Family and Peer Stress During Early Adolescence: Phenotypic and Genetic Moderation of Pubertal Development, Sleep and Internalizing Symptomatology

by

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ABSTRACT

Various physical and psychological forms of development take place during the transition from childhood to adolescence, including the onset of puberty, delayed sleep-wake behavior patterns, and increases in internalizing symptomatology. Theory suggests that pubertal processes influence the onset of internalizing symptoms, and this association may differ between boys and girls. The contextual amplification hypothesis suggests that pubertal development interacts with contextual or dispositional factors to impact risk for psychopathology. Family stress and peer stress are two critical factors during early adolescence that have potential moderating effects on the association between pubertal development and internalizing symptoms. In line with the biopsychosocial framework, the current study examined various psychosocial (e.g., family stress, peer stress) and biological (e.g., sleep, genetics) factors that may have effects on the puberty-internalizing relation. Participants were a racially/ethnically and socioeconomically diverse sample of twins who were part of an ongoing longitudinal study (N=818 children; Arizona Twin Project; Lemery-Chalfant et al. 2019). The current study examined the association between puberty and internalizing symptoms, with stress (i.e., family, peer) as potential moderators, and sleep indicators (i.e., objective and self-reported sleep) as potential mediators. Behavior genetic analyses explored the moderated heritability of family stress on internalizing symptoms. Findings revealed that greater pubertal development predicted higher internalizing symptoms in boys, but not girls. For girls, peer stress interacted with pubertal development to predict internalizing symptoms, but simple slopes were not significant. Sleep indicators were not significant mediators between pubertal

development and internalizing symptoms for boys or girls. Univariate twin models revealed environmental influences on internalizing symptoms, and both genetic and environmental influences on peer stress. Family stress did not significantly moderate the genetic and environmental influences of internalizing symptoms.

TABLE OF CONTENTS

CHAPTER 1

INTRODUCTION

The transition from childhood to adolescence is a pivotal period of development that involves rapid growth and maturation (i.e., puberty), learning, and adaptation to changes in environment (Dahl et al., 2018). Marked by changes in neural, biological, and psychosocial functioning, the stage of adolescence has been referred to as a sensitive window and period of plasticity during which an individual's experiences interact with their developmental changes to predict risk for psychopathology (Sisk & Gee, 2022). Puberty is a biological process that takes place during early adolescence (with pubertal onset ranging from ages 8-13 (most typically ages 10-11) for girls and 9-14 (most typically ages 11-12) for boys) in which hypothalamic-pituitary-gonadal (HPG) axis functioning results in neuroendocrine changes and leads to sexual maturation (Dorn & Biro, 2011). The onset of puberty promotes physical and psychological changes that increases risk for psychosocial disorders and physical health concerns (Patton & Viner, 2007). With the age of pubertal onset decreasing (Lee & Styne, 2013; Parent et al., 2013) and known lasting effects of puberty on physical and mental health in adolescence and early adulthood (e.g., increased stress, changes in circadian rhythm, internalizing behaviors; Mendle, 2014), it is critical to study the effects of puberty on these health processes at their onset (i.e., early adolescence).The process of puberty has been termed a "window of opportunity", as youth are in a period of increased vulnerability to changes in many processes related to health and well-being (Dorn et al., 2019).

During puberty, hormonal changes and physical maturation promote rapid brain development, which has been linked with changes in adolescents' social perceptions and experiences, and impact risk for internalizing disorders (Pfeifer and Allen, 2021). As a result, there is a large literature that has reported increases in internalizing symptoms, including anxiety and depressive symptoms, and clinical internalizing disorders during the transition from childhood to adolescence, particularly during the period of pubertal development (Costello et al., 2011; Graber & Sontag, 2009; Hammen & Rudolph, 2003). Indeed, a meta-analysis on the worldwide prevalence of mental disorders in children and adolescents concluded a 6.5% prevalence of anxiety disorders and 2.6% prevalence of depressive disorders (Polanczyk et al., 2015). Further, rates for depression seemingly increase from childhood to adolescence; for example, the 2.8% depression prevalence rate among children increases to a prevalence of 5.6% in adolescence (Costello et al., 2006; McLaughlin & King, 2015). Aside from diagnosed internalizing disorders, it may be even more important to examine the presence of sub-threshold symptom counts in normative community samples to detect the initial onset of these symptoms during early adolescence. In previous studies of community samples of youth ranging from 11 to 17 years old, anxious and depressive symptoms were prevalent in 8-25% of youth (Mazzone et al., 2007; Saluja et al., 2004; Wartberg et al., 2018). These studies underscore the importance of examining internalizing symptoms during early adolescence and the onset of pubertal development, as further understanding and identification of key predictors of internalizing symptoms can assist in informing targets for symptom preventative interventions.

