

Do Physical Activity and Sleep Health Predict Chronic Pain in Middle Childhood?

An Examination of Direct and Moderated Effects of Health Behaviors

by

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ABSTRACT

Chronic pain is common during childhood and has negative immediate and long-term implications for physical and mental health. Prior research points to physical activity and sleep as protective health-promoting behaviors predicting lower chronic pain intensity and disability during adulthood. No study has yet examined the interaction of physical activity and sleep parameters in prediction of chronic pain in a community sample of children. Guided by the biopsychosocial model of pediatric chronic pain, this study explored objectively assessed physical activity and sleep patterns at age 8 as predictors of childhood chronic pain at age 9 in a racially/ethnically and socioeconomically diverse sample of 896 twins participating in the longitudinal Arizona Twin Project. It was hypothesized that parameters of physical activity levels and sleep health would independently predict chronic pain one year later, and that sleep parameters would moderate the association between physical activity and chronic pain. Monthly chronic pain was common, with 57.1% of participants reporting at least one pain location. Headaches, stomachaches, and backaches were the most frequent chronic pain presentations. Participants exceeded physical activity guidelines but did not meet sleep recommendations for their age group. Multilevel modeling analyses revealed that physical activity and sleep parameters at age 8 did not predict chronic pain one year later, and that sleep parameters did not moderate the associations between physical activity and chronic pain. The present study provides evidence that the associations between objectively assessed physical activity and sleep and chronic pain are not yet evident during middle childhood in this community sample compared to patient samples who have existing pain, suggesting that these health behaviors may play distinct roles in pain

development versus pain management. They also point to the need to pinpoint the time frame during which these health behaviors become relevant and potentially interact to predict chronic pain development and maintenance. Longitudinal research tracking these health behaviors and pain using both subjective and objective methods as children transition into and through adolescence can help to identify optimal developmental stages at which to target prevention and intervention efforts to promote long term health.

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Introduction

Chronic pain is defined by the International Association for the Study of Pain (IASP) as the “unpleasant sensory and emotional experience associated with, or resembling, actual or potential tissue damage” (Raja et al., 2020). Chronic pain can be recurrent (\geq three episodes within three months) or persistent (lasting \geq 6 months) and is surprisingly prevalent in children, with rates reported in youth varying between 20-40% (Goodman & McGrath, 1991; Huguet & Miró, 2008; Perquin et al., 2000; Stanford et al., 2008). Experiencing pain is one of the primary reasons parents seek medical care for their children, and around 5% of children in the general population experience moderate to severe disability due to chronic pain (e.g., Roth-Isigkeit et al., 2005; Huguet & Miro, 2008).

The most common chronic pain manifestations in children are headaches (8-83%), abdominal pain (4-53%), and musculoskeletal pain (4-40%) (King et al., 2011), and are mostly considered benign in youth community samples. In addition to having immediate repercussions for physical, mental, and social health (Hunfeld et al., 2001), childhood chronic pain often persists into adulthood (e.g., Walker et al., 2010). Therefore it is important to identify modifiable risk and protective factors that predict the development and maintenance of pain before childhood chronic pain becomes a lifelong health concern (Borsook et al., 2018). Included among the modifiable lifestyle factors that may have implications for the development of and/or recovery from chronic pain are sleep health as well as physical activity and sedentary behavior (e.g., Whibley et al., 2019; Lemes et al., 2021). This study examined the longitudinal relations among these proposed health behaviors and childhood chronic pain to assess their protective potential.

Identifying modifiable risk and protective factors for childhood chronic pain is important because experiencing pain during childhood is associated with immediate impacts on social, physical, and emotional functioning, which is reflected in overall decreased quality of life (Hunfeld et al., 2001). For example, children in pain more frequently report negative affective states, difficulties with peer relationships, interpersonal stress, and show poorer school attendance and academic performance compared to kids without pain (Groenewald et al., 2020; McKillop & Banez, 2016; Carter & Threlkeld, 2012). Children who experience chronic pain also frequently experience comorbid mental health conditions such as mood and anxiety disorders and utilize significantly more health services compared to individuals who do not experience chronic pain (Foley et al., 2021).

Beyond its proximal effects, childhood chronic pain has significant long-term negative effects on mental and physical health (Palermo, 2000). Longitudinal studies suggest that children with chronic pain do not simply grow out of pain, but that pain during childhood sets the stage for ongoing chronic pain and related disability throughout adulthood (e.g., Murray et al., 2021; Murray et al., 2020; Groenewald et al., 2020). Once affected individuals develop and maintain pain throughout extended periods of their life, it is difficult to reverse such long-lasting maladaptation. Therefore, identifying promising targets for pain prevention efforts early in development can have an important impact on children's current and future mental and physical health. According to Palermo's (2012) theoretical framework for chronic pain in children and adolescents (Figure 1), health behaviors in childhood can be integrated into the biopsychosocial model of chronic pain (Turk, 1996).

A Theoretical Framework: The Biopsychosocial Model of Chronic Pain

The traditional conceptualization of chronic pain in medicine has frequently followed a dualistic view that chronic pain is a purely physiological phenomenon that is a direct result of tissue damage. However, theory and evidence from the field of health psychology suggest that the etiology and maintenance of chronic pain is dependent on a variety of factors beyond pure physiological contributions (Gatchel et al., 2007). The biopsychosocial model provides one guiding theoretical framework for understanding chronic pain in youth (Turk, 1996; Palermo, 2012). According to this model, chronic pain and pain-related disability are multidimensional and result from a dynamic interaction between biological, psychological, and sociocultural factors (Meints & Edwards, 2018). The biopsychosocial model is a generic model used to highlight biological, psychological, and social factors and is frequently applied across the lifespan even though biopsychosocial factors may vary by stage of development. To better understand the development and maintenance of childhood chronic pain, significant efforts have been directed toward refining the model and identifying factors that are associated with increased risk of pain-related disability and overall impairment in this phase of the lifespan.

Biological risk factors have received most of the attention in chronic pain research. For example, age is a risk factor, as prevalence rates of chronic pain and severe disability are higher for older adults compared to younger age groups (Elliott et al., 1999; Thomas et al., 2004). Further, there is considerable evidence suggesting that females are at increased risk of experiencing recurrent, more frequent, severe, and longer-lasting pain compared to males, starting in adolescence (Bartley and Fillingim, 2013; Bingefors and

Isacson, 2004; Racine et al., 2012; Hampton et al., 2015), possibly due in part to hormonal changes during puberty (Kloven et al., 2017) and increased vulnerability to pain risk factors such as internalizing problems (Gutman & McMaster, 2020). Further biological risk factors include injury, which results in chronic pain due to long-lasting changes within the peripheral and central nervous systems, setting the stage for chronic pain throughout adulthood (Carter & Threlkeld, 2012).

Psychosocial factors have received less attention than biological factors in chronic pain research and are typically viewed largely as reactions to pain in medical research (Meints & Edwards, 2021). However, existing literature suggests that psychosocial factors are important contributors to chronic pain in youth. Psychological risk factors for chronic pain include psychological distress and depressive mood (Cairns et al., 2003; Jagpal et al., 2021; Pincus et al., 2002), pain catastrophizing (Vervoort et al., 2006), as well as fear of pain (Simons et al., 2011). Beyond psychological risk factors, sociocultural risk factors such as lower socioeconomic status (Prego-Domínguez et al., 2021), perceived insufficiency of social support (Nicolson et al., 2020), and interpersonal stress (Meints & Edwards, 2018) have been identified as relevant predictors of chronic pain.

Though efforts to reduce risk factors have yielded some promising potential avenues for chronic pain prevention and intervention strategies, researchers have largely neglected a strengths-based approach to childhood chronic pain. Yet taking a strengths-based approach to identify modifiable protective factors for the development of pain and related disability in youth can help further the understanding of how childhood chronic pain can be prevented beyond reducing risk (Goubert & Trompetter, 2017; Huguet et al.,

2011). There is a relative lack of high-quality research utilizing a strengths-based approach and efforts to identify factors that predict the development of chronic pain have not capitalized on the potential value of protective factors in informing pain intervention and prevention programs for child health in general, and chronic pain in particular. In the current absence of evidence-based strategies for prevention or intervention, prevalence rates of child chronic pain appear to have increased over time (Gatchel et al., 2007; Tutelman et al., 2021).

The primary challenge of preventing pain chronification (i.e., progression from acute to chronic pain) in children is reorienting their attention to the engagement in activities that promote health and enhance daily quality of life (Andrews et al., 2016). It is likely that resilient youth and their caregivers are successful in reorienting behaviors towards such health-promoting activities, perhaps because coping styles and emotion regulation capacities allow them to engage in goal-directed activities that promote their health and well-being (Fisher & Palermo, 2016; Iddon et al., 2019). From a resilience perspective, acute pain management and coping efforts should focus both on recovery from pain and its emotional and cognitive effects, as well as on the sustainability of daily life by engaging in health-promoting, meaningful activities.

Two health behaviors that have been linked to sustained health over time are physical activity and sleep (The World Health Organization, 2022; International Association for the Study of Pain, 2021; National Sleep Foundation, 2022). Low levels of physical activity, high levels of sedentary behavior, and sleep disruptions have often been conceptualized as risk factors for the development of chronic pain (Chalkiadis, 2001), and there is also evidence in adult populations that high quantity and quality sleep as well

as physical activity may be protective against the development of chronic pain. As widely used as the biopsychosocial model of chronic pain is for defining risk and protective factors, health-promoting behaviors do not easily fit in any distinct category of this theoretical framework as they are arguably per se a blend of biological, psychological, and social influences (Palermo, 2012). Though they have thus far received relatively little empirical attention in the context of childhood chronic pain prevention and recovery, accruing evidence suggests that both physical activity and sleep are associated with chronic pain (e.g., Andrews et al., 2014). They are also modifiable health behaviors in youth (e.g., Stone et al., 1998; Åslund et al., 2018), thus placing them in the center of plausible targets for the prevention of chronic pain in children.

Physical Activity and Chronic Pain

The World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) identified insufficient physical activity as a leading risk factor for noncommunicable diseases and deaths worldwide (World Health Organization, 2022; Centers for Disease Control and Prevention, 2020; Carlson et al., 2018). Physical activity is defined as any bodily movement that results in energy and caloric expenditure following activation of skeletal muscles (Caspersen, 1985), and can be categorized as sedentary behavior (i.e., resting, sitting), as well as light (i.e., slow 2 mph walk), moderate (i.e., 3 mph brisk walk, 88 bpm stair climbing), and vigorous activity (i.e., 4 mph running, bicycling, 126 bpm jumping jacks). Aerobic exercise (e.g., physical activity that involves rhythmic movement of large muscles) has been linked to successful prevention and management of pediatric chronic pain (Hoffart & Wallace, 2014; U.S. Department of Health and Human Services, 2008). While some clinical and therapeutic

guidelines for treatment of chronic pain include recommendations for rest and inactivity when recovering from acute pain, the WHO recommends at least 60 minutes of moderate-to-vigorous physical activity daily, which may have specific benefits for reducing the likelihood of developing chronic pain (World Health Organization, 2022).

From a biological perspective, physical activity decreases risk for diseases in the musculoskeletal and cardiovascular systems through physiological mechanisms (Joyner & Green, 2009). Physical activity is also moderately influenced by genetics (Moore-Harrison & Lightfoot, 2010) with some evidence suggesting that age and sex are moderators of heritability (Moore-Harrison & Lightfoot, 2010). Further, the endorphin hypothesis posits that β -endorphins, which are endogenous opioids, are released and bind to receptor sites in the brain following physical exercise, which results in mood elevations and reduction in anxiety symptoms (Anderson & Shivakumar, 2013). Endogenous opioid activity has also been found to be associated with elevated positive affective states and reductions in pain (Harber and Sutton, 1984; Morgan, 1985; North et al., 1990; Thorén et al., 1990). Beyond biophysiological mechanisms, there are also psychosocial effects of physical activity. For example, physical activity has been linked to lower reports of pain risk factors such as depressive symptoms in children and adults (McKercher et al., 2009) as well as social benefits such as higher exposure to social interactions and teamwork (Duncan et al., 2005). Some studies further suggest that youth in pain who engage in routine aerobic activity report fewer health complaints, higher life satisfaction and better self-reported health (Swain et al., 2015).

Besides long-term benefits of physical activity, there is also evidence indicating that there are day-to-day bidirectional associations between physical activity and chronic

pain in both youth and adults, such that low physical activity increases risk for the development and exacerbation of chronic pain and chronic pain increases risk for low engagement in physical activity (Rabbitts et al., 2014). Rabbitts and colleagues (2014) examined the day-to-day relationships between pain and actigraphy-assessed activity in youth both with and without chronic pain. They found that higher pain intensity predicted reduced next-day vigorous physical activity for both healthy youth and youth with chronic pain, whereas higher physical activity levels predicted lower pain intensity at the end of the day only for youth with chronic pain. This suggests that youth who experience high pain intensity limit their physical activity, but also that higher levels of physical activity are associated with decreased levels of pain for those who are already experiencing chronic pain.

Landmark and colleagues (2013) extended these findings in adults using a longitudinal design by drawing on data from the HUNT Pain Study, which is a large population-based Norwegian study examining illness and health-related lifestyle factors in adults longitudinally since 1984 (Krokstad et al., 2013). They found that individuals who self-reported moderate to vigorous levels of physical activity at the baseline assessment experienced reduced intensity of pain one year later. Further, in a previous assessment from the HUNT study, low levels of physical activity predicted higher prevalence of chronic pain eleven years later and consistent levels of moderate activity were associated with more than 50% lower prevalence of chronic pain being present (Holth et al., 2008). These data suggest that a high level of physical activity is a potential protective factor against the worsening of chronic pain among adults. A recent meta-analysis by De Campos (2021) reviewed the effectiveness of prevention strategies to

reduce future negative impacts of low back pain in adults and found that combining exercise and psychoeducation is effective for reducing future pain intensity and associated disability. Taken together, these findings suggest that physical activity may reap benefits beyond the treatment of chronic pain and may also be relevant for the prevention of pain chronification, at least in adults. However, little high-quality research has examined the longitudinal associations between physical activity and chronic pain and potential protective role of physical activity in community samples of youth.

