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Physical Activity in Adolescents with and without ADHD: Longitudinal Associations with
Sleep, ADHD, and Internalizing Symptoms

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
Philosophy at Virginia Commonwealth University

By: Caroline Cusick Lowman, M.S.,

Bachelor of Science, University of Maryland, 2016
Master of Science, Virginia Commonwealth University, 2020

Co-Chairs: Rosalie Corona, Ph.D.
Professor of Psychology
Virginia Commonwealth University (VCU)

Joshua Langberg, Ph.D.
Professor of Psychology
Rutgers University
VCU Affiliate Faculty

Virginia Commonwealth University
Richmond, Virginia
May 16th, 2023

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Abstract

PHYSICAL ACTIVITY IN ADOLESCENTS WITH AND WITHOUT ADHD: LONGITUDINAL ASSOCIATIONS WITH SLEEP, ADHD, AND INTERNALIZING SYMPTOMS

By Caroline Cusick Lowman, M.S.

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of
Philosophy at Virginia Commonwealth University

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Major Director: Joshua Langberg, Ph.D.

Adolescents with Attention-Deficit/Hyperactivity Disorder (ADHD) are at risk for developing clinically significant sleep problems and comorbid internalizing symptoms. Physical activity (PA) has significant positive associations with a variety of health outcomes, including sleep and aspects of mental health. As such, the US Department of Health and Human Services recommends adolescents receive at least 60 minutes of moderate-to-vigorous PA (MVPA) per day. However, it is unknown whether adolescents with ADHD have different patterns of physical activity compared to their peers. Importantly, it may be that PA can serve as a buffer between ADHD symptoms and development of comorbid difficulties with sleep or internalizing symptoms. Accordingly, the first aim of the present study was to evaluate whether the PA behaviors of adolescents with ADHD differ compared to non-ADHD peers. The second aim was to evaluate whether there are bidirectional relationships between ADHD symptoms, PA, and sleep and internalizing symptoms, and explore whether PA serves as a protective factor. Results indicate that adolescents with ADHD engage in significantly less PA than their non-ADHD peers in middle school, but these differences become less pronounced following the transition to high

school. There were notable presentation and sex differences in levels of adolescent PA.

Adolescents with ADHD-I engaged in significantly less PA at T1 than adolescents in the comparison group, whereas adolescents with ADHD-C did not. Boys with ADHD engaged in less PA than boys without ADHD at T1 and T2, whereas sex differences largely did not emerge between girls with and without ADHD. Autoregressive cross-lagged panel models demonstrated significant associations between parent-reported sleep difficulties and symptoms of ADHD; ADHD symptoms and adolescent-reported sleep; PA and adolescent-reported sleep difficulties, symptoms of depression, and symptoms of anxiety; and symptoms of anxiety and symptoms of ADHD. Moderation analyses were not significant in the present study. Future research should examine samples of youth with higher levels and more variability of PA, greater demographic and socioeconomic diversity, and explore PA from a multidimensional perspective.

Physical Activity in Adolescents with and without ADHD: Longitudinal Associations with Sleep, ADHD, and Internalizing Symptoms

The overarching goal of this study was to increase understanding of physical activity (PA) in adolescents with Attention-Deficit/Hyperactivity Disorder (ADHD) and explore how PA is associated with ADHD symptoms, sleep, and internalizing symptoms. Accordingly, the introduction provides an overview of ADHD during adolescence and unique considerations during this developmental period. This is followed by a review of the literature on PA in adolescents with and without ADHD. Finally, evidence for associations between sleep difficulties and internalizing symptomology are discussed in regards to ADHD and PA. In each section, factors that contribute to sleep and internalizing symptoms in adolescents with ADHD are discussed along with associations with impairment. The introduction concludes with a discussion of study aims and how they build upon the existing literature.

Introduction

ADHD is one of the most common mental health disorders in childhood, with 6.0 million, or 9.8% of children receiving a diagnosis prior to the age of 18 (Bitsko et al., 2022). ADHD is a neurodevelopmental disorder characterized by developmentally inappropriate levels of inattentiveness, hyperactivity, and impulsivity (American Psychiatric Association, 2022). According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR), there are eighteen symptoms of ADHD broken into two categories: inattention and hyperactivity/impulsivity (American Psychiatric Association, 2022). In order to meet criteria for ADHD, six or more symptoms must be present in either or both categories (five or more for 17 and older), occur in two or more settings (e.g., at home, school, or work), and interfere with, or reduce the quality of, social, academic, or occupational functioning (American

Psychiatric Association, 2022). Based on the symptom domain presentation, children receive a diagnosis of Predominantly Inattentive Presentation (ADHD-I), Predominantly Hyperactive-Impulsive Presentation (ADHD-HI), or Combined Presentation (ADHD-C). Symptoms must be present before the age of twelve, and ADHD is most commonly diagnosed between the ages of 4 – 7 years old (Visser et al., 2014).

ADHD in Adolescence

ADHD is considered a lifespan disorder that persists from childhood to adolescence in 50%–80% of cases, and into adulthood in 35%–65% of cases (Owens et al., 2015). Adolescents with ADHD continue to experience elevated levels of hyperactivity, inattention, and impulsivity in adolescence relative to their typically developing peers (Sibley et al., 2012). In a longitudinal examination of ADHD across childhood into adolescence, Sibley and colleagues (2012) found that hyperactive/impulsive symptoms reduced across time, but symptoms of inattention remained stable, and functional impairment increased. ADHD is best understood when considering the intersection of developmental stage, symptomatology, and functional impairment (Adler et al., 2015). Adolescence is a unique and challenging developmental period, in that it is a time of distinct biological maturation, neurological changes, and societal role transitions (Sawyer et al., 2018). For example, puberty is associated with rapid reorganization of neural circuitry involved with planning, decision making, and reward sensitivity (Sisk & Foster, 2004; Sibley et al., 2014) and heightened emotional responses (Casey et al., 2010). Adolescents also experience increased academic pressures, peer influences, and expectations surrounding independence and autonomy (Dahl & Forbes, 2010). For youth with ADHD, these developmental challenges and transitions interact with ADHD symptoms and lead to functional impairment. The areas of functional

impairment most common in adolescents with ADHD are academic and interpersonal impairment (Loe & Feldman, 2007; Sasser et al., 2017).

Considerable research has focused on academic impairments in adolescents with ADHD (see Evans et al., 2020 for a review). Adolescents with ADHD perform significantly lower on standardized assessments of math, reading, and writing (Frazier et al., 2007; Molitor et al., 2016), have lower grade point averages (GPA) relative to peers (Molina et al., 2009), are less likely than non-ADHD peers to turn in completed homework, and are more likely to receive failing grades (Kent et al., 2011; Frazier et al., 2007). In regards to family functioning, several studies have found that families of both male and female adolescents with ADHD have higher levels of conflict (per mother-, father-, and adolescent-report) than families of typically developing adolescents (Barkley et al., 1992; Chang et al., 2013; Edwards et al., 2001; Markel & Wiener, 2014). Conflicts arise around topics such as academics, money, and rules (Markel & Wiener, 2014), and are more frequent when parental ADHD symptoms are also present (Babinski et al., 2016).

Treatments for adolescents with ADHD vary depending on which aspects of ADHD and associated impairments are being targeted. Possible treatments include stimulant medications (e.g., methylphenidate, amphetamines), non-stimulant medications (e.g., atomoxetine, guanfacine), school-based interventions, and behavioral therapies (e.g., behavioral parent training, organizational skills training, cognitive-behavioral therapy) (Wolraich et al., 2019). In a systematic review of the literature, Sibley and colleagues (2014) found that behavior therapies and medication had similar ranges of therapeutic effects on symptoms of ADHD in adolescents. One of the most common treatments is stimulant medications, which reduce symptoms of ADHD and in some cases, functional impairments (Corkum et al., 2020). Although notably, medication

nonadherence is a relevant concern with adolescents in particular (Barnard-Brak et al., 2020). Behavioral interventions target aspects of functional impairment, such as disruptions at home, school difficulties, and personal responsibilities (Sibley et al., 2014).

Notably, not all adolescents with ADHD experience negative outcomes. An important way to inform treatments for ADHD is to understand potential protective factors that may reduce risk for impairment. Protective factors serve to buffer an individual from negative outcomes by promoting positive adaptation (Luthar et al., 2000). Constructs examined as protective factors in adolescents with ADHD include individual, social, and familial factors (Dvorsky & Langberg, 2016). For example, in a sample of 194 adolescents (ages 13 – 18 years old) with ADHD, Schei and colleagues (2015) found that individual competencies (e.g., personal and social competence) and social support promoted quality of life and mediated the relationship between emotional/conduct problems and quality of life. Dvorsky and colleagues (2018) found that both parent- and adolescent-rated social support served as a protective factor against academic impairment in young adolescents (10-14 years old) with ADHD. Longitudinal studies have also found positive parenting (Chronis et al., 2007) and maternal adjustment/parenting skills (Latimer et al., 2003) during childhood to be associated with improved emotional and behavioral outcomes during adolescence. One construct that has remained unexplored as a protective factor for youth with ADHD is physical activity.

Physical Activity

Physical Activity During Adolescence. From the perspective of public health, physical activity (PA) is defined as “any bodily movement produced by skeletal muscles that results in a substantial increase in energy expenditure above resting metabolic rate and includes leisure time physical activity, exercise, sport, occupational work, and household and other chores”

(Caspersen et al., 1985). There is a plethora of research to support that regular PA during adolescence helps promote healthy development, reduces risk for a variety of physical and mental health conditions, and improves sleep (Physical Activity Guidelines Advisory Committee, 2018). PA Guidelines for Americans (US Department of Health and Human Services, 2018) recommends that children under the age of 18 engage in 60 minutes or more of moderate-to-vigorous PA (MVPA) daily. Despite this, national polling suggests that children and adolescents are not regularly physically active. Findings from the 2009 Centers for Disease Control and Prevention (CDC) Youth Risk Behavior Survey (YRBS) indicated that only 18% of high school students had been physically active for 60 minutes every day in the previous week (CDC, 2010).

Many factors influence PA habits during this time period. Examples include social norms, support from friends and family, and physical environmental factors (CDC, 2011). First, gender is highly correlated with PA levels, with males participating in more overall PA than females (CDC, 2005; Gordon-Larsen et al., 2000). Adolescent males also report a greater intention to be physically active in the future than females (Robbins et al., 2003). This is generally attributed to differences in rearing and social and cultural expectations (Rodrigues et al., 2010). Perceptions of parental support for PA (e.g., perceiving parents will help facilitate sports or other physical activities) has also been found to play a role in increased participation in both structured and unstructured PA in adolescents (Van der Horst et al., 2007). Peer support and joint involvement in PA is also particularly motivating for adolescents (Hamilton et al., 2017; Sallis et al., 2000). An adolescent's physical environment can act to encourage PA or as a barrier to being physically active (CDC, 2011). Barriers include low availability of safe locations to be

active, lack of access to exercise equipment, and relative cost of exercise-related activities (Ferreira et al., 2007; Smith et al., 2015).

Physical Activity and ADHD. Much of the research connecting ADHD and PA is related to PA as a potential treatment for symptoms of ADHD. Reviews and meta-analyses on the topic have come to mixed conclusions regarding the effects of PA on symptoms of inattention and hyperactivity, but there is preliminary evidence for improvement in executive functioning skills, overall functioning, and motor skills (Cerrillo-Urbina et al., 2015; Cornelius et al., 2017; Ng et al., 2017; Sun et al., 2022; Vysniauske et al., 2020).

As far as the general PA habits of adolescents with ADHD and how they compare to adolescents without ADHD, there are no studies examining differences in adolescent-specific samples. It is important to examine PA within a developmentally appropriate framework, as PA behaviors and habits change as youth transition from childhood into adolescence. For example, overall levels of PA decrease over the period of adolescence, which is associated with increases in sedentary behaviors (Llorente-Cantarero et al., 2020; Mitchell et al., 2012). Societal changes associated with adolescence also uniquely impacts PA, including less time spent in recess and/or loss of recess altogether (CDC, 2015; SHAPE America, 2016), less time in schooling curriculums dedicated to physical education (PE) class (CDC, 2015), and having increased autonomy on how time is spent outside of school and engagement in extracurriculars (Sawyer et al., 2018). Despite these differences, studies to date have often examined PA behaviors in combined samples of children and adolescents with ADHD, and with a wide range of methodologies used to evaluate PA.

For example, San Mauro Martín and colleagues (2018) found that in their sample of 89 children ages 8 – 16 years old, individuals in the ADHD group had significantly more days of

exercise at school, whereas those in the comparison group reported more days of exercise after school. The authors also found that the ADHD group had a higher total mean exercise score (mean number of minutes engaged in exercise per week), whereas differences were not significant when examining differences in the number of minutes of exercise/playtime either during or after school (San Mauro Martín et al., 2018). Párraga and colleagues (2019) examined outcomes in a sample of 160 children and adolescents (6-16 years old) with and without ADHD. They found that there were no group differences in hours per week playing sports (Párraga et al., 2019).

Tandon and colleagues (2019) examined outcomes in a large population-based sample of children and adolescents (6-18 years old) with ($n=4,267$) and without ($n=47,532$) ADHD. They found that youth in their sample with more severe ADHD symptoms had lower odds of participation in sports, but there were no significant differences in odds of engaging in PA based on ADHD status (Tandon et al., 2019). The authors also noted that only one-third of children with ADHD participated in daily PA, and only half participated in sports over the prior year (Tandon et al., 2019). Tandon and colleagues (2019) were the only authors to explore age effects in their sample, and they found that older children with ADHD were less likely to engage in daily PA compared to younger children with ADHD (Tandon et al., 2019). Zerón-Rugério and colleagues (2020) examined PA in a sample of 120 children and adolescents (6 – 16 years old), however, whereas all other studies reviewed have relied on self-report, the authors assessed physical activity through the use of an accelerometer device worn on the wrist. Interestingly, they found that overall, the ADHD group had significantly increased levels of PA compared to the non-ADHD group (Zerón-Rugério et al., 2020). Upon further analysis, they found that this

difference was driven by individuals in the ADHD-C subsample specifically (Zerón-Rugério et al., 2020).

Taken together, the evidence-base on the PA habits of youth with ADHD is mixed. Additionally, all studies examined samples of children and adolescents jointly making it difficult to draw conclusions about adolescents with ADHD. PA is an important component of health, and understanding the PA behaviors of adolescents with ADHD may help promote effective management of ADHD by building habits that protect against negative outcomes. Outcomes associated with ADHD that PA may serve as a buffer against are sleep difficulties and comorbid internalizing symptoms.

Sleep

Adolescent Sleep. Sleep functioning is an area of impairment common for adolescents with ADHD. Adolescence is a period of life associated with changes in sleep, when biopsychosocial factors often come together to limit the quantity and quality of sleep (Carskadon, 2011; Crowley et al., 2018). This includes neurophysiological factors, such as homeostatic pressure changes, which leads adolescents to not feeling tired until later in the evening (Owens et al., 2014), and a shift in circadian phase preference towards later bed-times and wake-times (i.e., “eveningness chronotype”; Colrain & Baker, 2011). Adolescents are also undergoing rapid cognitive and physical maturation, which requires more sleep than both children and adults in order to support these processes (Crowley et al., 2018). Taken together, adolescents have a biological need for more sleep and later sleep times. These biological changes interact with contextual factors that may not allow adolescents to obtain recommended amounts of sleep, such as early school start times, increased after school demands, and increased technology use (Owens et al., 2014; Crowley et al., 2018; Mei et al., 2018). This has led to a

pervasive issue in which adolescents are receiving insufficient and ill-timed sleep (Crowley et al., 2018; Maslowsky & Ozer, 2014).

Sleep in Adolescents with ADHD. There has been a long-standing interest in sleep functioning in individuals with ADHD. Behavioral sleep disturbances in this population were even once considered to be a core feature of the disorder in earlier versions of the DSM (Becker et al., 2015a). Relevant biological and social factors remain pertinent considerations, and are compounded by ADHD-specific difficulties, which leads to impaired sleep functioning in this population. The literature examining sleep patterns and behaviors in adolescents with ADHD has grown considerably in recent years, but remains limited relative to research in child and adult populations (Becker, 2019; Cortese et al., 2009). Research to date has utilized both population/community-based samples, examining “high” and “low” symptoms of ADHD (i.e., evaluated ADHD symptoms continuously) as well as samples of adolescents comprehensively diagnosed with ADHD.

Hysing and colleagues (2016) examined 9,846 adolescents between the ages of 16 – 19 years old, and compared self-reported sleep problems between those with high versus low self-reported ADHD symptoms. They found that adolescents with high ADHD symptoms reported shorter sleep duration and time in bed, longer sleep latency and wake after sleep onset, lower sleep efficiency, greater sleep need, and more daytime sleepiness than those with low ADHD symptoms (Hysing et al., 2016). Similarly, Lam and Yang (2008) examined a sample of 1,429 adolescents between the ages of 14 and 15 years old and found that increased symptoms of ADHD were associated with shorter sleep duration, even while controlling for body mass index (BMI) and snoring. However, the most compelling research for the unique problems in

individuals with ADHD comes from comparative studies with individuals comprehensively diagnosed with ADHD.

Lufi and Tzischinsky (2014) examined self-reported sleep problems in a sample of 30 adolescents diagnosed with ADHD compared with 28 adolescents without ADHD (ages 13-17). They found that participants in the ADHD group were significantly more likely to report needing more awakenings in the morning and longer onset sleep latency (Lufi & Tzischinsky, 2014). No group differences were found on daytime sleepiness (Lufi & Tzischinsky, 2014). In a larger sample, Becker and colleagues (2019) compared the sleep patterns of 162 adolescents diagnosed with ADHD with 140 comparison adolescents between the ages of 12 – 14 years old. After controlling for pubertal development, sex, medication use, and presence of an internalizing disorder, they found that those with ADHD experienced shorter school night sleep duration assessed via both sleep diary and actigraphy (Becker et al., 2019). Additionally, adolescents with ADHD had significantly higher rates of adolescent- and parent-reported daytime sleepiness and parent-reported difficulties initiating and maintaining sleep, and total sleep disturbance (Becker et al., 2019). In a large comprehensively diagnosed sample, Chiang and colleagues (2010) examined 325 adolescents with ADHD and 257 adolescents without ADHD (ages 10 – 17). Adolescents with ADHD were more likely than non-ADHD peers to have a variety of sleep problems, such as difficulties initiating sleep and daytime sleepiness (Chiang et al., 2010). These results were maintained while controlling for sex, age, psychiatric comorbidities, and medication use and were found across multiple informants. Chiang and colleagues (2010) also found that there were differences based on ADHD presentation. For example, only adolescents with ADHD-C had increased circadian rhythm problems and sleep talking as compared to adolescents without ADHD (Chiang et al., 2010). This study also highlighted interesting cultural

considerations. The authors had unexpected findings in which increased symptoms of ADHD were associated with earlier bedtimes and later rise times on school nights (Chiang et al., 2010). However, the authors noted that in Taiwan, where the sample was collected, youth with behaviors and qualities associated with ADHD are less likely to attend specialized time-intensive schools (i.e., “cram schools”; Chiang et al., 2010), which may account for some of the findings. This study underscores the importance of considering culturally relevant psychosocial factors when examining sleep functioning.

Research to date has primarily focused on subjective measures (e.g., parent- or self-reported questionnaires) of sleep functioning. Findings are mixed when examining objective measures of sleep (e.g., actigraphy and polysomnography [PSG]). Mullin and colleagues (2011) compared sleep of 14 adolescents (ages 11-17 years) with ADHD to 21 non-ADHD peers using both actigraphy and daily sleep diaries. There were no statistically significant differences on any measures of sleep functioning, including sleep duration, sleep efficiency, and sleep onset latency (Mullin et al., 2011). The authors noted that the ADHD group was worse on all objective sleep assessments and that sample size and statistical power was a limitation (Mullin et al., 2011). This is in contrast to findings by Becker and colleagues (2019), which found significant group differences on actigraphy-derived sleep duration, but no differences on actigraphy-derived sleep efficiency or sleep onset latency. Prehn-Kristensen and colleagues (2014) assessed 24 adolescent boys 10 – 14 years old with ADHD and examined sleep functioning via PSG. They found that adolescents with ADHD in their sample displayed increased sleep onset latency and reduced sleep efficiency compared to those without ADHD (Prehn-Kristensen et al., 2014). However, no differences were found on several other PSG-derived aspects of sleep functioning, such as total sleep time, number of night awakenings, and time in bed (Prehn-Kristensen et al., 2014). Taken

together, these results indicate that adolescents with ADHD experience worse sleep than their peers across a variety of sleep domains, although there are mixed results depending on the aspect of sleep examined and how sleep is assessed.

Impairments Associated with Sleep Difficulties. Sleep difficulties can be conceptualized as an impairment associated with ADHD, or a factor contributing to other areas of functional impairment. Research in samples of children with ADHD have found sleep difficulties to be associated with worse working memory (Sciberras et al., 2015), increased emotional and behavioral problems at school (Lucas et al., 2019), and lower quality of life (Craig et al., 2020; Sung et al., 2008). Less research has been conducted in adolescent samples, although similar patterns of findings have emerged. In a sample of 100 young adolescents (10 – 14 years old) with ADHD, Langberg and colleagues (2013) found that self-reported daytime sleepiness significantly predicted increased parent-rated academic impairment and homework problems and lower teacher-rated academic competence. These results were significant above and beyond symptoms of ADHD (Langberg et al., 2013). In a subset of the same sample, authors also found that above and beyond ADHD symptom severity, baseline sleep problems significantly predicted greater ODD symptoms, general externalizing behavior problems, and depressive symptoms one year later (Becker et al., 2015b). Taken together, these findings highlight the clinical importance of examining sleep functioning in samples of adolescents with ADHD.

Contributing Factors. Multiple underlying mechanisms have been theorized to contribute to increased sleep problems in youth with ADHD. There is growing evidence that ADHD is associated with a gene mutation in the CLOCK gene (Tauber et al., 2004), which is responsible for regulating circadian rhythm, in a way that is implicated with disrupted sleep

patterns (Kissling et al., 2008). With this, findings in both adults and children suggest that individuals with ADHD have a genetic predisposition for eveningness chronotypes (Durmuş et al., 2017). Additionally, there is overlap in the neurological circuits involved in arousal/sleep and attention (Owens et al., 2013). There is reason to believe that the orexin system, which is involved in both sleep-wake regulation and reward process, may be compromised in individuals with ADHD (Cortese et al., 2008). Konofal and colleagues (2014) conducted a pilot study of mazindol, an orexin receptor agonist, and found that it significantly improved ADHD symptoms in a sample of children with ADHD.

Psychotropic medication use has long been considered as a contributing factor to sleep problems in individuals with ADHD. As previously noted, one of the most common treatments for symptoms of ADHD in youth is medication (Corkum et al., 2020). Stimulant medications are the most commonly prescribed medications for youth with ADHD (Zuvekas & Vitiello, 2012). Stimulants work as a treatment for symptoms of ADHD by increasing levels of dopamine and/or norepinephrine in key areas of the brain associated with executive functions, however, they can also work to increase energy and alertness (Advokat et al., 2014). Kidwell and colleagues (2015) conducted a meta-analysis of the literature on stimulant ADHD medication use and sleep functioning. They found that stimulant medications impact aspects of sleep such as sleep onset latency, sleep efficacy, and total sleep time (Kidwell et al., 2015). Nevertheless, sleep problems exist outside of ADHD medication use in this population. For example, multiple studies have found no differences in sleep functioning among medicated versus non-medicated youth with ADHD (Becker et al., 2015b; Moreau et al., 2014). Youth with ADHD who are not taking stimulant medications also continue to have significantly higher rates of sleep problems in comparison to their peers (Cohen-Zion & Ancoli-Israel, 2004; Stein et al., 2012). Overall,

stimulant medication has a negative impact on sleep functioning, however, the high prevalence of sleep problems in youth with ADHD is not solely an artifact of stimulant medication use.

Multiple psychosocial factors come together to negatively impact sleep during adolescence, many of which are exacerbated in adolescents with ADHD. For example, adolescents with ADHD experience increased academic issues (Evans et al., 2020), and as a consequence, often need to spend more time in the evenings completing assignments than their peers. For example, Coghill and colleagues (2008) surveyed 910 families of youth (6-18 years old) with ADHD and 995 families of typically developing children. Parents of children with ADHD were significantly more likely to report that their child had homework difficulties, with issues becoming most apparent in the late afternoon and early evening hours leading to bedtime (Coghill et al., 2008). A series of studies in a sample of 302 adolescents (ages 12 – 14 years old) found that adolescents with ADHD had significantly greater technology use (e.g., TV/movie viewing, video game, and phone use; Bouchtein et al., 2019) and were more likely to consume caffeine in the afternoon and evening (Cusick et al., 2020) compared to adolescents without ADHD. Both studies, although notably cross-sectional, found unique associations between technology and caffeine use and worse sleep functioning in the ADHD group only (Bouchtein et al., 2019; Cusick et al., 2020). Longitudinal research is needed to understand the temporal ordering of effects. Nevertheless, it is clear that sleep during adolescence for youth with ADHD is a complex confluence of biological and contextual vulnerabilities.