Another salient change that takes place across the pubertal transition is a biological shift in circadian rhythm to later chronotypes (i.e., increased eveningness preference; Randler et al., 2009), which has resulted in delayed sleep-wake behavior patterns, less total sleep time, and increases in daytime sleepiness in adolescents (Gradisar et al., 2011). Simultaneously, increased cognitive capacity aligns with greater autonomy from caretakers starting in late childhood and results in youth being more independent in decision making, especially in terms of their own health-related decisions and setting their own daily sleep schedules (Collins, 1984; Hoyt et al., 2020). In addition, early adolescents have increased school demands and engage in higher amounts of electronic media use, resulting in poorer sleep outcomes (Cain & Gradisar, 2010; Clifford et al., 2020). As a result, poor sleep during adolescence has been deemed an international public health concern, as adolescents continue to get less than the recommended 9 hours per night of sleep (American Academy of Pediatrics, 2014; Gradisar et al., 2011; National Sleep Foundation, 2006). The examination of sleep as a key malleable factor affected by the pubertal transition may aid in understanding of the subsequent onset of internalizing symptomatology and provide an effective target of intervention.

Stress experiences are known to have a causal impact on various psychobiological health processes in childhood and adolescence (Grant et al., 2003). While direct effects of stress on health outcomes have been widely researched, less is known regarding stress as a moderator, but theory has suggested stress to be an important contextual variable throughout the process of pubertal development. One such theory is the *contextual amplification hypothesis*, which suggests that the interaction between pubertal processes

and contextual or dispositional factors may impact how pubertal timing is a risk for later psychopathology (Caspi et al., 1993; Caspi & Moffitt, 1991; Ge & Natsuaki, 2009). Further, *the biopsychosocial framework* would suggest looking at the various psychosocial (e.g., family stress, peer stress) and biological (e.g., sleep, genetics) that may impact how pubertal development effects the onset of psychopathology (Ullsperger & Nikolas, 2017). Despite this, no known study has included all of the above-mentioned critically related health factors in one model (i.e., puberty, stress, sleep, and internalizing symptoms; see Figure 1). In addition, a twin sample allows for the examination of how contextual stress may influence the genetic and environmental contributions to the development of internalizing symptoms which may assist in our etiological understanding of these symptoms and help determine effective preventative efforts. Therefore, understanding the links between these health factors is critical for developing best methods of intervention for children undergoing puberty.

Theories Linking Puberty to Internalizing Symptoms

There are various existing theories that consider the association between puberty and internalizing symptoms and studies testing these theories have produced mixed findings (for meta-analysis, see Ullsperger & Nikolas, 2017). The *maturational disparity hypothesis* is the most widely supported theory, and it posits that earlier physical development for both boys and girls places them at risk for internalizing psychopathology (Brooks-Gunn et al., 1985). The *social deviance hypothesis* extends the maturational disparity hypothesis and suggests that it is not only early developing adolescents, but also late developing adolescents, who are at risk for psychopathology (Petersen et al., 1988).

Third and most recently, *the gendered deviation hypothesis* theorizes that effects differ by gender, such that early maturing girls and late maturing boys are the two demographic groups most at risk for developing psychopathology (Brooks-Gunn et al., 1994; Sontag et al., 2011). The current study was guided by the gendered deviation theory such that I hypothesized that puberty-internalizing associations will differ by gender, with girls with greater pubertal development and boys with less pubertal development experiencing higher internalizing symptoms (Figure 1, *c* path).

Historically, greater pubertal development in boys has been linked with better adjustment, such that they become taller and stronger (Petersen et al., 1991). Conversely, pubertal development has been linked to various negative outcomes for girls, including less lean body shapes and increases in problems with body image and self-esteem (Petersen et al., 1991). A possible explanation is that girls have more risk factors (e.g., higher rumination, lower assertiveness, self-esteem reliant on approval from others; Mendle et al., 2020; Williams & Currie, 2000) than boys for developing symptomatology earlier on in adolescence, and these factors in addition to the social and biological challenges of adolescence makes them more prone to internalizing symptoms and disorders compared to boys (Nolen-Hoeksema & Gigrus, 1994). In addition, boys lack visible markers and an objective measurement of puberty that is equitable to breast development and menarche in girls, making it difficult to accurately report on whether puberty has begun and to differentiate between stages of pubertal development for boys (Deardorff et al., 2019). Therefore, the difference in frequency of internalizing symptoms in girls as compared to boys may stem from the biological (e.g., pubertal timing,

hormonal changes) *and* psychosocial (e.g., victimization, peer relationships) aspects of puberty that become apparent in early adolescence (Hayward & Sanborn, 2002).

