MVPA than girls. T1 depressive symptoms was significantly negatively correlated with T1 PA self-efficacy ($r=-0.16$) and significantly positively correlated with age ($r=0.11$) and T2 depressive symptoms ($r=0.59$). A significant negative correlation between T1 depressive symptoms and ethnicity ($r=-0.11$) indicated that African American participants reported fewer depressive symptoms than their peers at Time 1. T1 PA self-efficacy was positively correlated with T1 PA valuation ($r=0.61$), T1 peer social support ($r=0.32$), T2 PA self-efficacy ($r=0.49$), T2 PA valuation ($r=0.38$), and age

($r=0.13$). There was a significant negative correlation between T1 PA self-efficacy and sex, indicating that girls were reporting lower PA self-efficacy at time 1 than boys.

In addition to the positive correlation with T1 depressive symptoms, T2 depressive symptoms was significantly negatively correlated with T2 PA self-efficacy ($r=-0.12$), T2 peer social support ($r=-0.14$), and ethnicity ($r=-0.10$), indicating that African American participants were reporting fewer depressive symptoms than their peers at time 2. In addition to the significant correlations with T1 PA self-efficacy, T2 PA self-efficacy was significantly positively correlated with T1 PA valuation ($r=0.43$), T1 peer social support ($r=0.21$), T2 PA valuation ($r=0.69$), T2 peer social support ($r=0.42$), and significantly negatively correlated with age ($r=-0.12$).

Cross-Sectional Simple Mediation

The first part of the first aim was to test whether T1 MVPA was related to T1 depressive symptoms and whether T1 PA self-efficacy mediated this relation. Results from the multiple regression models are summarized in Tables 3.3-3.6, with Table 3.3 summarizing the direct, mediated, and specific effects, Tables 3.4-3.6 showing the details of the three regression models. The results indicated no significant relation between T1 MVPA and T1 depressive symptoms (c path; $B=0.000$, $SE=0.003$, $p=0.966$). There was a significant relation between T1 MVPA and T1 PA self-efficacy (a path; $B=0.006$, $SE=0.002$, $p=0.009$), with MVPA accounting for 6% of the variance in PA self-efficacy. Age and peer social support were also significantly and positively related to T1 PA self-efficacy ($ps<0.05$). Finally, there was a significant negative relation between T1 PA self-efficacy and T1 depressive symptoms (b path; $B=-0.262$, $SE=0.069$, $p<0.001$), with PA self-efficacy accounting for 0.07% of the variance in depressive symptoms. Furthermore,

age ($B=0.240$, $SE=0.101$, $p=0.017$) and ethnicity ($B=-0.319$, $SE=0.143$, $p=0.026$) were significantly related to T1 depressive symptoms over and above the other covariates, T1 PA self-efficacy, and T1 MVPA. The relation between T1 MVPA and T1 depressive symptoms after controlling for T1 PA self-efficacy (c' path) was not significant. Finally, the mediated effect of T1 MVPA on T1 depressive symptoms through T1 PA self-efficacy was not significant (ab path; $B=0.006$, $SE=0.002$, 95% CI: -0.003 to 0.000).

Cross-sectional moderated mediation

The second part of the first aim was to test whether T1 PA valuation moderated the effect of T1 PA self-efficacy on T1 depressive symptoms. The results of the moderation model are summarized in Table 3.7, and they indicated that the T1 PA Self-Efficacy X T1 PA Valuation interaction was not significantly related to T1 depressive symptoms ($B=0.089$, $SE=0.057$, $p=0.117$). Furthermore, T1 PA valuation ($B=0.015$, $SE=0.080$, $p=0.848$) was not significantly related to T1 depressive symptoms over and above the covariates, T1 PA self-efficacy, the T1 PA Self-Efficacy X T1 PA Valuation interaction, and T1 MVPA. Age, ethnicity, and T1 PA self-efficacy remained significant predictors of T1 depressive symptoms in the full model ($ps<0.05$).

An alternative, discrepancy score model, was conducted to determine whether the T1 MVPA and T1 depressive symptoms relation was mediated by a discrepancy between T1 PA self-efficacy and T1 PA valuation (T1 PA self-efficacy – T1 PA valuation; see Tables 3.8-3.10). In this model, there was no significant relation between T1 MVPA and T1 depressive symptoms (c path; $B=0.000$, $SE=0.003$, $p=0.966$). T1 MVPA was not significantly related to the discrepancy score (a path; $B=0.001$, $SE=0.002$, $p=0.694$), and the discrepancy score was not significantly related to T1 depressive symptoms (b path; -

0.121, SE=0.072, $p=0.090$). The mediated effect was not significant (ab path; $B=0.000$, SE=0.000, 95% CI: -0.001 to 0.001). The relation between T1 MVPA and T1 depressive symptoms controlling for the discrepancy score (c' path) was not significant ($B=0.000$, SE=0.003, $p=0.940$).

Variable stability across Time 1 to Time 2

The first part of the second aim was to test the stability of the predictor, mediator, and outcome variables over time by examining zero-order correlation coefficients from Time 1 to Time 2. Significant positive correlations were observed for T1 and T2 MVPA ($r=0.52$), T1 and T2 PA self-efficacy ($r=0.49$), T1 and T2 PA valuation ($r=0.51$), and T1 and T2 depressive symptoms ($r=0.59$).

Demi-longitudinal simple mediation

The second part of the second aim was to test whether T1 MVPA was related to T2 depressive symptoms and whether T2 PA self-efficacy mediated this relation. Results from the multiple regression models are summarized in Tables 3.11-3.14, with Table 3.11 summarizing the direct, mediated, and specific effects and Tables 3.12-3.14 showing the details of the three regression models. The results indicated no significant relation between T1 MVPA and T2 depressive symptoms (c path; $B=0.003$, SE=0.003, $p=0.244$). There was no significant relation between T1 MVPA and T2 PA self-efficacy (a path; $B=0.003$, SE=0.002, $p=0.262$). The only significant predictor of T2 PA self-efficacy was T1 PA self-efficacy ($B=0.438$, SE=0.051, $p<0.001$). The results showed no significant relation between T2 PA self-efficacy and T2 depressive symptoms (b path; $B=-0.006$, SE=0.078, $p=0.941$) or T1 MVPA and T2 depressive symptoms after controlling for T2 PA self-efficacy (c' path; $B=0.004$, SE=0.003, $p=0.240$). The only significant predictor of

T2 depressive symptoms was T1 depressive symptoms ($B=0.115$, $SE=0.010$, $p<.001$) accounting for 20% of the variance. Finally, the mediated effect of T1 MVPA on T2 depressive symptoms through T2 PA self-efficacy was not significant (ab path; $B=0.000$, $SE=0.000$, 95% CI: -0.001 to 0.001).