Physical Activity and Sleep. One of the many areas enhanced through regular PA is sleep functioning. A meta-analysis focused on the association between PA and sleep in adolescence and early adulthood (ages 14 – 24 years old) concluded that adolescents with higher subjective and objective levels of PA were more likely to experience better objectively- and

subjectively-measured sleep outcomes (Lang et al., 2016). In particular, the strongest associations were found between PA and sleep quality (Lang et al., 2016).

When systematically reviewing different assessment methodologies, Lang and colleagues (2016) found that subjective measures (e.g., self-report questionnaires, logs, surveys) were the most common mode of assessment for both PA and sleep. Interestingly, the authors found no evidence of self-report bias, as there were comparably strong effect sizes in studies using all self-report and those that examined objective measures of both sleep and PA (Lang et al., 2016). Notably, almost all studies were cross-sectional in nature. The only longitudinal study included that could evaluate the direction of effects found that morning PA intervention led to improved subjective and objective sleep outcomes for adolescents (Kalak et al., 2012). Recently published work also found preliminary support for bidirectional effects of sleep and PA in adolescents (Master et al., 2019).

The exact mechanisms connecting sleep and PA are not well-delineated, as the association likely involves a series of complex, bidirectional interactions between multiple physiological and psychological pathways (Chennaoui et al., 2015). However, broadly, shared pathways between sleep and PA include areas of energy conservation, body restoration, metabolic rate, cardiac functioning, and thermoregulation (Chennaoui et al., 2015). A psychologically-oriented theory proposes that sleep and PA are partially connected through subjective wellbeing and mood improvements (Gerber et al., 2014). For example, PA may lead to increased psychological well-being, and as a result, one becomes more satisfied with their sleep (Gerber et al., 2014; Chennaoui et al., 2015). With this, as someone becomes more sleep deprived, PA may also be one of the first behaviors abandoned (Gerber et al., 2014; Youngstedt, 2005).

Physical Activity and Sleep in Youth with ADHD. Despite the well-established connections between PA and improved sleep, and the ongoing concerns about sleep functioning in youth with ADHD, there has only been one study to examine the impact of PA on sleep in a sample of adolescents with ADHD. Li and colleagues (2021) examined sleep and levels of MVPA in 272 adolescents (10-17 years old) diagnosed with ADHD. In their sample they found significant, although weak, positive correlations between parent-reported sleep quality and parent-reported MVPA (Li et al., 2021). When examining relationships via path analyses, controlling for age and gender, the authors similarly found that MVPA was weakly and positively associated with sleep (Li et al., 2021). While this is the first study to examine this relationship in a sample of adolescents, the authors noted limitations in that all analyses were cross-sectional and utilized single item questions in order to assess both sleep and PA (Li et al., 2021). Research in this area could be strengthened by conducting longitudinal examinations of how these variables are associated with each other over time in order to better understand the directionality of these associations. Additionally, research examining PA as a moderating factor is needed to explore how PA may serve as a buffer for the development of sleep difficulties.

Anxiety/Depression

Adolescence and Anxiety/Depression. Comorbid internalizing symptoms, such as anxiety and depression, are another common concern for adolescents with ADHD. Broadly, adolescence is a period of increased vulnerability for the onset of internalizing mental health symptoms. When examining prevalence estimates of internalizing symptoms across childhood into adolescence, data shows that symptoms of anxiety and depression increase with age and peak during the period of adolescence between 12-17 years old (Bitsko et al., 2022). Notably, gender differences exist such that adolescent girls are at increased risk of developing anxiety

disorders and depression compared to boys (Bitsko et al., 2022; Essau et al., 2014). Biological and social changes interact during adolescence to account for the rise in internalizing symptoms during this period. Neurological changes occur in the limbic region and the prefrontal cortex during puberty, in which there is an imbalance between emotional response and cognitive abilities (Casey et al., 2008). This has been theorized as a potential mechanism for increased stress and internalizing symptomatology during adolescence (Casey et al., 2010). Cognitive maturation also occurs during this period, leading to enhanced social understanding and self-awareness (Thapar et al., 2012). These factors can then interact with a myriad of psychosocial stressors, such as peer victimization through bullying (Siegel et al., 2009; Sweeting et al., 2006), increased interpersonal conflict, and negative family relationships (Grant, 2013; Thapar et al., 2012) to lead to anxiety and depression.

Anxiety/Depression in Adolescents with ADHD. Mental health comorbidities are common for adolescents with ADHD. In a nationally representative sample of adolescents (13 – 18 years old), 92% of adolescents with ADHD met lifetime criteria for at least one other mental health disorder (Merikangas et al., 2010). Other lifetime estimates range from 40 – 80% depending on the sample and method for obtaining diagnostic information (Reale et al., 2017). Findings are mixed when looking specifically at comorbid anxiety and depressive disorders, and vary depending on how symptoms/diagnosis is evaluated. In an 8-year follow-up study of 436 youth comprehensively diagnosed with ADHD, Molina and colleagues (2009) found that adolescents (15 – 17 years old) with a childhood history of ADHD had significantly higher rates of having an anxiety and/or depression diagnosis (10.4%) compared to adolescents from the typically developing group (5.2%). Interestingly, there were no group differences at the continuous symptom level for anxiety, but there were significant differences for symptoms of

depression (Molina et al., 2009). In contrast, Costello and colleagues (2003) found significant concurrent co-occurrence between ADHD and having an anxiety disorder, but not between ADHD and having a depressive disorder. This in contrast not only to the findings of Molina and colleagues (2009), but also to several other studies indicating that childhood ADHD is associated with increased risk of depression (Biederman et al., 1996; Biederman et al., 2008; Smalley et al., 2007).

Smalley and colleagues (2007) longitudinally examined a population-based sample of youth in Finland and found that a lifetime diagnosis of ADHD was associated with a 2.4-fold risk increase for an anxiety disorder and 2.9-fold risk increase for a mood disorder during adolescence. Of the adolescents with ADHD (16 – 18 years old), 26.6% met criteria for an anxiety disorder, and 22.2% met criteria for a mood disorder (Smalley et al., 2007). Gau and colleagues (2010) assessed comorbidity prevalence rates in a sample of 296 Taiwanese adolescents (11 – 17 years old). The authors did not find significant differences in rates of having an anxiety disorder (with the exception of specific phobia) or a mood disorder diagnosis between adolescents with and without a lifetime diagnosis of ADHD (Gau et al., 2010). Rates of comorbid anxiety and mood disorders were generally high in the ADHD adolescent group nonetheless (38% and 24%, respectively; Gau et al., 2010). Bagwell and colleagues (2006) also did not find significant differences in rates of anxiety or mood disorders in adolescents with and without a history of childhood ADHD in a sample of 224 adolescents from the United States.

In summary, internalizing comorbidities are prevalent in adolescents with ADHD, and there is some evidence to suggest that this population is at increased risk of developing these disorders compared to typically developing peers. Future research would benefit from examining

anxiety and depression separately in order to better understand the unique vulnerabilities for adolescents with ADHD.

Impairments Associated with Comorbid Anxiety/Depression. Internalizing comorbidities in youth with ADHD can lead to a host of negative outcomes. Most of the research to date has examined the impact of comorbid internalizing symptoms in combined samples of children and adolescents. For example, research in a sample of 199 children and young adolescents (5 – 13 years old) found that parents of children with ADHD and a comorbid internalizing disorder reported worse family quality of life compared to parents of children with ADHD only (Armstrong et al., 2015). Similarly, Accardo and colleagues (2012) found that youth (ages 6 -18) with ADHD and comorbid anxiety had higher overall sleep disturbance scores than children with ADHD only. Youth with comorbid depression did not have higher overall sleep disturbance scores than children with ADHD only, but had significantly higher subscale scores in domains of sleep onset latency and sleep duration (Accardo et al., 2012). Biederman and colleagues (2008) found that adolescent girls with ADHD and comorbid depression had significantly higher odds of teen pregnancy compared to those with ADHD alone.

Finally, Becker and colleagues (2015c) examined how specific facets of comorbid anxiety and depression relate to peer functioning in adolescents (10 – 14 years old) with ADHD. The authors found that social anxiety, anhedonia, and negative self-evaluation in particular were associated with poorer social skills and social acceptance (Becker et al., 2015c). The presence of a comorbid diagnosis of depression, but not a comorbid anxiety diagnosis, was associated with poorer social functioning in their sample (Becker et al., 2015c). Overall, these findings point to the importance of further understanding internalizing symptoms in adolescents with ADHD as the presence of these comorbidities are associated with increased risk for negative outcomes.

Contributing Factors. Although many studies have investigated the prevalence of internalizing comorbidities in youth with ADHD, potential explanations for this co-occurrence have received less attention (Meinzer et al., 2014; Murray et al., 2022). There are several proposed theories as to why adolescents with ADHD may be at increased risk for developing comorbid symptoms of depression (Meinzer et al., 2014). One theory is that ADHD and depression are linked through shared distinct reward responsivity pathways (Meinzer et al., 2014). Both ADHD and depression have unique reactivity to pleasurable stimuli and rewards (Kato, 2007; Wood & Neale, 2010), and preliminary findings suggest that pathways of lower reward responsivity mediate the relationship between ADHD symptoms and symptoms of depression in adults with ADHD (Meinzer et al., 2012). Another theory relates to emotion dysregulation. Deficits in emotion regulation abilities have been included in theoretical models of both ADHD and depression (Meinzer et al., 2014). Seymour and colleagues (2012; 2014) found that emotion regulation behaviors mediated the relationship between ADHD and symptoms of depression in samples of youth with ADHD using both cross-sectional (Seymour et al., 2012) and longitudinal designs (Seymour et al., 2014). It has also been suggested that functional impairments associated with ADHD, such as in the domains of social, academic, and family functioning, may lead to feelings of demoralization, putting youth with ADHD at increased risk for developing symptoms of depression (Biederman et al., 1998; Meinzer et al., 2014). There is some evidence to support that difficulties with peer relationships (Bagwell et al., 2006; Humphreys et al., 2013), academic impairments, and parent-child conflict (Eadeh et al., 2017) may lead to symptoms of depression in adolescents with ADHD. Although notably, adolescents with ADHD continue to be at increased risk for symptoms of depression even when controlling for these impairments (Meinzer et al., 2013).

Few studies have examined factors contributing to comorbid anxiety in adolescents with ADHD, although psychosocial stressors associated with ADHD likely play a role (Becker & Fogleman, 2020). Theories have been proposed related to shared temperamental traits and family characteristics (Jarrett & Ollendick, 2008). Studies in children have found parental anxiety (Jarrett et al., 2016; Jarrett & Ollendick, 2008), greater parental control, and less positive parenting (Kepley & Ostrander, 2007; Pfiffner & McBurnett, 2006), are associated with comorbid anxiety in children with ADHD. There is also preliminary evidence to support bidirectional relationships between symptoms of ADHD and anxiety (Murray et al., 2022). In summary, this is a relatively understudied area, but there are likely multiple developmental pathways accounting for increased symptoms of anxiety and depression in adolescents with ADHD.

Physical Activity and Anxiety/Depression. Another area of functioning positively associated with PA is anxiety/depression. In a systematic review and meta-analysis of PA interventions and depression in adolescents (13 – 17 years old), Carter and colleagues (2016) found that increased PA was significantly associated with reduced depressive symptoms in both clinical and non-clinical samples. This finding is consistent with two other reviews of the literature which concluded that that PA may serve as a protective factor against depression in adolescents (Johnson & Taliaferro, 2011) and that there are moderate significant effects of increased PA on symptoms of depression in adolescents diagnosed with depression (Radovic et al., 2017). In regards to PA and symptoms of anxiety, there is less research, especially with adolescents, and more variability in methodologies making it difficult to draw firm conclusions (Biddle et al., 2019). However, multiple systematic reviews suggest that PA is associated with

small to moderate reductions in symptoms of anxiety in both clinical and nonclinical samples (Ahn & Fedewa, 2011; Ferreira-Vorkapic et al., 2015).

The proposed mechanisms underpinning the association between PA and mental health are complex. One of the proposed biological pathways is through the increased secretion of serotonin and endorphins associated with PA, which is known to have antidepressant and mood improving effects (Chaouloff, 1997). PA also impacts cortisol regulation, which can reduce physiological reactions to stress-inducing stimuli (Wipfli et al., 2011). There are also pathways identified via increased mitochondria efficiency in key areas of the brain, which leads to improved neural protein synthesis (Deslandes, 2014). Theories of psychosocial mechanisms suggest that increased PA and subsequent participation in PA-oriented activities, enhances self-esteem, feelings of autonomy, feelings of competence and confidence, and development of social supports (Biddle et al., 2019; Doré et al., 2020).

PA and Anxiety/Depression in Youth with ADHD. No studies have explicitly examined the relationship between PA and internalizing symptoms in youth with ADHD. However, amongst the many studies examining PA as an intervention for symptoms of ADHD, there were two studies which also explored how the intervention impacted comorbid internalizing symptoms in samples of adolescents with ADHD (Ahmed & Mohamed, 2011; Hernandez-Reif et al., 2001). Hernandez-Reif and colleagues (2001) examined the effects of participating in Tai Chi two times per week for five weeks in a sample of 13 adolescents (13 – 16 years old) with ADHD. They found significant reductions in symptoms of anxiety post intervention, and these reductions were maintained two weeks following the end of the intervention period (Hernandez-Reif et al., 2001). However, it is unclear whether these effects can be attributed to increased physical activity or other potential benefits of Tai Chi (e.g., stress

reduction; Hernandez-Reif et al., 2001). Ahmed and Mohamed (2011) examined the effects of participating in a 10-week aerobic-based exercise program in a sample of 84 adolescents (11 – 16 years old) with ADHD and compared outcomes to a randomly selected control group. No significant reductions were observed on the ‘emotional/oppositional behavior’ subscale between pre- and post-intervention, or between the intervention and control group (Ahmed & Mohamed, 2011). However, results are difficult to interpret as the subscale measured comprises both internalizing and externalizing behaviors (Ahmed & Mohamed, 2011). Overall, there is very little research on the relationship between PA and internalizing symptoms in the context of ADHD.

Statement of the Problem

Adolescents with ADHD are at increased risk for sleep problems and internalizing symptomatology. Comorbid internalizing symptoms and sleep functioning are clinically meaningful outcomes as both are associated with significantly increased functional impairment. There is ample evidence in normative populations that PA can be beneficial for both sleep and internalizing symptomatology. However, little is known about the PA habits of adolescents with ADHD and how they compare to adolescents without ADHD, and there is no longitudinal research evaluating how PA is associated with or impacts the development of comorbid mental health and sleep problems in the context of ADHD.

Present Study

Adolescence is a unique period of development both in regards to ADHD presentation and PA, and therefore it is important to understand how PA behaviors may uniquely manifest for adolescents with ADHD. This study builds upon and contributes to the existing literature in meaningful ways. The present study used data collected from a two-year longitudinal sample of

adolescents with and without ADHD, allowing for comparisons in outcomes over time and between groups. The first goal was to compare PA behaviors of youth with and without ADHD in an adolescent-specific sample. This is the first study to do so, as prior studies have only examined combined samples of children and adolescents. Further, previous studies have examined singular aspects of PA or relied solely on single item questions. In the present study, varying aspects of PA were examined, including an overall PA score using a well-validated measure to assess general levels of PA, as well as reports on the number of days spent engaging in at least 20 minutes, and at least 60 minutes of PA, in order to assess adherence to national recommendations for adolescents (US Department of Health and Human Services, 2018). As prior literature has found evidence for differences in PA based on ADHD presentations in children (Zerón-Rugério et al., 2020), differences between presentations were examined. Additionally, as there are documented differences in PA habits in normative samples of adolescents (CDC, 2005; Gordon-Larsen et al., 2000), sex differences between the ADHD and comparison groups were also explored.

The second goal was to examine longitudinal relations between symptoms of ADHD, PA, sleep, and comorbid internalizing symptoms, using well-validated measures from multiple informants. Longitudinal autoregressive cross-lagged models were utilized. This is an expansion of prior literature, which most often has examined cross-sectional associations. These analyses allowed for an examination of temporal effects and bidirectional relationships. Notably, this was the first study to have symptoms of ADHD and PA within the same model as sleep and internalizing symptoms. In order to understand how PA potentially acts as a buffer for the development of sleep difficulties and internalizing symptoms, PA was also examined as a

moderating factor in the second step of the cross-lagged models. This provides clinically meaningful information for those working with adolescents with ADHD or symptoms of ADHD.

Aims and Hypotheses

Aim 1: Evaluate whether there are differences in physical activity between adolescents with and without ADHD at three separate time points over a two-year period.

Hypothesis 1a: Adolescents with ADHD will have higher PA (using total PA score) compared to adolescents without ADHD at all time points. Adolescents with ADHD will also engage in at least 20 and 60 minutes of PA on significantly more days than adolescents in the comparison group at all time points.

Hypothesis 1b: Adolescents with ADHD-C will have higher levels of PA on all measures of PA compared to adolescents without ADHD and adolescents with ADHD- I at all time points.

Hypothesis 1c: Adolescents with ADHD-I will not have significantly different levels of PA, or engage in more days of PA, than adolescents in the comparison group.

Hypothesis 1d: Sex differences will emerge in that boys with ADHD will have significantly higher PA on all measures than boys without ADHD. There will be no significant differences on PA measures between girls with and without ADHD.

Aim 2: Explore bidirectional associations between (adolescent-reported) total PA, (parent-reported) ADHD symptoms, and (parent- and adolescent-reported) sleep difficulties and (adolescent-reported) internalizing symptoms (anxiety and depression).

Hypothesis 2a: ADHD symptoms will be positively associated with both parent- and adolescent-reported sleep difficulties, and these associations will be reciprocal over time.

Hypothesis 2b: PA will be negatively associated with both parent- and adolescent-reported sleep difficulties, and these associations will be reciprocal over time.

Hypothesis 2c: ADHD symptoms will be positively associated with symptoms of depression and anxiety, and these associations will be reciprocal over time

Hypothesis 2d: PA will be negatively associated with symptoms of depression and anxiety, and these associations will be reciprocal over time.

Aim 3: Examine whether the strength of the associations between ADHD symptoms and sleep difficulties and internalizing symptoms is buffered by level of PA.

Hypothesis 3a: The effect of ADHD symptoms on sleep difficulties over time will be buffered by PA. As such, for individuals with higher PA, ADHD symptoms would have less of an effect on parent- and adolescent-reported sleep difficulties.

Hypothesis 3b: The effect of ADHD symptoms on symptoms of depression and anxiety over time will be buffered by PA. As such, for individuals with higher PA, ADHD symptoms will have less of an effect on symptoms of depression and anxiety.

Method

Participants

Initial recruitment of participants included 302 adolescents (167 males, 135 females) in eighth grade (ages 12-14 years) recruited as part of a study evaluating sleep functioning across the transition from middle to high school. The study focused on ADHD and sleep and adolescents with ADHD and a comparison control were specifically recruited. Approximately half of the participants (53.6%; $n = 162$) were diagnosed with ADHD (57 female, 105 male) and half (46.4%; $n = 140$) were in the comparison group (78 females and 62 males). In the overall sample, most participants identified as White (81.8%), with the remaining participants identifying as Black (5.3%), Asian (4.6%), American Indian/Alaska Native (0.3%), or Biracial/Multiracial (7.9%). 4.6% of participants identified as Hispanic/Latinx (see Table 1). All

participants had an estimated IQ ≥ 80 based on the Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II; Wechsler, 2011). See Figure 1 for information on participant retention across time points.

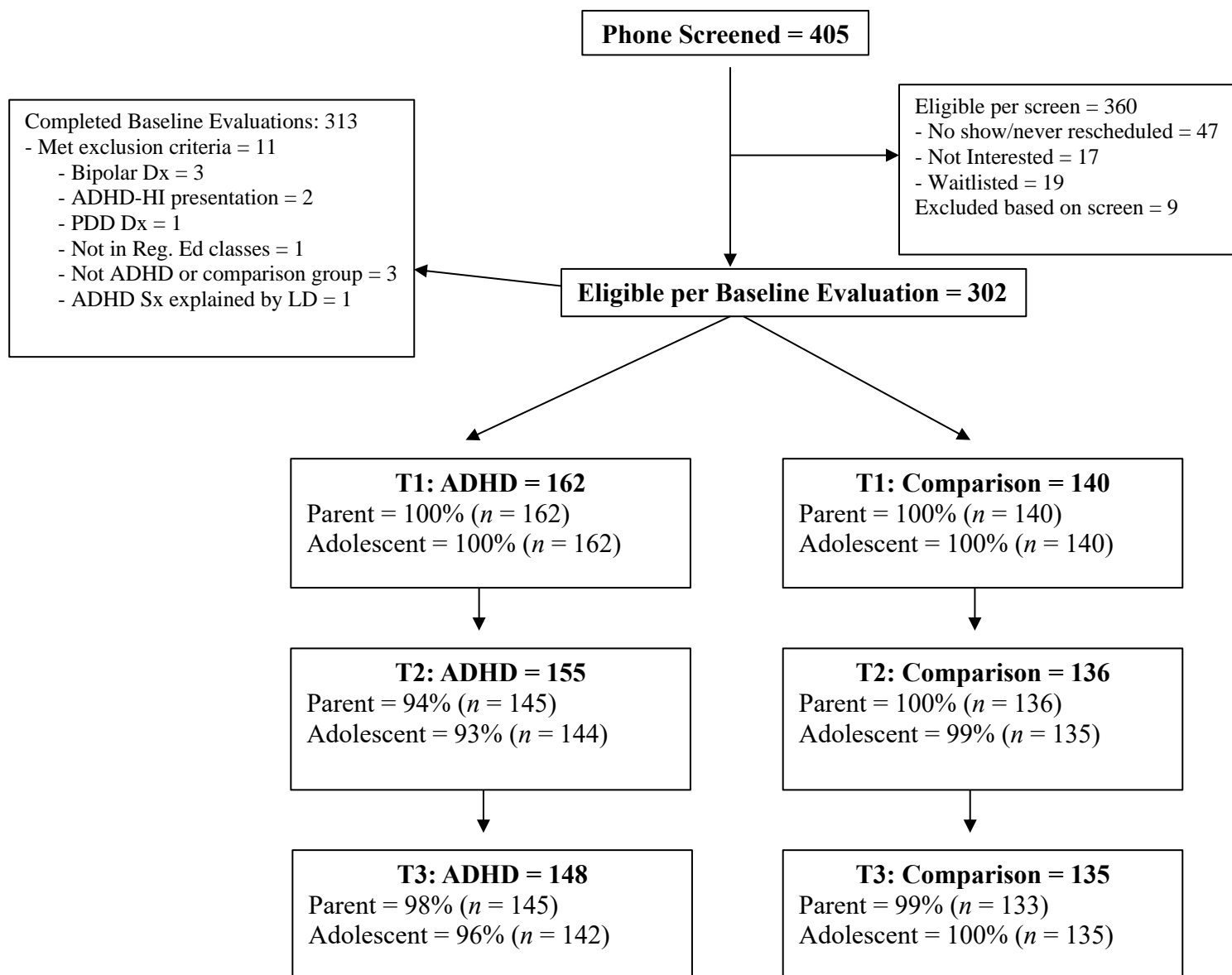
Table 1

Sample Characteristics at T1

	Total Sample (<i>N</i> = 302) <i>M</i> \pm <i>SD</i>	ADHD Group (<i>n</i> = 162) <i>M</i> \pm <i>SD</i>	Comparison Group (<i>n</i> = 140) <i>M</i> \pm <i>SD</i>
Age	13.2 \pm 0.4	13.2 \pm 0.4	13.2 \pm 0.4
Pubertal Development			
Female	3.07 \pm 0.6	3.11 \pm 0.6	3.05 \pm 0.7
Male	2.34 \pm 0.6	2.31 \pm 0.6	2.39 \pm 0.6
Primary Household Income	93,073 \pm 34,856	84,875 \pm 35,864	102,500 \pm 31,213
	<i>N</i> (%)	<i>n</i> (%)	<i>n</i> (%)
Sex			
Male	167 (55.3)	105 (64.8)	62 (44.3)
Female	135 (44.7)	57 (35.2)	78 (55.7)
ADHD Presentation			
Predominantly Inattentive	-	120 (74.1)	-
<i>Male</i>		76 (63.3)	
<i>Female</i>		44 (36.7)	
Combined	-	42 (25.9)	-
<i>Male</i>		29 (69.0)	
<i>Female</i>		13 (31.0)	
Race			
White	247 (81.8)	129 (79.6)	118 (84.3)
Black	16 (5.3)	12 (7.4)	4 (2.9)
Asian	14 (4.6)	4 (2.5)	10 (7.1)
American Indian/Alaskan	1 (0.3)	1 (0.6)	0 (0)
Biracial/Multiracial	24 (7.9)	16 (9.9)	8 (5.7)
Hispanic/Latinx	14 (4.6)	7 (4.3)	7 (5.0)
Any Medication Use	120 (39.7)	105 (64.8)	15 (10.7)
Comorbid psychiatric diagnoses ^a	107 (35.4)	74 (45.7)	33 (23.6)
Any externalizing (ODD/CD)	41 (13.6)	35 (21.6)	6 (4.3)
Any anxiety	73 (24.2)	46 (28.4)	27 (19.3)
Any depression	24 (7.9)	16 (9.9)	8 (5.7)

Note. SD=Standard Deviation; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; Income presented in US dollars.