Empirical evidence of puberty-internalizing relations has suggested mixed findings, such that some studies concluded sex differences and others did not find any significant sex differences. A recent systematic review concluded that advanced pubertal status was linked to risk of depression among adolescent girls, but not boys, and that moderators of the link between puberty and depression mainly included environmental stressors (e.g., negative life events, peer victimization; Stumper & Alloy, 2021). Other empirical work has not found evidence of sex differences, but rather, one study concluded comparable elevated anxiety and depressive symptoms in boys similar to that of girls during puberty (Susman & Dorn, 2013). In this way, boys may be experiencing the stressful biological and psychosocial changes at a similar level to that of girls, but their experiences may more likely go unnoticed because their pubertal maturation generally starts later and lacks easily identifiable physical markers compared to girls (Deardorff et al., 2019). Such mixed findings suggest further research is needed to examine the sex differences in these puberty-internalizing associations to determine whether adolescent boys and girls are experiencing similar or different effects of puberty on the development of internalizing symptoms. Potential moderators and mediators (e.g., stress, sleep) in the link between puberty and internalizing symptoms require attention to help in understanding why sex differences may or may not be present.

Stress Moderating the Association Between Pubertal Development and Internalizing Symptoms

As mentioned previously, the *contextual amplification model* posits that puberty-stress interactions are context dependent (e.g., family environment, peer relationships) and have effects on youth development (Caspi et al., 1993; Ge et al., 2001; Ge & Natsuaki, 2009). In two samples of predominantly White adolescents, more advanced pubertal status and greater recent stressful events (e.g., peer problems, harsh parenting) have been identified as independent predictors of higher levels of anxiety and depressive symptoms in adolescents, and have also been shown to have interactive effects such that earlier pubertal timing and greater stress predicted higher levels of anxiety and depression (Ge et al., 2001; Winer et al., 2016). Further examination of specific types of stress (i.e., family stress, peer stress) evident during the transition to adolescence and how effects of puberty and stress to internalizing symptoms might differ for boys and girls remains a necessary direction of research.

Family Stress as a Moderator

Stress in the family context (e.g., parent psychopathology, harsh parenting, parental rejection) has been studied as an influential moderator of the associations between pubertal development and the development of internalizing symptoms and disorders in youth. Evidence has been found in support of the contextual-amplification model such that earlier pubertal development predicted greater depression one year later for predominantly White, socioeconomically diverse school-aged youth (*M*age = 12.41 years) who had experienced recent maternal depression and family stress, as compared to

youth who were not exposed to these risk contexts (Rudolph & Troop-Gordon, 2010). For both boys and girls, earlier pubertal timing interacted with maladaptive parenting behaviors in the prediction of internalizing outcomes (Benoit et al., 2013; Winer et al., 2016). A longitudinal study of Canadian youth found parental rejection (measured at age 12-13 years) moderated the associations between pubertal onset (at age 12-13 years) and subsequent depressive symptoms (at age 16-17), such that youth with high parental rejection had the strongest associations between early pubertal onset and depressive symptoms, as compared to their peers with lower levels of parental rejection (Benoit et al., 2013). Another study found that harsh parental discipline moderated the associations between earlier pubertal timing and anxiety symptoms, but not depressive symptoms in a sample of predominantly White children and adolescents (*Ma*ge = 12.13 years; Winer et al., 2016). Specifically, those who reported high levels of harsh discipline had stronger associations between early pubertal timing and anxiety symptoms, as compared to those experiencing lower levels of harsh discipline (Winer et al., 2016). Overall, these studies demonstrate the importance of understanding the moderating role of family risk factors on associations between puberty and internalizing outcomes, but no study to date has looked at how these various family factors may collectively moderate the puberty-internalizing pathway. By examining the effects of multiple types of family stress together in a single composite, the current study aims to understand how overall family stress is impacting the relation between pubertal development and internalizing symptoms (Figure 1, W_1 on *c* path).

Research findings regarding sex differences in the moderating effects of family stress on the association between puberty and internalizing symptoms are sparse and inconsistent. A previously mentioned study found no sex differences such that high levels of depressive symptoms were found in early maturing boys and girls who experienced parental rejection (Benoit et al., 2013). In addition, three-way interactions were used to examine the interaction of puberty, harsh parental discipline, and child sex, on depressive and anxiety symptoms, and these results were non-significant, suggesting that contextual amplification of earlier pubertal timing may exist in both boys and girls (Winer et al., 2016). Aside from these studies, other research has reported significant sex differences. For example, results from a study examining the development of internalizing symptoms suggested that earlier pubertal timing predicted higher internalizing symptoms in girls, but not boys, experiencing high levels of parental psychological control (e.g., invalidating feelings), compared to youth without that family stress risk factor (Arim & Shapka, 2008). For boys, early maturation predicted lower internalizing symptoms for those with higher levels of parental psychological control (Arim & Shapka, 2008). Therefore, it remains important to examine the sex differences in the moderating effects of family stress on the association between puberty and internalizing symptoms. The *gendered deviation hypothesis* would suggest the examination of early maturing girls and late maturing boys, such that they may be at higher risk for internalizing symptom development as compared to their on-time maturing peers (Brooks-Gunn et al., 1994; Sontag et al., 2011). Thus, it may be that adolescents with higher family stress may demonstrate stronger associations between lesser pubertal development and greater

internalizing symptoms for boys, and greater pubertal development and greater internalizing symptoms for girls, as compared to their peers with lower levels of family stress.