Demi-longitudinal moderated mediation

The third part of the second aim was to test whether T2 PA valuation moderated the aforementioned mediated effect. Table 3.15 summarizes the results of the moderation model, which indicated that the T2 PA Self-Efficacy X T2 PA Valuation interaction was not significantly related to T2 depressive symptoms ($B=0.021$, $SE=0.060$, $p=0.732$). Furthermore, T2 PA valuation ($B=0.021$, $SE=0.095$, $p=0.829$) was not significantly related to T1 depressive symptoms over and above the covariates, T2 PA self-efficacy, the T2 PA Self-Efficacy X T2 PA Valuation interaction, and T1 MVPA. T1 depressive symptoms remained the only significant predictor of T2 depressive symptoms in the full model ($B=0.114$, $SE=0.010$, $p<0.001$) accounting for 20% of the variance.

An alternative, discrepancy score model, was conducted to determine whether the T1 MVPA and T2 depressive symptoms relation was mediated by a discrepancy between T2 PA self-efficacy and T2 PA valuation (T2 PA self-efficacy – T2 PA valuation). In this model (see Tables 3.16-3.18), there was no significant relation between T1 MVPA and T2 depressive symptoms (c path; $B=0.003$, $SE=0.003$, $p=0.244$). T1 MVPA was not significantly related to the discrepancy score (a path; $B=-0.001$, $SE=0.002$, $p=0.706$), and the discrepancy score was not significantly related to T2 depressive symptoms (b path; $B=-0.023$, $SE=0.084$, $p=0.781$). The mediated effect was not significant (ab path; $B=0.000$, $SE=0.000$, 95% CI: 0.000 to 0.000). The relation between T1 MVPA and T2 depressive

symptoms controlling for the discrepancy score (c' path) was not significant ($B=0.003$, $SE=0.003$, $p=0.258$). Again, the only predictor of T2 depressive symptoms was T1 depressive symptoms ($B=0.115$, $SE=0.010$, $p<0.001$) accounting for 21% of the variance.

Sensitivity analyses

Upon reviewing the distribution for T1 MVPA, it was noted that the data were positively skewed as is common for count data. Although there are no assumptions about the distribution of predictor variables in Ordinary Least Squares regression, a sensitivity analysis was conducted wherein T1 MVPA was square root transformed. The results did not change, and this indicates minimal to no effect of the skewed data on model results.

Diagnostic analyses of the regression models suggested outliers were present in the dataset. The statistical models were conducted with these 29 participants removed, and the mediation results did not change. However, the negative relation between ethnicity and T1 depressive symptoms was not significant with these individuals removed.

Additional sensitivity analyses tested the models with 1) all the covariates removed and 2) with an alternative measure of self-efficacy (relapse prevention self-efficacy; Sallis, Pinski, Grossman, Patterson, & Nader, 1988; Wilson et al., 2005), and these showed no differences in the overall pattern of results. Finally, a change-model (MacKinnon, 2008), wherein change in MVPA predicted change in depressive symptoms as mediated by change in PA self-efficacy was conducted, and there were no significant effects.

Racial Differences in Depressive Symptoms

As mentioned above, there was a significant, negative zero-order correlation between ethnicity and T1 depressive symptoms ($r=-0.11$), and in the cross-sectional regression models, ethnicity was consistently related to T1 depressive symptoms, over and above the other covariates and variables of interest ($Bs = -0.29$ to -0.32 , $SEs = 0.14$ to 0.15). These results indicate that the African American participants reported significantly fewer depressive symptoms than their peers. Therefore, in Table 3.19 the differences in mean depressive symptoms scores by ethnicity were examined with the full sample and after the depressive symptoms outliers were removed. There were significant differences in depressive symptoms at T1 ($t=-2.32$, $p=0.021$) with African American youth endorsing fewer depressive symptoms than their peers ($M=8.41$, $SD=6.90$ versus $M=10.21$, $SD=8.11$, respectively). The same pattern was observed at T2 ($t=-1.99$, $p=0.047$) with an average depression score of 11.28 ($SD=10.38$) for African American participants versus 13.62 ($SD=11.59$) for other racial groups.

Table 3.1. Descriptive statistics on final sample (n=409)

<i>Time 1 Variables and Covariates</i>					
	Mean (SD)	Range	Skewness	Kurtosis	Cronbach's α
MVPA	41.79 (24.34)	0-169.90	1.55	4.00	
CDI	8.93 (7.30)	0-38	1.38	2.17	0.87
PA Self-Efficacy	2.27 (0.36)	1-3	-0.12	-0.27	0.69
PA Valuation	2.28 (0.48)	1-3	-0.43	-0.51	0.85
Peer Social Support	2.31 (0.28)	1-3	0.37	-0.27	0.81
<i>Time 2 Variables and Covariates</i>					
MVPA	37.12 (26.79)	1.43-132.40	1.03	0.77	
CDI	11.95 (10.78)	0-46	1.29	0.85	0.93
PA Self-Efficacy	2.24 (0.42)	1-3	-0.21	-0.65	0.81
PA Valuation	2.16 (0.52)	1-3	-0.10	-0.93	0.88
Peer Social Support	2.34 (0.31)	1-3	-0.03	0.37	0.81

Notes. CDI=Children's Depression Inventory; MVPA=Moderate to Vigorous Physical Activity; PA=Physical Activity; SD=Standard Deviation

Table 3.2. Zero-order correlations (n=409)