In contrast to the view that high levels of physical activity are always beneficial for health and chronic pain, some studies examining the new onset of chronic pain have identified vigorous activity to be a risk factor for the development of chronic pain. For example, Jones and colleagues (2003) examined predictive factors of children's self-reported widespread body pain in English children and found that subjectively reported high levels of sports activity was positively correlated with widespread pain at the baseline assessment and predicted new onset of widespread pain at the one year follow up assessment. Further, Paananen and colleagues (2010) examined predictive factors of chronic pain in a birth cohort of Finnish adolescents and found that female participants who self-reported being highly physically active experienced a higher incidence of persistent multiple musculoskeletal pains two years later, whereas sedentary behavior predicted higher 2-year persistence and number of musculoskeletal pains in male but not female participants.

Most of the research on children's physical activity has focused on self- or proxy-reported activity. However, children's self-report is constrained by their attention and awareness of different activity levels throughout the day. Some studies suggest that there

are discrepancies between parents' and children's reports of the child's physical activity and sedentary behavior, reporting weak to no correlations between parent-report and actigraphy assessed sedentary behavior and moderate activity (Sarker et al., 2015; Sumonja & Jevtic, 2019). In contrast to self-report, objective assessments provide a more reliable and valid estimate of physical activity (Alfano et al., 2015); however, objective assessments are less often used in pain-related research in children and existing research is therefore limited.

Wedderkopp and colleagues (2009) objectively measured physical activity using accelerometry in 361 healthy Danish children recruited from state schools in Denmark when they were 9 and 12 years old. For children who experienced pain at age 9, there was no significant relationship between physical activity and back pain three years later. In contrast, longitudinal analyses revealed a significant association between low activity at age 9 and increased low back pain at age 12 in children who did not experience back pain at the baseline assessment. These findings suggest that while physical activity may not be related to reductions in existing chronic back pain, high levels of physical activity in healthy children may protect against incident back pain being present three years later.

Despite the lack of research in youth examining whether physical activity protects against the onset of chronic pain, a closer look at proposed underlying mechanisms between physical activity and chronic pain suggests that activity is a plausible protective factor for the onset of chronic pain. For example, aerobic exercise influences hormone secretion through physiological pathways that function as biological buffers for chronic pain (Deslandes et al., 2009; Lima et al., 2017), as the improved coronary blood flow and enhanced cardiac function resulting from aerobic activity have been linked to improved

mood and cognition (Nocon et al., 2008; Silverman & Deuster, 2014). Further, physical activity optimizes stress reactivity through the HPA axis and the sympathetic nervous system, which results in reductions of emotional, physiological, and metabolic reactivity to stress. It thereby protects against maladaptive effects of behavioral and metabolic responses to stress, which prevents the potential onset of chronic illness (Silverman & Deuster, 2014). In contrast, low physical activity and high sedentary behavior are associated with reduced muscle strength and lower bone mineral content, which contribute to chronic pain in youth (Wedderkopp et al., 2009), and is therefore often conceptualized as a risk factor for the development of chronic pain.

In summary, there is evidence that physical inactivity and sedentary behavior are associated with increased risk of chronic pain during childhood. While physical activity is often highlighted as a treatment or target of intervention for chronic pain, data from more recent studies point to high levels of physical activity as a potential protective behavior that could be utilized as a target of prevention in children (e.g., De Campos et al., 2021). Physical activity has frequently been measured using self-report or proxy-report in children; however, more recent studies highlight the utility of objective actigraphy assessments, which provide a more accurate and reliable assessment of physical activity in youth compared to self- or proxy-report. The links between physical activity and pediatric chronic pain have been evaluated in samples that present with a variety of pain diagnoses; however, the links between physical activity and chronic pain have rarely been examined in community samples of youth who have not been selected based on their chronic pain status.

Further, most existing studies have examined physical activity as sedentary behavior versus moderate-to-vigorous activity. Paying closer attention to the distinction between light, moderate, and vigorous activity may advance the understanding of how physical activity levels relate to the onset of chronic pain in children. The distinction between activity levels is important, as a small body of literature points at extreme levels of physical activity being associated with negative health outcomes (e.g., Eijsvogels et al., 2018). Further, there appear to be important sex differences relevant for the relationship between physical activity and chronic pain which should be further explored (Umeda & Kim, 2019). Not only physical activity, but also other health-promoting behaviors, particularly sleep, are relevant for pain.

Sleep Health and Chronic Pain

Sleep is critically important for cognitive, psychological, and physical functioning, and poor sleep has been linked with compromised emotional and behavioral functioning in healthy children and youth with persistent pain (O'Brien & Gozal, 2004; Owens, 2009; Taras & Potts-Datema, 2005). While there are a variety of definitions of sleep quality and other isolated domains of sleep (e.g., Ohayon et al., 2017), sleep health is an infrequently used term to describe the summary of sleep characteristics. Buysse (2014) provides one definition of sleep health in youth that describes it as a “multidimensional pattern of sleep-wakefulness, adapted to individual, social and environmental demands, that promotes physical and mental well-being.” Characteristics of good sleep health include adequate duration, high efficiency (ratio of total sleep time to time spent in bed), appropriate timing, regularity, subjective satisfaction, and sustained alertness during waking hours, though clear recommendations only exist for sleep

duration. According to the American Academy of Sleep Medicine, children aged six to twelve years should sleep between nine and twelve hours per day to promote optimal health (Paruthi et al., 2016). Instead of investigating sleep domains such as sleep duration in isolation, studying all domains of sleep health may provide a more comprehensive understanding of how different sleep characteristics contribute to health outcomes.

Sleep is commonly assessed via subjective assessments, including questionnaires regarding general sleep habits and diary reports on the previous night's sleep timing and quality (Fabbri et al., 2021). However, these methods are limited in their ability to accurately assess sleep in children (Alfano et al., 2015); children are too young to self-report on their sleep and parents may not have an adequate understanding of their children's sleep quality and related difficulties and tend to overestimate their child's sleep duration (Perpétuo et al., 2020). In contrast, using objective methods of sleep assessment removes some of the problems of self-reports, particularly for children. Methods to objectively assess sleep include polysomnography, non-REM EEG frequency spectral analysis, and actigraphy. Actigraphy is one of the most prominent methods to objectively measure sleep in children, as it has relatively low burden to the user and is inexpensive, allowing data from large cohorts to be collected for relatively long periods of time. It involves wearing a device that measures movement as a proxy for wakefulness (Acebo & LeBourgeois, 2006). Therefore, actigraphy is the objective method most suited to measure sleep quality outside of a laboratory. One shortcoming of actigraphy is that activity is only a proxy for sleep and not sleep itself. The most reliable and validated measure of sleep with actigraphy is total sleep time, and the best-supported use of actigraphy is to characterize the sleep/wake cycle rhythm over multiple days. For use as

an indicator of sleep, actigraphy is best thought of as a measure of sleep quantity or timing rather than as a measure of the quality or depth of sleep.

Unlike adults who have substantial control over their sleep health, children and adolescents live within a complex and dynamic system that exerts significant influence over their sleep health over which they have very limited control. This system is shaped by biopsychosocial influences, which are particularly important to consider when examining sleep in children. Biological factors include hormone secretion to maintain homeostatic sleep-wake cycles and circadian rhythms (Gnocchi & Bruscalupi, 2017; Borbély et al., 1982). Further, there is a strong genetic influence on sleep-wake patterns during middle childhood, with genetic contributions being highest for sleep initiation and maintenance compared to sleep timing (Sletten et al., 2013). Psychosocial contributions to sleep include family, peer, academic and mental health factors (Becker et al., 2015). Family factors are important to consider when examining sleep in children, as parents play an important role in structuring their children's health behaviors. For example, a chaotic family environment (e.g., parent marital problems, sibling conflict) may be detrimental to a child's sleep functioning (Breitenstein et al., 2018). Further, findings from a representative diary study of 2,400 US youth suggest that stricter household rules are associated with earlier bedtimes and longer parent-reported sleep duration on weekdays (Adam et al. 2007). It is further well-established that sleep and mental health are intertwined in children (Astill et al., 2012), and sleep disturbances are considered symptoms of several psychiatric disorders according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013).

The links between sleep and psychiatric disorders also point to the link between sleep and chronic pain. Mental health concerns such as internalizing symptoms are a risk factor for chronic pain, and there is also substantial evidence suggesting that children who experience poor sleep also commonly report experiencing chronic pain (Harrison et al., 2016). In a population-based study of 3812 Australian children, self-reported sleep difficulties at age ten were associated with a higher risk of developing persistent pain two to three years later (Incedon et al., 2016). A small diary study of children with sickle cell disease (Valrie et al., 2007) provides further evidence that high pain intensity predicts poor sleep quality and that poor sleep quality in turn predicts high pain the following day. Similar diary studies of children with juvenile idiopathic arthritis (Bromberg et al., 2012) and adolescents with persistent pain (Lewandowski et al., 2010) suggest that although pain intensity did not predict sleep quality, poor sleep quality was a predictor of high pain intensity the following day. In contrast, a prospective study of children and adolescents with headaches (Connelly & Bickel, 2011) found that although a large percentage (68%) of the sample identified the subjective feeling of a lack of sleep as a headache trigger, objective measurements of shorter sleep duration and the increased number of night awakenings did not predict subsequent headache occurrences two weeks afterwards.

Palermo and Kiska (2005) found that subjectively reported sleep disturbances are related to pain symptoms among youth experiencing chronic pain, which in turn also related to reduced daily functioning and lower quality of life. Palermo and colleagues (2011) extended their findings in a follow-up study and found that youth with chronic pain reported significantly lower sleep efficiency and more frequent waking at night using actigraphy assessment compared to healthy counterparts. There is further evidence

that insufficient sleep contributes to increased pain sensitivity and may initiate pain episodes in adults (Lautenbacher et al., 2006; Smith et al., 2007), whereas high quality sleep promotes immune system function that circumvents systemic inflammation that is often present for individuals with chronic pain symptoms (Motivala & Irwin, 2007). Some cross-sectional studies suggest that most children with chronic pain conditions experience clinical levels of proxy-reported sleep disturbances compared to community populations, which has a profound effect on daily functioning (Long et al., 2008; Meeske et al., 2006). Despite this evidence highlighting the importance of gaining sufficient high-quality sleep, almost 40% of school-aged children do not meet the National Sleep Foundations' recommendations for sleep duration specific to their age group (Centers for Disease Control & Prevention, 2020). This pattern only worsens as children transition from childhood to adolescence, with studies suggesting that only 5% of high school students (3% of girls, 7% of boys) in the United States meet the recommended amounts of sleep (Hirshkowitz et al., 2015).

These observed relations between pain characteristics and sleep health are often described as bidirectional. Studies using both behavioral assessments (e.g., Bloom et al., 2002; Butbul et al., 2011) and polysomnography (e.g., Armoni Domany et al., 2019; Olsen et al., 2013) in children with persistent pain generally identify bidirectional relationships between high pain levels and disrupted sleep. In addition, even after controlling for other factors, high pain is predictive of disrupted sleep patterns. For example, a study of children with chronic headaches revealed that after controlling for age, sex, and race, longer headache duration predicted higher sleep anxiety and more bedtime resistance on the Children's Sleep Habits Questionnaire (CSHQ; Miller et al.,

2003). More frequent headaches than predicted symptoms of parasomnia, sleep walking, and bruxism (i.e., the clenching or grinding teeth during sleep). These bidirectional associations are frequently reported for children with already existing pain, but it is unclear whether high quality and quantity sleep may also protect against the onset of chronic pain in children.

In summary, the available literature highlights the importance of experiencing sufficient high-quality sleep for chronic pain management and prevention. Most of the literature examines the association between sleep and chronic pain in adults; the existing knowledge on this association in children is based on few studies that used low-quality methods to measure sleep health. Existing research on self- and proxy-reported sleep disturbances and insufficient sleep suggests that sleep problems are frequently associated with negative health outcomes and may be risk factors for the development of chronic pain during childhood. While there is a bidirectional association between sleep and pain, there is strong evidence that poor sleep may be contributing to worsening of pain in individuals who are already experiencing chronic pain. Similar to physical activity, the available literature highlights the utility and validity of objective sleep assessments using actigraphy compared to subjective or proxy reports. Further, health behaviors are often evaluated in isolation or as additive factors predicting health outcomes. However, health promoting behaviors do not occur in isolation in real life. Rather, as suggested by the biopsychosocial model that outlines the dynamic nature of biopsychosocial influences in prediction of health outcomes, health behaviors co-occur and interact to predict health outcomes. Examining the interaction of health behaviors can enhance our understanding

of how to optimize health-promoting behaviors for the prevention of chronic pain in youth.

Interaction of Health Behaviors

Typically, specific health behavior domains are considered either in isolation or as additive factors, but health behaviors may in fact interact as predictors of health outcomes. As outlined above, two health behaviors that may combine to influence health, including the risk for pain, are physical activity and sleep. In adult samples, there is evidence that sleep disturbances and physical inactivity interact to predict the onset of pain two years later for individuals aged fifty and older (Whibley et al., 2020). Whibley and colleagues (2020) observed an association between physical activity and decreased likelihood of the onset of pain; however, this association was only observed for individuals who reported low levels of sleep disturbances. While no studies have examined the interactive relation between sleep and physical activity in the context of childhood chronic pain, the interactive relation has been examined in the context of risk factors for developing chronic pain, such as internalizing symptoms. For example, Ogawa and colleagues (2019) examined the interactive effect of subjectively assessed sleep duration and physical activity on internalizing symptoms in youth and found that there were main effects of sleep duration and amount of physical activity predicting internalizing symptoms. In addition, they also found a significant interaction of sleep and physical activity suggesting that meeting the recommended amounts of sleep compensated for deficits in physical activity.