Peer Stress as a Moderator

Another salient form of psychosocial stress for early adolescents is peer stress, or stress related to difficulties with peer relations. Peer stress, including victimization, rejection, and problems getting along with peers, in childhood and adolescence has been linked with concurrent, as well as long-term outcomes into adulthood, in multiple realms of well-being including physical health, academic functioning, social relations, and mental health (McDougall & Vaillancourt, 2015; Stapinski et al., 2015). A few studies have examined the role of peer stress as a moderator of associations between pubertal development and internalizing symptoms. For both boys and girls, those with greater instances of peer problems had stronger associations between earlier pubertal timing and higher anxiety and depressive symptoms, compared to those with less peer problems (Winer et al., 2016). Further, peer stress has been examined as a moderator of the association between puberty and depression in a sample of predominantly White, socioeconomic diverse adolescents (*M*age = 12.39 years) such that, for adolescents with high levels of peer stress (i.e., peer relationships were more stressful than supportive), earlier pubertal timing for girls and late timing for boys predicted higher depression levels one year later (Conley & Rudolph, 2009). These results align with contextual amplification and the latter study is in support of the gendered deviation hypothesis. Therefore, the current study extended these findings from previous studies and examined

internalizing symptoms as the outcome of the interaction between pubertal development and peer stress (Figure 1, W_2 on *c* path).

Similar to studies examining family stress, the peer stress literature has produced inconsistent findings regarding significance and direction of results, resulting in a lack of clarity regarding sex differences. A previously mentioned study that did not find significant sex differences when family stress was the moderator of the association between puberty and internalizing symptoms, also found a lack of sex differences when peer stress was examined as a moderator, such that earlier pubertal timing was related to the highest levels of anxiety and depressive symptoms for boys and girls with greater peer problems (Winer et al., 2016). In support of sex differences, another study found that sex moderated the mediational pathway from puberty to peer stress and peer stress to depression, such that longitudinal associations between puberty and depression were mediated by peer stress, with stronger associations in girls compared to boys (Conley et al., 2012). In addition, directionality of results for the path from puberty to peer stress was opposite for girls and boys, such that earlier pubertal timing predicted more peer stress in girls, but less peer stress in boys (Conley et al., 2012). It is important to note, though, that this study examined peer stress as a mediator rather than a contextual moderator. Overall, the effects of puberty and peer stress on internalizing symptom development in girls seems to be more consistent, while earlier pubertal development in boys has demonstrated mixed results. Additional research can continue to examine the potential sex differences in the role of peer stress as a moderator in youth at the onset of puberty and effects on internalizing symptom outcomes.

Sleep as a Mediating Process Between Puberty and Internalizing Symptoms

The biopsychosocial and contextual model of sleep in adolescence posits that sleep is an integral factor in adolescent development and has bidirectional associations with biological, psychosocial, and contextual factors (Becker et al., 2015). Although there are not yet any studies that have examined sleep as a mediator between puberty and internalizing symptoms, several studies suggest that pubertal development is associated with sleep-wake behaviors (Diao et al., 2020; Hoyt et al., 2018). For example, a prior study found that early pubertal timing was cross-sectionally linked with later bedtimes and shorter sleep durations in Chinese adolescents (Diao et al., 2020). In a study of socioeconomically and ethnically diverse adolescent girls, earlier pubertal timing was longitudinally associated with shorter sleep durations, (Hoyt et al., 2018). In turn, there is a large literature that has demonstrated that poor sleep was prospectively associated with higher internalizing symptoms in adolescence (Nunes et al., 2020; Pieters et al., 2015; Shimizu et al., 2021). For example, more sleep problems (e.g., problems going to bed, problems falling asleep) in a Netherlands sample of predominantly White early adolescents (*M*age = 13.96 years) prospectively predicted greater internalizing symptoms one year later (Pieters et al., 2015). In a socioeconomically diverse sample of high school students (68% White/European American, 32% Black/African American), longitudinal associations between sleep-wake behaviors during late childhood (age 9) to anxiety and depressive symptoms at age 18 were found, such that youth with higher sleep-wake problems in childhood had higher levels of anxiety and depressive symptoms in adolescence (Shimizu et al., 2021). While no known studies have examined the exact

mediational model in the current study (Figure 1, *ab* path), one related study examined a mediation pathway from puberty to sleep to alcohol use and found that pubertal development was positively associated with sleep problems and later bedtimes, which, in turn, predicted higher levels of alcohol use (Pieters et al., 2010). Internalizing symptoms is another problem behavior that first presents during the transition to adolescence and, therefore, is a critical outcome to explore in this mediational pathway.