	T1 Dep	T1 MVPA	T1 PASE	T1 PAV	T1 SS	T2 Dep	T2 MVPA	T2 PASE	T2 PAV	T2 SS	Age	Sex	Ethnicity	BMI
T1 Dep	-	0.04	-0.16*	-0.09	-0.07	0.59*	-0.02	-0.21*	-0.11*	-0.21*	0.11*	0.02	-0.11*	-0.01
T1 MVPA		-	0.15*	0.09	-0.01	0.07	0.52*	0.15*	0.12*	0.05	0.01	-0.26*	-0.04	-0.24*
T1 PASE			-	0.61*	0.32*	-0.09	0.13*	0.49*	0.38*	0.24*	0.13*	-0.12*	-0.03	-0.02
T1 PAV				-	0.33*	-0.06	0.12*	0.43*	0.51*	0.22*	0.08	-0.10*	0.00	-0.02
T1 SS					-	-0.12*	0.02	0.21*	0.15*	0.32*	-0.03	0.01	0.00	0.01
T2 Dep						-	-0.02	-0.12*	-0.05	-0.14*	0.06	0.05	-0.10*	-0.09
T2 MVPA							-	0.22*	0.20*	0.15*	0.06	-0.38*	-0.06	-0.26*
T2 PASE								-	0.69*	0.42*	0.02	-0.12*	-0.04	0.00
T2 PAV									-	0.39*	0.02	-0.15*	-0.02	0.04
T2 SS										-	-0.13*	-0.02	0.07	0.02
Age											-	-0.23*	-0.06	0.00
Female												-	0.09	0.10*
Ethnicity													-	0.01
BMI														-

Notes. BMI=Body Mass Index, Dep=Depressive Symptoms, MVPA=Moderate to Vigorous Physical Activity, PASE=Physical Activity Self-Efficacy, PAV=Physical Activity Valuation, SS=Peer Social Support, T=Time

Table 3.3. Estimates, standard errors, confidence intervals, and effect sizes for specific paths and mediated effects for cross-sectional simple mediation (Aim 1), controlling for covariates

	Coefficient	Std. Error	Lower 95% CL	Upper 95% CL	r^2
<i>Direct effect</i>					
T1MVPA→T1DEP (c)	0.00	0.00	-0.01	0.01	.10
<i>Mediated Path*</i>					
T1MVPA→T1PASE→ T1DEP (ab)	-0.002	0.001	-0.003	0.000	.03
<i>Specific Paths</i>					
T1MVPA→T1PASE (a)	0.01	0.002	0.002	0.01	.064
T1PASE→T1DEP (b)	-0.26	0.07	-0.40	-0.13	.001
T1MVPA→ T1DEP (c')	0.002	0.003	-0.005	0.008	.14

Notes. DEP=Depressive symptoms; MVPA=Moderate-vigorous physical activity; PASE = Self-efficacy for physical activity; Bold type indicates significant ($p<.05$)

*Asymmetric confidence intervals obtained from PRODCLIN

Table 3.4. T1 depression regressed on to T1 MVPA and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	1.01	1.22	0.407	-1.38	3.39	
BMI	0.002	0.02	0.868	-0.02	0.02	
Age	0.20	0.01	0.053	-0.02	0.39	
Sex	0.12	0.14	0.373	-0.15	0.39	
Ethnicity	-0.31	0.15	0.031*	-0.60	-0.03	.0001
School (Dummy Coded) [#]	-0.91 to 0.09	0.07 to 0.43	0.031 to 0.785			
T1 SS Friends	-0.13	0.07	0.050	-0.26	-0.00	
T1 MVPA	0.00	0.00	0.967	-0.01	0.01	

[#]Significant differences between: School 1 vs 12, School 4 vs 12; School 5 vs 12

Table 3.5. T1 PA self-efficacy regressed on to T1 MVPA and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	-2.04	0.88	0.021	-3.77	-0.30	
BMI	0.00	0.01	0.681	-0.01	0.02	
Age	0.17	0.07	0.026	0.02	0.31	.0001
Sex	-0.12	0.10	0.214	-0.32	0.01	
Ethnicity	-0.03	0.11	0.813	-0.23	0.18	
School (Dummy Coded) [#]	-0.51 to 0.34	0.21 to 0.32	0.039 to 0.843			
T1 SS Friends	0.32	0.049	<0.001	0.23	0.42	.0001
T1 MVPA	0.006	0.002	0.009	0.002	0.010	.064

[#]Significant differences between: School 8 vs 12

Table 3.6. T1 depressive symptoms regressed on to T1 MVPA, T1 PA self-efficacy, and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r ²
Intercept	0.48	1.21	0.693	-1.89	2.84	
BMI	0.003	0.01	0.804	-0.02	0.02	
Age	0.24	0.10	0.017	0.04	0.44	.0005
Sex	0.09	0.14	0.506	-0.18	0.36	
Ethnicity	-0.32	0.14	0.026	-0.60	-0.04	.00006
School (Dummy Coded) [#]	-0.92 to 0.06	0.29 to 0.42	0.030 to 0.861			
T1 SS Friends	-0.05	0.07	0.504	-0.18	0.09	
T1 MVPA	0.002	0.003	0.601	-0.005	0.008	
T1 PA Self-efficacy	-0.26	0.07	<0.001	-0.40	-0.13	.0007

[#]Significant differences between: School 1 vs 12, School 4 vs 12, School 5 vs 12

Table 3.7. T1 depressive symptoms regressed on to T1 MVPA, T1 PA self-efficacy, T1 PA valuation, T1 PA self-efficacy x T1 PA Valuation, and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	0.46	1.201	0.705	-1.91	2.82	
BMI	0.004	0.01	0.694	-0.02	0.03	
Age	0.24	0.10	0.019	0.04	0.43	.0005
Sex	0.08	0.14	0.552	-0.19	0.35	
Ethnicity	-0.29	0.14	0.041	-0.58	-0.01	.00006
School (Dummy Coded) [#]	-0.91 to 0.07	0.29 to 0.43	0.032 to 0.835			
T1 SS Friends	-0.05	0.07	0.472	-0.19	0.09	
T1 MVPA	0.002	0.003	0.580	-0.005	0.008	
T1 PA Self-efficacy	-0.26	0.08	0.002	-0.42	-0.10	.00028
T1 PA Valuation	0.02	0.08	0.848	-0.14	0.17	
T1 PA Self-efficacy X T1 PA Valuation	0.09	0.06	0.117	-0.02	0.20	