^aPresence of comorbid mental health diagnosis based on parent or adolescent report (only parents were administered ODD and PTSD modules) during the diagnostic interview.

Figure 1*CONSORT Diagram of Participant Retention Across Time Points***Procedures**

All procedures were approved by an Institutional Review Board. Participants were initially recruited during the fall semester of their 8th grade year at two sites in the Southeastern and Midwestern United States. Potential participants were recruited via flyers provided to schools and emails sent out by school administrators. The study sought to recruit approximately

equal numbers of adolescents with and without ADHD. Therefore, two sets of recruitment materials were used, with one set specifically directed toward youth with attention difficulties and/or ADHD.

Parents who contacted the investigators in response to recruitment activities through local schools completed an eligibility phone screening. Those meeting the screening criteria were scheduled for an evaluation to determine eligibility. To be eligible for the ADHD group, participants had to meet full DSM-5 diagnostic criteria for either ADHD-I or ADHD-C based on the Parent Children's Interview for Psychiatric Syndromes (P-ChIPS; Weller et al., 2000). To be eligible for the comparison group, participants were required to have fewer than four symptoms of ADHD in each domain (i.e., inattention and hyperactivity/impulsivity) on the P-ChIPS. To be eligible for either group, participants had to have an estimated IQ ≥ 80 , take core classes in a regular education setting, and could not have a diagnosis of autism, bipolar disorder, a dissociative disorder, a psychotic disorder, or an organic sleep disorder per parent report.

Parent and adolescent ratings were collected online during the in-person baseline assessment (T1; Fall of 8th grade; August – December 2016 and 2017) and final assessment (T3; fall of 10th grade; August – December 2017 and 2018) visit using Research Electronic Data Capture (REDCap; Harris et al., 2009). Diagnostic interviews were also collected at T1 and T3 time points. The intermediate time point used in this study (T2; fall of 9th grade; August – December 2018 and 2019) was conducted entirely online via completion of questionnaires on REDCap.

Measures

Vanderbilt ADHD Rating Scale. The Vanderbilt ADHD Rating Scale – Parent Version (VARSP; Wolraich, 2003) is a parent-report measures that asks respondents to rate the

frequency of occurrence of each of the 18 DSM-IV symptoms of ADHD. Parents rate each symptom on a four-point Likert scale (e.g., 0 = *never*, 3 = *very often*). Items are summed in order to create a total ADHD score, with higher scores indicating increased symptoms of ADHD. Scores can range from 0 to 54. Internal consistencies in the present study were: T1 $\alpha = .95$, T2 $\alpha = .95$, and T3 $\alpha = .94$.

Exercise Questionnaire. The Exercise Questionnaire is a self-report measure that was adapted for this study by using items from both the adolescent version of the Physical Activity Questionnaire (PAQ-A; Kowalski et al., 1997a; Kowalski et al., 1997b) and the physical activity questions from the Centers for Disease Control and Prevention's Youth Risk Behavior Survey (YRBS; Brener et al., 2002). Seven items from the PAQ-A assess frequency of physical activity at different times of the day/week, over the prior seven days (e.g. *In the last 7 days, on how many days right after school, did you do sports, dance, or play games in which you were very active?; ...on how many days on the weekend...; ...on how many evenings...*). Items are summed in order to create a total PA score, with higher scores indicating higher levels of physical activity. Scores can range from 0 to 28. Convergent validity has been demonstrated between the PAQ-A and other self-report measures of PA (r s ranging from .57 to .73), structured recall interview of PA ($r = .59$), and motion sensor data ($r = .33$) (Kowalski et al., 1997b). Internal consistencies in the present study were: T1 $\alpha = .73$, T2 $\alpha = .73$, and T3 $\alpha = .72$. Two items from the YRSB were also assessed, which asks participants to report the number of days out of the prior 7 days in which they "*exercised or participated in physical activity that made you sweat and breathe hard*" for at least 20 minutes and at least 60 minutes. Scores can range from 0 to 7.

Sleep Disturbance Scale for Children. The Sleep Disturbance Scale for Children (SDSC; Bruni et al., 1996) is a parent-rated measure of sleep functioning validated in youth ages

6-15 years old. For the present study, *t*-scores from the difficulties initiating and maintaining sleep subscale (seven items) were used, which specifically assesses the frequency of a number of sleep-related behaviors in the past 6 months, such as typical total sleep time (1= *9-11 hours*, 2= *8-9 hours*, 3= *7-8 hours*, 4= *5-7 hours*, 5= *less than 5 hours*), sleep onset latency (1= *less than 15 min*, 2= *15-30 min*, 3= *30-45 min*, 4= *45-60 min*, 5= *more than 60 min*) and difficulties falling and staying asleep (5-point Likert scale, 1 = *never*; 5= *always*). Scores can range from 38 to 100. Scores greater than 70 are in the clinical range. Bruni and colleagues (1996) conducted a psychometric evaluation of the SDSC in a sample of 1157 caregivers and found internal consistencies ranging from .71 to .79, a test-retest reliability of .71, and a diagnostic accuracy for sleep disorders of .91. Studies have yet to establish convergent validity between the SDSC and corresponding adolescent-reports of sleep functioning. However, in the present study, the SDSC difficulties initiating and maintaining sleep subscale was significantly correlated ($ps < .05$) with adolescent-reported sleep wake problems (via the Sleep Habits Survey, see below), T1 $r = .35$, T2 $r = .29$, and T3 $r = .35$. In analyses comparing the SDSC to actigraphy, it was found that the SDSC showed sensitivity of 80.6% and specificity of 37.9%, with no significant differences in the ability to detect sleep disturbances compared to actigraphy (Herwanto et al., 2018). Internal consistencies for the difficulties initiating and maintaining sleep subscale in the present study were: T1 $\alpha = .77$, T2 $\alpha = .72$, and T3 $\alpha = .74$.

Sleep Habits Survey. The Sleep Habits Survey (SHS; Wolfson & Carskadon, 1998) is a self-report measure of sleep functioning validated for use in youth ages 10-19 years old (Wolfson et al., 2003). Validity testing established that reports on the SHS were significantly correlated with daily sleep diaries and actigraphy (Wolfson et al., 2003). Items in the survey are summed, with higher scores indicating worse functioning in the corresponding area of sleep. For the

present study, the 10-item sleep-wake difficulties subscale was used, which specifically assesses the frequency of a number of sleep-related problems in the past two weeks, such as falling asleep in class, staying up late, and having a hard time falling asleep. Items for this subscale are rated on a 5-point Likert scale (1 = *never*; 5 = *every day/night*). Scores can range from 10 to 50. Internal consistencies for the sleep-wake difficulties subscale in the present study were: T1 $\alpha = .76$, T2 $\alpha = .72$, and T3 $\alpha = .73$.

Revised Child Anxiety and Depression Scale. The Revised Child Anxiety and Depression Scale (RCADS; Chorpita et al., 2005) assesses DSM-based anxiety and depression symptoms on a 4-point scale (0 = *never*, 4 = *always*). The RCADS has been validated for use with students in 3rd through 12th grade. Designed for youth self-report, the RCADS has demonstrated excellent reliability and validity in clinical and non-clinical samples (Chorpita et al., 2005; Piqueras et al., 2017). The long version was administered at T1 and T3 and the 25-item short version (Ebesutani et al., 2012) was administered at T2. For the present study, the total anxiety and total depression subscales from the self-report short versions of the RCADS were used. Additionally, items related to sleep functioning were removed from the subscales for the purposes of this study. The depression subscale had two items removed (i.e., “*I have trouble sleeping*”, “*I am tired a lot*”), leaving eight items in the subscale. Scores can range from 0 to 32. The anxiety subscale had one item removed (i.e., “*I feel scared if I have to sleep on my own*”), leaving fourteen items in the subscale. Scores can range from 0 to 56. Higher scores indicate higher symptoms of anxiety (e.g., “*I worry about making mistakes*”) and depression (e.g., “*I feel sad or empty*,” “*Nothing is much fun anymore*”). Internal consistencies for the depression subscale in the present study were: T1 $\alpha = .82$, T2 $\alpha = .84$, and T3 $\alpha = .88$. Internal consistencies for the anxiety subscale in the present study were: T1 $\alpha = .88$, T2 $\alpha = .83$, and T3 $\alpha = .86$.

Covariates

Biological Sex. Participants reported their biological sex at the initial baseline visit.

Pubertal Development. The Physical Development Scale (PDS; Petersen et al., 1988) is a validated self-report measure assessing pubertal development in adolescents. There are separate forms for males and females to complete; a mean score of five items pertaining specifically to physical indicators of puberty was calculated for analyses. Higher scores are associated with more physical maturation associated with pubertal development.

Medication Use. A modified version of the Services for Children and Adolescents Parent Interview (Jensen et al., 2004) was used, which is a clinician-administered interview that asks parents whether their children are receiving a variety of pharmacological and nonpharmacological treatments and adapted for this study to include sleep medication. In the present study, current medication use for attention, emotional/behavioral difficulties, or sleep problems was included as a binary (yes/no) variable.

Data Analyses

Preliminary Data Analysis

Descriptive statistics including means, standard deviations, and correlations of primary study variables were first examined. Data were then checked for univariate and multivariate outliers, and skewness and kurtosis were also evaluated for primary study variables (see Table 2). Data were considered normal if skewness was found to be within the range of +2.0 to -2.0, and kurtosis within -7.0 to +7.0 (Bryne, 2010; Hair et al., 2010). Linearity and normality were also assessed by generating histograms and Q-Q plots between variables. Additionally, multicollinearity was assessed by examining bivariate correlations between all study variables. As suggested by Tabachnick and Fidell (2007), $r = .80$ was used as a cutoff to assess for

multicollinearity. Homogeneity of variance was assessed using the Levene's test for Equality of Variances. If the homogeneity of variance failed to pass the recommended significance value of .05, the Welsh and Brown-Forsythe tests were used.

Missing Data

Little's Missing Completely at Random (MCAR) test was used to ensure that data is not missing from subjects in a systematic manner. All models were estimated in Mplus 8.8, which has the capability of handling missing data through either multiple imputation or full-information maximum likelihood estimation, and includes robust estimation procedures. Analyses were based on maximum likelihood estimation (MLR) with robust standard errors. MLR computes mean adjusted maximum likelihood estimates for non-normally distributed continuous data (Muthén & Muthén, 2017). With missing data, MLR is used to obtain robust estimates and it is also recommended for small and medium sample sizes (Muthén & Asparouhov, 2002; Yuan & Bentler, 2000).

Analytic Plan

Aim 1: T-tests and Cohen's *d* effect sizes were calculated to examine ADHD/comparison group differences in PA variables. A Cohen's *d* effect sizes of .2 is considered small, a value of .5 is considered medium, and a value of .8 is considered large (Cohen, 1988). One-way Analysis of Variance (ANOVA) tests were conducted in order to determine whether or not there were significant differences on the PA variables between the ADHD-I, ADHD-C, and comparison groups. Corresponding eta-squared (η^2) effect sizes were also calculated. Effect sizes up to .01 are considered small, effect sizes between .02 and .06 are considered medium, and effect sizes .07 and higher are considered large (Miles & Shevlin, 2001). In the case where ANOVA tests yielded a significant *F*-statistic, post-hoc tests were conducted to evaluate which differences

between pairs of means are statistically significant. Specifically, Tukey's Honest Significant Different (HSD) test was implemented, which accounts for type I errors when comparing each pair of group means (Howell, 2016). T-tests and Cohen's *d* effect sizes were also calculated to examine sex differences on PA variables between the ADHD and comparison groups.

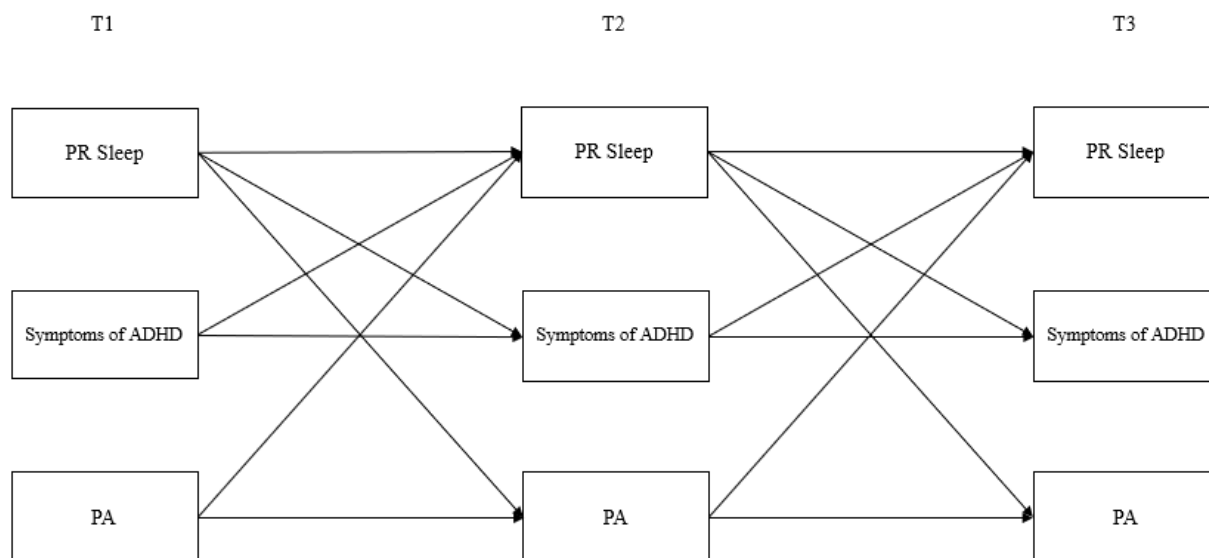
Aim 2: Longitudinal autoregressive cross-lagged models (ARCL; Cole & Maxwell, 2003; Curran & Bollen, 2001) were used to examine the associations between (adolescent-reported) total PA score, (parent-reported) ADHD symptoms, and parent-reported sleep difficulties (Figure 2)/adolescent-reported sleep difficulties (Figure 3)/(adolescent-reported) symptoms of depression (Figure 4)/(adolescent-reported) symptoms of anxiety (Figure 5). Separate models were examined for parent-reported sleep difficulties, adolescent-reported sleep difficulties, symptoms of depression, and symptoms of anxiety. Sex, pubertal development, and medication status were included as covariates on all models.

In ARCL models, scores at time (*t*) account for score deviation at a previous time (*t* – 1; (Curran & Bollen, 2001). More precisely, the autoregressive effects describe the stability of individual differences from one wave to the next; whereas the cross-lagged effects examine the effect of one construct on another measured at a later occasion. A feature of the ARCL model is that the cross-lagged effects are estimated controlling for prior level of the construct being predicted (i.e., autoregressive paths). For example, the variance in T2 sleep difficulties that is predicted by T1 ADHD symptoms is the residual variance controlling for previous levels of T1 sleep difficulties (i.e., the stable portion). Previously referred to as a “residual change model” (Cole & Maxwell, 2003; Gollob & Reichardt, 1987), the ARCL model allows one to rule out the possibility that a cross-lagged effect is due simply to the fact that the predictor and outcome were correlated at T1. In the models, significant paths from T1 ADHD symptoms to T2

sleep/depression/anxiety or T2 ADHD symptoms to T3 sleep/depression/anxiety would establish that ADHD symptoms predict sleep difficulties/depression/anxiety (testing hypotheses 2a/2c). Significant paths from T1 PA to T2 sleep/depression/anxiety or T2 PA to T3 sleep/depression/anxiety would establish that PA symptoms predict sleep difficulties/depression/anxiety (testing hypotheses 2b/2d).

Figure 2

Example ARCL Panel Model Examining Longitudinal Associations Between Parent-Reported Sleep Difficulties, Symptoms of ADHD, and Total PA

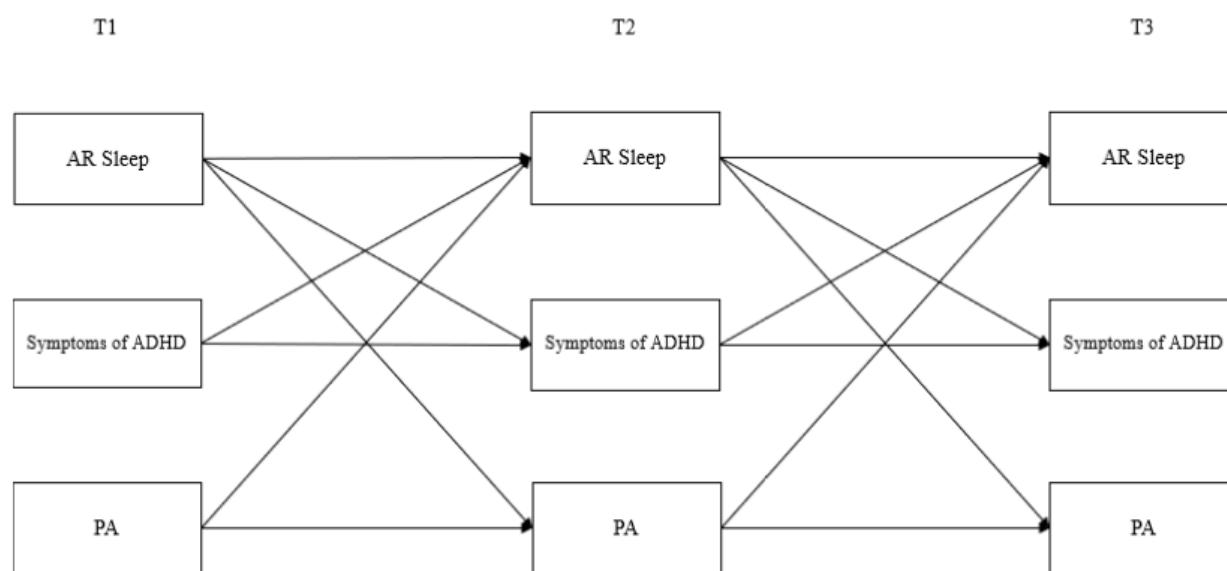


Note. ARCL = autoregressive cross-lagged; PR = Parent-Reported; PA = Physical Activity;

Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

Figure 3

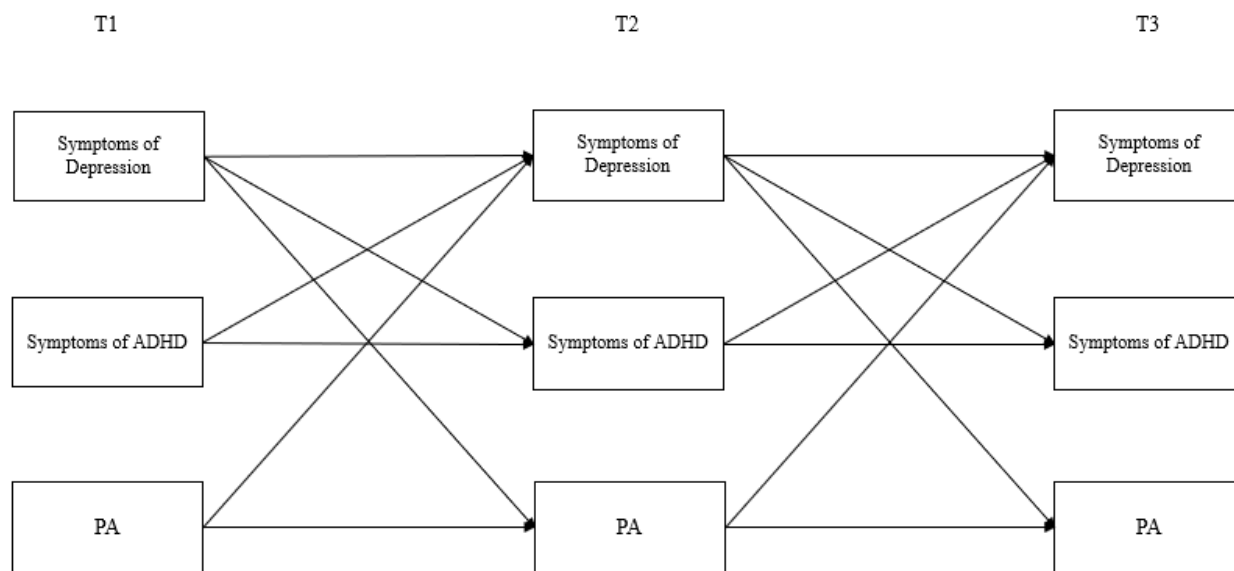
Example ARCL Panel Model Examining Longitudinal Associations Between Adolescent-Reported Sleep Difficulties, Symptoms of ADHD, and Total PA



Note. ARCL = autoregressive cross-lagged; AR = Adolescent-Reported; PA = Physical Activity; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

Figure 4

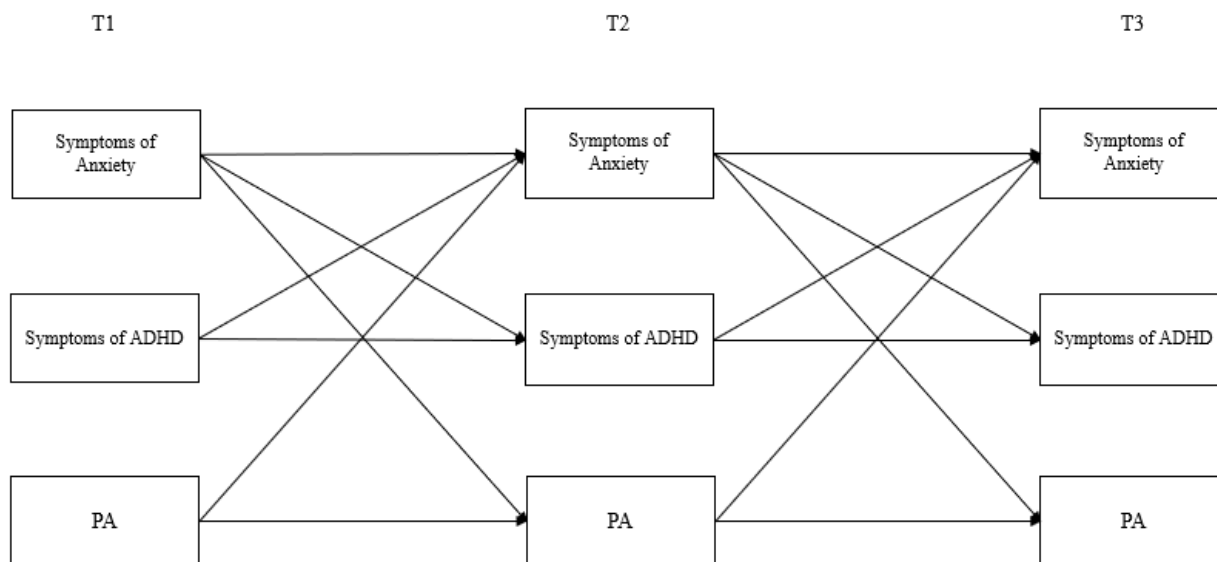
Example ARCL Panel Model Examining Longitudinal Associations Between Symptoms of Depression, Symptoms of ADHD, and Total PA



Note. ARCL = autoregressive cross-lagged; PA = Physical Activity; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

Figure 5

Example ARCL Panel Model Examining Longitudinal Associations Between Symptoms of Anxiety, Symptoms of ADHD, and Total PA



Note. ARCL = autoregressive cross-lagged; PA = Physical Activity; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

All longitudinal ARCL models were examined with equality constraints to determine the extent to which within-construct and cross-construct effects were significantly different across time points. A series of hierarchically nested models were examined in order to determine which path coefficients can or cannot be constrained (i.e., which parameters do or do not differ over time). These models were nested such that equality constraints were placed on each set of parameters one at a time (i.e., first autoregressive paths, then cross-lagged paths) and the more restrictive model (i.e., allowing more degrees of freedom) was compared to the previous unconstrained model. At each step, the model fit was compared with that of the previous step

using a chi-square difference test to evaluate whether path constraints resulted in decremented fit in order to test for stability across time.

The unconstrained and constrained models were compared using the Satorra-Bentler Chi-Square ($S-B\Delta\chi^2$) difference test and a comparison of model fit indices. Model fit was assessed with the Comparative Fit Index (CFI) and the Root Mean Square Error of Approximation (RMSEA) given that χ^2 is known to be sensitive to sample size (McDonald & Ho, 2002). Values between .90 - .95 or above for the CFI and .08 or below for the RMSEA (Bentler, 2007; Kline, 2005; McArdle & Nesselroade, 2014; Wang & Wang, 2019) indicate that the model adequately fits the data. The CFI is a goodness-of-fit index is a proportion fit metric, whereas the RMSEA is a badness-of-fit index that is not in a proportion metric (West et al., 2012). The CFI compares the specified model to the null model. The null model assumes zero covariance among the observed variables; thus, the CFI indicates the ratio of improvement from the null to the specified model (Wang & Wang, 2019). The RMSEA assesses the lack of fit of the specified model to the population, adjusting for the model degrees of freedom. Additionally, the RMSEA provides a 90% confidence interval for the calculated RMSEA value (Wang & Wang, 2019). Using the Satorra-Bentler Chi-Square ($S-B\Delta\chi^2$) difference test, significant χ^2 difference test indicates that the imposed constraint leads to decrement in model fit and should be rejected. A non-significant χ^2 test will indicate that the model with imposed constraint fits the data comparably and more parsimoniously than the unconstrained model (Kelloway, 2014; Wang & Wang, 2019).