While extant research has demonstrated links between pubertal development and sleep, as well as sleep and the development of internalizing symptoms, a large part of this literature has relied on self-reports of sleep, but some researchers have used objective sleep measurement. To highlight a few studies that have used actigragh sleep, a cross-sectional study of adolescents (ages 13-18) who were further along in pubertal development demonstrated greater amounts of wake after sleep onset and lower sleep durations as compared to less mature adolescents (Short et al., 2012). In contrast, in a sample of Hispanic and White youth (*Mage*= 8.41 years), more advanced pubertal development was associated with longer sleep durations and higher sleep efficiency (Lecarie et al., 2022a). These opposing results highlight the importance of studying puberty-sleep associations at all stages of pubertal development, as results may differ with age. In terms of associations between sleep and internalizing outcomes, a study of a community sample of youth (69% European American; 31% African American) found that shorter sleep duration at age 10 was associated with higher anxiety and depressive symptoms at age 13 (Kelly & El-Sheikh, 2014). Actigraph-based sleep is a valid and reliable sleep measurement in youth (Meltzer et al., 2016). Importantly, rather than

subjective reports of sleep problems, objective measurement of sleep allows for the calculation of various sleep indicators including sleep quantity, quality, timing, and variability. In addition to adolescent-reported sleep problems, actigraphy sleep was examined as an intermediary process between pubertal development and internalizing symptoms in the current study (Figure 1, *ab* path), as sleep may be a malleable factor and potential method of intervention for children starting to go through pubertal development.

Moderating Effects of Stress on Puberty to Sleep and Sleep to Internalizing Symptoms

Although stress and sleep have both been studied as factors contributing to the onset of internalizing symptoms, few studies have taken both stress and sleep into account when examining internalizing symptom outcomes, and these studies have not directly considered the rate of pubertal development. The biopsychosocial framework would support the examination of the psychosocial (e.g., family stress, peer stress) and biological factors (e.g., puberty, sleep) relevant to the onset of internalizing symptoms in one all-encompassing model (Ullsperger & Nikolas, 2017). One study of a community sample of predominantly White adolescent girls (*M*age = 12.4 years) examined the interaction of three variables; reward processing, stressful life events (i.e., school and family problems, friendship and romantic relationship difficulties), and sleep problems in the prediction of depressive symptoms, and found that those with lower reward response were at higher risk of developing depressive symptoms if they also had high levels of stress and sleep problems (Burani et al., 2021). Another recent study of female adolescents aged 15-17 years examined objective sleep duration as a mediator of the

association between stressful life events and anxiety and depressive symptoms and found that within-person increases in stressful life events (i.e., peer relationships, academics, and health) were associated with greater variability in sleep duration, and higher sleep duration variability predicted greater anxiety symptoms (Vidal Bustamante et al., 2020). These studies include stress, sleep, and internalizing variables, but do not consider the role of pubertal development, and have samples of older adolescents as compared to the early adolescent sample of the current study. In addition, rather than examining stress as a predictor in these associations, the current study focused on stress as a contextual variable that may moderate these associations.

Sex-stratified literature on this topic is limited, but evidence suggests that females have greater increases in sleep problems and higher instances of internalizing symptoms after the onset of puberty as compared to males (Nunes et al., 2020). The current study built on these findings by including pubertal development as a key predictor of sleep and internalizing symptomatology, examining peer stress and family stress as unique stress moderators of the associations between pubertal development and sleep (W_3 and W_4 on *a* path, Figure 1), as well as sleep and internalizing symptoms (W_5 and W_6 on *b* path, Figure 1), and conducting these analyses in both boys and girls to understand whether sex differences exist.

Stress as a Moderator of the Heritability of Internalizing Symptoms

The *contextual-amplification model* suggests that genetics are biological dispositional factors that may impact development (Caspi et al., 1993). In this way, examining the heritability of internalizing symptoms may aid in understanding the extent

to which the development of these symptoms is more genetically or environmentally-based. With a twin sample, quantitative genetic ACE models can be used to estimate genetic and environmental influences on variances and covariances. Monozygotic (MZ) twins share 100% of their segregating DNA, while dizygotic (DZ) twins share 50% of their segregating DNA, on average. The shared environment consists of all non-genetic factors contributing to similarities between twins, while the nonshared environment includes environmental influences contributing to differences between MZ and DZ cotwins, as well as measurement error. Therefore, any differences between MZ twins are due solely to nonshared environmental influences, while DZ twin differences may be genetic or environmental.

Regarding the heritability of internalizing symptoms, family sibling clustering, or ongoing sibling similarity, of internalizing symptoms has been identified and accounted for due to the heritability of internalizing symptoms and the shared environmental effects in early childhood (Daniel et al., 2019). A review article found support for a strong genetic influence of both the etiology and stability of internalizing psychopathology, and small to insignificant gender differences, across childhood and adolescence (Franić et al., 2010). A recent study similarly concluded that genetic influences of internalizing symptoms are constant during adolescence, while environmental influences varied with age such that shared environmental effects of internalizing symptoms decreased across development (Patterson et al., 2018). Another study found early pubertal status moderated internalizing symptoms and was associated with increased genetic influences of internalizing symptoms in girls, but not boys (Corley et al., 2015). With the heritability

of internalizing symptoms increasing or staying consistent and shared environmental effects decreasing during adolescence, this leaves room for the potential increasing effects of nonshared environmental effects (e.g., unique stressors experienced by each twin).