[#]Significant differences between: School 1 vs 12, School 5 vs 12

Table 3.8. Estimates, standard errors, confidence intervals, and effect sizes for specific paths and mediated effects for cross-sectional discrepancy score model (Aim 1c), controlling for covariates

	Coefficient	Std. Error	Lower 95% CL	Upper 95% CL	r^2
<i>Direct effect</i>					
T1MVPA→T1DEP (c)	0.00	0.00	-0.01	0.01	
<i>Mediated Path*</i>					
T1MVPA→T1DISC→ T1DEP (ab)	-0.0001	0.0003	-0.001	0.001	
<i>Specific Paths</i>					
T1MVPA→T1DISC (a)	0.001	0.002	-0.004	0.005	
T1DISC→T1DEP (b)	-0.12	0.07	-0.26	0.01	
T1MVPA→ T1DEP (c')	0.0002	0.003	-0.006	0.007	

Notes. DEP=Depressive symptoms; MVPA=Moderate-vigorous physical activity; PASE = Self-efficacy for physical activity; PAV = Valuation of physical activity; DISC=PASE – PAV

*Asymmetric confidence intervals obtained from PRODCLIN

Table 3.9. T1 discrepancy score (PA self-efficacy minus PA valuation) regressed on to T1 MVPA and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	-0.58	0.86	0.500	-2.26	1.10	
BMI	0.000	0.008	0.971	-0.01	0.02	
Age	0.06	0.07	0.379	-0.08	0.20	
Sex	-0.001	0.10	0.993	-0.19	0.19	
Ethnicity	-0.08	0.10	0.435	-0.28	0.12	
School (Dummy Coded)#	-0.40 to 0.14	0.21 to 0.31	0.079 to 0.993			
T1 SS Friends	-0.005	0.05	0.916	-0.10	0.09	
T1 MVPA	0.001	0.002	0.694	-0.004	0.005	

#No significant differences

Table 3.10. T1 depression regressed on to T1 MVPA, T1 discrepancy score (PA self-efficacy minus PA valuation), and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	0.94	1.22	0.440	-1.44	3.32	
BMI	0.002	0.01	0.864	-0.02	0.02	
Age	0.20	0.10	0.044	0.005	0.40	.0004
Sex	0.12	0.14	0.372	-0.15	0.39	
Ethnicity	-0.32	0.15	0.026	-0.61	-0.04	.00005
School (Dummy Coded) [#]	-0.91 to 0.05	0.29 to 0.43	0.031 to 0.886			
T1 SS Friends	-0.13	0.07	0.048	-0.26	-0.001	.0002
T1 MVPA	0.000	0.003	0.940	-0.006	0.007	
T1 Discrepancy Score	-0.12	0.07	0.090	-0.26	0.02	

[#]Significant differences between: School 4 vs 12, School 5 vs 12

Table 3.11. Estimates, standard errors, confidence intervals, and effect sizes for specific paths and mediated effects for demi-longitudinal simple mediation model (Aim 2a), controlling for covariates

	Coefficient	Std. Error	Lower 95% CL	Upper 95% CL	r^2
<i>Direct effect</i>					
T1MVPA→T2DEP (c)	0.003	0.003	-0.002	0.009	
<i>Mediated Path*</i>					
T1MVPA→T2PASE→ T2DEP (ab)	-0.000	0.000	-0.001	0.001	
<i>Specific Paths</i>					
T1MVPA→T2PASE (a)	0.003	0.002	-0.002	0.007	
T2PASE→T2DEP (b)	-0.006	0.08	-0.16	0.15	
T1MVPA→ T2DEP (c')	0.004	0.003	-0.002	0.009	

Notes. DEP=Depressive symptoms; MVPA=Moderate-vigorous physical activity; PASE = Self-efficacy for physical activity

*Asymmetric confidence intervals obtained from PRODCLIN

Table 13.2. T2 depression regressed on to T1 MVPA and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	2.02	1.25	0.105	-0.43	4.47	
BMI	-0.01	0.01	0.360	-0.03	0.01	
Age	0.01	0.10	0.901	-0.19	0.22	
Sex	0.09	0.14	0.542	-0.19	0.37	
Ethnicity	-0.20	0.15	0.19	-0.50	0.10	
School (Dummy Coded)#	-0.47 to 0.21	0.30 to 0.43	0.161 to 0.676			
T1 SS Friends	-0.07	0.07	0.341	-0.21	0.07	
T1 Depr. Symptoms	0.12	0.01	<0.001	0.10	0.13	.225
T1 MVPA	0.003	0.003	0.244	-0.002	0.009	

#No significant differences

Table 3.13. T2 PA self-efficacy regressed on to T1 MVPA and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	<i>r</i> ²
Intercept	0.91	0.89	0.306	-0.83	2.65	
BMI	0.008	0.008	0.328	-0.01	0.02	
Age	-0.08	0.07	0.262	-0.23	0.06	
Sex	-0.15	0.10	0.136	-0.34	0.05	
Ethnicity	-0.07	0.10	0.486	-0.40	0.81	
School (Dummy Coded) [#]	-0.07 to 0.19	0.21 to 0.31	0.420 to 0.878			
T1 SS Friends	0.09	0.05	0.079	-0.01	0.19	
T1 PA Self-efficacy	0.44	0.05	<0.001	0.34	0.54	.0065
T1 MVPA	0.003	0.002	0.262	-0.002	0.007	