Gillis and El-Sheikh (2019) further examined self-reported physical activity as a moderator of risk for actigraphy-assessed sleep over one week and psychological

symptoms in youth and found that sleep and physical activity interacted to predict internalizing and externalizing symptoms. More specifically, both poor-quality and short-duration sleep interacted with low levels physical activity to predict high internalizing and externalizing problems, which other studies have found to be comorbid with chronic pain (Vinall et al., 2016). Their study also provided further evidence that higher levels of physical activity may serve as a protective factor for maladaptive psychological outcomes, and vice versa, that high quality sleep may buffer against the negative effects of physical inactivity. Taken together, these findings suggest that sleep and physical activity may interact to predict pain among youth and that the combined effects of health behaviors should be evaluated.

The Present Study and Hypotheses

The available literature suggests that health-promoting behaviors such as physical activity and sleep are efficacious for the prevention of chronic pain in adult populations. Moreover, previous research has aimed to address how sleep and physical activity interventions impact chronic pain outcomes in adults (e.g., Whibley et al., 2021) and have established the importance of physical activity and sleep as predictors of physical and psychological function of adults with chronic pain (Geneen et al., 2017; Zambelli et al., 2021). However, research examining the role of such health-promoting behaviors for chronic pain prevention in children is relatively scant. Existing research on children predominantly includes self- and proxy-report assessments of childhood health-promoting behaviors is often retrospective and lacks sufficient attention to detail in highlighting the important nuances within each health behavior. Further, despite evidence that both sleep and physical activity are important predictors of chronic pain (Andrews et

al., 2014; Tang & Sanborn, 2014), particularly for female adults in pain (Fillingim et al., 2009; Keogh & Eccleston, 2006), no study has yet examined the interaction of these health-promoting behaviors in prediction of chronic pain in children. Thus, the proposed study aimed to extend the existing literature by utilizing objective actigraphy assessments to examine how the health-promoting behaviors of physical activity and sleep relate to next-year onset, maintenance, and worsening of childhood chronic pain. Further, the interaction of objectively assessed physical activity and sleep health to predict childhood chronic pain was explored. The current study aimed to test the following hypotheses in a community sample of twin children:

Aim 1. The first aim was to assess objectively measured physical activity and sleep domains at age 8 as independent predictors of next-year chronic pain.

Hypothesis 1a: It was hypothesized that sedentary behavior will show a positive linear relation with chronic pain, such that children who engage in higher levels of sedentary behavior will experience next-year onset, maintenance, and worsening of chronic pain.

Hypothesis 1b: It was hypothesized that higher levels of moderate and vigorous activity will predict lower levels of subsequent chronic pain. It was expected that moderate activity will have a negative linear relationship with chronic pain. In addition, it was explored whether the relationships between vigorous activity and chronic pain are curvilinear, as some literature suggests that extreme levels of vigorous activity are related to higher levels of chronic pain (e.g., Paananen et al., 2010; Eijsvogels et al., 2018).

Hypothesis 1c: It was hypothesized that longer sleep duration and efficiency will predict lower subsequent chronic pain. In addition, it was explored whether the relationship between sleep duration and chronic pain is curvilinear based on data linking too much

and too little sleep with poor health outcomes (e.g., Pasch et al., 2012; American Psychiatric Association, 2013), and whether less explored sleep domains such as sleep latency and midpoint variability will show a linear relation with chronic pain.

Aim 2. The second aim was to determine whether the interaction between sleep and physical activity at age 8 is associated with age 9 differences in childhood chronic pain (i.e., onset, maintenance, and worsening). It was expected that sleep health domains will be significant moderators of the relation between physical activity and next-year chronic pain (Figure 2). Two competing interaction models were tested:

Hypothesis 2a: It was hypothesized that the non-compensatory model will yield the best fit with our data, such that the relation between health-promoting patterns of activity (i.e., higher levels of physical activity, lower levels of sedentary behavior) and lower levels of pain will be stronger among children who have better versus poorer quality of sleep (Figure 3).

Hypothesis 2b: It was hypothesized that the compensatory model will yield the best fit with our data, such that the relation between health-promoting patterns of activity (i.e., lower levels of physical activity, higher levels of sedentary behavior) and lower levels of chronic pain will be stronger among children who have poorer versus better quality of sleep (Figure 4).

The firmest predictions could be made regarding the relations between pain and sedentary behavior, moderate-to-vigorous activity, sleep duration, and sleep efficiency; however, exploratory analyses were also conducted to disaggregate other physical activity levels and sleep metrics as possible predictors and moderators in the relationship with pain.

Due to evidence in US samples suggesting higher prevalence rates and reports of pain and sleep problems in females (e.g., Fillingim et al., 2009; Keogh & Eccleston, 2006), exploratory analyses were conducted to examine whether sex moderates the relationship between physical activity, sleep health, and childhood chronic pain.

Method

Participants

This study utilized the Arizona Twin Project sample, which is a community-based cohort of twin pairs and their primary caregivers who were recruited via birth records and followed longitudinally since they were ten months of age ($N=896$ children; Arizona Twin Project; Lemery-Chalfant et al., 2013). Data for the current study was collected in 2017. Demographic characteristics of the sample are presented in Table 1. At the age 8 visit ($M_{age} = 8.43$ years, $SD = 0.68$), participants were 51.2% female, and the sample consisted of 58.8% non-Hispanic White/Euro American, 23.7% Hispanic/Latino, 3.4% Black/African American, 3.4% Asian/Asian American, 2.7% Native American, 0.9% Native Hawaiian, and 6% bi-/multi racial twins. Family incomes ranged from 7.4% of families living in poverty and 22.3% living near the poverty line, to 16.3% being lower to middle class and 53.9% being middle to upper class. Primary caregiver education ranged widely; 0.6% had less than high school education, 9.2% had a high school or equivalent education, 26.9% had some college education, 36.6% had a college degree, and 23.1% had a graduate or professional degree.

Procedure

Approval by the Institutional Review Board was obtained prior to data collection for both phases of this study. Written informed consent was provided by primary

caregivers prior to all assessments and all participating twins verbally assented at each wave of data collection. For the assessments of this study, interested and eligible families were enrolled in a study wave focused on sleep and health (i.e., age eight assessment), which included intensive assessments of daily activities such as physical activity and sleep, as well as the twins' home environment and indicators of health such as chronic pain. At the eight-year assessment, data was collected via surveys, two home visits, and one week of daily assessments, which entailed objective measurement of physical activity and sleep using actigraphy wrist-worn accelerometers and daily diaries.

During the first home visit, trained research assistants administered questionnaires, took biological assessments, facilitated cognitive and interaction tasks, and explained actigraphy and daily diary procedures to be completed during the week-long study period following the home visit. The study week entailed objective measurements of daily physical activity and sleep, as well as daily diaries each night for seven consecutive days and assessment tables tracking bedtimes and waketimes that were completed for each twin separately by the primary caregiver.

Throughout the study week, research assistants contacted the twins' parents every morning to address any questions and ensure that actigraphy measurements and daily diary procedures were followed adequately. Within a week after completion of the study week, research assistants returned for a second home visit and retrieved actigraphy watches and paper daily diaries. Approximately one year later, families were contacted to participate in an annual follow-up assessment (i.e., age nine assessment), which entailed a single home visit and completion of questionnaires by caregivers and twins. At this home visit, trained research assistants administered questionnaires, took biological assessments,

and both twins and caregivers reported on their health-related symptoms. Families received monetary compensation for participation at each wave of data collection.

Measures

Objective Physical Activity. Physical activity was measured objectively using actigraphy at age eight. The twins were instructed to wear the wrist-based accelerometer (Motion Logger Micro Watch, Ambulatory Monitoring, Inc., Ardsley, NY, USA) on their non-dominant wrist in a free-living standard environment for seven consecutive days and nights except during water-based activities and high contact sports that might damage the accelerometer. Validation studies have revealed that accelerometer data from wrist-based accelerometers such as the Motion Logger Micro Watch are a valid and reliable measure of activity in middle childhood if the accelerometer is worn for four or more days in a free-living environment (Barreira et al., 2015; Crouter et al., 2016). Accelerometers were initialized and assigned to twins using Micrologger Microwatch software.

Motion during waking hours was measured in one-minute epochs and activity was quantified using the proportional integration measure in the Sadeh algorithm on ActionWare-2 (Version 2.7.1; Ambulatory Monitoring). Participants with less than three days of actigraphy data were excluded from analyses. As identified in validation studies of physical activity thresholds for activity in youth, physical activity thresholds and intervals were set based on age-specific cut points for youth to estimate physical activity (Duncan et al., 2020) and adjusted from 15-second to 60-second epoch length. Daily sedentary behavior (i.e., engaging in activities requiring very low energy expenditure such as resting or sitting) was identified as less than 100 acceleration counts (< 1.5 Metabolic equivalent of task units (METs)). Light activity (i.e., engaging in low energy

expenditure such as a slow 2 mph walk) was identified as less than 2292 acceleration counts (1.5 – 2.99 METs). Moderate activity (i.e., brisk 3 mph brisk walk or 88 bpm stair climbing) was identified as less than 4011 acceleration counts (3 – 5.99 METs). Vigorous activity (i.e., 4 mph running, bicycling, 126 bpm jumping jacks) was identified as more than 4012 acceleration counts (> 6 METS).

Objective Sleep. Objective actigraphy data to assess sleep were collected at age eight using the Motion Logger Micro Watch, a wrist-based accelerometer (Ambulatory Monitoring, Inc, Ardsley, NY USA) which was worn on the children’s non-dominant wrist for seven days and nights. Activity during bedtime and night hours was measured in 60-second epochs using a zero-crossing mode, and periods of sleep and waking were determined using the Sadeh algorithm in ActionWare-2 (Version 2.7.1; Ambulatory Monitoring). Actigraphy data is a nonintrusive and reliable measure of objective sleep parameters within a child’s free-living environment when sleep data are collected over at least five consecutive nights and validated by subjective sleep diaries (Acebo et al., 1999). Bedtimes were cross-validated by primary-caregiver daily reports of their children’s bedtime using a bedtime assessment table. To determine the time when a child attempted to sleep and first fell asleep, parent-report of their children’s bedtime was used in conjunction with actigraph-detected ambient light and movement during bedtime hours. Participants with less than three nights of actigraphy data were excluded from analyses.

Extracted objective sleep parameters included sleep duration, efficiency, latency, and sleep midpoint variability. Sleep duration was defined as total time spent asleep (in hours) excluding periods of wakefulness and latency during the total sleep period. Sleep

efficiency was defined as the ratio of time spent asleep to total time spent in bed (in hours), with total time in bed including time spent asleep, bouts of wakefulness, and latency prior to sleep onset. Latency was defined as the amount of time spent in bed (in minutes) before falling asleep. Sleep midpoint variability was defined as the variability of the halfway point between waketime and bedtime. Bedtime was determined by a combination of parent-reported of when their child went to bed, in conjunction with actigraph-detected light in the room and motion during bedtime.

Pediatric Chronic Pain. Childhood chronic pain was defined as any report of child pain occurring at least monthly or more and was assessed at both the 8-year and 9-year assessments. Parents reported on their twins' pain at up to 5 different body locations, including headache, stomachache, lower back pain, widespread pain, and temporomandibular pain. Parents rated child pain on a 1 (never/rarely) to 5 (about every day) scale. The assessments of child subclinical pain were recoded, such that any pain sites endorsed as occurring at least "once a month or more" were given a value of "1" whereas any sites of pain rated as "rarely/never" were coded as "0". Previous work has established the validity of assessing childhood chronic pain in body sites using this 1-5 scale in children (Mikkelsen et al., 1997; Stanford et al., 2008). A summary score of all possible nonclinical pain locations occurring monthly or more was created for each twin. At the age 9 assessment, primary caregivers also completed survey assessments of their twin's clinical levels of pain for twins who experienced any monthly pain. Clinical pain was assessed through self-report for five locations for each twin, including headache, stomachache, lower back pain, widespread pain, and temporomandibular pain (Dionne et

al., 2008; Drossman, 2016; Dworkin et al., 1992; Headache Classification Subcommittee of the International Headache Society, 2004; Wolfe et al., 2010).

Data Analysis

Prior to conducting main analyses for the study, person-level sleep (mean sleep duration, efficiency, latency, and midpoint variability) and physical activity (sedentary behavior, light activity, moderate activity, vigorous activity) parameters were aggregated within person to create mean scores for each twin. Skewness and kurtosis of main study variables and potential covariates (i.e., vacation, child sex, age, race, ethnicity, BMI, and family SES) were examined, and skewed and kurtotic variables (i.e., skewness > 2.0, kurtosis > 7.0) were normalized and transformed prior to analyses. Outliers were winsorized to three standard deviations above or below the mean. Next, descriptive statistics for and intercorrelations among study variables were computed.

After computing mean activity and sleep scores across the week, there are two remaining possible sources of variance in this sample of twins nested within families that could lead to clustering: differences between individuals within the family (Level 1) and differences between families (Level 2). Clustering on the family level is expected for physical activity and sleep domains and was therefore accounted for in analyses. Multilevel modeling to test study hypotheses was conducted in MPlus (Version 7.0; Muthén & Muthén, 2012) using the *type=complex* command for error correction due to the nested structure of the sample. Analyses were conducted to examine the main effects and interactions between person-level average sleep and physical activity measures in relation to childhood chronic pain. The focus of the current study was on the phenotypic associations between health-promoting behaviors and chronic pain, with the unique

advantage that only a few community samples of children have included objective actigraphy data among community-dwelling youth. Thus, although twin data were available, quantitative genetic models were not fit as part of this study.

Results

Missing Data

Out of the 749 participants who participated in one or both age 8 and age 9 assessments, data were available for 438 twins (48.9%) who participated in both waves, whereas 99 twins (11%) participated at the age 8 assessment only, and 212 twins (23.7%) participated at the age 9 assessment only, with new participants being recruited for the age 9 assessment. Chi-square and one-way ANOVA analyses of variance indicated that participation across the three groups did not differ based on race, ethnicity, or SES (all p s > 0.05). However, missing data were non-random in this sample, given that actigraphy assessments of health behaviors were only conducted for in-state participants who were able to receive study materials in the mail. Full information maximum likelihood (FIML) estimation in MPlus was used to handle missing data.