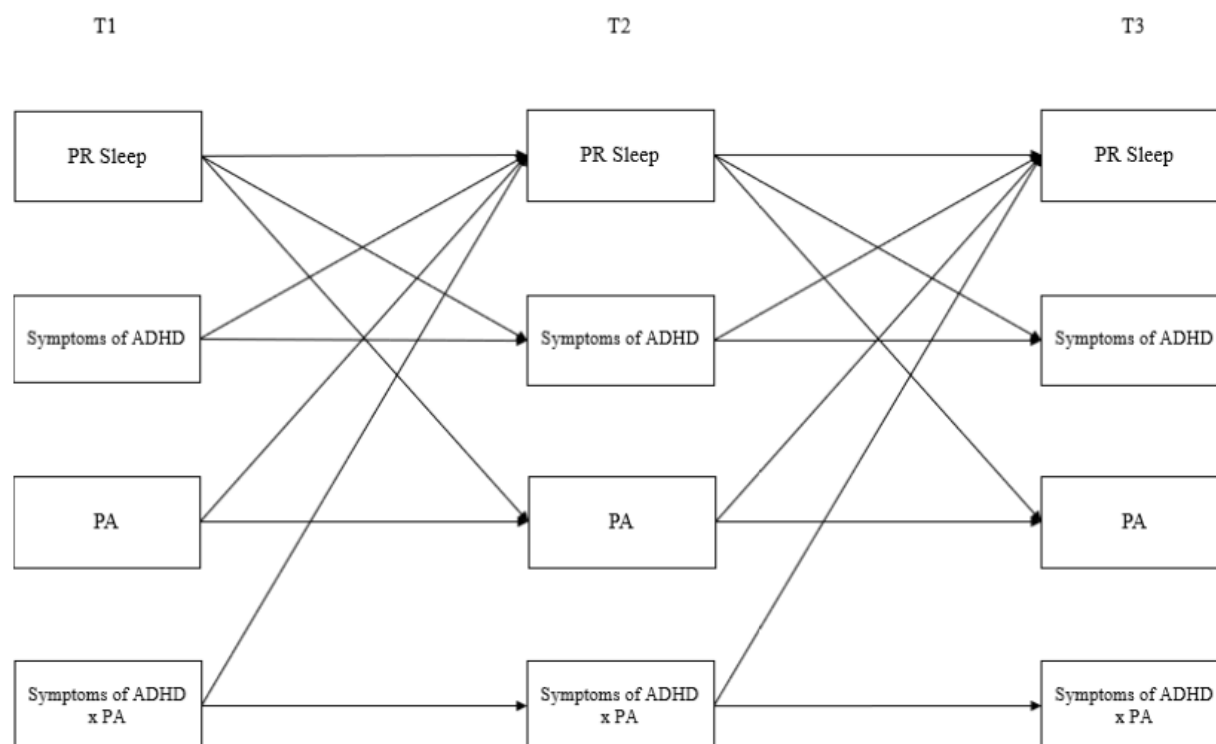
As previously noted, biological sex, pubertal development, and medication status were included as covariates as they are theoretically relevant and linked with the variables of interest. In addition, as there is evidence to support bidirectional associations between sleep and internalizing symptomology, sensitivity analyses were conducted as the final step for each of the

three models. Sensitivity analyses are conducted in order to establish the robustness or consistency of results under varying assumptions (Thabane et al., 2013). In the present study, sensitivity analyses were conducted to evaluate the degree to which effects in the sleep and internalizing models could withstand controlling for internalizing symptoms and sleep, respectively.

Aim 3: Using the baseline model that was established in Aim 2, the third aim assessed the potential interactive effect of total PA with ADHD symptoms for predicting sleep difficulties/internalizing symptoms. Following model-testing recommendations (e.g., Cohen et al., 2014), continuous variables were mean-centered prior to creating interaction terms to reduce multicollinearity and to aid in the interpretation of significant interactions. A centered predictor variable (i.e., Symptoms of ADHD), a centered moderator variable (i.e., PA) and a moderator X predictor interaction term (i.e., Symptoms of ADHD x PA) were entered at each of the time points. These variables were used to predict changes in the outcome variable, either parent-reported sleep difficulties (Figure 6), adolescent-reported sleep difficulties (Figure 7), adolescent-reported symptoms of depression (Figure 8), or adolescent-reported symptoms of anxiety (Figure 9) at each subsequent time point.

Figure 6

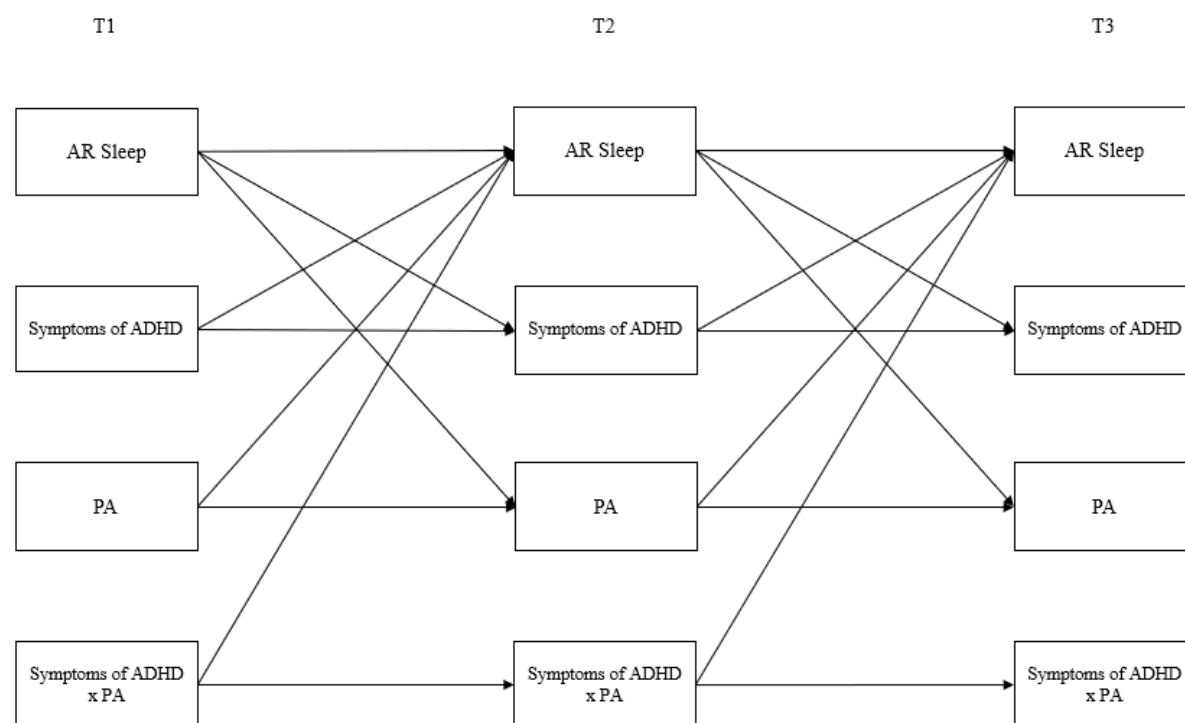
Example ARCL Panel Model Examining Longitudinal Associations Between Parent-Reported Sleep Difficulties, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. ARCL = autoregressive cross-lagged; PR = Parent-Reported; PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

Figure 7

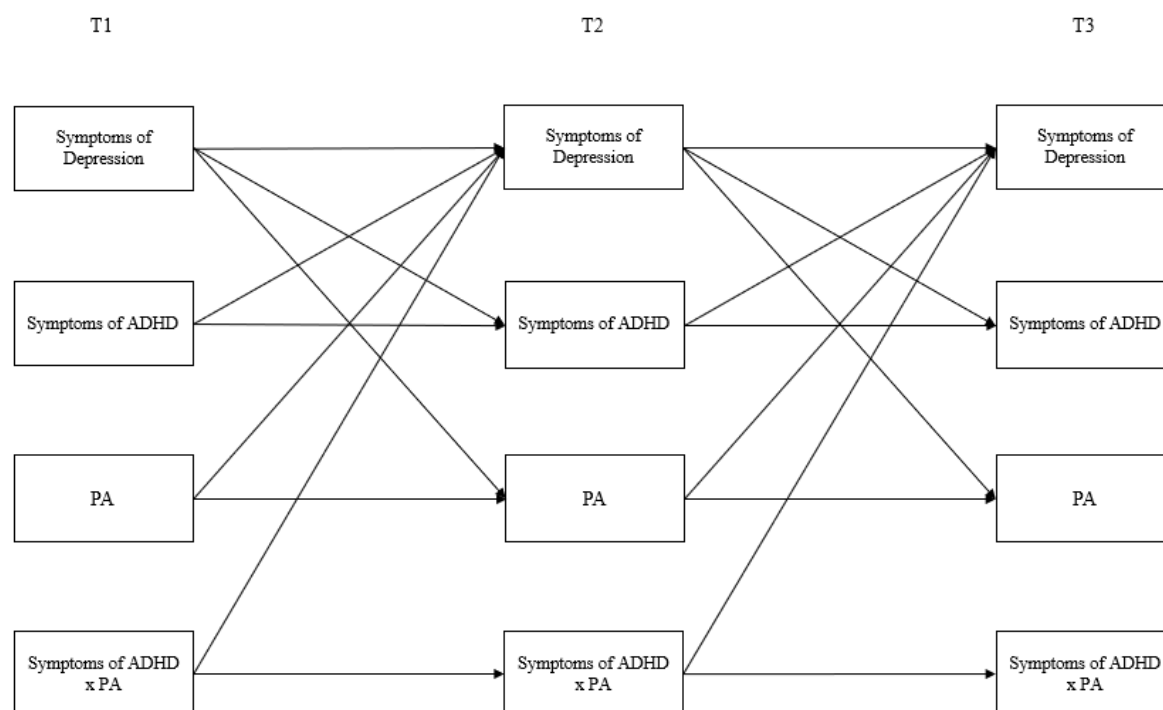
Example ARCL Panel Model Examining Longitudinal Associations Between Adolescent-Reported Sleep Difficulties, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. ARCL = autoregressive cross-lagged; AR = Adolescent-Reported; PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

Figure 8

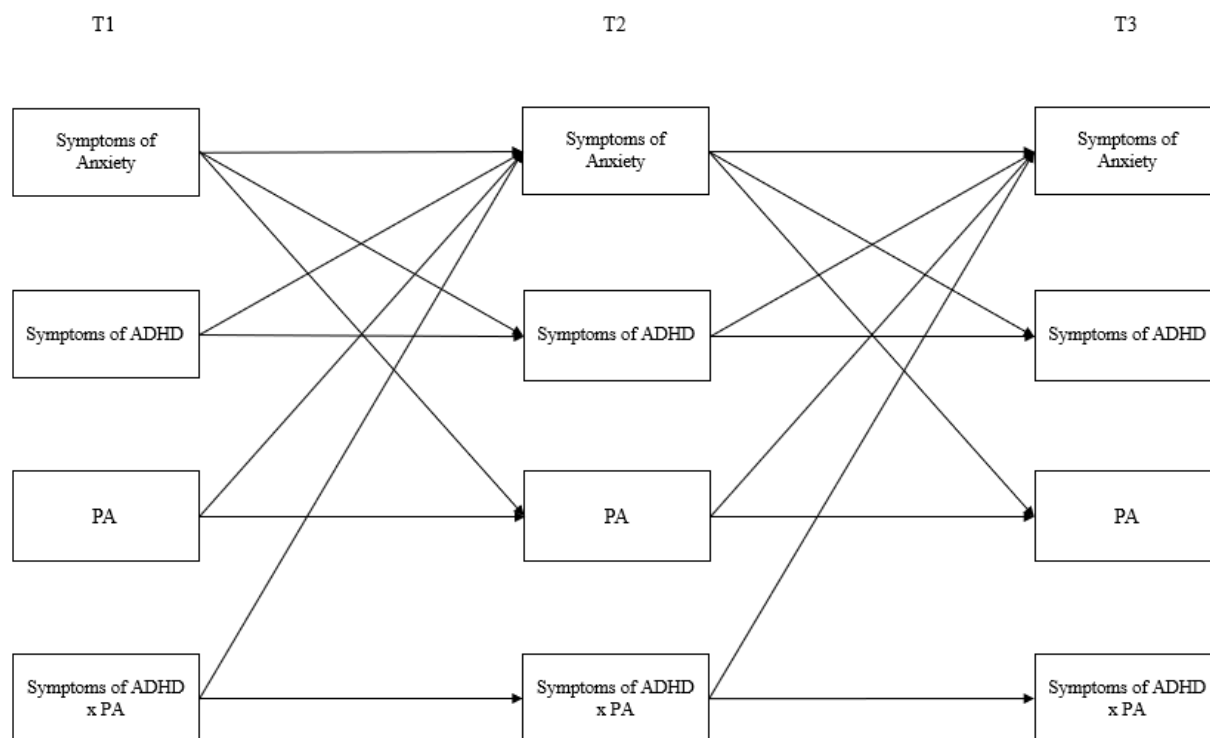
Example ARCL Panel Model Examining Longitudinal Associations Between Symptoms of Depression, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. ARCL = autoregressive cross-lagged; PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

Figure 9

Example ARCL Panel Model Examining Longitudinal Associations Between Symptoms of Anxiety, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. ARCL = autoregressive cross-lagged; PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable; Covariates and correlations between measures within each time point are included but not shown in the figure to reduce complexity.

These analyses also followed a series of hierarchically nested models for examining equality constraints for (1) the autoregressive (stability) path coefficients and (2) cross-lagged path coefficients, and evaluated χ^2 difference tests as well as comparison of model fit indices. Specifically, model constraints were tested for the interaction effect path coefficients. If the constrained model demonstrates a significant χ^2 difference test or decreased model fit, this indicates significant differences in the strength of associations between the interactive effect (Symptoms of ADHD x PA) and either sleep/internalizing symptoms across time. In the presence

of a significant interaction, subsequent computational tools would be used to plot and interpret the findings (Preacher et al., 2006). Specifically, the simple intercepts and simple slopes for the effects of the predictor on the outcome at specified values of the moderator (i.e., one standard deviation above and below the mean), as well as the region of significance tests would be examined. A visual plot of the interaction would be produced by imputing the resulting tables into the graphical user interface of SPSS (Bauer & Curran, 2005; Cohen et al., 2014). Sensitivity analyses would also be considered in the final step, which would determine the robustness of effects when controlling for internalizing symptoms or sleep (for the sleep and internalizing models, respectively).

Results

Missing Data

Extensive efforts were made to minimize attrition and missing data over the course of the longitudinal study. Overall, 90.0% of youth and 88.7% of parents from the initial sample provided data at all three assessment time points. There were two participants missing partial data at T1, although these participants had complete data at T2 and T3. Approximately 7.9% ($n = 24$) were missing data at T2, and approximately 11.3% ($n = 34$) were missing data at T3, with 4.6% ($n = 14$) missing data at both T2 and T3. Little's MCAR test for all primary study variables at all time points resulted in nonsignificant p -values, which demonstrated that data were missing in a random fashion.

In model estimation, missing data were handled through use of full-information maximum likelihood estimates, which is the default in Mplus for dealing with missing data (Graham et al., 2006; Wang & Wang, 2019). Maximum likelihood estimation methods can accommodate missing data, allowing analyses to make use of all available data so that any

participant with at least one time point can be included (Schafer & Graham, 2002). This procedure is more efficient and less biased in comparison to traditional approaches such as listwise or pairwise deletion (Arbuckle, 1996; Collins et al., 2001; Little & Rubin, 1989).

Descriptive Statistics – Primary Study Variables

Means, standard deviations, and normality statistics (i.e., skewness and kurtosis) for all primary study variables are presented in Table 2. Differences in adolescent- and parent-reported sleep difficulties between the ADHD and comparison group can be found in Becker and colleagues (2019). Assumption checks indicated that there were no violations of the homogeneity of variance assumption. Additionally, visual inspection of histograms and Q-Q scatterplots indicated normal distribution of variables. Nearly all correlations between PA, symptoms of ADHD, parent- and adolescent-reported sleep difficulties, and symptoms of depression and anxiety were statistically significant. For example, total PA was significantly negatively associated with parent- and adolescent-reported sleep difficulties, symptoms of ADHD, and symptoms of depression and anxiety at T1. Whereas, at T3, total PA was only significantly associated with adolescent-reported sleep difficulties and symptoms of depression and anxiety.

Table 2*Descriptive Statistics and Correlations Within Time Points for Primary Study Variables*

Variable	1	2	3	4	5	6	7	8	<i>M</i>	<i>SD</i>	α	Skew	Kurt
<i>Time point 1</i>													
1. Days of 20 mins PA/week	--	.83**	.68**	-.14*	-.19**	-.11	-.28**	-.19**	3.75	2.23	--	-0.13	-1.12
2. Days of 60 mins PA/week		--	.70**	-.18**	-.19**	-.13*	-.30**	-.25**	3.40	2.24	--	0.06	-1.20
3. Total PA			--	-.13*	-.17**	-.13*	-.23**	-.16**	8.80	5.17	.73	0.33	-0.53
4. Symptoms of ADHD				--	.45**	.31**	.26**	.17**	15.00	11.56	.95	0.54	-0.63
5. PR Sleep Difficulties					--	.35**	.31**	.22**	62.19	13.86	.77	0.90	0.28
6. AR Sleep Difficulties						--	.57**	.46**	17.14	5.63	.76	1.14	1.39
7. Symptoms of Depression							--	.74**	3.45	3.47	.82	1.34	1.74
8. Symptoms of Anxiety								--	6.75	6.37	.88	1.60	2.48
<i>Time point 2</i>													
1. Days of 20 mins PA/week	--	.89**	.71**	-.06	-.25**	-.13*	-.25**	-.25**	3.97	2.27	--	-0.28	-1.03
2. Days of 60 mins PA/week		--	.74**	-.07	-.25**	-.11	-.25**	-.27**	3.63	2.37	--	-0.13	-1.26
3. Total PA			--	-.06	-.18**	.41	-.25**	-.26**	8.13	5.14	.73	0.17	-0.95
4. Symptoms of ADHD				--	.35**	.21**	.18**	.17**	13.45	10.06	.95	0.87	0.42
5. PR Sleep Difficulties					--	.29**	.29**	.22**	60.32	12.24	.72	0.95	0.53
6. AR Sleep Difficulties						--	.47**	.26**	17.19	5.16	.72	1.48	5.39
7. Symptoms of Depression							--	.72**	2.71	3.27	.84	1.76	4.08
8. Symptoms of Anxiety								--	5.02	4.90	.83	1.51	2.56
<i>Time point 3</i>													
1. Days of 20 mins PA/week	--	.88**	.72**	-.03	-.09	-.07	-.18**	.14*	3.50	2.27	--	-0.05	-1.18
2. Days of 60 mins PA/week		--	.74**	-.07	-.13*	-.10	-.18**	-.17**	3.13	2.26	--	0.17	-1.14
3. Total PA			--	-.04	-.10	-.13*	-.20**	-.20**	6.96	4.85	.72	0.49	-0.43
4. Symptoms of ADHD				--	.38**	.26**	.22**	.23**	12.33	9.58	.94	1.06	1.05
5. PR Sleep Difficulties					--	.35**	.31**	.29**	60.03	12.21	.74	0.99	0.64
6. AR Sleep Difficulties						--	.57**	.46**	17.56	5.42	.73	0.84	0.36
7. Symptoms of Depression							--	.75**	4.05	4.36	.88	1.54	2.09
8. Symptoms of Anxiety								--	6.11	5.82	.86	1.57	2.73

Note. PA = Physical Activity; ADHD = Attention-Deficit/Hyperactivity Disorder; PR = Parent-Reported; AR = Adolescent-Reported; Skew = Skewness; Kurt = Kurtosis; Days of at least 20- and 60-minutes PA/week range = 0-7; Total PA range = 0-28; Symptoms of ADHD range = 0-54; PR Sleep Difficulties range = 38-100; AR Sleep Difficulties range = 10-50. Symptoms of Depression range = 0-32. Symptoms of Anxiety range = 0-56.

Descriptive Statistics – Covariates

Sex differences emerged on several of the primary study variables. Male participants had significantly higher symptoms of ADHD at all time points, significantly higher total PA scores at T1 and T2, and significantly more days engaging in at least 20 and at least 60 minutes of PA per week at T2. Female participants had significantly higher symptoms of depression (at all three time points) and significantly higher symptoms of anxiety (at T2 and T3). There were no significant sex differences at any time point on adolescent- or parent-reported sleep difficulties.

Significant differences also emerged on primary variables based on medication status. Youth taking medication for either attention, emotional/behavioral difficulties, or sleep had significantly higher symptoms of ADHD (at T1 and T2), parent-reported sleep difficulties (at T1 and T2), adolescent-reported sleep difficulties (at T1), and symptoms of depression (at T1 and T2). Participants not taking medication had significantly higher total PA scores (at T1 and T2) and more days engaging in at least 20 and at least 60 minutes of PA per week at T1. No significant differences emerged at T3 between youth who were taking medication versus those who were not.

Pubertal development was significantly correlated with a majority of the primary study variables. Pubertal development was negatively correlated with total PA at T1 and T3, negatively correlated with number of days engaging in at least 20 minutes of PA per week at T2, negatively correlated with symptoms of ADHD at all three time points, positively correlated with adolescent-reported sleep difficulties at T1 and T3, positively correlated with symptoms of depression at all time points, and positively correlated with symptoms of anxiety at T1 and T2. Pubertal development was not significantly correlated with parent-reported sleep difficulties.

Aim 1

T-tests were first conducted in order to assess whether there were differences in the mean number of days in which participants engaged in PA for at least 20 minutes, at least 60 minutes, and the total PA score based on ADHD status (i.e., ADHD group versus no ADHD comparison group) at each of the three time points (see Table 3).

Average number of days participating in at least 20 minutes of PA significantly differed based on ADHD status at T1, $t(297) = 2.43, p = .02$ and T2, $t(276) = 2.15, p = .03$. Effect sizes were small at T1 and T2, $d = .28$ and $.26$, respectively. At T1 and T2, the number of days engaged in at least 20 minutes of PA per week was significantly lower in the ADHD group compared to the comparison group. At T3, there were no significant differences.

Number of days participating in at least 60 minutes of PA was also examined, importantly as the US Department of Health and Human Services recommends that youth engage in at least 60 minutes of PA daily. At T1, when participants were in 8th grade, 11.0% of the total sample engaged in at least 60 minutes of PA every day of the week. Specifically, 12.1% of the comparison group compared to 10.0% of the ADHD group were meeting daily PA recommendations. At T2, when participants were in 9th grade, 15.1% of the total sample met daily PA recommendations, 16.3% of the comparison group and 14.0% of the ADHD group. At T3, when participants were in 10th grade, 9.9% of the total sample met daily PA recommendations, 9.8% of the comparison group and 10.1% of the ADHD group. Differences in the percentages of participants meeting recommendations between groups were not statistically significant. Average number of days participating in at least 60 minutes of PA, however, did significantly differ based on ADHD status at T1, $t(298) = 2.69, p = .008$, and T2, $t(276) = 2.25, p = .03$. Effect sizes were small to medium at T1 and T2, $d = .31$ and $.27$, respectively. At T1 and

T2, the mean number of days engaged in at least 60 minutes of PA per week was significantly lower in the ADHD group compared to the comparison group. At T3, there were no significant differences.

Finally, mean total PA score was examined. This is a composite score from the PAQ-A, with possible scores ranging from 0 to 28. In the present sample, scores ranged from 0 – 24 at T1, 0 – 20 at T2, and 0 – 22 at T3. The mean total PA score significantly differed based on ADHD status at T1 only, $t(298) = 2.15, p = .03$, with a small effect size, $d = .25$. Youth with ADHD had significantly lower total PA scores compared to youth in the comparison group.