[#]No significant differences

Table 3.14. T2 depressive symptoms regressed on to T1 MVPA, T2 PA self-efficacy, and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	2.00	1.25	0.111	-0.46	4.46	
BMI	-0.01	0.01	0.367	-0.03	0.01	
Age	0.01	0.10	0.888	-0.19	0.22	
Sex	0.08	0.14	0.549	-0.19	0.36	
Ethnicity	-0.20	0.15	0.187	-0.50	0.10	
School (Dummy Coded)#	-0.48 to - 0.10	0.30 to 0.43	0.159 to 0.745			
T1 SS Friends	0.06	0.07	0.391	-0.21	0.08	
T1 Depr. Symptoms	0.11	0.01	<0.001	0.10	0.13	.204
T1 PA Self-efficacy	-0.01	0.08	0.906	-0.16	0.14	
T1 MVPA	0.004	0.003	0.240	-0.002	0.009	
T2 PA Self-efficacy	-0.01	0.08	0.941	-0.16	0.15	

#Significant differences between: School 1 vs 12, School 4 vs 12, School 5 vs 12

Table 3.15. T2 depressive symptoms regressed on to T1 MVPA, T2 PA self-efficacy, T2 PA valuation, T2 PA self-efficacy x T2 PA Valuation, and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r^2
Intercept	2.01	1.26	0.111	-0.46	4.47	
BMI	-0.01	0.01	0.374	-0.03	0.01	
Age	0.01	0.10	0.891	-0.19	0.22	
Sex	0.09	0.14	0.545	-0.19	0.37	
Ethnicity	-0.21	0.15	0.177	-0.51	0.09	
School (Dummy Coded)#	-0.46 to 0.20	0.30 to 0.44	0.152 to 0.704			
T1 SS Friends	-0.07	0.08	0.343	-0.22	0.08	
T1 Depr. Symptoms	0.11	0.01	<0.001	0.10	0.14	.205
T1 PA Self-efficacy	-0.04	0.09	0.674	-0.21	0.13	
T1 PA Valuation	0.05	0.09	0.561	-0.12	0.22	
T1 MVPA	0.003	0.003	0.251	-0.002	0.01	
T2 PA Self-efficacy	-0.02	0.10	0.815	-0.21	0.17	
T2 PA Valuation	0.02	0.10	0.829	-0.17	0.21	
T2 PA Self-efficacy X T2 PA Valuation	0.02	0.06	0.732	-0.10	0.14	

#No significant differences

Table 3.16. Estimates, standard errors, confidence intervals, and effect sizes for specific paths and mediated effects for cross-sectional discrepancy score model (Aim 2c), controlling for covariates

	Coefficient	Std. Error	Lower 95% CL	Upper 95% CL	r^2
Direct effect					
T1MVPA→T2DEP (c)	0.003	0.003	-0.002	0.009	
Mediated Path*					
T1MVPA→T2DISC→ T2DEP (ab)	0.000	0.000	0.000	0.000	
Specific Paths					
T1MVPA→T2DISC (a)	-0.001	0.002	-0.005	0.003	
T2DISC→T2DEP (b)	-0.02	0.08	-0.19	0.14	
T1MVPA→ T2DEP (c')	0.003	0.003	-0.002	0.009	

Notes. DEP=Depressive symptoms; MVPA=Moderate-vigorous physical activity; PASE = Self-efficacy for physical activity; PAV = Valuation of physical activity; DISC=PASE – PAV

*Asymmetric confidence intervals obtained from PRODCLIN

Table 3.17. T2 discrepancy score (PA self-efficacy minus PA valuation) regressed on to T1 MVPA and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	<i>r</i> ²
Intercept	0.22	0.79	0.777	-1.33	1.78	
BMI	-0.01	0.01	0.404	-0.02	0.01	
Age	-0.02	0.07	0.719	-0.15	0.11	
Sex	0.11	0.09	0.206	-0.06	0.28	
Ethnicity	-0.12	0.09	0.187	-0.31	0.06	
School (Dummy Coded)	-0.12 to 0.52	0.18 to 0.28	0.063 to 0.862			
T1 SS Friends	0.047	0.04	0.374	-0.05	0.12	
T1 PA Self-efficacy	0.29	0.06	<0.001	0.17	0.40	.0111
T1 PA Valuation	-0.28	0.06	<0.001	-0.39	-0.17	.0124
T1 MVPA	-0.001	0.002	0.706	-0.005	0.003	

#No significant differences

Table 3.18. T2 depression regressed on to T1 MVPA, T2 discrepancy score (PA self-efficacy minus PA valuation), and covariates

	Coefficient	Std. Error	p-value	Lower 95% CL	Upper 95% CL	r ²
Intercept	2.01	1.26	0.111	-0.46	4.48	
BMI	-0.01	0.01	0.354	-0.03	0.01	
Age	0.01	0.10	0.887	-0.19	0.22	
Sex	0.09	0.14	0.526	-0.19	0.37	
Ethnicity	-0.21	0.15	0.17	-0.51	0.09	
School (Dummy Coded)#	-0.47 to 0.22	0.30 to 0.44	0.152 to 0.728			
T1 SS Friends	-0.07	0.07	0.344	-0.22	0.08	
T1 Depr. Symptoms	0.11	0.01	<0.001	0.10	0.13	.206
T1 PA Self-efficacy	-0.04	0.08	0.668	-0.20	0.13	
T1 PA Valuation	0.05	0.08	0.566	-0.12	0.21	
T1 MVPA	0.003	0.003	0.258	-0.002	0.01	
T1 Discrepancy score	-0.02	0.08	0.781	-0.19	0.14	

#No significant differences

Table 3.19. Difference in mean depressive symptoms scores by ethnicity

	African American	Other	t-value	p-value
T1 Dep. Symptoms, M (SD)	8.41 (6.90)	10.21 (8.11)	-2.32	0.021
T2 Dep. Symptoms, M (SD)	11.28 (10.38)	13.62 (11.59)	-1.99	0.047

CHAPTER 4

DISCUSSION

The purpose of the present study was to investigate the effect of accelerometry measured physical activity on depressive symptoms in underserved adolescents and examine related mechanisms of this effect. Specifically, a moderated mediation model was tested. In this model PA self-efficacy was hypothesized to mediate the effect of MVPA on depressive symptoms, and PA valuation was hypothesized to moderate the effect of PA self-efficacy on depressive symptoms. In other words, it was proposed that PA self-efficacy serves as a mechanism by which MVPA impacts depressive symptoms, and this effect would be stronger for youth who have high PA valuation. This model was tested cross-sectionally and longitudinally across two time points. In both sets of analyses, the results suggested that there was no direct effect of MVPA on depressive symptoms and no mediated effect via PA self-efficacy. However, as predicted, in the cross-sectional model MVPA was significantly and positively related to PA self-efficacy, and PA self-efficacy was significantly and negatively related to depressive symptoms. Additionally, the results indicated that PA valuation did not moderate the effect of PA self-efficacy on depressive symptoms. Finally, the present study found that, after controlling for relevant covariates and predictors, there was a significant relation between ethnicity and depressive symptoms, suggesting that African American youth endorsed significantly fewer depressive symptoms than their peers.