The onset and severity of internalizing symptoms differs between individuals, and understanding the role of genetics in these individual differences may assist in determining effective preventative interventions or treatments by matching treatments to identified genetic predictors (Lemery-Chalfant et al., 2018). In this way, it may be helpful to know whether children are more genetically sensitive to stress in the environment (e.g., family stress, peer stress) or less sensitive to these contexts to then determine a best method of intervention in the prevention of high stress and resulting increases in internalizing symptoms. Extending the ACE models examining heritability of a trait, moderation of heritability models can be used to examine whether genetic and environmental influences of a trait vary by a moderator (Purcell, 2002; van der Sluis et al., 2012). In this way, stress experiences can be examined as a moderator of the genetic and environmental influences of internalizing symptoms, such that stress experiences might change the expression of genes important for the development of internalizing symptoms. While some studies have concluded lower internalizing heritability estimates as a result of stressful events (i.e., parental divorce and peer victimization; Brendgen et al., 2015; Hicks et al., 2009), other studies have determined higher heritability of depressive symptoms for adolescents who experienced a greater number of stressful life events (Lau & Eley, 2008). Specifically, stressful life events moderated the genetic

influences of depression and anxiety in adolescent girls, such that higher exposure to stressful life events increased the genetic variance (Silberg et al., 2001). Therefore, while internalizing psychopathology has strong genetic influences, it is not yet known how family stress and peer stress might alter the genetic and environmental influences, as research thus far is quite limited and has produced inconsistent findings.

Current Study

The current study tested four study aims. The first three aims were phenotypic analyses and were conducted separately for boys and girls. Phenotypic analyses examined family and peer stress as moderators of pubertal development to internalizing symptoms, and sleep indicators as mediators, while behavior genetic analyses focused on the role of family and peer stress as moderators on the genetic and environmental contributions of internalizing symptoms. The first aim (*c* path, Figure 1) examined whether pubertal development (at age 10) predicted internalizing symptoms (at age 11), while controlling for initial internalizing symptoms (at age 10). In line with the *gendered deviation hypothesis*, I hypothesized that girls with more advanced pubertal development and boys with less advanced pubertal development would have higher internalizing symptoms (Brooks-Gunn et al., 1994; Sontag et al., 2011).

The second aim (W_1 and W_2 on *c* path, Figure 1) examined whether stress (i.e., family stress and peer stress at age 10) moderated the relation between pubertal development (at age 10) and internalizing symptoms (at age 11), while controlling for initial internalizing symptoms (at age 10). Regarding family stress, I hypothesized that girls with high family stress would have stronger positive associations between greater

pubertal development and higher internalizing symptoms, as compared to girls in low family stress environments (Benoit et al., 2013; Winer et al., 2016). Given existing theory and empirical evidence, I predicted that boys with high family stress would have stronger negative associations between pubertal development and internalizing symptoms, as compared to boys with low family stress (Ge et al., 2001; Sontag et al., 2011). Regarding peer stress, I hypothesized that youth with higher peer stress would have stronger positive associations for girls and negative associations for boys between pubertal development and internalizing symptoms, as compared to youth with lower peer stress experiences (Conley & Rudolph, 2009).

The third aim was an exploratory analysis that tested mediation pathways from (a*b*, Figure 1) pubertal development to internalizing symptoms indirectly through both self-reported sleep (i.e., sleep hygiene, sleep insomnia, and daytime sleepiness at age 10) and objective sleep (i.e., duration, efficiency, midpoint, and midpoint variability at age 10). A secondary exploratory aim tested a moderated mediation model with family and peer stress as moderators of the associations between pubertal development and sleep (W_3 and W_4 on *a* path, Figure 1), and as a moderator on the path from sleep to internalizing symptoms (W_5 and W_6 on *b* path, Figure 1) for three sleep variables (i.e., sleep duration, sleep insomnia, daytime sleepiness) because these have been widely researched sleep variables in adolescents (Meltzer et al., 2012a; Meltzer et al., 2013).

The fourth study aim examined whether family stress moderated the genetic and environmental influences of internalizing symptoms (Figure 2a). I hypothesized that

family stress would moderate the genetic and environmental influences on internalizing symptoms for all youth (Hicks et al., 2009).

While the Purcell (2002) model is appropriate if the moderator does not differ between twins (i.e., family-level moderator; family stress), it may lead to inflated false positive rates if the moderator differs between twins (i.e., individual-level moderator; peer stress), if the moderator is correlated across twins, or if the moderator and outcome are correlated. Potential false positive results may occur if there is moderation of the covariance between the moderator and outcome because this moderation is modeled as the moderation on the variance components unique to the trait (van der Sluis et al., 2012). Therefore, an extension to the Purcell (2002) model has been suggested to alleviate this issue, such that the moderator values for both twins are entered into the means model of the outcome for each twin (Figure 2b). In this way, moderation is modeled on the residual outcome variance, which does not overlap with the moderator (Burt et al., 2014). This model removes any potential gene-environment correlation confounds and is more parsimonious than a bivariate moderation of heritability model (van der Sluis et al., 2012). With a sample size of 1,000 twin pairs, this model can be robustly estimated, but is sensitive to start values and local minima (van der Sluis et al., 2012). Due to lack of an adequate sample size, this method was not able to be implemented to test how the second moderator (i.e., peer stress), moderates the genetic and environmental components of the phenotype of interest (i.e., internalizing symptoms; Figure 2b).