Descriptive Statistics and Analysis of Covariates

All sleep variables (i.e., sleep duration, efficiency, latency, and midpoint variability) as well as vigorous physical activity were winsorized to three standard deviations above and below the mean, and sleep latency was further log transformed. Descriptive statistics for all main study variables and covariates are depicted in Table 1. When examining descriptive statistics of physical activity variables, it was apparent that light activity absorbed most of the variance we would expect to see for sedentary behavior. This may be due to methodological limitations associated with applying small

threshold differences between sedentary behavior and light activity in actigraphy data. Given that the currently available literature most frequently includes light activity within sedentary behavior, sedentary behavior and light activity were combined in all subsequent analyses and are henceforth referred to as sedentary behavior.

At the age 8 assessment, children's sleep duration was on average 8 hours and 7 minutes ($M = 8.12$, $SD = 0.71$) per night, and around 90% of the time spent in bed was spent asleep ($M = 90.07$, $SD = 5.47$). On average, it took participants around 21 minutes to fall asleep ($M = 2.86$, $SD = 0.63$) and the participants' sleep midpoint varied by 34 minutes ($M = 0.57$, $SD = 0.29$). Further, per 24 hours of actigraphy assessment excluding sleep periods, participants engaged in 10 hours and 30 minutes of sedentary behavior ($M = 10.48$, $SD = 2.34$), one hour and 57 minutes of moderate activity ($M = 1.96$, $SD = 1.15$), and 55 minutes of vigorous activity ($M = 0.91$, $SD = 0.81$). On average, twins reported one area of monthly recurring pain at the age 9 assessment ($M = 1.08$, $SD = 1.22$), with 42.9% indicating no pain areas, 26.6% indicating one pain area, 16.8% indicating two pain areas, 8.7% indicating three pain areas, 3.5% indicating 4 pain areas, and 1.5% indicating 5 pain areas.

Table 2 shows the intercorrelations among all study variables. Age 9 monthly recurring pain was not related to age 8 sedentary behavior ($r = -0.04$, ns), moderate activity ($r = 0.04$, ns), or vigorous activity ($r = 0.03$, ns). Similarly, age 9 monthly recurring pain was not related to age 8 sleep duration ($r = -0.03$, ns), sleep efficiency ($r = -0.03$, ns), sleep latency ($r = -0.03$, ns), or sleep midpoint variability ($r = -0.04$, ns). Higher sedentary behavior was significantly correlated with shorter sleep duration ($r = -0.18$, $p < 0.01$), higher sleep efficiency ($r = 0.11$, $p < 0.01$), longer sleep latency ($r = 0.14$, $p <$

0.01), and higher sleep midpoint variability ($r = 0.17, p < 0.01$). Moderate activity was not significantly associated with sleep duration ($r = -0.05, ns$), sleep latency ($r = -0.07, ns$), and sleep midpoint variability ($r = -0.07, ns$); however, there was a significant negative correlation between moderate activity and sleep efficiency ($r = -0.14, p < 0.01$). Vigorous activity was not significantly associated with sleep duration ($r = -0.03, ns$), sleep efficiency ($r = -0.08, ns$), or sleep latency ($r = -0.05, ns$), or sleep midpoint variability ($r = -0.07, ns$). Point-biserial correlations revealed that vacation status was not significantly associated with vigorous activity ($r = -0.05, ns$), sleep duration ($r = -0.01, ns$), sleep efficiency ($r = -0.04, ns$), sleep latency ($r = 0.05, ns$), and sleep midpoint variability ($r = -0.08, ns$), though children on vacation engaged in significantly less sedentary behavior ($r = -0.15, p < 0.01$) and moderate activity ($r = -0.23, p < 0.01$) compared to those who were not on vacation during the assessment.

Preliminary analyses included bivariate correlations between age 9 monthly recurring pain and individual potential covariates (i.e., vacation, child age, sex, ethnicity, BMI, and family SES). Age 9 recurring pain was not related to age 8 family SES ($r = -0.04, ns$), age ($r = 0.04, ns$), or BMI ($r = 0.05, ns$). Point-biserial correlations were conducted to examine the association between chronic pain and sex as well as vacation status and revealed that chronic pain was not significantly correlated with child sex ($r = 0.03, ns$) or vacation status ($r = 0.08, ns$). A one-way ANOVA revealed that chronic pain did not significantly differ based on race/ethnicity, $F(10, 721) = 1.46, p = 0.15$.

Thus, of the proposed covariates, only sex and SES were included in final analyses due to the known sex differences between males and females in the onset and maintenance of chronic pain (Keogh & Eccleston, 2006). SES was included in analyses

as an auxiliary variable and covariate to account for the non-random missingness of data in the sample, and because it has previously been reported that children from families with lower SES are at higher risk of developing pain (Morris et al., 2021). Child chronic pain at the age 8 assessment was included as a covariate in all analyses to adjust for initial levels of pain. Due to the significant correlations between vacation status and physical activity levels, vacation status was regressed out of physical activity parameters for all Aim 1 and Aim 2 analyses. All models for aims 1 and 2 were repeated, including all potential covariates (i.e., vacation, sex, age, race/ethnicity, BMI, and family SES), and without controlling for age 8 chronic pain. These supplementary models yielded similar patterns of results to those generated in the primary analyses.

Aim 1 Analyses

Prior to testing moderation, the direct association of each physical activity and sleep parameter with monthly recurring pain was examined. Age 8 sedentary behavior (Table 3), moderate activity (Table 4), and vigorous activity (Table 5) did not significantly predict age 9 monthly recurring pain. Age 9 monthly recurring pain was not associated with age 8 sedentary behavior ($\beta = 0.001$ [95% CI: -0.048, 0.049]; $SE = 0.025$; *ns*), moderate activity ($\beta = -.002$ [95% CI: -0.101, 0.097]; $SE = 0.048$; *ns*), or vigorous activity ($\beta = 0.049$ [95% CI: -0.078, 0.176]; $SE = 0.065$; *ns*). Monthly recurring pain at age 8 and sex were significant predictors of age 9 monthly recurring pain.

Similar to physical activity, age 8 sleep duration (Table 6), sleep efficiency (Table 7), sleep latency (Table 8), and sleep midpoint variability (Table 9) did not significantly predict age 9 monthly recurring pain. Age 9 monthly recurring pain was not associated with age 8 sleep duration ($\beta = -0.026$ [95% CI: -0.191, 0.138]; $SE = 0.084$; *ns*), sleep

efficiency ($\beta = 0.005$ [95% CI: -0.012, 0.023]; $SE = 0.009$; *ns*), sleep latency ($\beta = 0.005$ [95% CI: -0.139, 0.150]; $SE = 0.074$; *ns*), or sleep midpoint variability ($\beta = -0.023$ [95% CI: -0.419, 0.373]; $SE = 0.202$; *ns*). Monthly recurring pain at age 8 and sex were significant predictors of age 9 monthly recurring pain.

To examine whether these associations changed when examining new onset of chronic pain, the sample was restricted to twins who did not report any chronic pain at age 8. Results did not vary from those reported for the entire sample.

Aim 2 Analyses

Following the examination of the direct association of each physical activity and sleep parameter with monthly recurring pain, interaction models were tested to examine whether sleep parameters moderated the association between physical activity parameters and chronic pain.

Sleep Parameters X Sedentary Behavior Predicting Pain

In Model 1, main effects of sleep duration ($\beta = -0.195$ [95% CI: -1.140, 0.750]; $SE = 0.482$; *ns*) and sedentary behavior ($\beta = -0.002$ [95% CI: -0.047, 0.042]; $SE = 0.023$; *ns*) were not significant, and there was no significant interaction between sleep duration and sedentary behavior ($\beta = -0.017$ [95% CI: -0.101, 0.068]; $SE = 0.043$; *ns*) in the prediction of age 9 recurrent pain (Table 10).

In Model 2, main effects of sleep efficiency ($\beta = -0.061$ [95% CI: -0.145, 0.023]; $SE = 0.043$; *ns*) and sedentary behavior ($\beta = -0.001$ [95% CI: -0.050, 0.047]; $SE = 0.025$; *ns*) were not significant, and there was no significant interaction between sleep efficiency and sedentary behavior ($\beta = -0.006$ [95% CI: -0.013, 0.001]; $SE = 0.004$; *ns*) in the prediction of age 9 recurrent pain (Table 11).

In Model 3, main effects of sleep latency ($\beta = -0.389$ [95% CI: -0.984, 0.206]; $SE = 0.304$; *ns*) and sedentary behavior ($\beta = -0.001$ [95% CI: -0.050, 0.048]; $SE = 0.025$; *ns*) were not significant, and there was no significant interaction between sleep latency and sedentary behavior ($\beta = -0.038$ [95% CI: -0.097, 0.020]; $SE = 0.030$; *ns*) in the prediction of age 9 recurrent pain (Table 12).

In Model 4, main effects of sleep midpoint variability ($\beta = -0.370$ [95% CI: -2.057, 1.318]; $SE = 0.861$; *ns*) and sedentary behavior ($\beta = 0.003$ [95% CI: -0.044, 0.051]; $SE = 0.024$; *ns*) were not significant, and there was no significant interaction between sleep midpoint variability and sedentary behavior ($\beta = -0.034$ [95% CI: -0.194, 0.127]; $SE = 0.082$; *ns*) in the prediction of age 9 recurrent pain (Table 13).

Sleep Parameters X Moderate Physical Activity Predicting Pain

In Model 5, main effects of sleep duration ($\beta = 0.091$ [95% CI: -0.169, 0.352]; $SE = 0.133$; *ns*) and moderate activity ($\beta = -0.005$ [95% CI: -0.101, 0.092]; $SE = 0.049$; *ns*) were not significant, and there was no significant interaction between sleep duration and moderate activity ($\beta = 0.060$ [95% CI: -0.082, 0.202]; $SE = 0.072$; *ns*) in the prediction of age 9 recurrent pain (Table 14).

In Model 6, main effects of sleep efficiency ($\beta = 0.033$ [95% CI: -0.001, 0.064]; $SE = 0.016$; *ns*) and moderate activity ($\beta < 0.001$ [95% CI: -0.097, 0.096]; $SE = 0.049$; *ns*) were not significant, and there was no significant interaction between sleep efficiency and moderate activity ($\beta = 0.015$ [95% CI: < 0.001, 0.030]; $SE = 0.008$; *ns*) in the prediction of age 9 recurrent pain (Table 15).

In Model 7, main effects of sleep latency ($\beta = 0.148$ [95% CI: -0.165, 0.461]; $SE = 0.160$; *ns*) and moderate activity ($\beta = 0.002$ [95% CI: -0.092, 0.096]; $SE = 0.048$; *ns*)

were not significant, and there was no significant interaction between sleep latency and moderate activity ($\beta = 0.071$ [95% CI: -0.055, 0.196]; $SE = 0.064$; *ns*) in the prediction of age 9 recurrent pain (Table 16).

In Model 8, main effects of sleep midpoint variability ($\beta = -0.037$ [95% CI: -0.855, 0.780]; $SE = 0.417$; *ns*) and moderate activity ($\beta = -0.005$ [95% CI: -0.102, 0.092]; $SE = 0.049$; *ns*) were not significant, and there was no significant interaction between sleep midpoint variability and moderate activity ($\beta = -0.005$ [95% CI: -0.341, 0.332]; $SE = 0.172$; *ns*) in the prediction of age 9 recurrent pain (Table 17).

Sleep Parameters X Vigorous Physical Activity Predicting Pain

In Model 9, main effects of sleep duration ($\beta = -0.018$ [95% CI: -0.239, 0.203]; $SE = 0.065$; *ns*) and vigorous activity ($\beta = 0.048$ [95% CI: -0.079, 0.175]; $SE = 0.113$; *ns*) were not significant, and there was no significant interaction between sleep duration and vigorous activity ($\beta = 0.007$ [95% CI: -0.156, 0.171]; $SE = 0.083$; *ns*) in the prediction of age 9 recurrent pain (Table 18).

In Model 10, main effects of sleep efficiency ($\beta = 0.017$ [95% CI: -0.009, 0.042]; $SE = 0.013$; *ns*) and vigorous activity ($\beta = 0.054$ [95% CI: -0.076, 0.183]; $SE = 0.066$; *ns*) were not significant, and there was no significant interaction between sleep efficiency and vigorous activity ($\beta = 0.012$ [95% CI: -0.009, 0.032]; $SE = 0.010$; *ns*) in the prediction of age 9 recurrent pain (Table 19).

In Model 11, main effects of sleep latency ($\beta = 0.020$ [95% CI: -0.231, 0.270]; $SE = 0.128$; *ns*) and vigorous activity ($\beta = 0.050$ [95% CI: -0.076, 0.176]; $SE = 0.064$; *ns*) were not significant, and there was no significant interaction between sleep latency and

vigorous activity ($\beta = 0.012$ [95% CI: -0.184, 0.208]; $SE = 0.100$; *ns*) in the prediction of age 9 recurrent pain (Table 20).

In Model 12, main effects of sleep midpoint variability ($\beta = 0.315$ [95% CI: -0.195, 0.826]; $SE = 0.260$, *ns*) and vigorous activity ($\beta = 0.054$ [95% CI: -0.069, 0.176]; $SE = 0.063$; *ns*) were not significant, and there was no significant interaction between sleep midpoint variability and vigorous activity ($\beta = 0.328$ [95% CI: -0.057, 0.714]; $SE = 0.197$; *ns*) in the prediction of age 9 recurrent pain (Table 21).