MVPA-Depressive Symptoms Relation

The results of the present investigation indicated no correlational or predictive relation between MVPA and depressive symptoms. It is important to note that power analyses indicated adequate power to detect the direct effect of MVPA on depressive symptoms at time one and across time points. Furthermore, a series of sensitivity analyses, in which alternative models were tested systematically, provided the same pattern of results as the primary analyses. The sensitivity analyses provided further evidence that the relation between MVPA and depressive symptoms was not significant in this population. Current literature supports the hypothesized models as it generally suggests that this relation exists both cross-sectionally and longitudinally (e.g., Fulkerson et al., 2004; Jerstad et al., 2010; Motl et al., 2004; Sallis et al., 2000). However, only three studies to date have examined this relation using objectively measured MVPA, and none of the studies with accelerometry estimates showed a relation between MVPA and depressive symptoms (Johnson et al., 2008; Toseeb et al., 2014; Wiles et al., 2012). The majority of existing studies have used self-reported PA data, which are known to be less accurate and more biased than objectively measured PA estimates (Bassett et al., 2008; Corder et al., 2009; Kohl et al., 2000). Previous research has shown that, compared to those with accelerometry estimates of PA, studies with self-report PA data tend to overestimate correlations of PA with psychosocial variables (e.g., Dishman, Darracott, & Lambert, 1992; Sallis et al., 2002). This may be due to shared method variance as well as more objective assessments being able to assess activities that are not included by participants when they self-report (Sallis et al., 2002). Considering the negative cognitive bias exhibited by depressed youth (Hankin & Abramson, 2001), it may be that the

tendency to under-report PA is even greater for depressed youth. However, no studies to date have investigated the bias in self-reported PA data for depressed adolescents.

Overall, taken with the existing literature, these present findings support the notion that, when measured with accelerometry, MVPA is perhaps not correlated with and does not predict depressive symptoms in adolescents, over and above other important predictors.

An alternative explanation for the lack of association between MVPA and depressive symptoms is that this relation may not exist in this particular population of young adolescents. In the current study the adolescents were younger than many previous studies with a mean age of 11.5. These youth were at an age before trends indicate increases in the prevalence of depressive symptoms (Hankin & Abramson, 2001; Twenge & Nolen-Hoeksema, 2002). On average, the participants endorsed depressive symptoms below the recommended diagnostic cut off for major depression (Craighead, Curry, & Ilardi, 1995; Hodges, 1990), and the distribution of depression scores observed in this study was similar to previous findings with middle school aged youth (Finch, Saylor, & Edwards, 1985; McLaughlin et al., 2007; Steele et al., 2006; Twenge & Nolen-Hoeksema, 2002). Similarly, this study did not show a sex difference in depressive symptoms in the zero-order correlations or in the multiple regression models, which is also likely due to the fact that these youth are at an age before existing literature suggests that sex differences are observed (Hankin & Abramson, 2001). It may be that the relation between PA and depressive symptoms does not emerge until later in adolescence when depression becomes more prevalent. In other words, the protective benefits of MVPA may not be apparent until depressive symptoms increase to a certain severity or prevalence level. In fact, across observational and intervention studies to date, the

majority have been conducted with older adolescents (12-18 year olds), and only a handful of studies provided specific evidence for a PA-Depression relation in young adolescents (Annesi, 2004; Neissaar & Raudsepp, 2011; Petty et al., 2009; Rothon et al., 2010; Stavrakakis et al., 2012). Moreover, in a seminal review, Sallis, Prochaska, and Taylor (2000) determined that depression was a correlate of physical activity for adolescents (ages 13-18) but not children (ages 3-12).

Second, the present sample consisted primarily (72%) of ethnic minority youth. Of the research conducted with younger adolescents, only the studies by Annesi (2004) and Petty et al. (2009) were conducted in the United States and used a majority African American sample (59% and 93%, respectively). While Annesi's results suggest his PA intervention reduced depressive symptoms, the findings are limited by the lack of a control group in his study. Petty and colleagues conducted a randomized controlled trial with 222 overweight children age 7-11 years old. Similar to the current study, the majority of their participants reported depressive symptoms in the non-clinical range. Their results showed an effect of the exercise intervention on depressive symptoms, with a high-dose (40 minutes per day of aerobic activity) exercise group endorsing significantly lower levels of depressive symptoms than a no intervention control group. These findings suggest that a structured PA program may reduce depressive symptoms for ethnically diverse youth. To date, there is limited evidence supporting the relation between PA and depressive symptoms in young adolescent populations, and there are few studies showing this association in young African American adolescents, in the absence of intervention. Therefore, it may be that an exercise intervention or PA at a certain intensity and duration is necessary to produce the reduction in depressive symptoms in

these youth. It will be important for future studies to investigate these relations more fully in this specific population, as African American youth may be at a greater risk for developing depression as they transition into middle and late adolescence (Van Voorhees et al., 2008) and intervening in early adolescence provides the opportunity to prevent depression.