Table 3

Means, Standard Deviations, and T-Test Results Across ADHD and Comparison Groups

	Comparison <i>M</i> ± <i>SD</i>	ADHD <i>M</i> ± <i>SD</i>	<i>t</i> Statistic	Cohen's <i>d</i> Effect Size
<i>Time point 1</i>	(<i>n</i> = 139)	(<i>n</i> = 160)		
Days of 20 mins PA/week	4.1 (2.1)	3.5 (2.3)	2.43*	.28
Days of 60 mins PA/week	3.8 (2.2)	3.1 (2.2)	2.69**	.31
Total PA	9.5 (5.0)	8.2 (5.3)	2.15*	.25
<i>Time point 2</i>	(<i>n</i> = 135)	(<i>n</i> = 143)		
Days of 20 mins PA/week	4.3 (2.2)	3.7 (2.3)	2.15*	.26
Days of 60 mins PA/week	4.0 (2.3)	3.3 (2.5)	2.25*	.27
Total PA	8.6 (5.1)	7.7 (5.2)	1.46	.18
<i>Time point 3</i>	(<i>n</i> = 133)	(<i>n</i> = 139)		
Days of 20 mins PA/week	3.7 (2.2)	3.3 (2.3)	1.58	.19
Days of 60 mins PA/week	3.4 (2.2)	2.9 (2.3)	1.69	.21
Total PA	7.4 (4.9)	6.5 (4.8)	1.55	.19

Note. ADHD = Attention-Deficit/Hyperactivity Disorder; PA = Physical Activity; Days of at least 20- and 60-minutes PA/week range = 0-7; Total PA range = 0-28.

* $p < .05$. ** $p < .01$. *** $p < .001$

In order to further understand differences in PA between the ADHD and comparison group, differences between ADHD presentations were analyzed. One-way ANOVAs assessed whether there were differences in the mean number of days in which participants engaged in PA for at least 20 minutes, at least 60 minutes, and the total PA score based on ADHD presentation

(i.e., ADHD-I, ADHD-C, versus no ADHD comparison group) at each of the three time points (see Table 4).

Number of days participating in at least 20 minutes of PA significantly differed based on ADHD presentation at T1, $F(2, 296) = 4.15, p = .02$, with a relatively small effect size, $\eta^2 = .03$. To determine which groups significantly differed, post hoc comparisons using the Tukey's HSD test were conducted. Significant differences emerged between the ADHD-I and comparison group only, in which youth in the ADHD-I group engaged in at least 20 minutes of PA on significantly fewer days per week than youth in the comparison group at T1. There were no significant differences between the ADHD-I and ADHD-C groups, or the ADHD-C and comparison groups on days of at least 20 minutes of PA at T1. One-way ANOVAs at T2 and T3 examining differences in number of days of 20 minutes of PA did not significantly differ between groups.

Next, when examining the number of days participating in at least 60 minutes of PA, there were significant differences based on ADHD presentation at T1, $F(2, 297) = 3.69, p = .03$, with a relatively small effect size, $\eta^2 = .02$. Tukey's HSD test again indicated that youth in the ADHD-I group engaged in at least 60 minutes of PA on significantly fewer days than youth in the comparison group at T1. There were no significant differences between the ADHD-I and ADHD-C groups, or the ADHD-C and comparison groups on the average number days of at least 60 minutes of PA at T1. One-way ANOVAs at T2 and T3 examining differences in number of days of 60 minutes of PA did not significantly differ between groups.

Finally, there were no significant presentation differences at any time point when examining total PA scores.

Table 4

Means, Standard Deviations, and One-Way ANOVA Results Across ADHD Presentations and Comparison Groups

	Comparison <i>M</i> ± <i>SD</i>	ADHD-I <i>M</i> ± <i>SD</i>	ADHD-C <i>M</i> ± <i>SD</i>	<i>F</i> Statistic	η^2 Effect Size
<i>Time point 1</i>	(<i>n</i> = 139)	(<i>n</i> = 118)	(<i>n</i> = 42)		
Days of 20 mins PA/week	4.1 (2.1)	3.3 (2.3)	3.9 (2.2)	4.15*	.03
Days of 60 mins PA/week	3.8 (2.2)	3.0 (2.3)	3.2 (2.1)	3.69*	.02
Total PA	9.5 (5.0)	8.0 (5.4)	8.7 (4.8)	2.57	.02
<i>Time point 2</i>	(<i>n</i> = 135)	(<i>n</i> = 108)	(<i>n</i> = 35)		
Days of 20 mins PA/week	4.3 (2.2)	3.7 (2.3)	3.7 (2.4)	2.32	.02
Days of 60 mins PA/week	4.0 (2.3)	3.3 (2.4)	3.3 (2.6)	2.51	.02
Total PA	8.6 (5.1)	7.4 (5.0)	8.7 (5.6)	2.05	.02
<i>Time point 3</i>	(<i>n</i> = 133)	(<i>n</i> = 104)	(<i>n</i> = 35)		
Days of 20 mins PA/week	3.7 (2.2)	3.1 (2.3)	3.8 (2.3)	2.45	.02
Days of 60 mins PA/week	3.4 (2.2)	2.8 (2.3)	3.3 (2.0)	2.31	.02
Total PA	7.4 (4.9)	6.1 (4.9)	7.7 (4.4)	2.60	.02

Note. ADHD-I = Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive

Presentation; ADHD-C = Attention-Deficit/Hyperactivity Disorder, Combined Presentation; PA = Physical Activity; Days of at least 20- and 60-minutes PA/week range = 0-7; Total PA range = 0-28.

* $p < .05$. ** $p < .01$. *** $p < .001$

Finally, sex differences between groups were also explored (see Table 5). Boys without ADHD were significantly more likely to engage in PA than boys with ADHD on all variables at T1 and T2 (d s ranging from .33 to .54). No significant differences based on ADHD/comparison status between boys emerged at T3. When comparing PA variables at all time points between girls with and without ADHD, girls without ADHD had significantly higher total PA scores than girls with ADHD at T1 only. No other significant differences emerged between girls with versus without ADHD.

Table 5

Means, Standard Deviations, and Sex Difference T-Test Results Between ADHD and Comparison Groups

<i>Time point 1</i>	Boys <i>M ± SD</i>		<i>t</i>	<i>d</i>	Girls <i>M ± SD</i>		<i>t</i>	<i>d</i>
	ADHD (<i>n</i> = 104)	Comparison (<i>n</i> = 61)			ADHD (<i>n</i> = 56)	Comparison (<i>n</i> = 78)		
Days of 20 mins PA/week	3.6 (2.3)	4.7 (1.8)	3.36***	.51	3.3 (2.4)	3.6 (2.2)	0.96	.17
Days of 60 mins PA/week	3.2 (2.3)	4.2 (2.1)	2.94**	.47	2.9 (2.1)	3.4 (2.2)	1.36	.24
Total PA	9.1 (5.4)	10.8 (4.8)	2.07*	.33	6.6 (4.7)	8.4 (4.9)	2.19*	.38
<i>Time point 2</i>	(<i>n</i> = 89)	(<i>n</i> = 61)			(<i>n</i> = 54)	(<i>n</i> = 74)		
Days of 20 mins PA/week	3.8 (2.4)	5.0 (1.9)	3.19**	.51	3.4 (2.3)	3.7 (2.2)	0.66	.12
Days of 60 mins PA/week	3.5 (2.5)	4.6 (2.0)	3.05**	.49	3.1 (2.3)	3.5 (2.3)	0.80	.14
Total PA	7.7 (5.3)	10.4 (4.6)	3.24**	.54	7.7 (5.1)	7.1 (4.7)	0.66	.12
<i>Time point 3</i>	(<i>n</i> = 90)	(<i>n</i> = 58)			(<i>n</i> = 49)	(<i>n</i> = 75)		
Days of 20 mins PA/week	3.3 (2.4)	4.1 (2.3)	1.94	.33	3.3 (2.2)	3.5 (2.1)	0.41	.08
Days of 60 mins PA/week	2.9 (2.3)	3.6 (2.3)	1.64	.28	2.8 (2.1)	3.2 (2.2)	0.91	.17
Total PA	7.0 (5.0)	8.2 (5.0)	1.37	.23	5.6 (4.3)	6.8 (4.7)	1.49	.27

Note. ADHD = Attention-Deficit/Hyperactivity Disorder; PA = Physical Activity; Days of at least 20-

and 60-minutes PA/week range = 0-7; Total PA range = 0-28.

* $p < .05$. ** $p < .01$. *** $p < .001$

Aim 2

Bidirectional Associations Between Parent-Reported Sleep Difficulties, Symptoms of

ADHD, and PA. A series of analyses in Mplus 8.8 were conducted to assess associations

between parent-reported sleep difficulties, symptoms of ADHD, and total PA across T1 to T3

(see Table 6). The models included sex, pubertal development, and medication status as

covariates. An unconstrained model was first run where all path coefficients were allowed to

vary across T1 to T3. This model fit the data well, $\chi^2(32) = 70.98$, $p < .001$, CFI = .968, RMSEA

= .064, 90% CI [.044, .084]. The unconstrained model was then compared to each of the

following constrained models in which autoregressive path coefficients were constrained to be

equal across T1 to T3 for: a) parent-reported sleep difficulties, b) symptoms of ADHD, and c)

PA. Comparisons included the Satorra-Bentler Chi-Square difference test ($S-B\Delta\chi^2$) and fit indices (i.e., CFI and RMSEA).

For parent-reported sleep difficulties, the constrained model (i.e., with path coefficients were constrained to be equal between adjacent time points assessing sleep difficulties) resulted in a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.55, p = .46$) and slight improvement in fit indices. This finding suggested the stability of parent-reported sleep difficulties over time. Parent-reported sleep difficulties at T1 were associated with parent-reported sleep difficulties at T2 ($\beta = .630, p < .001$), and from T2 to T3 ($\beta = .520, p < .001$). For symptoms of ADHD, the constrained model also resulted in a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 3.23, p = .07$), indicating stability in symptoms of ADHD over time. Symptoms of ADHD were significantly associated between time points 1 and 2 ($\beta = .802, p < .001$), and between time points 2 and 3 ($\beta = .728, p < .001$). Finally, the constrained model for the PA autoregressive paths resulted a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.04, p = .85$) with little change in fit indices. PA at T1 was significantly associated with PA at T2 ($\beta = .378, p < .001$) and similarly between T2 and T3 ($\beta = .404, p < .001$).

This model (i.e., with autoregressive paths constrained for parent-reported sleep difficulties, symptoms of ADHD, and PA) was then compared to a series of models in which the following cross-lagged paths were tested for equality constraints: d) symptoms of ADHD to parent-reported sleep difficulties, e) parent-reported sleep difficulties to symptoms of ADHD, f) PA to parent-reported sleep difficulties, g) parent-reported sleep difficulties to PA. The cross-lagged path constraints for symptoms of ADHD to parent-reported sleep difficulties was supported based on a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.65, p = .42$), and little change in fit indices, suggesting that constraining these cross-lagged path coefficients did not

significantly decrease model fit. Cross-lagged path constraints for parent-reported sleep difficulties to symptoms of ADHD showed a significant decrease in fit, as indicated by a significant χ^2 difference test, (S-B $\Delta\chi^2$ (1) = 5.61, p = .02), decreases in CFI from .969 to .965, and increases in RMSEA from .060 to .063. Therefore, this equality constraint was rejected and not carried forward in the next step. The cross-lagged path constraints for PA to parent-reported sleep difficulties was supported based on non-significant χ^2 difference tests, (S-B $\Delta\chi^2$ (1) = 0.39, p = .53) and improvements in fit indices. Finally, the constrained cross-lagged path from parent-reported sleep difficulties to PA was not supported, as evidenced by a significant χ^2 difference test, (S-B $\Delta\chi^2$ (1) = 4.11, p = .04), a decreased CFI from .970 to .967, and an increase in RMSEA from .058 to .060. Thus, in the final model, cross-lagged paths between parent-reported sleep difficulties to symptoms of ADHD and parent-reported sleep difficulties to PA were left unconstrained (i.e., freely estimated across time points). All other autoregressive and cross-lagged paths were constrained. The final model fit the data well, $\chi^2(37)$ = 74.43, p < .001, CFI = .970, RMSEA = .058, 90% CI [.039, .078]. All standardized paths for this final model are presented in Figure 10. Parent-reported sleep difficulties significantly predicted increases in symptoms of ADHD between T2 and T3 (β = .068, p = .04). No other cross-lagged paths were significant predictors at any of the time points.

Table 6

Comparison of Model Constraints for the Longitudinal Associations Between Parent-Reported Sleep

Difficulties, Symptoms of ADHD, and PA

Model		χ^2	df	CFI	RMSEA	CI	χ^2_Δ	Decision
<i>Unconstrained</i>		70.98	32	.968	.064	[.044, .084]	--	
<i>Auto-regressive paths</i>	a. PR Sleep	71.44	33	.969	.063	[.043, .083]	$\chi^2_\Delta(1) = 0.55, p = .46$	Retain
	b. ADHD	74.57	34	.967	.063	[.044, .083]	$\chi^2_\Delta(1) = 3.23, p = .07$	Retain
	c. PA	74.29	35	.968	.062	[.042, .081]	$\chi^2_\Delta(1) = 0.04, p = .85$	Retain
<i>Cross-lagged paths</i>	d. ADHD \rightarrow PR Sleep	74.64	36	.969	.060	[.041, .080]	$\chi^2_\Delta(1) = 0.65, p = .42$	Retain
	e. PR Sleep \rightarrow ADHD	80.83	37	.965	.063	[.044, .082]	$\chi^2_\Delta(1) = 5.61, p = .02$	Reject
	f. PA \rightarrow PR Sleep	74.43	37	.970	.058	[.039, .078]	$\chi^2_\Delta(1) = 0.39, p = .53$	Retain
	g. PR Sleep \rightarrow PA	78.82	38	.967	.060	[.041, .079]	$\chi^2_\Delta(1) = 4.11, p = .04$	Reject

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

Approximation; PR Sleep = Parent-Reported Sleep Difficulties; ADHD = Symptoms of Attention-

Deficit/Hyperactivity Disorder; PA = Total Physical Activity; Models presented above are

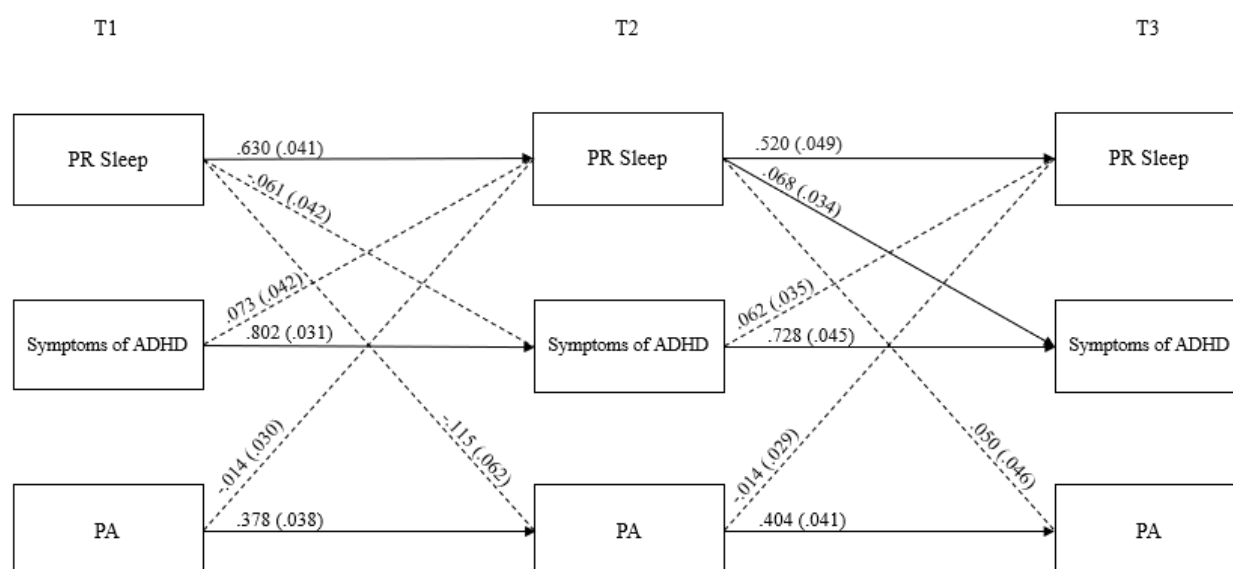
hierarchically nested, that is, constraints that are retained (or rejected) at initial steps are retained (or

rejected) for the subsequent models. The first model is the unconstrained (base) model. The

subsequent models test constraints on the autoregressive paths, and then the cross-lagged paths.

Figure 10

Longitudinal Associations Between Parent-Reported Sleep Difficulties, Symptoms of ADHD, and Total PA



Note. PR = Parent-Reported; PA = Physical Activity; Final model fit: $\chi^2(37) = 74.43, p < .001$, CFI = .970, RMSEA = .058, 90% CI [.039, .078]; Betas (β) are shown and standard errors are in parentheses. Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

Bidirectional Associations Between Adolescent-Reported Sleep Difficulties, Symptoms of ADHD, and PA. Another series of analyses were conducted to assess associations between adolescent-reported sleep difficulties, symptoms of ADHD, and total PA across T1 to T3 (see Table 7). The models included sex, pubertal development, and medication status as covariates. An unconstrained model was first run where all path coefficients were allowed to vary across T1 to T3. This model fit the data very well, $\chi^2(32) = 51.10, p = .02$, CFI = .979, RMSEA = .045, 90% CI [.019, .067]. The unconstrained model was then compared to each of

the following constrained models in which autoregressive path coefficients were constrained to be equal across T1 to T3 for: a) adolescent-reported sleep difficulties, b) symptoms of ADHD, and c) PA. Comparisons included the Satorra-Bentler Chi-Square difference test ($S-B\Delta\chi^2$) and fit indices (i.e., CFI and RMSEA).

For adolescent-reported sleep difficulties, the constrained model (i.e., with path coefficients were constrained to be equal between adjacent time points assessing sleep difficulties) resulted in a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.003, p = .96$) and fit indices improved. This finding suggested the stability of adolescent-reported sleep difficulties over time. Adolescent-reported sleep difficulties at T1 were associated with adolescent-reported sleep difficulties at T2 ($\beta = .425, p < .001$), and from T2 to T3 ($\beta = .386, p < .001$). Consistent with the prior model, constraining the autoregressive paths for symptoms of ADHD and PA resulted in non-significant χ^2 difference tests, ($S-B\Delta\chi^2 (1) = 1.73, p = .19$ and $S-B\Delta\chi^2 (1) = 0.02, p = .89$, respectively). Again, symptoms of ADHD and PA were stable over time.

This model (i.e., with autoregressive paths constrained for adolescent-reported sleep difficulties, symptoms of ADHD, and PA) was then compared to a series of models in which the following cross-lagged paths were tested for equality constraints: d) symptoms of ADHD to adolescent-reported sleep difficulties, e) adolescent-reported sleep difficulties to symptoms of ADHD, f) PA to adolescent-reported sleep difficulties, g) adolescent-reported sleep difficulties to PA. The cross-lagged path constraints for symptoms of ADHD to adolescent-reported sleep difficulties was supported based on a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.02, p = .90$), and improvements in fit indices, suggesting that constraining these cross-lagged path coefficients did not significantly decrease model fit. Cross-lagged path constraints for adolescent-reported sleep difficulties to symptoms of ADHD was also supported based on non-

significant χ^2 difference tests, ($S-B\Delta\chi^2(1) = 0.49, p = .48$) and little change in fit indices. The cross-lagged path constraints for PA to adolescent-reported sleep difficulties showed a significant decrease in fit as indicated by a significant χ^2 difference test, ($S-B\Delta\chi^2(1) = 4.23, p = .04$), decreases in CFI from .985 to .982, and increases in RMSEA from .035 to .038. Therefore, this equality constraint was rejected and not carried forward in the next step. Finally, the constrained cross-lagged path from adolescent-reported sleep difficulties to PA was supported by a non-significant χ^2 difference test, ($S-B\Delta\chi^2(1) = 1.11, p = .29$). Thus, in the final model, cross-lagged paths between PA and adolescent-reported sleep difficulties were left unconstrained (i.e., freely estimated across time points). All other autoregressive and cross-lagged paths were constrained. The final model fit the data very well, $\chi^2(38) = 50.59, p = .08$, CFI = .986, RMSEA = .033, 90% CI [.000, .056], with improvements in fit from the fully unconstrained model. All standardized paths for this final model are presented in Figure 11. Symptoms of ADHD significantly predicted adolescent-reported sleep problems between T1 and T2 ($\beta = .093, p = .04$) and between T2 and T3 ($\beta = .076, p = .04$). PA at T2 significantly predicted adolescent-reported sleep problems at ($\beta = -.129, p = .008$). No other cross-lagged paths were significant predictors at any of the time points.

Table 7

Comparison of Model Constraints for the Longitudinal Associations Between Adolescent-Reported Sleep Difficulties, Symptoms of ADHD, and PA

Model		χ^2	df	CFI	RMSEA	CI	χ^2	Decision
<i>Unconstrained</i>		51.10	32	.979	.045	[.019, .067]	--	
<i>Auto-regressive paths</i>	a. AR Sleep	47.94	33	.984	.039	[.006, .062]	$\chi^2_{\Delta}(1) = 0.003, p = .96$	Retain
	b. ADHD	49.63	34	.983	.039	[.008, .062]	$\chi^2_{\Delta}(1) = 1.73, p = .19$	Retain
	c. PA	49.74	35	.984	.038	[.000, .060]	$\chi^2_{\Delta}(1) = 0.02, p = .89$	Retain
<i>Cross-lagged paths</i>	d. ADHD \rightarrow AR Sleep	49.72	36	.985	.036	[.000, .058]	$\chi^2_{\Delta}(1) = 0.02, p = .90$	Retain
	e. AR Sleep \rightarrow ADHD	50.23	37	.985	.035	[.000, .057]	$\chi^2_{\Delta}(1) = 0.49, p = .48$	Retain
	f. PA \rightarrow AR Sleep	54.28	38	.982	.038	[.008, .060]	$\chi^2_{\Delta}(1) = 4.23, p = .04$	Reject
	g. AR Sleep \rightarrow PA	50.59	38	.986	.033	[.000, .056]	$\chi^2_{\Delta}(1) = 1.11, p = .29$	Retain

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

Approximation; AR Sleep = Adolescent-Reported Sleep Difficulties; ADHD = Symptoms of

Attention-Deficit/Hyperactivity Disorder; PA = Total Physical Activity; Models presented above are

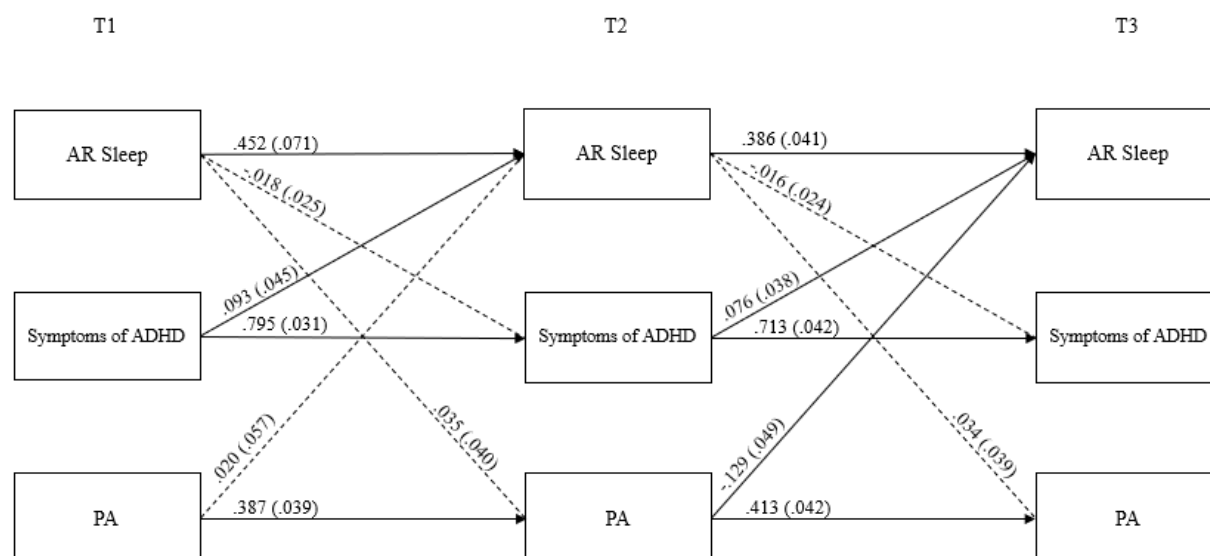
hierarchically nested, that is, constraints that are retained (or rejected) at initial steps are retained (or

rejected) for the subsequent models. The first model is the unconstrained (base) model. The

subsequent models test constraints on the autoregressive paths, and then the cross-lagged paths.

Figure 11

Longitudinal Associations Between Adolescent-Reported Sleep Difficulties, Symptoms of ADHD, and Total PA



Note. AR = Adolescent-Reported; PA = Physical Activity; Final model fit: $\chi^2(38) = 50.59$, $p = .08$, CFI = .986, RMSEA = .033, 90% CI [.000, .056]; Betas (β) are shown and standard errors are in parentheses. Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

Bidirectional Associations Between Symptoms of Depression, Symptoms of ADHD, and PA. A similar series of analyses were conducted to assess associations between symptoms of depression, symptoms of ADHD, and total PA across T1 to T3 (see Table 8). The models also included sex, pubertal development, and medication status as covariates. An unconstrained model was first run where all path coefficients were allowed to vary across T1 to T3. This model fit the data well, $\chi^2(32) = 60.07$, $p < .001$, CFI = .968, RMSEA = .059, 90% CI [.038, .080]. The unconstrained model was then compared to each of the following constrained models in which autoregressive path coefficients were constrained to be equal across T1 to T3 for: a) symptoms

of depression, b) symptoms of ADHD, and c) PA. Comparisons included the Satorra-Bentler Chi-Square difference test ($S-B\Delta\chi^2$) and fit indices (i.e., CFI and RMSEA).