Exploratory Analyses

Exploratory Analyses were conducted to examine whether sex moderated the association between health behaviors at age 8 and monthly recurring pain at age 9. Interaction models revealed that sex did not moderate the association health behaviors at age 8 and chronic pain at age 9. Finally, because the majority of the literature linking sleep and pain in children has relied on self-report of sleep duration and problems, further exploratory analyses were conducted to examine the association between subjective (parent-report of child sleep) and objective (actigraphy assessed) sleep parameters. Correlations between subjective and objective sleep parameters can be found in Table 22 and indicate that objective sleep duration problems and subjective sleep duration problems show a small but significant negative correlation ($r = -0.16$, $p < 0.001$). Correlations between subjective (parent- and self-report of child activity) and objective (actigraphy assessed) physical activity parameters revealed that parent-report of child physical activity (assessed via the TMCQ Physical Activity subscale) was positively correlated with actigraphy-assessed levels of moderate activity ($r = 0.17$, $p < 0.01$) and vigorous activity ($r = 0.14$, $p < 0.01$), but was uncorrelated with sedentary behavior ($r =$

0.03, $p = 0.50$). Child self-reported physical activity over the past 7 days was significantly positively correlated actigraphy-assessed levels of moderate activity ($r = 0.11$, $p < 0.05$) and vigorous activity ($r = 0.12$, $p < 0.05$), but was uncorrelated with sedentary behavior ($r = -0.03$, $p = 0.50$).

Discussion

Aims and Hypotheses

Identifying modifiable biopsychosocial risk and protective factors of chronic pain in youth is important to prevent chronic pain from becoming a lifelong health concern (Borsook et al., 2018). The present study was the first to examine parameters of sleep health as moderators of the relationship between physical activity levels and nonclinical chronic pain in a community sample of twin children. Physical activity and sleep health parameters were measured using objective actigraphy assessment, which is a more reliable and valid assessment of sleep onset and duration compared to subjective reports of sleep problems and is further better able to parse apart levels of physical activity compared to retrospective report of activity. It was hypothesized that sleep and physical activity at age 8 would predict next-year chronic pain, and that sleep health and physical activity parameters would interact to predict chronic pain the following year. The results were not consistent with hypotheses.

Chronic Pain Characteristics

Chronic pain was common in this community sample of youth with 57.1% of children endorsing at least one area of pain. Headaches, stomachaches, and backaches were the most common pain presentations. This frequency of common chronic pain locations is congruent with prevalence rates reported in other studies examining

nonclinical chronic pain presentations, with prevalence rates in large-scale epidemiological ranging between 4% and 88% depending on the location of chronic pain (King et al., 2011), though more recent studies have estimated the prevalence of general chronic pain presentations in pediatric populations to be high, with 46% of children experiencing some form of chronic pain (Miró et al., 2022). These studies estimating prevalence rates of chronic pain in youth took a similar approach of measuring chronic pain through self- or parent-report of significant pain problems experienced in the past 3 months or more by endorsing the frequency of pain experienced across several body sites. Further, longitudinal studies examining chronic pain in youth frequently examined prospective associations between sleep and activity with changes in pain using a one-to-two year follow up study design (e.g., Sil et al., 2020; Incledon et al., 2016), similar to the time frame of the present study. This suggests that the present community sample of twin children is comparable to previously reported samples of youth, both in the prevalence and locations of their chronic pain concerns as well as the method of assessment of recurring or persistent pain concerns using a longitudinal design.

Sleep Health and Chronic Pain

Participants of the present study did not meet the recommendations by the American Academy of Medicine of 9-12 hours of sleep for their age group (Paruthi et al., 2016). They slept an average of only 8 hours per night but demonstrated adequate sleep regularity with low sleep midpoint variability. Further, they experienced good sleep efficiency, with 90% of time in bed spent asleep, and took an average of 20 minutes to fall asleep. Recent studies suggest that many children are not meeting sleep guidelines; approximately 50% of school-aged children and 70% of adolescents are not meeting

sleep duration recommendations (Galland et al., 2019). Minges and colleagues (2016) assessed sleep characteristics using actigraphy in a community sample of 8-year-old children and found that children obtained an average of 9.2 hours per night. Thus, our sample obtained even less sleep in comparison to other similarly aged community samples, though their variability in sleep duration patterns was lower compared to other samples (Minges et al., 2016). Sleep efficiency was good (>85%) in the present sample and compares to other community samples of similarly aged children (Reed & Sacco, 2016). This suggests that our sample differs to other community samples in their sleep duration, but not other indicators of sleep health.

The available literature investigating the association between sleep health and chronic pain in community-dwelling youth is scarce, but more or less consistent in linking sleep with risk for future pain. Of note, each of the existing studies used youth or parent report of pain problems such as daytime tiredness, difficulty falling asleep, sleep problems, and short sleep duration. Though community samples of youth included in these studies are typically slightly older than the present sample, there are important similarities in the study design between the present study and the available literature. First, several studies examined chronic pain by pain location. For example, Paananen and colleagues (2010) assessed musculoskeletal pains over the past 6 months via child report of pain in different body locations, and coded responses into categories by presence of pain in number of body sites (i.e., 0, 1, 2, or > 2 sites). This study found that short sleeping time in girls but not boys predicted persistence of pain two years later. Second, most studies used a 1–2-year prospective study design. For example, Incledon and

colleagues (2016) found that sleep deficiency predicted parent-reported weekly pain problems one year later in Australian 10- to 12-year-old children.

Much of the available literature on the associations between sleep and chronic pain in youth are based on a Finnish sample of children aged 10 to 19 years old. In this sample, daytime sleepiness predicted the new onset of musculoskeletal pain at different body locations one year later for 10- to 11-year-old children (El-Metwally et al., 2007). Other studies examining this Finnish cohort of children also focused on sleep duration as opposed to sleep problems and found that lower sleep duration predicted neck and low back pain in 15- to 19-year-old adolescents (Auvinen et al., 2010). In contrast to these findings, one study (Jones et al., 2003) found no association between self-reported daytime tiredness and new onset of chronic pain one year later in a population-based study of 11- to 14-year-old English children.

What can account for the discrepant findings between the current study and the extant literature? Though the studies in the current body of literature have important similarities with the present study, they also differ to the present study in some important ways. First, the association between sleep and chronic pain has mostly been examined in early to late adolescence, as opposed to middle childhood. This is a crucial age difference, as adolescents experience significant pubertal changes in the homeostatic and circadian regulation of sleep that is associated with delayed sleep phases and changes in sleep routines (Galland et al., 2020; Carskadon et al., 1993, as well as the initiation of potential sex differences in the sleep-pain association (Hagenauer et al., 2009). Thus, studies examining the association between sleep health and chronic pain in adolescents

may not generalize to younger children who have not yet experienced such pubertal influences on their sleep health.

Further, in previous studies linking sleep problems to chronic pain, sleep was not assessed using objective actigraphy assessment but rather low-quality child self-report using short or single-item questionnaires (e.g., Incledon et al., 2016; El-Metwally et al., 2007; Mikkelsen et al., 1997) or via visual analog scales to indicate daytime sleepiness (Jones et al., 2003). As the exploratory analyses on the associations between subjective and objective indicators of sleep in the current study revealed that there was little to no alignment between these methods, replicating studies that suggest low agreement between self-report questionnaires and actigraphy assessment in youth (e.g., Arora et al., 2013; Girschik et al., 2012). Because it is objective, actigraphy is a more valid and reliable method than self-reports of assessing particular dimensions of sleep health during this developmental period.

In addition to alternative ways of measuring sleep health, some studies also used alternative ways to assess childhood chronic pain, such as mannequins to indicate pain sites (Jones et al., 2003). Compared to listing body sites, this method of assessing chronic pain in youth has been criticized due to its lack of standardization (McGrath, 1998) and subjectivity influencing the interpretation of pain locations and intensity (Varni et al., 1987). Further, studies frequently have examined the association between sleep problems and development of specific types of chronic pain such as musculoskeletal pain (El-Metwally et al., 2007; Paananen et al., 2010). This highlights sleep problems as a risk factor for the development and maintenance of certain chronic pain types, as opposed to sleep health as a protective factor for the development of a variety of pain presentations.

In conclusion, the available body of literature on the association between sleep health and chronic pain suggests that among early adolescents, indicators of sleep problems predict higher risk of subsequent chronic pain, with some indication of gender differences emerging during late adolescence. One explanation for why we did not observe these associations in our sample of school-aged children examining both the new onset and maintenance of chronic pain is because these associations may be more tentative very early in pubertal development. The current sample is 2+ years younger than samples from existing studies, which is likely a meaningful difference at this age as the transition from middle childhood to early adolescence is shaped by important socioemotional changes, hormonal alterations, as well as homeostatic and circadian rhythms impacting sleep health (Galland et al., 2020; Carskadon et al., 1993). Further, the available literature has relied entirely on self- or parent-report of sleep, which is likely capturing a phenomenon distinct from that captured through actigraphy. Subjective reports may be assessing not only sleep problems but other related phenomena (e.g., memory bias, perception errors), whereas actigraphy assessment captures parameters of sleep health in an objective way. Lastly, much of this literature is based on large, epidemiologically sized samples (particularly from the Finnish cohort), which have substantial power to detect even very small effects driving associations. Of note, many of the effect sizes in the epidemiological studies were quite robust (e.g., odds ratios of 1.53; El-Metwally et al., 2007), suggesting this explanation is unlikely to account for the discrepant findings.

Physical Activity and Chronic Pain

Participants were very active with 2 hours of moderate and 1 hour of vigorous activity on average per day. This level of activity goes above and beyond national averages as well as the recommendations of the Centers for Disease Control and Prevention (CDC), which recommends 60 minutes or more of moderate-to-vigorous activity per day for children. Recent studies suggest that only 28.1% of children (Saint-Maurice et al., 2021) and 15% of female and 23% of male adolescents meet these physical activity recommendations (Guthold et al., 2018). The physical activity levels observed in this study also are higher compared to other large international studies, which suggest that school-aged children spend an average of 60 minutes per day engaging in objectively assessed moderate-to-vigorous activity per day (Cooper et al., 2015). The high levels of moderate and vigorous activity observed in the present study may be driven by the fact that most participants are living in Arizona, where the weather allows for engagement in outdoor activities throughout the majority of the year. Favorable weather conditions have been shown to be associated with increased sports participation and better outcomes of physical activity interventions (Welch et al., 2018).

Beyond physical activity, participants engaged in an average of 10 hours and 30 minutes of sedentary behavior per day. In comparison to moderate and vigorous activity, recommendations for sedentary behavior are less defined, though the present findings suggest that such levels of sedentary behavior did not have any adverse nor positive effects on chronic pain at this age. These levels of sedentary behavior are slightly higher compared to other community samples, with the present sample spending around 65% of their waking hours in sedentary behavior, whereas other studies report that sedentary behavior accounted for an average of 50-60% of waking hours in other community

samples of youth (Cooper et al., 2015). However, these differences may be driven by sleep deficits, as a lack of sleep duration allows for more hours during the waking day to be engaged in sedentary behavior compared to sleep.

We did not observe an association between objectively assessed physical activity parameters and chronic pain in the present sample. The prior literature on the association between moderate to vigorous activity and pain in children is mixed. Some studies suggest that physical activity is protective against negative outcomes such as neck pain (Ståhl et al., 2008) and low back pain during mid adolescence (Wedderkopp et al., 2009). In contrast, extreme levels of physical activity have also been highlighted as a risk factor for chronic pain. For example, some studies provide evidence that high physical activity levels are associated with persistent chronic pain being present two years later, though this association does not hold for the new onset of chronic pain (Paananen et al., 2010). In addition, Jones and colleagues (2003) found that those who engaged in extreme levels of physical activity were at doubled risk of experiencing widespread pain one year later, and El-Metwally and colleagues (2007) further suggest that vigorous levels of physical activity predict traumatic musculoskeletal pain.

With regard to sedentary behavior, some studies suggest that it is a risk factor for the onset and maintenance of chronic pain, while others report null findings for the onset of chronic pain in youth. For example, Paananen and colleagues (2010) found that high sedentary behavior at age 16 predicts pain persistence two years later, and Jones and colleagues (2003) identified sedentary behavior to be a risk factor for the new onset of widespread body pain being present one year later in 11- to 14-year-old children. In contrast to Jones' (2003) findings on the new onset of chronic pain, some studies report

null findings suggesting that sedentary behavior does not predict the new onset of chronic pain two years later (Paananen et al., 2010). There are notable similarities between studies. First, several studies examined this association in similar age groups of school-aged children between 8 and 12 years old (e.g., Wedderkopp et al., 2003; Ståhl et al., 2008). Further, studies generally distinguish between sedentary behavior and higher activity levels, with most studies suggesting that high sedentary behavior is a risk factor for chronic pain being present one to two years later.

Despite important similarities, the present study also differed from much of the available literature in important ways. First, most studies have focused on early to mid-adolescence (e.g., Paananen et al., 2010; Jones et al., 2003). Though this age difference is only a few years, the transition from childhood to adolescence is a developmental period shaped by significant declines in physical activity and a shift towards higher sedentary behavior that is frequently associated with higher levels of adiposity in adolescence (Kwon et al., 2015). Second, the timing of follow up assessments varies widely. Instead of conducting a 12-month follow up, many studies examined this association either cross-sectionally (e.g., Wedderkopp et al., 2003) or over a longer span of time, with some studies utilizing 3- to 4-year longitudinal designs (e.g., Ståhl et al., 2008). Third, virtually all the available literature has focused on a specific type of pain when examining the association between physical activity and pain in youth, such as back pain (Wedderkopp et al., 2003), musculoskeletal pain (El-Metwally et al., 2007), or neck pain (Ståhl et al., 2008).

Perhaps most important, physical activity has almost exclusively been assessed using self-report of activity levels retrospectively (e.g., Paananen et al., 2010), and is

often poorly defined such as vigorous activity level being indicated by breathlessness following exercise (e.g., El-Metwally et al., 2007). Though high-quality objective assessment is often not feasible for large cohort studies, prior literature suggests that such subjective retrospective report of activity is a poor indicator of physical activity in youth (Riddoch et al., 2007). For example, Wedderkopp and colleagues (2003) examined the association between physical activity and pain in 8- to 10-year-old children, and then compared whether this association differed based on whether activity was self-reported or assessed using actigraphy. Congruent with the findings of the present study, they found no associations between pain and actigraphy assessment of activity, but also observed weak inconsistent correlations between objective and subjective assessment of activity. This suggests that retroactive subjective reports of physical activity in youth are biased and may therefore be a poor indicator of different physical activity levels.