PA Self-Efficacy-Depressive Symptoms Relation

Despite not finding a direct effect of MVPA on depressive symptoms, the present investigation found a statistically significant negative relation between PA self-efficacy and depressive symptoms at time 1 and, thus, suggests that PA self-efficacy is associated with depressive symptoms cross-sectionally. It should be noted that the effect size for this relation was quite small, with PA self-efficacy explaining 0.01% of the variance in depressive symptoms. Again, it may be that this relation would be stronger in an older population with a higher prevalence of clinical depression and thus more variance in reported depressive symptoms. The present findings also corroborated the literature suggesting MVPA is positively correlated with self-reported PA self-efficacy (Dishman et al., 2006; Sallis et al., 2000). Therefore, although causal inferences cannot be concluded, the correlational effects in this study overall are consistent with current literature suggesting that PA is associated with PA self-efficacy, which in turn is related to fewer depressive symptoms. The findings from the cross-sectional models provide preliminary support for existing theoretical models, such as the “mastery hypothesis” that suggests physical activity that builds self-efficacy may be associated with fewer depressive symptoms (Annesi, 2005; MacMahon, 1990; Martinsen, 2008; Rethon et al., 2010; Ryan, 2008; Sonstroem & Morgan, 1989). Previous studies (see review by

Trumpeter & Wilson, 2013; Wilson, 2015) indicate that there is a direct relation between both global and PA-specific self-esteem and depressive symptoms in adolescents and, more generally, that cognitive factors (such as PA self-efficacy, general self-worth) mediate the PA-depression relation. However, the existing evidence is quite limited, and this study adds to past literature by showing the relation between depressive symptoms and PA self-efficacy, a construct that is particularly relevant for PA interventions (Biddle & Asare, 2011).

It should be noted that neither of these effects were observed in the longitudinal model. More specifically, in the longitudinal model, T1 MVPA did not predict T2 PA self-efficacy, after controlling for T1 PA self-efficacy and the other covariates. This finding was in contrast to the existing literature which provides robust evidence suggesting self-reported PA and PA interventions are associated with increased PA self-efficacy (Annesi, 2004; Dishman et al., 2005; Goldfield et al., 2007; Haerens et al., 2008; Lubans et al., 2008; Lytle et al., 2009; Neissaar & Raudsepp, 2011; Stein et al., 2007; Taymoori & Lubans, 2008; van Stralen et al., 2011). These studies have shown the effect of PA on PA self-efficacy in the context of interventions or that integrate strong behavior change elements. The increase in PA self-efficacy, therefore, may only be due to an increase in PA or may only be observable after an intervention, rather than PA at one time point predicting a change in PA self-efficacy at a later time point. Likewise, T2 PA self-efficacy was not associated with T2 depressive symptoms over and above the effects of T1 depressive symptoms and the other covariates, which may suggest that this effect may have been too small to hold up in the longitudinal model. Another factor that may have contributed to the lack of findings in the longitudinal model is the time period

between the two waves of data. It could have been that the relatively short time period of five months did not match the timing of these effects (MacKinnon, 2008) and therefore was too short to detect the effect of MVPA on PA self-efficacy and, in turn, the effect of PA self-efficacy on depressive symptoms. The studies by Neissaar & Raudsepp (2011) as well as Stein and colleagues (2007) were conducted over two-year periods, suggesting a longer time lag between assessments is needed to detect these longitudinal associations.

Moderating Effect of PA Valuation

The present study did not support the hypothesized moderating effect of PA valuation on the PA Self-Efficacy-Depressive Symptoms relation¹. The interaction between PA valuation and PA self-efficacy was not significantly associated with depressive symptoms in the cross-sectional or longitudinal models. Although existing theory provides strong rationale for such an effect (Bandura, 1978; Harter et al., 1992), no studies to date have investigated the proposed notion that the extent to which one values PA may interact with PA self-efficacy to influence depressive symptoms in young adolescents. Marsh (1986) investigated the effect of importance ratings by domain-specific self-esteem ratings in a university sample. He found that for the physical abilities domain, the effect of importance interacted with physical self-esteem to contribute significantly to global self-esteem. In contrast, the present findings indicate that PA self-efficacy is associated with depressive symptoms regardless of the extent to which PA is

¹ Exploratory, simple mediation analyses (cross-sectional and longitudinal) were conducted in which PA valuation was included as the mediator of the relation between MVPA and depressive symptoms. The pattern of results was the same as the models with PA self-efficacy as the mediator.

valued by the individual. Developmentally, sixth grade is a major transition year (Simmons et al., 1987). During this period, capacity for self-evaluation increases, and a major developmental objective is identity formation and understanding one's values (Cole et al., 1997; Eccles et al., 1989). Major changes in social and biological factors during this transition year contribute to the formulation of values (Eccles et al., 1989; Harter et al., 1998; Rodriguez et al., 2003; Wigfield & Eccles, 2002; Wilson et al., 2002). Thus, given that values are just beginning to develop, values during this transition year may not be solidified to the point that they can be accurately reported or their influence on other processes can be detected.

African American Youth Reported Fewer Depressive Symptoms

The results of this study also suggest that African American youth exhibit fewer depressive symptoms on average than their peers. This finding was generally consistent with the existing literature, which indicates that young adolescent (middle school aged) African American adolescents may experience lower or comparable levels of depressive symptoms than their peers (Costello et al., 1996; Kennard et al., 2006; Latzman et al., 2011; McLaughlin et al., 2007; Saluja et al., 2004; Schraedley et al., 1999) and, in early adolescence, that these trends are similar across sex (McLaughlin et al., 2007; Schraedley et al., 1999). On the other hand, older African American adolescents (ages 12-18) endorse greater levels of depression and are at greater risk for a major depressive episode (Anderson & Mayes, 2010; Van Voorhees et al., 2008). Furthermore, other researchers have argued that racial differences in depression may differ as a function of social context (Anderson & Mayes, 2010). For example, previous studies have shown that African Americans residing in majority Caucasian environments display greater depressive

symptoms than those residing in predominantly African American communities (Walsemann, Bell, & Maitra, 2011; Wight, Aneshensel, Botticello, & Sepulveda, 2005). Therefore, the youth in this study may be at an age before racial disparities emerge or may be protected by social factors associated with living in primarily African American communities.