For symptoms of depression, the constrained model resulted in a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.01, p = .94$) and fit indices improved. This finding suggested the stability of symptoms of depression over time. Symptoms of depression at T1 were associated with symptoms of depression at T2 ($\beta = .583, p < .001$), and from T2 to T3 ($\beta = .421, p < .001$). As with prior models, constraining the autoregressive paths for symptoms of ADHD and PA resulted in non-significant χ^2 difference tests, ($S-B\Delta\chi^2 (1) = 1.51, p = .22$ and $S-B\Delta\chi^2 (1) = 0.01, p = .93$, respectively), indicating stability in these variables over time.

This model (i.e., with autoregressive paths constrained for symptoms of depression, symptoms of ADHD, and PA) was then compared to a series of models in which the following cross-lagged paths were tested for equality constraints: d) symptoms of ADHD to symptoms of depression, e) symptoms of depression to symptoms of ADHD, f) PA to symptoms of depression, g) symptoms of depression to PA. The cross-lagged path constraints for symptoms of ADHD to symptoms of depression was supported based on a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.01, p = .91$), and little change in fit indices, suggesting that constraining these cross-lagged path coefficients did not significantly decrease model fit. Cross-lagged path constraints for symptoms of depression to symptoms of ADHD also did not show a significant decrease in fit, ($S-B\Delta\chi^2 (1) = 0.19, p = .66$). Finally, the cross-lagged path constraints for PA to symptoms of depression and symptoms of depression to PA were both supported based on non-significant χ^2 difference tests, ($S-B\Delta\chi^2 (1) = 0.002, p = .97$ and $S-B\Delta\chi^2 (1) = 0.88, p = .35$, respectively) and further improvements in model fit. Thus, in the final model, all autoregressive and cross-lagged paths were constrained. The final model fit the data very well, $\chi^2(39) = 66.80, p$

= .004, CFI = .973, RMSEA = .049, 90% CI [.028, .069], with improvements in fit from the fully unconstrained model. All standardized paths for this final model are presented in Figure 12. PA negatively predicted symptoms of depression between T1 and T2 ($\beta = -.133, p < .001$) and T2 and T3 ($\beta = -.100, p < .001$). No other cross-lagged paths were significant predictors at any of the time points.

Table 8

Comparison of Model Constraints for the Longitudinal Associations Between Symptoms of Depression, Symptoms of ADHD, and PA

Model		χ^2	df	CFI	RMSEA	CI	χ^2	Decision
<i>Unconstrained</i>		60.07	32	.968	.059	[.038, .080]	--	
<i>Auto-regressive paths</i>	a. Dep	63.98	33	.970	.056	[.035, .077]	$\chi^2_{\Delta}(1) = 0.01, p = .94$	Retain
	b. ADHD	65.54	34	.970	.056	[.035, .076]	$\chi^2_{\Delta}(1) = 1.51, p = .23$	Retain
	c. PA	65.43	35	.971	.054	[.033, .074]	$\chi^2_{\Delta}(1) = 0.01, p = .93$	Retain
<i>Cross-lagged paths</i>	d. ADHD \rightarrow Dep	65.89	36	.971	.053	[.032, .073]	$\chi^2_{\Delta}(1) = 0.01, p = .91$	Retain
	e. Dep \rightarrow ADHD	65.78	37	.972	.051	[.030, .071]	$\chi^2_{\Delta}(1) = 0.19, p = .66$	Retain
	f. PA \rightarrow Dep	65.74	38	.973	.050	[.028, .069]	$\chi^2_{\Delta}(1) = 0.002, p = .97$	Retain
	g. Dep \rightarrow PA	66.80	39	.973	.049	[.028, .069]	$\chi^2_{\Delta}(1) = 0.88, p = .35$	Retain

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

Approximation; Dep = Symptoms of Depression; ADHD = Symptoms of Attention-

Deficit/Hyperactivity Disorder; PA = Total Physical Activity; Models presented above are

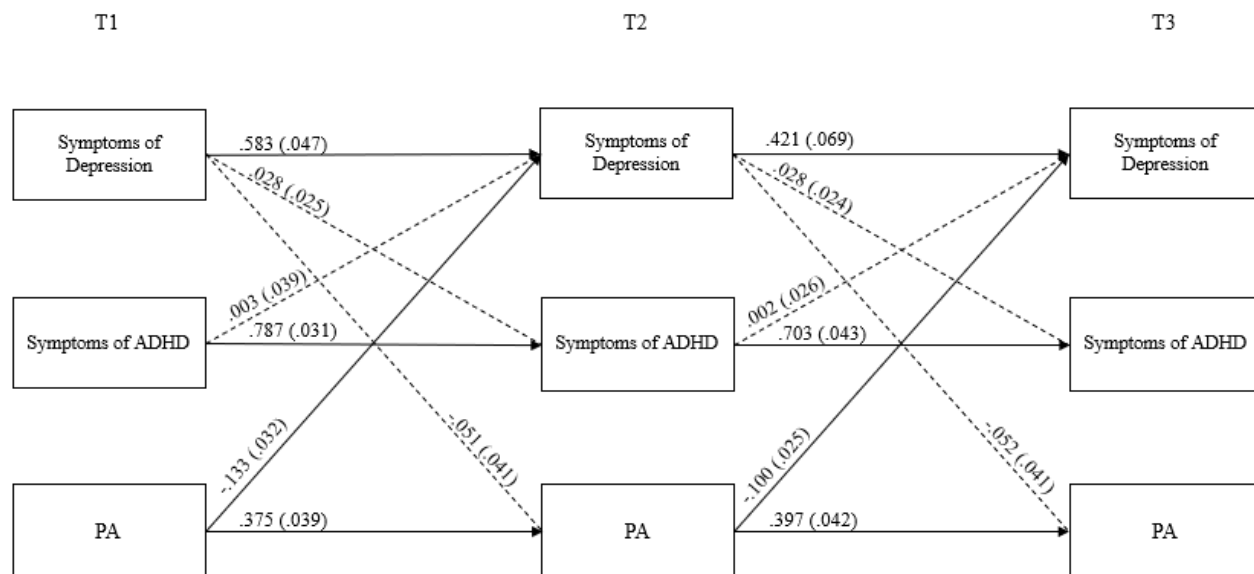
hierarchically nested, that is, constraints that are retained (or rejected) at initial steps are retained (or

rejected) for the subsequent models. The first model is the unconstrained (base) model. The

subsequent models test constraints on the autoregressive paths, and then the cross-lagged paths.

Figure 12

Longitudinal Associations Between Symptoms of Depression, Symptoms of ADHD, and Total PA



Note. PA = Physical Activity; Final model fit: $\chi^2(39) = 66.80$, $p = .004$, CFI = .973, RMSEA = .049, 90% CI [.028, .069]; Betas (β) are shown and standard errors are in parentheses.

Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

Bidirectional Associations Between Symptoms of Anxiety, Symptoms of ADHD, and PA. Finally, analyses were conducted to assess associations between symptoms of anxiety, symptoms of ADHD, and total PA across T1 to T3 (see Table 9). The models also included sex, pubertal development, and medication status as covariates. An unconstrained model was first run where all path coefficients were allowed to vary across T1 to T3. This model fit the data well, $\chi^2(32) = 74.29$, $p < .001$, CFI = .960, RMSEA = .067, 90% CI: [.047, .087]. The unconstrained model was then compared to each of the following constrained models in which autoregressive path coefficients were constrained to be equal across T1 to T3 for: a) symptoms of anxiety, b)

symptoms of ADHD, and c) PA. Comparisons included the Satorra-Bentler Chi-Square difference test ($S-B\Delta\chi^2$) and fit indices (i.e., CFI and RMSEA).

For symptoms of anxiety, the constrained model resulted in a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 2.37, p = .12$) and little change in fit indices. This finding suggested the stability of symptoms of anxiety over time. Symptoms of anxiety at T1 were associated with symptoms of anxiety at T2 ($\beta = .592, p < .001$), and from T2 to T3 ($\beta = .419, p < .001$). Consistent with prior models, constraining the autoregressive paths for symptoms of ADHD and PA resulted in non-significant χ^2 difference tests, ($S-B\Delta\chi^2 (1) = 1.52, p = .22$ and $S-B\Delta\chi^2 (1) = 0.06, p = .80$, respectively), indicating stability in these variables over time.

This model (i.e., with autoregressive paths constrained for symptoms of anxiety, symptoms of ADHD, and PA) was then compared to a series of models in which the following cross-lagged paths were tested for equality constraints: d) symptoms of ADHD to symptoms of anxiety, e) symptoms of anxiety to symptoms of ADHD, f) PA to symptoms of anxiety, g) symptoms of anxiety to PA. The cross-lagged path constraints for symptoms of ADHD to symptoms of anxiety was supported based on a non-significant χ^2 difference test, ($S-B\Delta\chi^2 (1) = 0.10, p = .32$), and little change in fit indices, suggesting that constraining these cross-lagged path coefficients did not significantly decrease model fit. Cross-lagged path constraints for symptoms of anxiety to symptoms of ADHD also did not show a significant decrease in fit, ($S-B\Delta\chi^2 (1) = 1.11, p = .29$). Finally, the cross-lagged path constraints for PA to symptoms of anxiety and symptoms of anxiety to PA were both supported based on non-significant χ^2 difference tests, ($S-B\Delta\chi^2 (1) = 0.001, p = .97$ and $S-B\Delta\chi^2 (1) = 0.29, p = .59$, respectively) and further improvements in model fit. Thus, in the final model, all autoregressive and cross-lagged paths were constrained. The final model fit the data well, $\chi^2(39) = 81.12, p < .001$, CFI = .961,

RMSEA = .060, 90% CI [.042, .079]. All standardized paths for this final model are presented in Figure 13. PA negatively predicted symptoms of anxiety between T1 and T2 ($\beta = -.123, p < .001$) and T2 and T3 ($\beta = -.108, p < .001$). Additionally, symptoms of anxiety positively predicted symptoms of ADHD between T1 and T2 ($\beta = .058, p = .02$) and T2 and T3 ($\beta = .048, p = .02$). All other cross-lagged paths were not significant predictors at any of the time points.

Table 9

Comparison of Model Constraints for the Longitudinal Associations Between Symptoms of Anxiety, Symptoms of ADHD, and PA

Model		χ^2	df	CFI	RMSEA	CI	χ^2	Decision
<i>Unconstrained</i>		74.29	32	.960	.067	[.047, .087]	--	
<i>Auto-regressive paths</i>	a. Anx	76.69	33	.959	.067	[.047, .087]	$\chi^2_{\Delta}(1) = 2.37, p = .12$	Retain
	b. ADHD	78.28	34	.958	.066	[.047, .086]	$\chi^2_{\Delta}(1) = 1.52, p = .22$	Retain
	c. PA	78.14	35	.960	.065	[.045, .084]	$\chi^2_{\Delta}(1) = 0.06, p = .80$	Retain
<i>Cross-lagged paths</i>	d. ADHD \rightarrow Anx	79.22	36	.959	.064	[.045, .083]	$\chi^2_{\Delta}(1) = 0.10, p = .32$	Retain
	e. Anx \rightarrow ADHD	80.59	37	.959	.063	[.044, .082]	$\chi^2_{\Delta}(1) = 1.11, p = .29$	Retain
	f. PA \rightarrow Anx	80.66	38	.960	.062	[.043, .080]	$\chi^2_{\Delta}(1) = 0.001, p = .97$	Retain
	g. Anx \rightarrow PA	81.12	39	.961	.060	[.042, .079]	$\chi^2_{\Delta}(1) = 0.29, p = .59$	Retain

Note. CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; df =

degrees of freedom; Anx = Symptoms of Anxiety; ADHD = Symptoms of Attention-

Deficit/Hyperactivity Disorder; PA = Total Physical Activity; Models presented above are

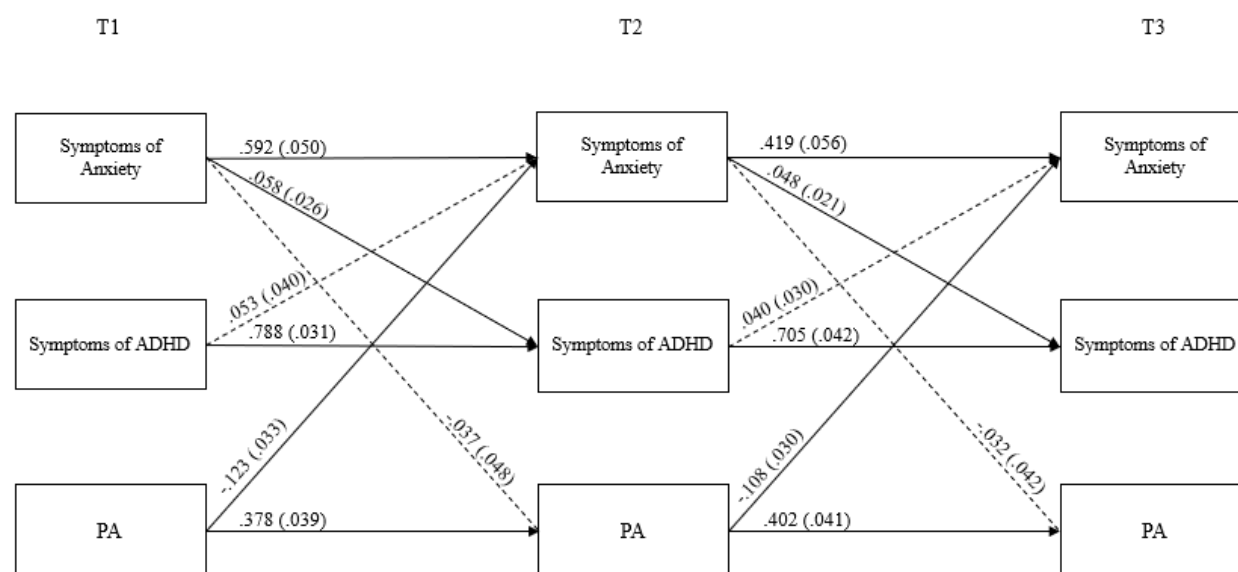
hierarchically nested, that is, constraints that are retained (or rejected) at initial steps are retained (or

rejected) for the subsequent models. The first model is the unconstrained (base) model. The

subsequent models test constraints on the autoregressive paths, and then the cross-lagged paths.

Figure 13

Longitudinal Associations Between Symptoms of Anxiety, Symptoms of ADHD, and Total PA



Note. PA = Physical Activity; Final model fit: $\chi^2(39) = 81.12, p < .001$, CFI = .961, RMSEA = .060, 90% CI [.042, .079]; Betas (β) are shown and standard errors are in parentheses.

Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

Sensitivity Analyses. In order to evaluate the degree to which sleep difficulties and symptoms of depression and anxiety effect the significant findings, sensitivity analyses were conducted. First, the variable for symptoms of depression was added as a covariate into the final model for longitudinal associations between parent-reported sleep difficulties, symptoms of ADHD, and PA. The significant cross-lag path established in the final model (i.e., parent-reported sleep difficulties at T2 predicting symptoms of ADHD at T3) was no longer significant following the inclusion of the additional covariate. Then, the variable for symptoms of anxiety

was added as a covariate into the model (without symptoms of depression included). Again, the previously significant path was no longer significant.

Symptoms of depression and then symptoms of anxiety were added as covariates into the final model for longitudinal associations between adolescent-reported sleep difficulties, symptoms of ADHD, and PA. The significant cross-lag paths established in the final model (i.e., symptoms of ADHD at T1 [and T2] predicting adolescent-reported sleep difficulties at T2 [and T3] and PA at T2 predicting adolescent-reported sleep difficulties at T3) were no longer significant following the inclusion of each of the additional covariates.

Next, variables for parent-reported sleep difficulties, then adolescent-reported sleep difficulties, and then symptoms of anxiety were each added as covariates into the final model for longitudinal associations between symptoms of depression, symptoms of ADHD, and PA. Significant cross lagged paths (i.e., PA at T1 [and T2] predicting symptoms of depression at T2 [and T3]) were maintained following the inclusion of each of the additional covariates.

Finally, variables for parent-reported sleep difficulties, then adolescent-reported sleep difficulties, and then symptoms of depression were each added as covariates into the final model for longitudinal associations between symptoms of anxiety, symptoms of ADHD, and PA. The significant cross-lag paths between symptoms of anxiety at T1 (and T2) predicting symptoms of ADHD at T2 (and T3) were no longer significant with the inclusion of each of the additional covariates (added and tested separately). The significant cross-lag paths between PA at T1 (and T2) predicting symptoms of anxiety at T2 (and T3), were maintained following the inclusion of parent-reported sleep difficulties, as well as adolescent-reported sleep difficulties. When symptoms of depression were added into the model, cross-lagged paths between PA and anxiety were no longer significant.

Associations with Covariates. Medication use was significantly associated with increased parent- and adolescent-reported sleep difficulties, symptoms of ADHD, and symptoms of depression at T1 only. Pubertal development was significantly and positively associated with symptoms of depression and anxiety at T1 and T3. Sex was significantly associated with symptoms of depression and anxiety at T3, in that girls were more likely to have increased symptoms of depression at anxiety. No other associations between covariates and primary study outcomes were significant at any of the time points.

Aim 3

PA Moderating the Association Between Symptoms of ADHD and Parent-Reported Sleep Difficulties. The moderating role of PA on the relation between symptoms of ADHD and parent-reported sleep difficulties was assessed. Centered predictor (i.e., symptoms of ADHD), centered moderator (i.e., PA), and interaction (i.e., symptoms of ADHD x PA) variables were used in the analyses as exogenous variables at each time point. The unconstrained model fit the data well, $\chi^2(64) = 102.16, p = .002$, CFI = .967, RMSEA = .045, 90% CI [.028, .061]. Next, tests were conducted on each of the four autoregressive paths to see if any could be constrained to be equal across waves. As shown in Table 10, all of the autoregressive paths could be constrained to be equal, except for the PA interaction term (ADHD x PA), based on a χ^2 difference test, $(S-B\Delta\chi^2(1) = 7.44, p = .006)$, a decrease in the CFI from .967 to .961, and an increase in RMSEA from .044 to .047.

This model was then compared to the following models with cross-lagged paths constrained equal across waves for e) symptoms of ADHD to parent-reported sleep difficulties, f) parent-reported sleep difficulties to symptoms of ADHD, g) PA to parent-reported sleep difficulties, h) parent-reported sleep difficulties to PA, and i) ADHD x PA interaction term to

parent-reported sleep difficulties. Similar patterns emerged in the model constrain process when examining the model using non-centered variables without the inclusion of the interaction term (Aim 2), in which cross-lagged paths from parent-reported sleep difficulties to symptoms of ADHD and from parent-reported sleep difficulties to PA were allowed to vary freely (per significant χ^2 difference tests) and cross-lagged paths from symptoms of ADHD to parent-reported sleep difficulties and from PA to parent-reported sleep difficulties were best kept constrained (per non-significant χ^2 difference tests). Notably, the χ^2 difference test conducted following the constraint of the cross-lagged paths from the ADHD x PA interaction term to parent-reported sleep difficulties was not significant ($S-B\Delta\chi^2(1) = 1.03, p = .31$). This indicates stability in the interaction term, which means that there are not significant differences in the strength of associations between the interactive effect of PA and symptoms of ADHD and parent-reported sleep difficulties across time (i.e., PA does not significantly moderate the relationship between symptoms of ADHD and parent-reported sleep difficulties). With this, the cross-lagged paths from the ADHD x PA interaction term to parent-reported sleep difficulties from T1 to T2 ($\beta = .013, p = .76$) and from T2 to T3 ($\beta = .011, p = .76$) in the final model were also not significant (see Figure 14).

Table 10

Comparison of Model Constraints for the Longitudinal Associations Between Parent-Reported Sleep

Difficulties, Symptoms of ADHD, PA, and PA Moderation Interaction

Model		χ^2	df	CFI	RMSEA	CI	χ^2	Decision
<i>Unconstrained</i>		102.16	64	.967	.045	[.028, .061]	--	
<i>Auto-regressive paths</i>	a. PR Sleep	102.73	65	.967	.044	[.027, .060]	$\chi^2_{\Delta}(1) = 0.45, p = .50$	Retain
	b. ADHD	105.17	66	.966	.045	[.028, .060]	$\chi^2_{\Delta}(1) = 2.46, p = .12$	Retain
	c. PA	105.26	67	.967	.044	[.027, .059]	$\chi^2_{\Delta}(1) = 0.03, p = .86$	Retain
	d. ADHD x PA	112.78	68	.961	.047	[.031, .062]	$\chi^2_{\Delta}(1) = 7.44, p = .006$	Reject
<i>Cross-lagged paths</i>	e. ADHD \rightarrow PR Sleep	105.91	68	.967	.043	[.026, .059]	$\chi^2_{\Delta}(1) = 0.67, p = .41$	Retain
	f. PR Sleep \rightarrow ADHD	111.54	69	.963	.046	[.029, .061]	$\chi^2_{\Delta}(1) = 6.00, p = .01$	Reject
	g. PA \rightarrow PR Sleep	106.02	69	.968	.043	[.025, .058]	$\chi^2_{\Delta}(1) = 0.21, p = .65$	Retain
	h. PR Sleep \rightarrow PA	110.08	70	.965	.044	[.027, .059]	$\chi^2_{\Delta}(1) = 4.33, p = .04$	Reject
	i. ADHD x PA \rightarrow PR Sleep	106.94	70	.968	.043	[.025, .058]	$\chi^2_{\Delta}(1) = 1.03, p = .31$	Retain

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

Approximation; PR Sleep = Parent-Reported Sleep Difficulties; ADHD = Symptoms of Attention-

Deficit/Hyperactivity Disorder; PA = Total Physical Activity; ADHD x PA = PA moderation

interaction; Models presented above are hierarchically nested, that is, constraints that are retained (or

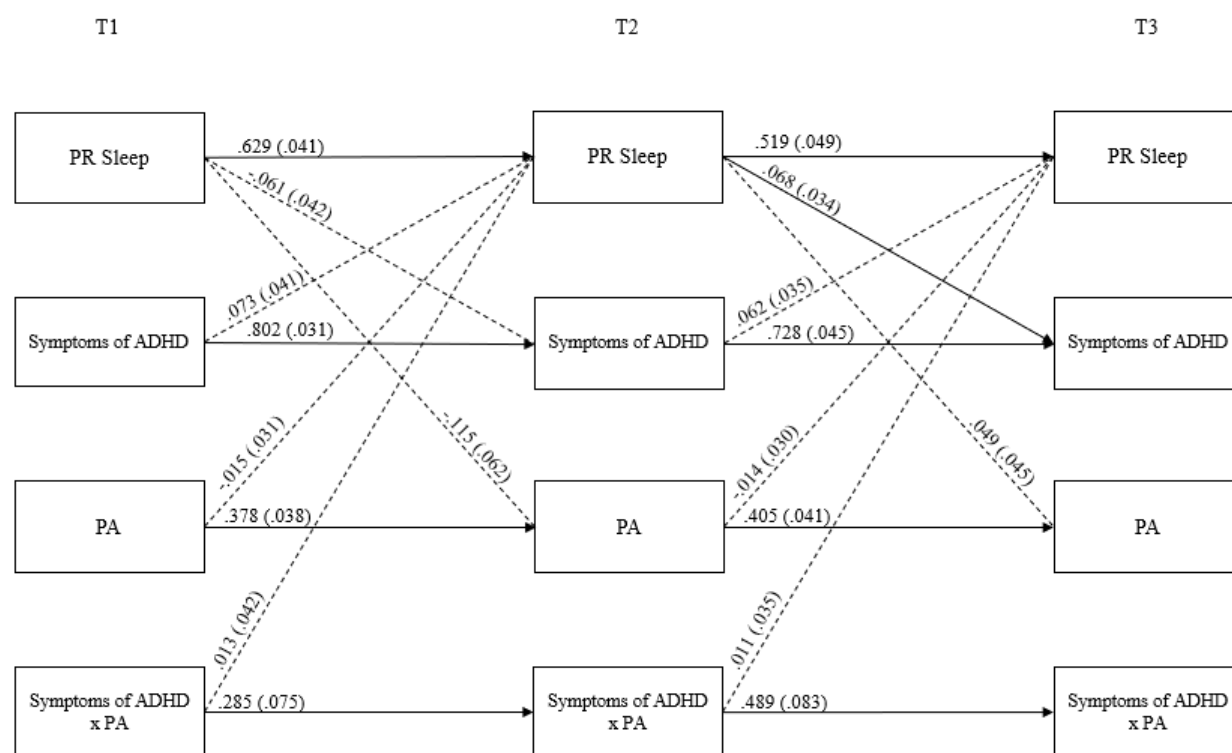
rejected) at initial steps are retained (or rejected) for the subsequent models. The first model is the

unconstrained (base) model. The subsequent models test constraints on the autoregressive paths, and

then the cross-lagged paths.