In conclusion, the available body of literature on the association between physical activity and chronic pain is mixed, with some indication that sedentary behavior is a risk factor for, and physical activity a protective factor against the maintenance of chronic pain during childhood. While the associations between moderate activity and persistent chronic pain mostly hold, evidence regards the relation of moderate activity and new onset of chronic pain is inconsistent. Potential explanations for why we did not observe associations between sedentary behavior, physical activity, and chronic pain in the current study include that prior studies used longer follow-up designs, focused on single chronic pain sites, and examined chronic pain during early to mid-adolescence, a period that is shaped by important developmental changes in sedentary behavior and physical activity patterns. Further, most of the available literature has relied on retrospective self-

report of activity levels, and activity levels were often poorly defined. Similar to sleep health, subjective reports of activity levels are often inaccurate due to individuals' limited ability to recall information accurately as well as propensity to respond to items in a manner that is socially desirable.

Effect of the Interaction Between Sleep and Activity on Child Chronic Pain

Higher sedentary behavior was associated with shorter sleep duration, higher sleep efficiency, longer sleep latency and higher sleep variability. This indicates that children who were less physically active and engaged in higher sedentary behavior slept for a shorter amount of time, took longer to fall asleep, and had sleep that was less regular relative to less active/more sedentary children; however, they spent more time asleep while in bed. In contrast, moderate activity was not associated with sleep duration, latency, or midpoint variability; however, those who engaged in more versus less moderate activity also experienced less time asleep in comparison to the time they spent in bed. In comparison to moderate activity, vigorous activity was not associated with sleep duration, efficiency, and latency; however, it was associated with lower midpoint variability, indicating that twins who engaged in higher versus lower levels of vigorous activity demonstrated more regular sleep. This suggests that each physical activity parameter relates to sleep parameters differently, underscoring the importance of examining them as separate indicators of health promoting behaviors with potentially separate mechanisms in their relation to sleep and other health outcomes.

This study was the first to examine this potential moderated association in the prediction of chronic pain among youth. We did not observe that sleep health parameters moderated the association between physical activity levels and childhood chronic pain.

Other studies have investigated the interaction of physical activity and sleep parameters in youth in the context of risk factors for chronic pain, including internalizing and externalizing symptoms (e.g., Gillis & El-Sheikh, 2019; Ogawa et al., 2019). These studies consistently revealed significant main and interaction effects, suggesting that internalizing and externalizing problems are more severe for youth who experience poor-quality sleep in conjunction with low physical activity levels (Gillis & El-Sheikh, 2019). This is in line with the dual-risk approach, which suggests that risk aggregates in the presence of existing vulnerabilities and thus increases the risk of negative health outcomes (Sameroff, 1983; McEwen, 1998). Importantly, protective effects of physical activity are evidenced by adolescents with higher levels of physical activity presenting with lower internalizing and externalizing problems, regardless of their sleep quality or quantity (Gillis & El-Sheikh, 2019).

This study by Gillis and El-Sheikh (2019) differs from the present study not only in their outcome of interest, but also in three other important ways: Though sleep parameters were examined using objective actigraphy assessment, physical activity was measured using a self-report questionnaire. Further, participants were adolescents as opposed to youth in middle childhood, which may point to developmental differences in the interaction of physical activity and sleep in prediction of health outcomes. Lastly, they used a cross-sectional design, as opposed to examining associations using a 1-year longitudinal design. These discrepancies in the findings point to the potential role of developmental periods and the measurement of physical activity and sleep in their interactive prediction of health outcomes.

The interaction of sleep and physical activity has also been studied in adults with chronic pain. Non-restorative sleep and lack of physical activity have been found to be independent predictors of chronic pain using a 1-year longitudinal design, suggesting that they are important short-term risk factors for the development of chronic pain in young adults (Lindell & Grimby-Ekman, 2022). Other studies examining the interrelationship between sleep and physical activity revealed that physical activity is associated with enhanced sleep duration and efficiency, particularly for adults suffering from disease (Dolzal et al., 2017). Intervention studies in adult patients with chronic pain further suggest that physical activity is associated with lower sleep disturbances and lower pain intensity for adult patients in chronic pain, which were found to last for one year (Wiklund et al., 2018). Physical activity programs and sleep health interventions such as CBT-I are recommended for reducing chronic pain intensity and improving physical function in adult patients with chronic pain conditions (Todd et al. 2021). Even small amounts of activity have been found to be advantageous, especially in adult patients who transition from a sedentary to a more active lifestyle (Siddique et al., 2017). These studies point to promising interaction effects of sleep and physical activity in prediction of chronic pain during adulthood, though these findings have not been replicated in younger age groups with either developing or established chronic pain.

Sex Differences

For exploratory analyses, we did not observe any sex differences in chronic pain or any moderation by sex in the present study. Sex differences in pain and risk factors for pain (e.g., depression) differ by age and may become more pronounced at older age when puberty and hormonal changes contribute to physiological mechanisms underlying health

behaviors drive chronic pain and related outcomes (Athnaiel et al., 2023; Vincent & Tracey, 2008). For example, Jones and colleagues (2003) examined persistent pain in 10- to 12-year-old children and found that receiving insufficient sleep is associated with persistent musculoskeletal pain two years later in female participants, but not male participants. Sex differences may be due to several biological, psychological, and sociocultural factors. For example, it is possible that prevalence rates of chronic pain conditions are higher in women because of hormonal differences during puberty (LeResche et al., 1997). Further, psychosocial mechanisms may contribute to sex differences in pain experiences. For example, gender roles are associated with pain responses, as males tend to adopt the masculine gender norm of tolerating increased levels of pain whereas females tend to accept pain as a normal part of life (Myers et al., 2003). In addition, factors such as problem-focused coping, catastrophizing, social support, and affective distress may contribute to sex differences observed in pain sensitivity and tolerance as well as the prevalence of chronic pain reports (Unruh et al., 1999; Sullivan et al., 2000; Lynch et al., 2007; El-Shormilisy et al., 2015). Several studies examining chronic pain following traumatic injury also point to important sex differences, suggesting that females are at higher risk of experiencing recurring pain following traumatic injury (e.g., Bartley and Fillingim, 2013).

Insights from the Existing Literature on Established Chronic Pain Across the Lifespan

Many studies on chronic pain across the lifespan focus on already established pain conditions, such as fibromyalgia (Olsen et al., 2013) or osteoarthritis (Whibley et al., 2021). Though studies on clinical pain conditions consistently establish associations

between health-promoting behaviors and lower symptom severity, they focus on already existing pain that has specific underlying mechanisms, which differ based on chronic pain condition (Courtney et al., 2017). In addition, participants in this sample were around two years younger than participants in most studies examining chronic pain in youth, which may account for major differences in observed associations. The period between age 8 and 14 is a developmental period when children are advancing towards early adolescence, which is characterized by changes in environmental factors such as increased peer relationships and development of independence (Eccles, 1999), factors that may in turn impact parents' understanding of their child's pain experience. Further, much of the available literature has examined chronic pain intensity and pain-related disability as compared to presence of pain by location (e.g., Brosbe et al., 2022; Nelson et al., 2018), thereby highlighting a different aspect of the chronic pain experience as opposed to presence of chronic pain by location.

The current study focused on identifying the role of health behaviors for risk for developing pain in a community sample of children. However, most of the literature in the field has evaluated the relation between these health behaviors and pain in youth and adults with established chronic pain, and most often with diagnosed pain conditions. The association between health behaviors and established chronic pain is well-established from adolescence throughout adulthood, suggesting that physical activity and sleep play an important role for tertiary pain prevention that is focused on management of already existing pain conditions. However, the role of physical activity and sleep is less robust for primary pain prevention that is focused on preventing the development of chronic pain in the first place.

Though the literature on the associations of sleep and physical activity behaviors for new onset of pain is scarce, there is evidence in the literature on adults that these associations may become more pronounced during adolescence and/or young adulthood. For example, a study of Finnish adult workers suggests that low levels of physical activity, more specifically less than 30 minutes of activity per day, significantly predicted the new onset of knee pain one year later (Miranda et al., 2002). Further, Finan and colleagues (2014) argue that sleep disturbances increase the risk for new onset of chronic pain because sleep disturbance impairs processes involved in recovery from physical damage. Though these studies utilize similar follow-up designs and methods to assess chronic pain, they differ in their assessment of health behaviors and currently do not address developmental trajectories relevant to the differential association between health behaviors and already established pain versus new onset of pain. This remains an area ripe for new research.

Strengths and Limitations

This study is characterized by both strengths and limitations. Strengths of the present study include the use of objective actigraphy assessment to examine physical activity and sleep parameters over the course of one week, which allows for reliable and valid assessments of continuous health behaviors in the free-living environment. Further, we were able to examine a variety of chronic pain presentations rather than focusing on a single pain presentation, which is a more comprehensive representation of the pain experience in community samples of youth. This study further examined an age group of children that is underrepresented in the currently available literature, as most of the current research focuses on samples that are approaching adolescence or are adolescents.

By examining a racially/ethnically and socioeconomically sample of 8- and 9-year-old children, we are able to address important gaps in the available literature regarding the role of health behaviors for chronic pain based on different developmental trajectories. The longitudinal design further allows promising avenues to continue studying both cross-sectional but also longitudinal associations between health behaviors and chronic pain as the twins approach adolescents and are impacted by pubertal changes. Overall, this study addressed an important gap in the literature by examining the interaction of physical activity and sleep health parameters in a community sample of youth, utilizing a one-year longitudinal design.

Limitations of the present study include that off-wrist periods (i.e., times when the watch was taken off) were not controlled for, which may lead to inaccurate assessment of physical activity levels. Further, we were unable to evaluate whether the single week of physical activity and sleep measurement is consistent and representative over the following year's health behaviors, particularly during a stage of development that is highly dynamic. The coding of the actigraphy data for physical activity levels is further limited at this age because it was seemingly unable to differentiate between sedentary behavior and light activity due to small count threshold differences. Differentiating between sedentary behavior and light activity is important, as there may be important underlying mechanisms driving their differential associations with health outcomes. Further, we relied on the parent report of child pain. Though this is consistent with the assessment of chronic pain in the available literature for this age group, it may be biased by how the parent interprets and reports their child's pain experiences. We were further

unable to examine pain intensity and associated disability, as well as whether the pain was related to traumatic injury.

Directions for Future Research

Future studies may examine day-to-day associations between health behaviors and chronic pain in addition to longitudinal associations, particularly during the transition from childhood to adolescence. Little to no research has focused on the association between stomachaches and physical activity or sleep, however, some research suggests that another health promoting behavior – diet – is a relevant factor to consider when examining chronic abdominal pain (Friesen et al., 2021). Therefore, diet should also be included as an additional health behavior due to its connection to abdominal pain in youth, and it may provide a more comprehensive overview of the dynamic nature and interactions of health promoting behaviors.

Further, pain patterns may be examined to better investigate location, intensity, and quality of pain in relation to pain-related disability and mental and physical health outcomes. In particular, examining the role of health behaviors in the development or exacerbation of different types of pain (e.g., pain related to headaches versus musculoskeletal pain) may provide a more nuanced perspective on whether and how to intervene in a more tailored manner. Congruent with much of the available literature, there should be a focus on pain intensity in addition to body locations of pain to better understand the differential role of health behaviors for intervention to decrease pain intensity versus prevention of chronic pain. Utilizing multi-method assessments (i.e., actigraphy *and* self and parent report) is valuable to examine both quality and quantity of health behaviors to better understand their possible protective potential for pain.

Using actigraphy assessment of physical activity and sleep health, we were able to obtain an important snapshot of the children's health behaviors over one week. However, it is important to further understand whether they can sustain these levels of physical activity and sleep health over longer periods of time. Such a long-term approach would help us to better understand whether regular and consistent engagement in health behaviors is protective over time during these critical developmental periods and pinpoint timing of when health behaviors relate to health outcomes such as chronic pain. The regularity and consistency of exercise in adults has been shown to be crucial to reap health benefits. For example, evidence from exercise programs suggests that regular engagement in exercise yielded the largest improvements in symptoms and quality of life (Herbert et al., 2020). For patients with already existing pain, beginning with low levels of activity intensity and progressing steadily is important to not exacerbate pain and stiffness and yield greater activity adherence, thus leading to overall improvement in quality of life and ability to complete activities of daily living (Ambrose & Golightly, 2015).

This consistency of engagement in health promoting activities may be particularly important to achieve during middle childhood as this is a period of development when health promoting behaviors are likely dynamic, influenced by a variety of environmental factors such as SES that are related to access to recreational exercise space, neighborhood crime, and ability to receive medical care when injured (Henderson et al., 2015).

Studying health behaviors longitudinally over several time points during the transition from childhood to adolescence to adulthood may therefore not only allow us to better

understand what characteristics of health behaviors are important, but also pinpoint the timing of when beneficial associations are established.

At this age, it may further be important to differentiate between traumatic and non-traumatic injury and how it relates to pain in youth. Literature points to neural mechanisms underlying pain conditions being impacted following experience of trauma, thereby leading to increased vulnerability of developing chronic pain following exposure to trauma, particularly for females (Bartley and Fillingim, 2013; Courtney et al., 2017, Stabell et al., 2014; Palacios-Ceña et al., 2016; Moeller-Bertram et al., 2014). Further, much of the available literature to activity potentially being associated with more strain on the body through traumatic injury, and individuals who experience poor quality and quantity sleep are at increased risk of being involved in an event of injury (Huang & Ihm, 2021). In turn, some studies suggest that sleep disturbance following traumatic injury is associated with increased risk of developing chronic pain as a result of the injury (Smith et al., 2018). In the context of injury-related pain and health behaviors, health behaviors may accrue over time to impact health outcomes (Sameroff, 1983; McEwen, 1998). For example, consistently engaging in maladaptive health following traumatic injury may lead to wear and tear (Li et al., 2018), possibly impacting the capacity to recover from injury and develop protective mechanisms against the development of chronic pain during developmentally critical periods.