Strengths and Limitations

The results of this study should be interpreted with caution. Although the use of multiple imputation to handle missing data provided unbiased parameter estimates and standard errors (Dong & Peng, 2013; Schafer & Olsen, 1988), as noted in the methods section, the missing MVPA and depressive symptom data were imputed in separate imputation models. While MVPA data were used to predict missing depressive symptoms data, the reverse was not true. Therefore, the relations between MVPA and depressive symptoms may have been underestimates of the true relations in the population. The observed effect size of the relation between MVPA and depressive symptoms, both cross-sectionally and longitudinally, was still large enough to provide adequate power in spite of this statistical limitation. An additional weakness of this study was the limited number of and short time lag between data collection time points. Longitudinal data enable researchers to establish temporal precedence and better support causal mediation models (MacKinnon et al., 2002). However, ideally three or more time points that are strategically spaced to match the hypothesized timing of effects would be utilized (MacKinnon, 2008). It is also noted that the reliability of the PA self-efficacy measure at time 1 was adequate but somewhat low (Cronbach's $\alpha = 0.69$). Measurement error in predictor variables can lead to bias in regression coefficients and standard errors and

incorrect confidence intervals and significance tests (Cohen et al., 2003). Therefore, the observed relation between T1 PA self-efficacy and depressive symptoms could be somewhat inaccurate due to the measurement error in the PA self-efficacy measure. Additionally, the clustered nature of the data (individuals nested within school) calls for a multi-level modeling approach. However, because the data were only available for 11 schools, a multi-level model would limit power and, perhaps, mask meaningful relations, and school was included as a control variable.

It is further noted that the results of this study may have been affected by the measures used to assess depressive symptoms and PA. First, depressive symptoms were measured by a commonly used self-report inventory. Because youth may under-report depressive symptomology (Hodges, 1990), the accuracy of the estimated relations of this variable with PA may have been affected. Second, despite the existing evidence of a dose response effect of PA on depressive symptoms in adolescents, with high intensity exercise typically being associated more strongly with depression (Crews, Lochbaum, & Landers, 2004; Petty et al., 2009), the present study utilized a measure of PA that combined both moderate- and vigorous- intensity activity. If, as these previous studies suggest, it is vigorous PA that is associated with reduced depressive symptoms, the composite MVPA variable may have dampened the association between PA and depression in the present study.

Future Directions

The present study added to the existing literature by including the use of accelerometry measured PA data and the primarily ethnic minority, young adolescent sample. Accelerometry estimated PA provides a more accurate, unbiased picture of

engagement in PA and the ability to more accurately estimate associations with psychosocial constructs (Bassett et al., 2008; Dishman et al., 1992; Kohl et al., 2000; Sallis et al., 2002). Including the present study, there are only four studies to date that have used objectively measured PA in examining relations with depressive symptoms. Given the potential impact of PA interventions on preventing and treating depression, future studies that utilize more objective measures of PA would provide a more accurate understanding of the relation between PA and depression.

The present study was one of the first to target youth in early adolescence, before the rise in the prevalence of depression (Hankin & Abramson, 2001; Twenge & Nolen-Hoeksema, 2002). Moreover, of the studies with young adolescents to date (Annesi, 2004; Neissaar & Raudsepp, 2011; Petty et al., 2009; Rothon et al., 2010; Stavrakakis et al., 2012), only Annesi and Petty and colleagues included African American youth. Therefore, the over-representation of ethnic minorities in the present sample extends our understanding of the PA-depressive symptoms relation in these underserved and understudied youth. More research is clearly needed to understand the relations between PA, PA self-efficacy, and depressive symptoms in this at-risk population in longitudinal studies starting at a developmental point before the rise in depression and tracking the developmental trajectory across adolescence. Future research designed to track the trajectory of the effect of PA in early adolescence on later depressive symptoms would add to the field's understanding of the potential for early intervention and prevention, the causal mechanisms involved in these relations, and factors that may moderate intervention effects. For example, social support is a known protective factor for depression (Stice et al., 2011), and physical activity interventions may impact depression

by increasing access to social interaction and social support from peers (MacMahon, 1990; Phillips et al., 2001; Wilson, 2015). Future studies investigating more comprehensive models that include multiple mediators would better inform future interventions. Additionally, sex differences in depression are well established, but they do not emerge until later in adolescence (Hankin & Abramson, 2001). Similarly, ethnic differences in depression are apparent in older adolescents (Van Voorhees et al., 2008). While the present study controlled for sex and ethnicity, future research that tracked these relations throughout a longer timespan could explicitly investigate sex and ethnic differences in the relations between PA, cognitions, and depression outcomes.

Furthermore, it is important to continue to investigate these models, given that PA is perhaps more acceptable than traditional mental health intervention approaches (Dunn & Weintraub, 2008). In fact, other researchers have shown that children's cognitive capacities and, thus, the effectiveness of cognitive-behavioral interventions increase with age (Stice et al., 2009). Therefore, PA-based interventions may be a prevention strategy appropriate across a wide age range, unlike other evidence-based interventions such as cognitive-behavioral therapy. Beyond their potential impact on depression, interventions that specifically target PA self-efficacy also promote continued involvement in PA (Bandura, 1978; Lubans et al., 2008). Thus, such interventions have the potential to impact both adolescent mental and physical health. For example, enhanced self-efficacy for PA may simultaneously reduce depressive symptoms while increasing ongoing PA behavior (Wilson, 2015). This is an important approach as physical inactivity and depression are both associated with obesity (Anderson, Cohen, Naumova, Jacques, & Must, 2007; Goodman & Whitaker, 2002; Janssen & Leblanc, 2010). Obese youth have

lower self-perceptions such as low self-efficacy and low self-esteem (De Bourdeaudhuij et al., 2005; Wang, Wild, Kipp, Kuhle, & Veugelers, 2009), which may contribute to the higher rates of depression experienced by these youth (Ali et al., 2010; Melnyk et al., 2006; Roth, Munsch, Meyer, Isler, & Schneider, 2008; Sheslow, Hassink, Wallace, & DeLancey, 1993). Interventions that integrate approaches for mental and physical health may be more effective and efficient than those targeting single outcomes, i.e. decreasing obesity and depression in youth (Wilson, 2015). Future studies that deliberately target and track multiple outcomes over the long-term have the greatest potential for informing early intervention efforts aimed at improving overall well-being across the lifespan. These will be particularly important in ethnic minority youth who are at the highest risk for negative mental and physical health outcomes.

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