Figure 14

Longitudinal Associations Between Parent-Reported Sleep Difficulties, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. PR = Parent-Reported; PA = Physical Activity; Symptoms of ADHD x PA = PA

Moderator Interaction Variable; Final model fit: $\chi^2(70) = 106.94$, $p = .003$, CFI = .968, RMSEA = .043, 90% CI [.025, .058]; Betas (β) are shown and standard errors are in parentheses.

Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

PA Moderating the Association Between Symptoms of ADHD and Adolescent-Reported Sleep Difficulties. Next, the moderating role of PA on the relation between symptoms of ADHD and adolescent-reported sleep difficulties was assessed. Centered predictor (i.e., symptoms of ADHD), centered moderator (i.e., PA), and interaction (i.e., symptoms of ADHD x

PA) variables were used in the analyses as exogenous variables at each time point. The unconstrained model fit the data well, $\chi^2(64) = 88.79, p = .02, CFI = .973, RMSEA = .036, 90\% CI [.014, .053]$. Next, tests were conducted on each of the four autoregressive paths to see if any could be constrained to be equal across waves. As shown in Table 11, all of the autoregressive paths could be constrained to be equal, except for the PA interaction term (ADHD x PA), based on a χ^2 difference test, (S-B $\Delta\chi^2(1) = 7.40, p = .007$), a decrease in the CFI from .977 to .970, and an increase in RMSEA from .033 to .037.

This model was then compared to the following models with cross-lagged paths constrained equal across waves for e) symptoms of ADHD to adolescent-reported sleep difficulties, f) adolescent-reported sleep difficulties to symptoms of ADHD, g) PA to adolescent-reported sleep difficulties, h) adolescent-reported sleep difficulties to PA, and i) ADHD x PA interaction term to adolescent-reported sleep difficulties. Similar patterns emerged in the model constrain process when examining the model using non-centered variables without the inclusion of the interaction term (Aim 2), in which cross-lagged paths from PA to adolescent-reported sleep difficulties were allowed to vary freely (per significant χ^2 difference tests) and all other cross-lagged paths were best kept constrained (per non-significant χ^2 difference tests). Notably, the χ^2 difference test conducted following the constraint of the cross-lagged paths from the ADHD x PA interaction term to adolescent-reported sleep difficulties was not significant (S-B $\Delta\chi^2(1) = 0.22, p = .64$). This indicates stability in the interaction term, which means that there are not significant differences in the strength of associations between the interactive effect of PA and symptoms of ADHD and adolescent-reported sleep difficulties across time (i.e., PA does not significantly moderate the relationship between symptoms of ADHD and adolescent-reported sleep difficulties). With this, the cross-lagged paths from the ADHD x PA interaction term to

adolescent-reported sleep difficulties from T1 to T2 ($\beta = -.013, p = .76$) and from T2 to T3 ($\beta = -.011, p = .76$) in the final model were also not significant (see Figure 15).

Table 11

Comparison of Model Constraints for the Longitudinal Associations Between Adolescent Reported Sleep Difficulties, Symptoms of ADHD, PA, and PA Moderation Interaction

Model		χ^2	df	CFI	RMSEA	CI	χ^2	Decision
<i>Unconstrained</i>		88.79	64	.973	.036	[.014, .053]	--	
<i>Auto-regressive paths</i>	a. AR Sleep	86.48	65	.976	.033	[.008, .051]	$\chi^2_{\Delta}(1) = 0.003, p = .96$	Retain
	b. ADHD	87.90	66	.976	.033	[.009, .051]	$\chi^2_{\Delta}(1) = 1.43, p = .23$	Retain
	c. PA	88.14	67	.977	.033	[.006, .050]	$\chi^2_{\Delta}(1) = 0.02, p = .90$	Retain
	d. ADHD x PA	95.25	68	.970	.037	[.016, .053]	$\chi^2_{\Delta}(1) = 7.40, p = .007$	Reject
<i>Cross-lagged paths</i>	e. ADHD \rightarrow AR Sleep	88.29	68	.978	.032	[.001, .049]	$\chi^2_{\Delta}(1) = 0.03, p = .86$	Retain
	f. AR Sleep \rightarrow ADHD	88.91	69	.978	.031	[.000, .049]	$\chi^2_{\Delta}(1) = 0.50, p = .48$	Retain
	g. PA \rightarrow AR Sleep	92.60	70	.975	.033	[.009, .050]	$\chi^2_{\Delta}(1) = 4.10, p = .04$	Reject
	h. AR Sleep \rightarrow PA	89.33	70	.979	.031	[.000, .048]	$\chi^2_{\Delta}(1) = 0.16, p = .69$	Retain
	i. ADHD x PA \rightarrow AR Sleep	89.55	71	.980	.030	[.000, .047]	$\chi^2_{\Delta}(1) = 0.22, p = .64$	Retain

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

Approximation; AR Sleep = Adolescent-Reported Sleep Difficulties; ADHD = Symptoms of

Attention-Deficit/Hyperactivity Disorder; PA = Total Physical Activity; ADHD x PA = PA

moderation interaction; Models presented above are hierarchically nested, that is, constraints that are

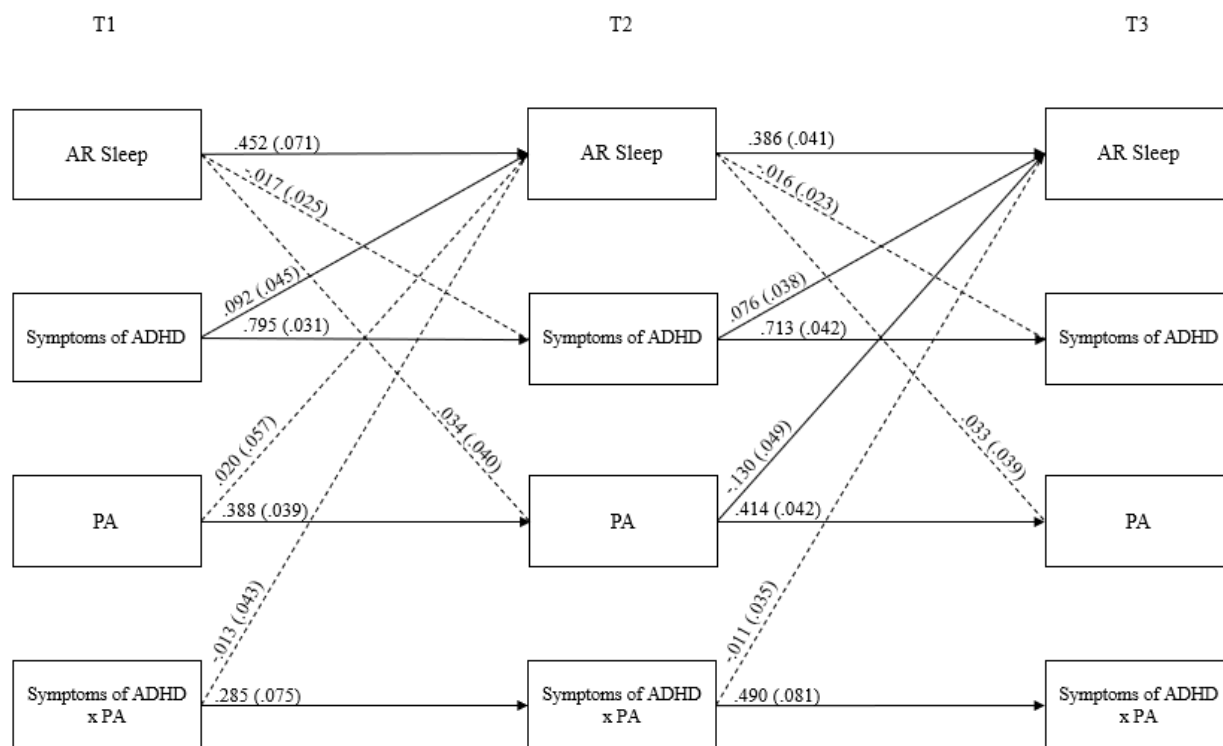
retained (or rejected) at initial steps are retained (or rejected) for the subsequent models. The first

model is the unconstrained (base) model. The subsequent models test constraints on the autoregressive

paths, and then the cross-lagged paths.

Figure 15

Longitudinal Associations Between Adolescent-Reported Sleep Difficulties, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. AR = Adolescent-Reported; PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable; Final model fit: $\chi^2(71) = 89.55$, $p = .07$, CFI = .980, RMSEA = .030, 90% CI [.000, .047]; Betas (β) are shown and standard errors are in parentheses.

Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

PA Moderating the Association Between Symptoms of ADHD and Symptoms of Depression. The moderating role of PA on the relation between symptoms of ADHD and symptoms of depression was then assessed. Centered predictor (i.e., symptoms of ADHD), centered moderator (i.e., PA), and interaction (i.e., symptoms of ADHD x PA) variables were

used in the analyses as exogenous variables at each time point. The unconstrained model fit the data well, $\chi^2(64) = 97.22, p < .001$, CFI = .967, RMSEA = .042, 90% CI [.024, .058]. Next, tests were conducted on each of the four autoregressive paths to see if any could be constrained to be equal across waves. As shown in Table 12, all of the autoregressive paths could be constrained to be equal, except for the PA interaction term (ADHD x PA), based on a χ^2 difference test, (S-B $\Delta\chi^2(1) = 7.55, p = .006$), a decrease in the CFI from .969 to .963, and an increase in RMSEA from .040 to .043.

This model was then compared to the following models with cross-lagged paths constrained equal across waves for e) symptoms of ADHD to symptoms of depression, f) symptoms of depression to symptoms of ADHD, g) PA to symptoms of depression, h) symptoms of depression to PA, and i) ADHD x PA interaction term to symptoms of depression. Similar patterns emerged in the model constrain process when examining the model using non-centered variables without the inclusion of the interaction term (Aim 2), in which all cross-lagged paths were best kept constrained (per non-significant χ^2 difference tests). Notably, the χ^2 difference test conducted following the constraint of the cross-lagged paths from the ADHD x PA interaction term to symptoms of depression was not significant (S-B $\Delta\chi^2(1) = 0.87, p = .35$). This indicates stability in the interaction term, which means that there are not significant differences in the strength of associations between the interactive effect of PA and symptoms of ADHD and symptoms of depression across time (i.e., PA does not significantly moderate the relationship between symptoms of ADHD and symptoms of depression). With this, the cross-lagged paths from the ADHD x PA interaction term to symptoms of depression from T1 to T2 ($\beta = -.035, p = .29$) and from T2 to T3 ($\beta = -.023, p = .30$) in the final model were also not significant (see Figure 16).

Table 12

Comparison of Model Constraints for the Longitudinal Associations Between Symptoms of Depression, Symptoms of ADHD, PA, and PA Moderation Interaction

	Model	χ^2	df	CFI	RMSEA	CI	χ^2_{Δ}	Decision
	<i>Unconstrained</i>	97.22	64	.967	.042	[.024, .058]	--	
<i>Auto-regressive paths</i>	a. Dep	96.74	65	.969	.041	[.022, .057]	$\chi^2_{\Delta}(1) = 0.01, p = .91$	Retain
	b. ADHD	98.03	66	.969	.040	[.022, .057]	$\chi^2_{\Delta}(1) = 1.26, p = .26$	Retain
	c. PA	98.18	67	.969	.040	[.021, .056]	$\chi^2_{\Delta}(1) = 0.01, p = .92$	Retain
	d. ADHD x PA	105.50	68	.963	.043	[.026, .059]	$\chi^2_{\Delta}(1) = 7.55, p = .006$	Reject
<i>Cross-lagged paths</i>	e. ADHD → Dep	98.68	68	.970	.039	[.020, .055]	$\chi^2_{\Delta}(1) = 0.74, p = .39$	Retain
	f. Dep → ADHD	98.86	69	.971	.038	[.019, .054]	$\chi^2_{\Delta}(1) = 0.19, p = .67$	Retain
	g. PA → Dep	99.07	70	.971	.037	[.018, .054]	$\chi^2_{\Delta}(1) = 0.37, p = .55$	Retain
	h. Dep → PA	100.09	71	.971	.037	[.018, .053]	$\chi^2_{\Delta}(1) = 0.84, p = .36$	Retain
	i. ADHD x PA → Dep	101.19	72	.971	.037	[.018, .053]	$\chi^2_{\Delta}(1) = 0.87, p = .35$	Retain

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

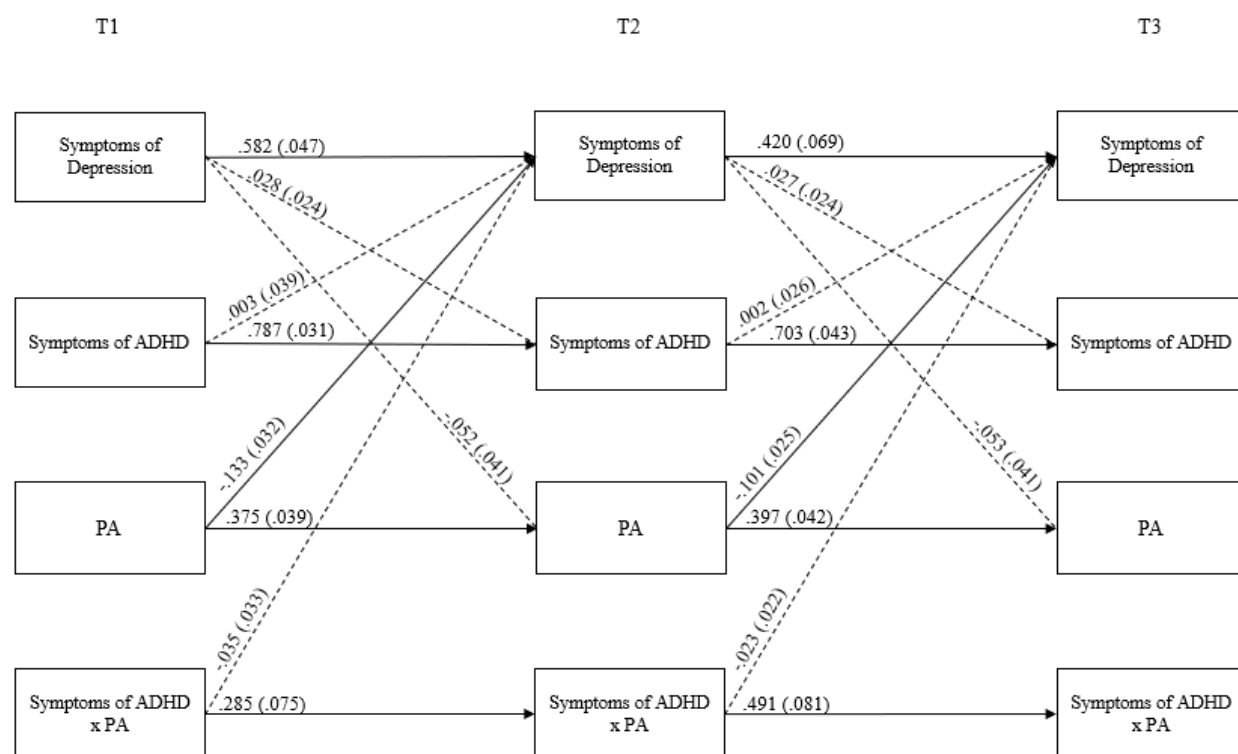
Approximation; Dep = Symptoms of Depression; ADHD = Symptoms of Attention-

Deficit/Hyperactivity Disorder; PA = Total Physical Activity; ADHD x PA = PA moderation

interaction; Models presented above are hierarchically nested, that is, constraints that are retained (or rejected) at initial steps are retained (or rejected) for the subsequent models. The first model is the unconstrained (base) model. The subsequent models test constraints on the autoregressive paths, and then the cross-lagged paths.

Figure 16

Longitudinal Associations Between Symptoms of Depression, Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable; Final model fit: $\chi^2(72) = 101.19, p = .01, CFI = .971, RMSEA = .037, 90\% CI [.018, .053]$; Betas (β) are shown and standard errors are in parentheses. Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

PA Moderating the Association Between Symptoms of ADHD and Symptoms of Anxiety. Finally, the moderating role of PA on the relation between symptoms of ADHD and symptoms of anxiety was assessed. Centered predictor (i.e., symptoms of ADHD), centered moderator (i.e., PA), and interaction (i.e., symptoms of ADHD x PA) variables were used in the

analyses as exogenous variables at each time point. The unconstrained model fit the data well, $\chi^2(64) = 105.09, p < .001$, CFI = .960, RMSEA = .047, 90% CI [.030, .062]. Next, tests were conducted on each of the four autoregressive paths to see if any could be constrained to be equal across waves. As shown in Table 13, all of the autoregressive paths could be constrained to be equal, except for the PA interaction term (ADHD x PA), based on a χ^2 difference test, (S-B $\Delta\chi^2$ (1) = 7.39, $p = .007$), a decrease in the CFI from .959 to .953, and an increase in RMSEA from .046 to .049.

This model was then compared to the following models with cross-lagged paths constrained equal across waves for e) symptoms of ADHD to symptoms of anxiety, f) symptoms of anxiety to symptoms of ADHD, g) PA to symptoms of anxiety, h) symptoms of anxiety to PA, and i) ADHD x PA interaction term to symptoms of anxiety. Similar patterns emerged in the model constrain process when examining the model using non-centered variables without the inclusion of the interaction term (Aim 2), in which all cross-lagged paths were best kept constrained (per non-significant χ^2 difference tests). Notably, the χ^2 difference test conducted following the constraint of the cross-lagged paths from the ADHD x PA interaction term to symptoms of anxiety was not significant (S-B $\Delta\chi^2$ (1) = 1.04, $p = .31$). This indicates stability in the interaction term, which means that there are not significant differences in the strength of associations between the interactive effect of PA and symptoms of ADHD and symptoms of anxiety across time (i.e., PA does not significantly moderate the relationship between symptoms of ADHD and symptoms of anxiety). With this, the cross-lagged paths from the ADHD x PA interaction term to symptoms of anxiety from T1 to T2 ($\beta = -.014, p = .69$) and from T2 to T3 ($\beta = -.011, p = .69$) in the final model were also not significant (see Figure 17).

Table 13

Comparison of Model Constraints for the Longitudinal Associations Between Symptoms of Anxiety,

Symptoms of ADHD, PA, and PA Moderation Interaction

Model		χ^2	df	CFI	RMSEA	CI	χ^2	Decision
<i>Unconstrained</i>		105.09	64	.960	.047	[.030, .062]	--	
<i>Auto-regressive paths</i>	a. Anx	107.87	65	.958	.047	[.031, .063]	$\chi^2_{\Delta}(1) = 2.33, p = .13$	Retain
	b. ADHD	109.19	66	.958	.047	[.031, .062]	$\chi^2_{\Delta}(1) = 1.28, p = .26$	Retain
	c. PA	109.39	67	.959	.046	[.030, .062]	$\chi^2_{\Delta}(1) = 0.07, p = .79$	Retain
	d. ADHD x PA	116.59	68	.953	.049	[.033, .064]	$\chi^2_{\Delta}(1) = 7.39, p = .007$	<i>Reject</i>
<i>Cross-lagged paths</i>	e. ADHD → Anx	110.46	68	.959	.046	[.030, .061]	$\chi^2_{\Delta}(1) = 0.90, p = .34$	Retain
	f. Anx → ADHD	111.79	69	.958	.046	[.029, .061]	$\chi^2_{\Delta}(1) = 1.15, p = .29$	Retain
	g. PA → Anx	112.10	70	.959	.045	[.029, .060]	$\chi^2_{\Delta}(1) = 0.52, p = .47$	Retain
	h. Anx → PA	112.68	71	.959	.045	[.028, .060]	$\chi^2_{\Delta}(1) = 0.28, p = .60$	Retain
	i. ADHD x PA → Anx	113.83	72	.959	.044	[.028, .059]	$\chi^2_{\Delta}(1) = 1.04, p = .31$	Retain

Note. df = degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of

Approximation; Anx = Symptoms of Anxiety; ADHD = Symptoms of Attention-Deficit/Hyperactivity

Disorder; PA = Total Physical Activity; ADHD x PA = PA moderation interaction; Models presented

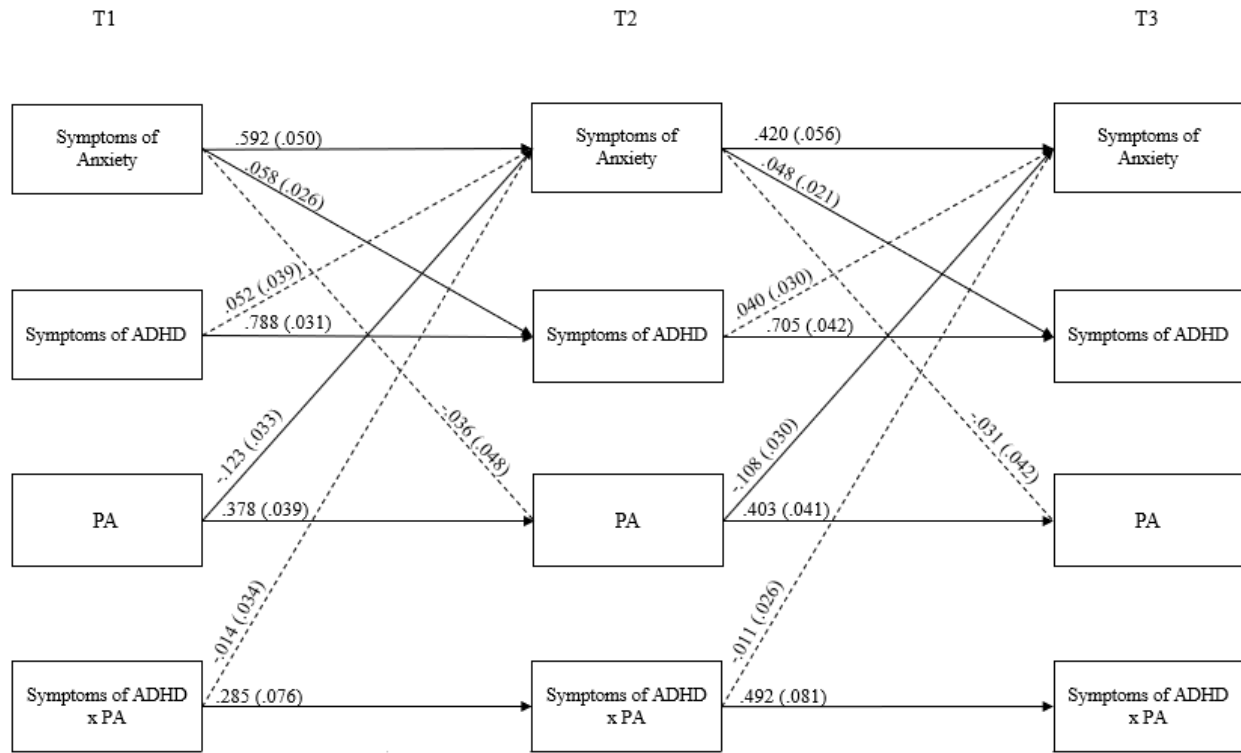
above are hierarchically nested, that is, constraints that are retained (or rejected) at initial steps are

retained (or rejected) for the subsequent models. The first model is the unconstrained (base) model.

The subsequent models test constraints on the autoregressive paths, and then the cross-lagged paths.

Figure 17

Longitudinal Associations Between Symptoms of Anxiety Symptoms of ADHD, Total PA, and PA Moderator Interaction Variable



Note. PA = Physical Activity; Symptoms of ADHD x PA = PA Moderator Interaction Variable;

Final model fit: $\chi^2(72) = 113.83, p = .001, CFI = .959, RMSEA = .044, 90\% CI [.028, .059];$

Betas (β) are shown and standard errors are in parentheses. Correlations between variables within each time point and covariates were included in the model, but are not shown in the figure to reduce complexity. The dotted lines represent non-significant paths; the solid lines indicate significant paths.

Sensitivity Analyses. As there were no significant moderating effects in any of the models, sensitivity analyses were not conducted.

Discussion

This study builds upon prior literature by examining multiple aspects of PA and associations with sleep and internalizing symptoms in a large sample of adolescents with and without ADHD across the transition from middle to high school. This is the first study to compare differences in PA behaviors in an adolescent-only sample with and without ADHD, as well to examine differences between ADHD presentations and sex differences. Autoregressive cross-lagged (ARCL) panel models were used to comprehensively examine longitudinal associations between symptoms of ADHD, PA, sleep difficulties, and internalizing symptoms, while also accounting for the impacts these variables have on each other over time.