Final Conclusions

This study was the first to examine the interaction of objectively assessed physical activity and sleep health parameters in the prediction of new onset and maintenance of chronic pain in a community-sample of middle-school aged children, with relatively high

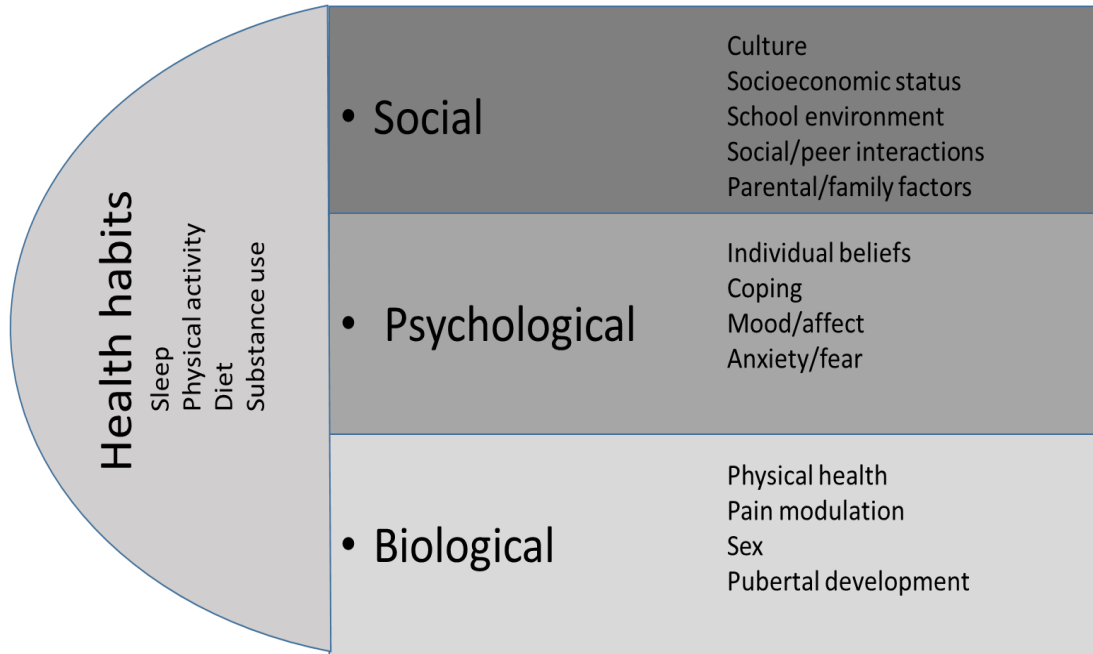
pain prevalence. Though chronic pain in youth is frequently examined using similar metrics of pain following similar follow-up timeframes, there is only a small body of prior literature examining the relationship between sleep, physical activity, and the onset and development of chronic pain in a community sample of youth. The existing literature pointed to a promising role of these health behaviors for the onset and worsening of chronic pain among children; however, we did not observe that objectively assessed physical activity and/or sleep health predicted chronic pain one year later.

The current findings suggest that actigraphy assessment of sleep health may be assessing a phenomenon distinct from subjectively assessed sleep problems, as self-report of sleep is impacted by the individual's own perception of their sleep patterns, which is confounded by socioemotional and contextual factors. Thus, future research may benefit from multi-method assessment of health behaviors to better parse apart discrepancies between subjective and objective measures in association with health outcomes. The present study also highlights the need to study children throughout the course of development to pinpoint the timing of when these associations emerge, and further tailor the biopsychosocial model of chronic pain to younger age groups. Middle childhood is a period of development that is understudied in the context of chronic pain, even though it precedes important developmental changes that may be contributing to later associations between health behaviors and chronic pain outcomes.

The present study provides evidence that the association between health behaviors and chronic pain is not yet evident in this community sample of 8- to 9-year-old children compared to similarly aged patient samples who already have existing pain. Thus, children at risk of developing pain and children with already established chronic pain

need to be examined as separate groups to better understand when and how chronic pain can be prevented before it grows to be a lifelong health concern. Existing findings highlight that physical activity and sleep health play an important role for tertiary pain prevention of already existing pain conditions, though the role of these health behaviors is not consistently established for primary pain prevention in 8- to 9-year-old children. Finally, the interaction of biopsychosocial influences such as sleep health and physical activity with other health behaviors needs to receive more attention in child research to better understand their role for the new onset versus maintenance of already established pain.

Figure 1. Biopsychosocial Model Tailored to Pediatric Chronic Pain



Reproduced from Tonya M. Palermo, *Cognitive-Behavioral Therapy for Chronic Pain in Children and Adolescents*, Oxford University Press, Inc. New York, USA, p. 13. 2012.

Figure 2. Aim 2 Examining the Moderating Effect of Sleep Health on the Relationship Between Physical Activity and Childhood Chronic Pain

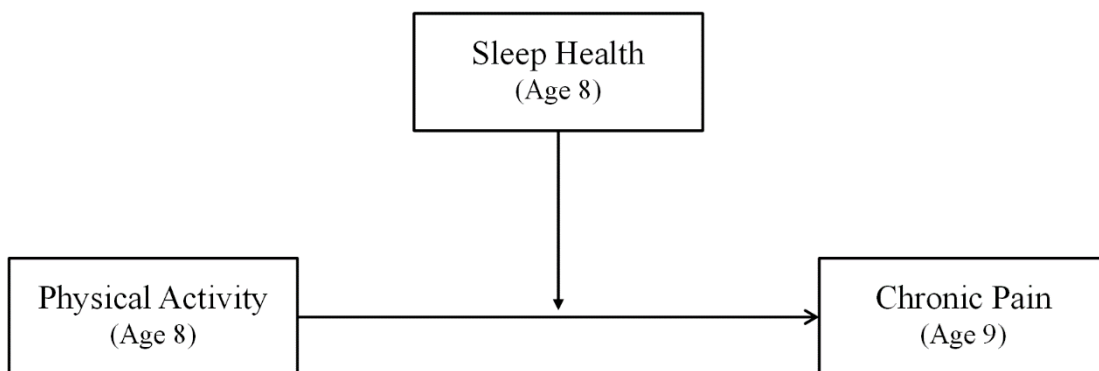


Figure 3. Non-compensatory Model of the Moderating Effect of Sleep Health on the Relationship Between Physical Activity and Childhood Chronic Pain

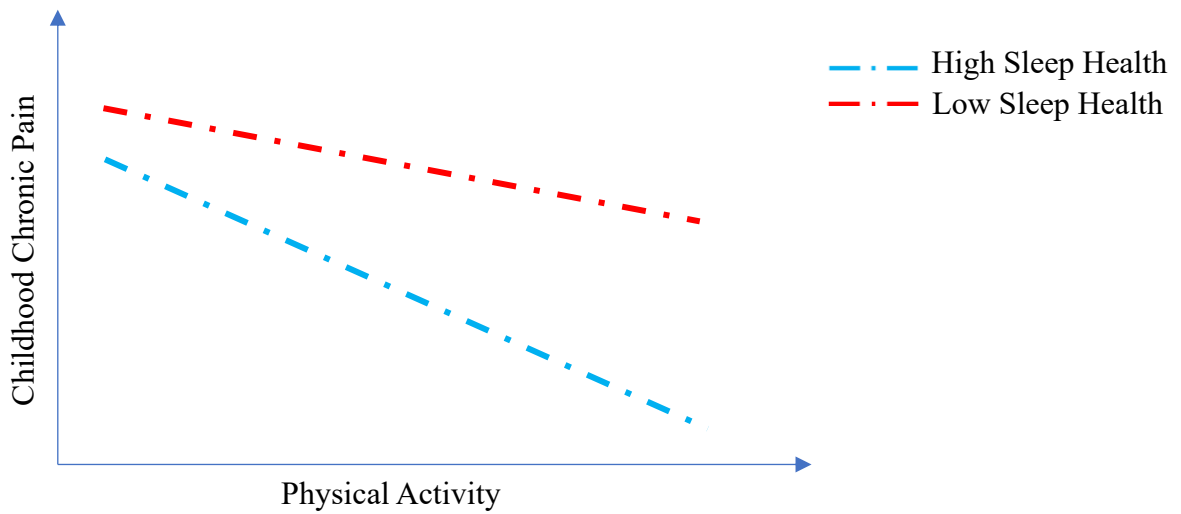


Figure 4. Compensatory Model of the Moderating Effect of Sleep Health on the Relationship Between Physical Activity and Childhood Chronic Pain

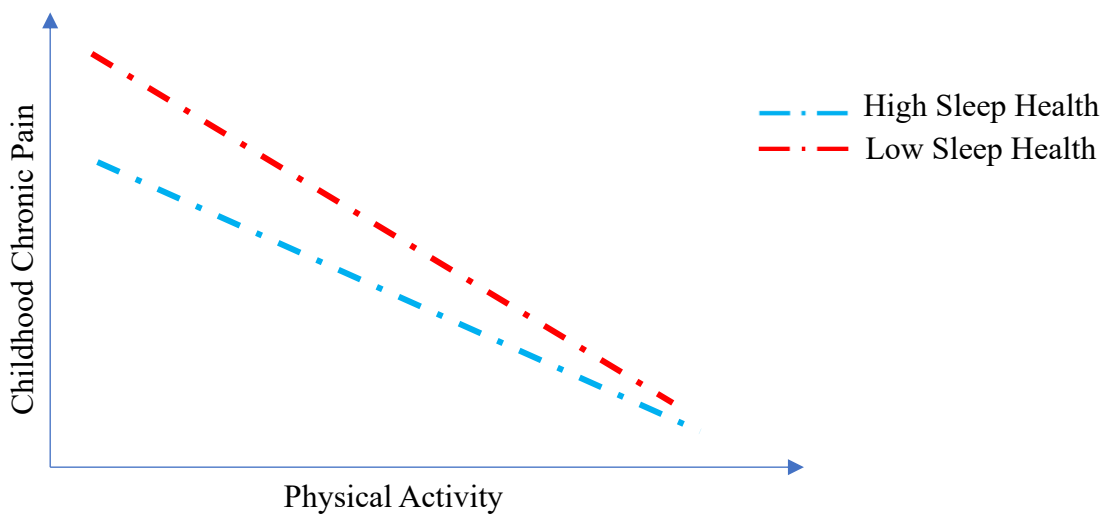


Table 1. Participant Demographics and Descriptive Statistics

	<i>n</i>	<i>%</i>	<i>M</i>	<i>SD</i>	<i>Skew</i>	<i>Kurtosis</i>
Sex						
Male	452	48.8				
Female	474	51.2				
Ethnicity						
Non-Hispanic White/Euro American	396	58.8				
Hispanic/Latino	160	23.7				
Asian/Asian American	23	3.4				
Black/African American	26	3.9				
Native American	18	2.7				
Native Hawaiian	6	0.9				
Bi-/Multi Racial	41	6.0				
Other	4	0.6				
Vacation (8 year)						
Summer/Vacation Participation	194	28.1				
Not Summer/ Vacation	497	71.9				
Income-to needs Ratio (8 year)						
Living in Poverty	42	7.4				
Near the Poverty Line	126	22.3				
Lower Middle Class	92	16.3				
Middle to Upper Class	304	53.9				
Primary Caregiver Education (8 year)						
Less than high school	4	0.6				
High school or equivalent	62	9.2				
Some college	181	26.9				
College degree	247	36.6				
2+ years of graduate school	24	2.6				
Graduate or professional degree	156	23.1				
Age (8 year)	702		8.43	0.68	-0.18	-0.38
Age (9 year)	801		9.72	0.93	0.32	-0.01
BMI (8 year)	589		16.76	2.91	1.99	5.89
BMI (9 year)	630		17.78	3.69	1.79	4.50
Monthly Recurring Pain (8 year)	657		0.77	1.06	1.44	1.58
Monthly Recurring Pain (9 year)	748		1.08	1.22	1.09	0.59
Sedentary Behavior (8 year) in hours	514		10.48	2.34	-0.18	1.72
Moderate Activity (8 year) in hours	514		1.96	1.14	0.58	-0.17
Vigorous Activity (8 year) in hours	514		0.91	0.81	1.25	1.27
Sleep Duration (8 year) in hours	537		8.12	0.71	-0.38	0.14
Sleep Efficiency (8 year) in percentage	537		90.07	5.47	-0.87	0.54
Sleep Latency (8 year) in minutes	537		20.51	15.51	1.86	3.75
Sleep Midpoint Variability (8 year) in hours	537		0.57	0.29	0.11	0.21

Note. Sex (0 = male, 1 = female). Income-to-needs ratio is calculated by dividing the income by the federal guideline for poverty by family size. BMI = Body Mass Index, calculated by weight divided by the square of height. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days.

Table 2. Intercorrelations Among Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Sedentary Behavior (8 year)	-											
2. Moderate Activity (8 year)	-.50**	-										
3. Vigorous Activity (8 year)	-.52**	.51**	-									
4. Sleep Duration (8 year)	-.18**	-.05	-.03	-								
5. Sleep Efficiency (8 year)	.11*	-.14**	-.08	.65**	-							
6. Sleep Latency (8 year)	.14**	-.07	-.05	-.21**	-.12**	-						
7. Sleep Midpoint Variability (8 year)	.17**	-.07	-.07	-.22**	-.04	.10*	-					
8. Age (8 year)	.22**	-.11*	-.03	-.25**	-.02	.05	.28**	-				
9. Sex	.03	.02	-.09*	.17**	.15**	-.02	.02	.02	-			
10. SES (8 year)	-.12*	.01	.05	.19**	.11*	-.10*	-.15**	-.09*	.05	-		
11. Vacation	-.15**	-.23**	-.05	-.01	-.04	.05	-.08	-.01	-.02	.05	-	
12. BMI (8 year)	.09*	-.05	.02	-.19**	-.15**	-.04	.02	.15**	.02	-.13**	.02	-
13. Monthly Chronic Pain (9 year)	-.04	.04	.03	-.03	.03	-.03	-.04	-.04	.03	-.04	.08	.05

Note. * $p < .05$. ** $p < .01$ Activity and Sleep Parameters are based on actigraphy data collected using a Motion logger Microwatch over 7 days. Sex (0 = male, 1 = female). Ethnicity (1=Non-Hispanic White/Euro American, 2=Hispanic/Latino, 3=Asian/Asian American, 4=Black/African American, 5=Native American, 6=Native Hawaiian, 7=Bi-/Multi Racial, 8=Other). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. Vacation = participation during summer or holiday break (0 = not on vacation, 1 = on vacation). BMI = Body Mass Index, calculated by weight divided by the square of height. Recurring Pain = presence of monthly recurring pain at up to 5 body sites.