CHAPTER 2

METHOD

Participants

Participants are families from the Arizona Twin Project, which is an ongoing longitudinal study of twins including those who were initially recruited from birth records in the state of Arizona, or who now live in Arizona (Lemery-Chalfant et al., 2019). Families were initially recruited through state birth records for participation in the first assessment wave when twins were 12 months old and additional families were recruited through online advertising at subsequent study waves to allow for a sample size with the power necessary to conduct twin modeling. The analyses for the current study aims included data from the 10 year (785 children; M_{age}= 10.88, *SD*= 1.15; data collection took place May 2018 to July 2021) and 11 year (628 children; M_{age}= 11.63, *SD*= 1.04; 55% participated pre-COVID onset (marked by 3/25/2020), 45% participated post-COVID onset; data collection took place April 2019 to June 2022) study waves. For the 11 year wave, 7 new families were recruited into the study. Of these two study waves, 594 children participated in both waves.

The community-based subsample consisted of 818 children (51% female; 32% monozygotic (MZ) twin pairs, 36% same-sex dizygotic (DZ), 32% opposite-sex DZ) who participated in the 10 year and/or 11 year study waves. Twins were diverse in race/ethnicity with 25.7% Hispanic/Latino, 2.6% Asian/Asian American, 3.3% Black/African American, 3.0% Native American, 0.5% Native Hawaiian/Pacific Islander, 56.5% non-Hispanic White, 6.8% biracial or multiracial (4.6% of these twins report

Hispanic/Latino as one of their racial/ethnic origins), and 1.6% other ethnicity. Families were also diverse in socioeconomic backgrounds with 6.6% of families living in poverty, 21.6% living near the poverty line, 22.7% lower middle class, and 49.1% middle to upper class (i.e., based on income to needs and according to the U.S. Census Bureau federal poverty threshold). In terms of education, primary caregivers reported having completed a college degree (37.8; 35.4% secondary caregiver), two or more years of graduate school (4.2; 2.5%), completed graduate or professional degree (21.4; 20.6%), completed some college (28.2; 23.2%), a high school degree or equivalent (7.3; 16.4%), or less than a high school degree (1.1; 1.9%).

Procedure

Institutional Review Board approval was obtained prior to the start of each study wave. Primary caregiver informed consent and children's assent were obtained before each wave of data collection. This study utilized data from the 10 year and 11 year waves to capture early adolescence, including the onset of pubertal development and internalizing symptoms. Families were compensated for their participation at each study wave.

At the 10 year study wave, families were contacted to participate in an assessment involving online questionnaires, one home visit, and a week of daily assessments, including actigraphy-based sleep measurement. During the home visit, two trained research assistants administered questionnaires to twins regarding their pubertal development, mental health symptoms, and sleep. Twins answered questions about puberty in private, but could ask questions to the study staff if any clarifications were

needed. Biological measurements including twin height, weight, waist circumference, and percent body fat were collected during the home visit. Primary caregivers (95% mothers) completed questionnaires related to their twins' health, pubertal development, and stress at home and with peers.

During the study week, twins wore wrist-based accelerometers (Ambulatory Monitoring, Inc, Ardsley, NY USA) for approximately 7 (M=6.81, SD = .86) consecutive nights to measure their sleep. During this week, primary caregivers also reported on twin wake times and bedtimes via assessment tables to cross-reference actigraph-assessed measures. Study staff contacted families before and throughout their study week to ensure that all procedures were being followed and to answer any questions.

At the 11 year study wave, twins and their primary caregivers participated in a home visit (56% in person, 44% virtual) in which trained research assistants collected physical health assessments and administered questionnaires assessing twins' self-report of puberty, mental health, and sleep. Following the onset of COVID, data collection methods were moved to a virtual Zoom platform, such that research assistants conducted questionnaires with twins via Zoom. Physical health assessments were not collected for participants who completed virtual home visits.