Importantly, PA was examined in three separate ways for the present study. Participants reported on the number of days they engaged in at least 20 and 60 minutes of PA; *“On how many of the past 7 days did you exercise or participate in physical activity for at least 20 [at least 60] minutes that made you sweat and breathe hard”*. These questions were taken from the CDC’s Youth Risk Behavior Survey, which is administered nationally every two years (CDC, 2010) and maps on to US Department of Health and Human Services’ PA Guidelines for Americans. The recommendation states that children under the age of 18 should engage in 60 minutes or more of moderate-to-vigorous PA (MVPA) (i.e., makes you sweat or breathe hard) every day (US Department of Health and Human Services, 2018). Accordingly, the present study was also able to evaluate the percentage of participants with and without ADHD in the sample meeting this recommendation. In addition, a total PA composite score was also examined, which was taken from the PAQ-A, a well-validated measure of PA in adolescents (Kowalski et al., 1997a; Kowalski et al., 1997b). This measure assesses more general levels of PA over the prior week, including the frequency and timing of engaging in sports, dancing, playing games, and

other activities in which participants were physically active. Major findings of the study are reviewed briefly in the subsequent sections, followed by a discussion of implications, limitations, and future directions.

When participants were in 8th, 9th, and 10th grades, 10.0%, 14.0%, and 10.1%, of the ADHD group were meeting daily PA recommendations, respectively. These percentages were not significantly different than the percentages of those in the comparison group. These figures are slightly lower than national averages (18% of high school students; CDC, 2010), and averages from another study examining adherence to PA recommendations in youth with ADHD (17.8%; Wang et al., 2022). Significant differences in PA behaviors in participants with and without ADHD did emerge. Specifically, during 8th grade (T1), youth in the comparison group engaged in more PA than youth with ADHD by all measures. They engaged in PA for at least 20 and 60 minutes on more days of the week and had higher overall PA scores. This pattern of differences was also found when the participants were in 9th grade (T2) when examining the average number of days engaging in at least 20 and 60 minutes of PA. At both time points, the comparison group engaged in approximately one additional day of PA per week relative to the ADHD group. By the time participants were in 10th grade (T3), there were no significant differences between groups. When examining differences in PA based on ADHD presentation, participants with ADHD-I engaged in significantly less PA than those in the comparison group at T1 only. No significant differences were found when comparing the ADHD-C group to either the ADHD-I or the comparison group at any time point. Boys with ADHD engaged in significantly less PA than boys in the comparison group at T1 and T2, whereas girls with and without ADHD engaged in similar levels of PA.

This study also evaluated longitudinal associations between symptoms of ADHD, PA, sleep, anxiety, and depression. Longitudinal ARCL models revealed that symptoms of ADHD predicted increases in adolescent-reported sleep difficulties, PA predicted decreases in adolescent-reported sleep difficulties (from T2 to T3 only), and PA predicted decreases in symptoms of depression and anxiety across all time points. Finally, PA was examined as a potential buffer for the development of sleep difficulties and internalizing symptoms. PA did not significantly moderate the associations between symptoms of ADHD and sleep or internalizing outcomes.

PA Differences in Adolescents with and without ADHD

Findings that adolescents in the ADHD group overall engaged in less PA than their non-ADHD peers are consistent with some of the previous literature in combined samples of children and adolescents (Mercurio et al., 2021; San Mauro Martín et al., 2018; Tandon et al., 2019). Multiple factors are hypothesized as potentially contributing to this, including deficits in motor skills and coordination, executive functioning, and/or symptoms of inattention. Motor problems and developmental coordination difficulties have been well-documented in children with ADHD, including deficits in hand-eye coordination, gross and fine motor skills, and balance (Pitcher et al., 2003; Rosa Neto et al., 2015; Soorya & Halpern, 2009). These difficulties persist in adolescents, although become less pronounced over time (Fliers et al., 2008). Although motor skills were not assessed in the present study, it is possible this could impact engagement in PA, particularly for younger adolescents with ADHD. For example, they may be less inclined to engage in physical activities that require a degree of competence in motor skills. Indeed, research has found that adolescents who perceive themselves to have less motor competence have less motivation to engage in PA (De Meester et al., 2016).

ADHD in adolescence is also associated with deficits in skills such as planning and time management (Martel et al., 2007; Toplak et al., 2008), and it may be that these difficulties negatively impact engagement in PA. Participation in PA outside of school may be most relevant and affected for adolescents with ADHD, as incorporating these activities into their schedules would require some degree of independent planning and time management. This assertion is consistent with findings from San Mauro Martín and colleagues (2018), who examined PA in a combined sample of children and adolescents. They found that youth in the comparison group engaged in more days of exercise after school, whereas youth in the ADHD group had more days of exercise during school (San Mauro Martín et al., 2018). They also found that overall PA was higher in the ADHD group, which was inconsistent with the findings of the current study. However, this may be because their sample consisted of younger children ($M_{\text{age}} = 10.4$ and 9.5 years old for the ADHD and comparison groups, respectively), who may have more opportunities to engage in PA during the school day (i.e., recess) compared to adolescents (San Mauro Martín et al., 2018). The current study did not assess time of day differences or compare PA differences during versus outside of school. However, results highlight that there may be unique challenges in engaging in PA for adolescents with ADHD that could be related to managing time and balancing responsibilities outside of school.

Another potential explanation relates to symptoms of inattention in particular. In the present study, differences in PA were examined based on ADHD presentation. It was found that adolescents with ADHD-I engaged in significantly less PA than those in the comparison group at T1, whereas no significant differences were found when comparing the ADHD-C group to either the ADHD-I or the comparison group. It is important to note that the ADHD-C group had a smaller sample size compared to the other two groups. Although having equal sample sizes is not

an assumption of the ANOVA, performing a one-way ANOVA with unequal sample sizes can result in reduced statistical power to detect significant effects (Kim, 2017). Nonetheless, significant differences did emerge between the ADHD-I and comparison group on the number of days engaging in at least 20 and 60 minutes of PA at T1. Prior research suggests that symptoms of inattention in particular may play a role in the PA behaviors of adolescents. Khalife and colleagues (2014) found that symptoms of inattention at ages 7 - 8 were prospectively associated with physical inactivity at age 16. Similarly, Selinus and colleagues (2021) found that symptoms of inattention during childhood (9 - 12 years old) predicted less PA in adolescence (15 years old). The connection between symptoms of inattention and PA is not yet well understood, but authors theorize that since physical activities require increased concentration, perception, and self-directedness, these activities may be more challenging for children experiencing high levels of inattention (Khalife et al., 2014).

It was predicted that adolescents with ADHD-C would experience higher levels of PA than youth in the comparison and ADHD-I groups, since hyperactive-impulsive symptoms are associated with feelings of restlessness, fidgetiness, and overall increased motor movements (Gawrilow et al., 2014). As previously noted, it is important to consider that in the current study significant differences between the ADHD-C group and the other two groups may not have been discovered due to low power. With this, it remains unclear to what degree PA habits differ for youth with ADHD-C versus ADHD-I and how that may compare to youth without ADHD. Zerón-Rugério and colleagues (2020) examined PA differences in youth with and without ADHD and compared across presentations, finding increased levels of PA only in the ADHD-C group when compared to youth without ADHD. Notably however, they utilized an objective measure of PA (accelerometry) in their study. It is possible that youth with ADHD-C have more

motor movements throughout the day, however, this may not necessarily translate into engagement in specific physical activities (i.e., sports, walking, etc.). Selinus and colleagues (2021) found that symptoms of hyperactivity/impulsivity during childhood predicted more PA (via single item self-report assessing frequency of PA that leads to becoming sweaty/breathing hard) during adolescence. This study examined a normative sample of youth, and future research may benefit from disentangling symptoms of inattention versus hyperactivity in adolescents with ADHD-C when examining PA outcomes in this population.

Interestingly, PA differences between the ADHD and comparison group may be driven by sex differences. In the present study, boys in the comparison group engaged in significantly more PA than boys in the ADHD group, whereas very few differences emerged between girls with and without ADHD. There is a wealth of literature supporting that boys engage in PA more than girls, and these differences become more pronounced during adolescence (Robbins et al., 2003; Telford et al., 2016). Evidence suggests these differences are present for several reasons, including societal expectations, physiological differences (i.e., higher body fat and lower cardiovascular fitness in girls), and differences in perceived competence and physical abilities (Telford et al., 2019). Prior research has shown that boys with ADHD engage in more PA than girls with ADHD (Tandon et al., 2019). However, this is the first study to compare the PA habits of boys and girls with ADHD compared to boys and girls without ADHD. An area of future research may be to further explore how biological and societal differences interact with lifestyle behaviors in boys versus girls with ADHD.

A final pattern that emerged is that differences in PA between youth in the ADHD and comparison groups became less pronounced as they got older. Specifically, there were significant differences on all PA variables at T1, and then on two out of three variables at T2, and then no

significant differences by T3. This may reflect PA decreasing overall from 8th to 10th grade, coinciding with less variability in the data. PA often decreases throughout adolescence when compared to childhood, as the period of adolescence is associated with decreased opportunities for PA during the school day and increased sedentary behaviors (Llorente-Cantarero et al., 2020; CDC, 2015; Mitchell et al., 2012). The present study followed adolescents until 10th grade (approximately 16 years old), and research is needed to determine whether these developmental trends continue, particularly across the transition to emerging adulthood. Indeed, research on PA habits in adults with ADHD has yielded mixed results (Bijlenga et al., 2013; Björk et al., 2018; Spencer et al., 2014), although one study found that adults with ADHD engaged in significantly less PA on multiple measures compared to adults without ADHD (Björk et al., 2018). However, it is unclear to what degree this is a reflection of overall decreases in healthy lifestyle behaviors (i.e., worse eating behaviors, increased smoking and drinking) found in the sample (Björk et al., 2018).

Associations Between Sleep Difficulties, Symptoms of ADHD, and PA

The second aim of the study was to examine longitudinal and bidirectional associations between sleep, symptoms of ADHD, and PA. Two different models of sleep difficulties were examined, one with adolescent-reported sleep difficulties (from the sleep-wake difficulties subscale of the SHS) and one with parent-reported sleep difficulties (from the difficulties initiating and maintaining sleep subscale of the SDSC). ADHD symptoms were parent-reported (via Vanderbilt Rating Scale) and PA was self-reported (overall PA score from PAQ-A).

Parent-reported sleep difficulties predicted increased ADHD symptoms from T2 to T3, which is consistent to a degree with prior research finding that impaired sleep can worsen ADHD symptomatology (Hysing et al., 2016; Lam & Yang, 2008). In the model with adolescent-reported

sleep difficulties, ADHD symptoms predicted adolescent-reported sleep difficulties from T1 to T2 and from T2 to T3. This is consistent with prior research finding that ADHD symptomology predicts a variety of negative sleep outcomes in adolescent-only samples (Hysing et al., 2016; Johnson et al., 2006; Sivertsen et al., 2015). This study is unique in that it found evidence in support of these associations across raters and utilized longitudinal data, allowing for an exploration of bidirectional associations. However, significant associations were only found from ADHD symptoms to adolescent-reported sleep difficulties. This is notably inconsistent with the growing body of research in youth with ADHD that has found bidirectional associations between symptoms of ADHD and sleep difficulties (Weiss et al., 2015).

A novel finding of the present study is that PA predicted decreased adolescent-reported sleep difficulties from T2 to T3, above and beyond the impacts of ADHD symptomology on sleep difficulties. This association was also above and beyond the impacts of sex, pubertal development, and medication use. This adds to the literature on the associations between PA, ADHD, and sleep functioning in adolescents, which thus far consists of one study (Li et al., 2021). Specifically, Li and colleagues (2021) found parent-reported PA positively predicted parent-reported sleep quality in a sample of adolescents with ADHD (Li et al., 2021). Findings from the present study build upon these findings by evaluating associations above and beyond symptoms of ADHD and using well-validated questionnaires and a multi-rater model. Overall, these findings highlight the beneficial effects of PA and how PA remains important for sleep functioning even in the presence of varying levels of ADHD symptomology.

Associations Between Internalizing Symptoms, Symptoms of ADHD, and PA

This study also evaluated longitudinal and bidirectional associations between internalizing symptoms, symptoms of ADHD, and PA. Symptoms of depression and anxiety

were both assessed via self-report, using subscales from the RCADS. Notably, items related to sleep functioning were removed from the depression and anxiety subscales, in order to evaluate unique associations without sleep difficulties as a potential confound in the assessment of internalizing symptomology.

This is the first study to directly examine associations between internalizing symptoms, ADHD symptomology, and PA within a sample of adolescents. From T1 to T2 and T2 to T3, PA predicted decreases in both symptoms of depression and anxiety. This was above and beyond the influence of symptoms of ADHD on depression and anxiety, and controlling for sex, pubertal status, and medication use. Additionally, symptoms of anxiety positively predicted symptoms of ADHD across time. Overall, these results are consistent with several observational and intervention studies documenting that PA is associated with decreases in symptoms of depression (Carter et al., 2016; Johnson & Taliaferro, 2011) and anxiety (Ahn & Fedewa, 2011; Ferreira-Vorkapic et al., 2015) in adolescents. In the present study, reciprocal relationships were not established, in that neither symptoms of depression nor anxiety predicted PA. This is inconsistent with prior literature, which has found bidirectional links between PA and symptoms of depression (Jerstad et al., 2010; Stavrakakis et al., 2012) and PA and anxiety (Gunnell et al., 2016) in adolescents. Overall, as with sleep difficulties, the results of the present study highlight the continued importance of PA, as it serves to protect against the development of internalizing symptomology, even in the presence of comorbid symptoms of ADHD. Additionally, the finding that symptoms of anxiety positively predicted symptoms of ADHD supports the growing literature establishing bidirectional associations between these variables in adolescents (Murray et al., 2022).

Exploring Moderation

The present study also evaluated whether PA served as a buffer between ADHD and sleep difficulties and internalizing symptoms. In all models in the present study, the addition of the interaction variable (i.e., symptoms of ADHD x PA) resulted in improved model fit, indicating that PA did not significantly moderate the paths from symptoms of ADHD to sleep difficulties, symptoms of depression, or symptoms of anxiety. This was in contrast to the original study hypotheses. Since previous research has demonstrated the positive impacts of PA on sleep functioning and internalizing symptoms, it was predicted that PA may serve as protective factor against the existing pathways between symptoms of ADHD and sleep difficulties and/or internalizing symptoms. It may be that despite the beneficial effects of PA, associations between symptoms of ADHD and sleep/internalizing are not impacted at the levels of PA found in this sample. As there are many hypothesized neurobiological links between ADHD and sleep difficulties (Cortese et al., 2008; Owens et al., 2013) and between ADHD and internalizing symptomology (Jarrett & Ollendick, 2008; Meinzer et al., 2014), it may be that low levels of PA cannot counteract these associations. It is also possible that in the present study, there was not enough variability in PA in order to see meaningfully positive impacts. Indeed, research on PA interventions that yield positive results often have youth engaging in frequent, intense, and consistent levels of PA (Ahn & Fedewa, 2011; Carter et al., 2016), and national guidelines for adolescents encourages at least 60 minutes of MVPA daily (US Department of Health and Human Services, 2018). In order to further explore and understand this issue, future research should utilize a larger sample, with greater variability and higher levels of PA, in order to better determine to what degree PA can act as a buffer for the development of outcomes such as sleep difficulties and internalizing symptomology.

Limitations

Although the findings of this study provide important information, several limitations should be discussed. First, there are certain characteristics of the sample that likely influenced results. As previously mentioned, the sample had overall low levels of PA and little variability within their PA habits. This may not allow for the exploration of the true effects of PA, which is more likely to manifest when PA is frequent and consistent. In addition, the sample size was also relatively small for ARCL analyses. The sample size may have limited the ability to detect smaller effects and reduced power to find significant interaction effects.

Other important characteristics include the socioeconomic and racial/ethnic makeup of the sample. The sample primarily consisted of White adolescents from relatively high-income families. This significantly limits the generalizability of these findings, particularly to financially disadvantaged or racially/ethnically diverse adolescents. Opportunities for PA for adolescents can largely depend on time, opportunity, and access. There are many environmental determinants to engaging in PA, including school and community sports access, home access to fitness equipment, and access to safe outdoor spaces (Gordon-Larsen et al., 2000). These environmental factors often intersect with sociocultural factors, such as race/ethnicity and socioeconomic status. Research has shown MVPA and organized sports participation to be lower in Black and Hispanic adolescents compared to White adolescents (Black, 2022; CDC, 2010; Van der Horst et al., 2007), although use of community recreation centers is higher in Black youth and associated with increased likelihood of PA engagement (Gordon-Larsen et al., 2000). Family income and lower SES is also associated with less engagement and opportunities for PA in adolescents (Black, 2022; Brodersen et al., 2007; CDC, 2010; Van Der Horst, 2007).

In the present study, there were insufficient numbers of participants to evaluate how race/ethnicity and SES may have impacted the observed relationships. With this, as the sample was primarily White and from high-income families, it is likely the results of the study can only extend to youth with more access and opportunities for PA. Differences in PA habits between the ADHD and comparison groups could be more pronounced when examining youth from sociocultural groups who have less PA opportunities. It is also possible that youth with less access to other forms of treatment (i.e., therapy and/or medical care for sleep difficulties and/or internalizing symptomology) may rely more heavily on PA as a mechanism for maintaining positive health outcomes. Future research with more culturally diverse adolescents would provide valuable information on how to best support PA engagement in different populations of adolescents with and without ADHD. Overall, as PA is highly associated with socioeconomic and cultural characteristics, the results of the present study should be interpreted with caution.

Another notable limitation relates to the way ADHD was examined in the ARCL models. For the purposes of this study, ADHD symptoms were examined using the full range of symptoms across the whole sample, rather than conducting analyses in a way that separated based on adolescents with and without a clinical diagnosis of ADHD. This was done to increase variability, and to explore how symptoms of ADHD broadly are associated with PA, sleep difficulties, and internalizing symptomology. Even adolescents who do not meet clinical threshold for ADHD may still have impairment associated with subthreshold ADHD symptomology (Sibley et al., 2012), and therefore it is important to examine how varying levels of ADHD symptoms (rather than clinical levels only) are associated with outcomes. The overarching goal of the present study was to explore how these variables are connected within the same model, rather than explore ADHD versus non-ADHD differences in the ARCL models.

However, it is nonetheless important to note that the results of the ARCL models may look different in samples of only youth diagnosed with ADHD. It is possible that certain associations could be stronger, and/or the buffering effects of PA could be more relevant for youth with a clinical diagnosis of ADHD.

Finally, ADHD symptoms were examined together as a total score, and separate symptom dimensions (i.e., inattention, hyperactivity, impulsivity) were not explored in the ARCL models. This is a notable limitation as there is reason to believe that symptoms of inattention and symptoms of hyperactivity may have different impacts on PA and motor movements (Khalife et al., 2014; Selinus et al., 2021; Zerón-Rugério et al., 2020). There is also some evidence that different dimensions of ADHD are uniquely associated with sleep functioning (Chiang et al., 2010) and internalizing symptomology (Schatz & Rostain, 2006; Trani et al., 2014). Further exploring how different ADHD symptom dimensions differentially impact associations between ADHD, PA, and sleep functioning and/or internalizing symptomology will aid in better understanding the mechanisms behind these associations.

Despite these limitations, this study makes an important contribution to the understanding of PA habits in adolescents with ADHD, and how they compare to adolescents without ADHD. It also contributes to the literature on the connections between ADHD, PA, sleep difficulties, and comorbid internalizing symptomology in adolescents. Although the present study cannot indicate causal associations between the primary study variables, the longitudinal bidirectional design provides information about the possible direction of effects from 8th grade to 10th grade. The general pattern of significant and nonsignificant results can also provide useful insight for future longitudinal and intervention research.

Future Directions

Several areas for future research have thus far been outlined, including collecting a larger sample of adolescents who engage in a greater amounts of PA, and exploring associations in youth with diverse racial/ethnic and socioeconomic identities. In addition, BMI and the degree to which obesity may be related to PA and ADHD should also be explored. A meta-analysis by Cortese and colleagues (2016) found that ADHD in childhood/adolescence is associated with increased risk for obesity, as well as increased risk for being classified as overweight. Studies included in the meta-analysis did not specifically examine whether there were differences in associations between children versus adolescents, however, the link between ADHD and obesity persists into adulthood (Cortese et al., 2016). Exploring the degree to which weight management difficulties impacts PA behaviors is particularly relevant for youth with ADHD. In the present study, BMI was not collected, and it is unclear how this may have impacted the results. If adolescents in the ADHD group had significantly higher BMIs than adolescents in the comparison group, it could potentially account for why youth in the ADHD group engaged in less PA. BMI is particularly relevant when considering sleep difficulties as well, and data suggests there is complex interplay between obesity and sleep functioning in youth with ADHD (Hvolby, 2015).

The impact of medication use on associations between ADHD, PA, sleep functioning, and internalizing symptomology is a highly relevant and complex issue that warrants attention in future research. For the purposes of this study, medication use (for attention, emotional/behavior difficulties, or sleep problems) was included as a covariate on all ARCL models and examined as a binary yes/no variable. The absence of medication use was significantly associated with increased levels of PA at T1 and T2. It was beyond the scope of the study to disentangle the

unique effects of different types of medication; however, it is an important consideration when understanding PA habits in youth with ADHD. Various psychotropic medications could impact either behaviors related to PA or physiological responses to PA. Only one study thus far has evaluated associations between PA and medication use in youth with ADHD. Kim and colleagues (2011) explored differences in the likelihood of engaging in regular PA between medicated and unmedicated children with ADHD. They found no significant differences in PA between medicated and unmedicated children in their sample (Kim et al., 2011). However, medication use did emerge as a protective factor for obesity in children with ADHD (Kim et al., 2011), which could indirectly impact PA. Future research would benefit from further exploring to what degree PA is influenced by medication use in adolescents with ADHD.

Future research should also explore different aspects of PA and alternative ways to measure PA. There are several ways in which PA can be defined and measured, including intentional engagement in fitness-related activities (e.g., sports, exercise), engagement in activities that leads to increased energy expenditure (e.g., walking, completing chores or occupational tasks), and/or focusing on markers of high intensity exercise (Caspersen et al., 1985). There are a wide variety of aspects of PA to explore that may be relevant for youth with ADHD, as well as several different methodologies to collect this information. The current study used retrospective self-report measures, assessing both the broad definition of PA (i.e., any activity in which participants were physically active) and more intense and intentional PA (i.e., MVPA or activities that make you breath/sweat hard). Assessing MVPA in particular is common in PA research, as national PA guidelines are constructed around MVPA specifically (US Department of Health and Human Services, 2018). Importantly, accelerometry can be used to capture MVPA, and can collect information on light-intensity physical movements that occur

throughout the day (Lee & Shiroma, 2014). Both of these types of PA could be relevant for youth with ADHD but may manifest differently depending on symptomology. As previously noted, there are mixed results in the literature when examining PA behaviors of youth with and without ADHD. Inconsistent findings may reflect important differences in how PA is measured across studies (e.g., motor movements throughout the day versus specific physical activities; Zerón-Rugério et al., 2020). A notable strength of the use of accelerometry is that it does not rely on self-report and is not subject to errors or recall bias. The use of objective measures of sleep functioning (via actigraphy) has similar strengths. Sleep measured via self-report and actigraphy can capture different, but equally important, aspects of sleep functioning.

Overall, as there is limited research examining the PA habits of adolescents with ADHD, opportunities exist for future research to comprehensively examine PA from a multidimensional perspective. This could include using a large sample of diverse adolescents, who engage in a variety of levels of PA. Distinct and clinically meaningful information could be yielded by examining a combination of days of MVPA, overall PA scores, amount of light-intensity PA, as well as daily reports of PA (all assessed via both self-report and accelerometry).

Implications and Conclusion

Results from this study indicate that adolescents, particularly younger adolescents, engage in less PA than their non-ADHD peers. The findings also suggest that individual characteristics put certain adolescents with ADHD at risk for less PA, including sex and ADHD presentation. Although PA did not moderate the associations between ADHD symptoms and sleep difficulties or internalizing symptomology in the present study, ARCL models found PA to predict decreased sleep difficulties, symptoms of depression, and symptoms of anxiety. This was significant above and beyond the impact of symptoms of ADHD on these outcomes. Although

there are oftentimes barriers to engaging in PA, it can nonetheless be a more cost effective and accessible way to maintain positive health outcomes. If adolescents with ADHD are in fact at risk for engaging in less PA, then PA behaviors should be considered and thoughtfully incorporated into treatment planning by clinicians and providers who work with this population. Results suggest this may be particularly relevant for adolescents with ADHD and comorbid depression. Ensuring that adolescents with ADHD engage in healthy levels of PA is crucial, not only because of the known long-term health benefits of PA, but also to potentially protect against the development of sleep difficulties and comorbid internalizing symptomology.

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Vita

Caroline N. Lowman (née Cusick) graduated with high honors from University of Maryland, College Park in 2016. She received her Bachelor of Science in Psychology, with a minor in Neuroscience. She worked as a full-time clinical research coordinator at Cincinnati Children's Hospital from 2016 - 2018. She began the doctoral program in Clinical Psychology at Virginia Commonwealth University (VCU) in August 2018. She received a Master of Science in Psychology from VCU in May 2020. After completing her clinical internship at Children's National Hospital, Caroline will graduate with her doctorate in Clinical Psychology from VCU in May 2024.