Table 3. *Sedentary Behavior (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Sedentary Behavior (age 8)	0.001	0.025	0.975	-0.048, 0.049
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.207, 0.386
Sex	0.187	0.087	0.032	-0.016, 0.358
SES (age 8)	-0.088	0.061	0.149	-0.207, 0.031

Note. Results are from the standardized model. Activity Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

3 **Table 4.** *Moderate Activity (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Moderate Activity (age 8)	-0.002	0.048	0.966	-0.101, 0.097
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.237, 0.443
Sex	0.188	0.087	0.031	0.008, 0.168
SES (age 8)	-0.089	0.060	0.140	-0.156, 0.021

Note. Results are from the standardized model. Activity Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 5. *Vigorous Activity (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Vigorous Activity (age 8)	0.049	0.065	0.450	-0.078, 0.176
Chronic Pain (age 8)	0.295	0.046	< 0.001	0.205, 0.385
Sex	0.194	0.088	0.028	-0.021, 0.367
SES (age 8)	-0.091	0.061	-1.501	-0.210, 0.028

Note. Results are from the standardized model. Activity Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

☞ **Table 6.** *Sleep Duration (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Sleep Duration (age 8)	-0.026	0.084	0.755	-0.191, 0.138
Chronic Pain (age 8)	0.296	0.045	< 0.001	0.207, 0.384
Sex	0.193	0.085	0.024	0.025, 0.360
SES (age 8)	-0.083	0.064	0.193	-0.207, 0.042

Note. Results are from the standardized model. Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 7. *Sleep Efficiency (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Sleep Efficiency (age 8)	0.005	0.009	0.549	-0.012, 0.023
Chronic Pain (age 8)	0.295	0.046	< 0.001	0.206, 0.385
Sex	0.178	0.086	0.039	0.009, 0.347
SES (age 8)	-0.092	0.062	0.136	-0.214, 0.029

Note. Results are from the standardized model. Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

64 **Table 8.** *Sleep Latency (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Sleep Latency (age 8)	0.005	0.074	0.943	-0.139, 0.150
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.207, 0.386
Sex	0.187	0.087	0.031	0.017, 0.358
SES (age 8)	-0.088	0.061	0.149	-0.208, 0.032

Note. Results are from the standardized model. Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 9. *Sleep Midpoint Variability (age 8) Predicting Chronic Pain (age 9)*

	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Sleep Midpoint Variability (age 8)	-0.023	0.202	0.911	-0.419, 0.373
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.206, 0.385
Sex	0.187	0.087	0.032	0.016, 0.357
SES (age 8)	-0.089	0.061	0.143	-0.208, 0.030

Note. Results are from the standardized model. Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

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Table 10. *Sleep Duration as a Moderator of the Association between Sedentary Behavior (age 8) and next-year Chronic Pain (age 9)*

	Model 1			
	β	<i>SE</i>	<i>p</i>	<i>95% CI</i>
Sedentary Behavior (age 8)	-0.002	0.023	0.928	-0.047, 0.042
Sleep Duration (age 8)	-0.195	0.482	0.686	-1.140, 0.750
Sedentary Behavior X Sleep Duration	-0.017	0.043	0.700	-0.101, 0.068
Chronic Pain (age 8)	0.295	0.046	< 0.001	0.206, 0.384
Sex	0.193	0.086	0.024	0.025, 0.362
SES (age 8)	-0.084	0.063	0.188	-0.208, 0.041

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 11. *Sleep Efficiency as a Moderator of the Association between Sedentary Behavior (age 8) and next-year Chronic Pain (age 9)*

	Model 2			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Sedentary Behavior (age 8)	-0.001	0.025	0.960	-0.050, 0.047
Sleep Efficiency (age 8)	-0.061	0.043	0.158	-0.145, 0.023
Sedentary Behavior X Sleep Efficiency	-0.006	0.004	0.100	-0.013, 0.001
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.206, 0.385
Sex	0.190	0.085	0.025	0.024, 0.356
SES (age 8)	-0.088	0.063	0.161	-0.211, 0.035

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

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Table 12. *Sleep Latency as a Moderator of the Association between Sedentary Behavior (age 8) and next-year Chronic Pain (age 9)*

	Model 3			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Sedentary Behavior (age 8)	-0.001	0.025	0.957	-0.050, 0.048
Sleep Latency (age 8)	-0.389	0.304	0.200	-0.984, 0.206
Sedentary Behavior X Sleep Latency	-0.038	0.030	0.196	-0.097, 0.020
Chronic Pain (age 8)	0.299	0.045	< 0.001	0.210, 0.389
Sex	0.182	0.088	0.038	0.010, 0.354
SES (age 8)	-0.087	0.061	0.157	-0.207, 0.034

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 13. *Sleep Midpoint Variability as a Moderator of the Association between Sedentary Behavior (age 8) and next-year Chronic Pain (age 9)*

	Model 4			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Sedentary Behavior (age 8)	0.003	0.024	0.885	-0.044, 0.051
Sleep Midpoint Variability (age 8)	-0.370	0.861	0.668	-2.057, 1.318
Sedentary Behavior X Midpoint Variability	-0.034	0.082	0.682	-0.194, 0.127
Chronic Pain (age 8)	0.297	0.046	< 0.001	0.207, 0.387
Sex	0.187	0.087	0.032	0.016, 0.358
SES (age 8)	-0.090	0.061	0.143	-0.210, 0.030

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

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Table 14. *Sleep Duration as a Moderator of the Association between Moderate Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 5			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Moderate Activity (age 8)	-0.005	0.049	0.925	-0.101, 0.092
Sleep Duration (age 8)	0.091	0.133	0.493	-0.169, 0.352
Moderate Activity X Sleep Duration	0.060	0.072	0.405	-0.082, 0.202
Chronic Pain (age 8)	0.294	0.046	< 0.001	0.205, 0.384
Sex	0.191	0.086	0.026	0.023, 0.359
SES (age 8)	-0.083	0.063	0.186	-0.207, 0.040

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 15. *Sleep Efficiency as a Moderator of the Association between Moderate Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 6			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Moderate Activity (age 8)	< .001	0.049	0.995	-0.097, 0.096
Sleep Efficiency (age 8)	0.033	0.016	0.045	-0.001, 0.064
Moderate Activity X Sleep Efficiency	0.015	0.008	0.055	< 0.001, 0.030
Chronic Pain (age 8)	0.297	0.046	< 0.001	0.207, 0.387
Sex	0.179	0.085	0.036	0.012, 0.346
SES (age 8)	-0.092	0.062	0.135	-0.213, 0.029

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

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Table 16. *Sleep Latency as a Moderator of the Association between Moderate Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 7			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Moderate Activity (age 8)	0.002	0.048	0.969	-0.092, 0.096
Sleep Latency (age 8)	0.148	0.160	0.355	-0.165, 0.461
Moderate Activity X Sleep Latency	0.071	0.064	0.271	-0.055, 0.196
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.206, 0.386
Sex	0.192	0.088	0.028	0.020, 0.364
SES (age 8)	-0.088	0.061	0.148	-0.207, 0.031

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 17. *Sleep Midpoint Variability as a Moderator of the Association between Moderate Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 8			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Moderate Activity (age 8)	-0.005	0.049	0.918	-0.102, 0.092
Sleep Midpoint Variability (age 8)	-0.037	0.417	0.929	-0.855, 0.780
Moderate Activity X Midpoint Variability	-0.005	0.172	0.978	-0.341, 0.332
Chronic Pain (age 8)	0.296	0.046	< 0.001	0.206, 0.387
Sex	0.188	0.087	0.031	0.017, 0.358
SES (age 8)	-0.089	0.061	0.143	-0.209, 0.030

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

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Table 18. *Sleep Duration as a Moderator of the Association between Vigorous Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 9			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Vigorous Activity (age 8)	0.048	0.065	0.462	-0.079, 0.175
Sleep Duration (age 8)	-0.018	0.113	0.874	-0.239, 0.203
Vigorous Activity X Sleep Duration	0.007	0.083	0.931	-0.156, 0.171
Chronic Pain (age 8)	0.294	0.046	< 0.001	0.204, 0.384
Sex	0.200	0.087	0.022	0.029, 0.370
SES (age 8)	-0.086	0.064	0.180	-0.211, 0.039

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 19. *Sleep Efficiency as a Moderator of the Association between Vigorous Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 10			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Vigorous Activity (age 8)	0.054	0.066	0.418	-0.076, 0.183
Sleep Efficiency (age 8)	0.017	0.013	0.206	-0.009, 0.042
Vigorous Activity X Sleep Efficiency	0.012	0.010	0.262	-0.009, 0.032
Chronic Pain (age 8)	0.297	0.046	< 0.001	0.206, 0.387
Sex	0.191	0.087	0.028	0.021, 0.362
SES (age 8)	-0.099	0.063	0.116	-0.222, 0.024

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

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Table 20. *Sleep Latency as a Moderator of the Association between Vigorous Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 11			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Vigorous Activity (age 8)	0.050	0.064	0.440	-0.076, 0.176
Sleep Latency (age 8)	0.020	0.128	0.879	-0.231, 0.270
Vigorous Activity X Sleep Latency	0.012	0.100	0.904	-0.184, 0.208
Chronic Pain (age 8)	0.295	0.046	< 0.001	0.205, 0.385
Sex	0.195	0.089	0.027	0.022, 0.369
SES (age 8)	-0.091	0.061	0.137	-0.212, 0.029

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 21. *Sleep Midpoint Variability as a Moderator of the Association between Vigorous Activity (age 8) and next-year Chronic Pain (age 9)*

	Model 12			
	β	<i>SE</i>	<i>p</i>	95% <i>CI</i>
Vigorous Activity (age 8)	0.054	0.063	0.389	-0.069, 0.176
Sleep Midpoint Variability (age 8)	0.315	0.260	0.226	-0.195, 0.826
Vigorous Activity X Midpoint Variability	0.328	0.197	0.095	-0.057, 0.714
Chronic Pain (age 8)	0.299	0.046	< 0.001	0.209, 0.389
Sex	0.200	0.088	0.023	0.027, 0.372
SES (age 8)	-0.090	0.061	0.137	-0.209, 0.029

Note. Results are from the standardized model. Activity and Sleep Parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Monthly Recurring Pain = Presence of monthly recurring pain at up to 5 body sites. Sex (0 = male, 1 = female). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education.

Table 22. *Correlations between Subjective and Objective Sleep Parameters at Age 8*

	1	2	3	4	5	6	7	8	9	10
1. Sleep Duration	-									
2. Sleep Efficiency	.65**	-								
3. Sleep Latency	-.21**	-.12**	-							
4. Sleep Midpoint Variability	-.18**	-.04	.14**	-						
5. CSHQ Duration	-.16**	-.12*	.04	.05	-					
6. CSHQ Bedtime Resistance	-.12*	-.13**	-.03	.11*	.35**	-				
7. CSHQ Sleep Anxiety	-.03	-.02	-.09	-.05	.26**	.40**	-			
8. CSHQ Night Wakings	-.11*	-.14**	.02	-.04	.23**	.45**	.36**	-		
9. CSHQ Parasomnias	-.04	-.02	.08	-.01	.21**	.26**	.29**	.36**	-	
10. CSHQ Disordered Breath	-.08	-.02	.09	.14**	.05	.16**	.02	.19**	.24**	-
11. CSHQ Daytime Sleepiness	-.12*	.04	.13**	.22**	.36**	.28**	.23**	.16**	.28**	.18**

Note. * $p < .05$. ** $p < .01$ Objective sleep parameters are based on actigraphy data collected using a Motionlogger Microwatch over 7 days. Subjective sleep parameters were assessed using parent-report on the Children's Sleep Habits Questionnaire (CSHQ).

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APPENDIX A
IRB ACKNOWLEDGEMENTS

Appendix A: IRB Acknowledgements



EXEMPTION GRANTED

Kathryn Lemery
 CLAS-NS: Psychology
 480/727-6459
 Kathryn.Lemery@asu.edu

Dear [Kathryn Lemery](#):

On 9/20/2022 the ASU IRB reviewed the following protocol:

Type of Review:	Continuing Review
Title:	Genetic and Environmental Origins of the Development of Pain in Children
Investigator:	Kathryn Lemery
IRB ID:	STUDY00004309
Funding:	Name: HHS-NIH: National Institute of Child Health & Human Development (NICHD), Grant Office ID: FP00004748
Grant Title:	None
Grant ID:	None
Documents Reviewed:	None

The IRB determined that the protocol is considered exempt pursuant to Federal Regulations 45CFR46 (4) Data, documents, or specimens on 9/20/2022.

APPROVAL:CONTINUATION

[Kathryn Lemery](#)
 CLAS-NS: Psychology
 480/727-6459
 Kathryn.Lemery@asu.edu

Dear [Kathryn Lemery](#):

On 1/24/2023 the ASU IRB reviewed the following protocol:

Type of Review:	Continuing Review
Title:	Social and Genetic Contributions to Children’s Sleep, Health and Functioning
Investigator:	Kathryn Lemery
IRB ID:	STUDY00000637
Category of review:	
Funding:	Name: HHS-NIH: National Institute of Child Health & Human Development (NICHD), Grant Office ID: MGS 0400, Funding Source ID: R01HD079520
Grant Title:	None
Grant ID:	None
Documents Reviewed:	None

The IRB approved the protocol from 1/24/2023 to 7/23/2023 inclusive. Three weeks before 7/23/2023 you are to submit a new study if analysis will continue.

APPENDIX B
FUNDING ACKNOWLEDGEMENTS

Appendix B: Funding Acknowledgements

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