Measures

Child Internalizing Symptomatology

The Berkeley Puppet Interview (BPI; Measelle et al., 1998) was collected via child-report during the home visits at the 10 year and 11 year study waves. During this interview, children were asked questions in two stages: (1) which of two statements

applied to them (e.g., “You worry if other kids will like you,” or “You don’t worry if other kids will like you.”), and (2) how accurately the statement described them (“Sort of describes you,” or “Really describes you.”). Items were recoded into single items with higher scores meaning higher behavior problems (e.g., 1 = ‘Really describes you (You don’t worry if other kids will like you)’, 2 = ‘Sort of describes you (You don't worry if other kids will like you)’, 3 = ‘Sort of describes you (You worry if other kids will like you), and 4 = ‘Really describes you (You worry if other kids will like you)’). The possible scoring range of the BPI is a 1-6 scale. The internalizing composite consists of 26 items across depression, overanxious, and separation anxiety subscales. The internalizing composite at the 10 year wave (range= 1.00-5.04, $\alpha = .83$) was used as a baseline to control for the internalizing symptoms composite at the 11 year wave (range= 1.23-5.19, $\alpha = .86$). For the 11 year wave, the alphas were consistent across in-person home visits ($\alpha = .85$) and virtual home visits ($\alpha = .85$).

Pubertal Development

To measure pubertal status, twins and their primary caregivers completed the Pubertal Development Scale (PDS; Petersen et al., 1988) by rating pubertal indicators on a Likert scale from 1 (development has not yet started) to 4 (development is complete). The 5-item composite score captures growth in height, growth of body hair, and skin changes, as well as breast growth and menstruation for girls, and voice deepening and growth of facial hair for boys. The male and female composite scores were standardized separately. The PDS has been well validated and shown to be highly correlated with pubertal stages via physical exam (Conley et al., 2012; Shirtcliff et al., 2009). If

correlated, youth and caregiver reports can be averaged together to form a puberty composite index (Conley et al., 2012). As twin-report and primary caregiver-report PDS scores were moderately correlated in this sample (r = .45 for boys and r = .74 for girls; ps < .001), scores were averaged to form an overall composite of pubertal status, such that higher scores indicate more advanced pubertal development (α = .85 for girls, α = .62 for boys).

Sleep

Self-reported sleep. The Children's Report of Sleep Patterns (CRSP) is a 60-item self-report questionnaire, in which twins reported on various aspects of their sleep including their sleep patterns, sleep hygiene, sleep disturbances, and daytime sleepiness (Meltzer et al., 2012b; Meltzer et al., 2013). The CRSP is a valid and reliable self-report measure for school-aged children and adolescents (Brimeyer et al., 2013; Gamble et al., 2012). The Sleep Patterns scale includes questions about their wake times and bedtimes, subjective sleep quality, and sleep schedule variability during the last night, over a typical week and weekend, and overall sleep on most days. This scale is often used for descriptive purposes and was not used in the current study, as we instead relied on actigraphy data to capture sleep patterns over a typical week. The Sleep Hygiene scale (18 items) assesses caffeine use, activities before bed, electronic use, and sleep location on a Likert scale from 1 (never) to 5 (always). A mean score of the Sleep Hygiene scale was computed such that higher scores indicated poorer sleep hygiene. A Cronbach's alpha was not calculated for the Sleep Hygiene scale, as this scale captures different facets of sleep that would not be expected to behave similarly. The Sleep Disturbances

scale includes subscales related to bedtime fears and worries, restless legs, parasomnias, and insomnia. The insomnia subscale (e.g., trouble falling asleep at bedtime, wake up during night; 6 items; $\alpha = .68$) was used in the current study to measure sleep disturbances and higher mean scores indicated greater levels of insomnia. The Daytime Sleepiness scale includes 5 items related to daytime sleepiness (i.e., felt sleepy or fell asleep in different situations) on a Likert scale from 1 (never) to 5 (always). A mean score of the Daytime Sleepiness scale was computed such that higher scores indicated greater levels of sleepiness ($\alpha = .72$).

Actigraphy. Twins wore wrist-based accelerometers (Motion Logger Micro Watch; Ambulatory Monitoring, Inc, Ardsley, NY USA) on their non-dominant wrist for 7 consecutive days and nights to capture their sleep. Motion was be measured in one-minute epochs using a zero-crossing mode and sleep data was scored using the Sadeh algorithm in Action W-2 software version 2.7.1 program (Sadeh et al., 1994). Sleep indicators include duration (total time asleep in hours excluding any waking periods), efficiency (ratio of time spent asleep (duration) to total time in bed, with total time in bed including true sleep and waking periods), midpoint (midpoint between sleep start and end), and midpoint variability (within-person standard deviation estimate of sleep midpoint, averaged across all nights of the study week).

Actigraph sleep periods were validated against primary caregiver reports of twin bedtimes and wake times from daily assessment tables as a check of compliance. Compliance was high in this sample with 8.1% (N = 42 children) missing data due to watch malfunction and 2.1% (N = 11) of children not participating in the actigraphy

portion of the study. Of the children with actigraphy data, 76.0% (N = 393) of children had 7 or more nights of actigraphy data, 13.7% (N = 71) had 6 nights of data, 5.0% (N = 26) had 5 nights of data, 2.7% (N = 14) had 4 nights of data, 2.5% (N = 13) had 3 nights of data, and 7.7% (N = 40) of children wore the watch for less than three nights of their study week. Children excluded from analyses (N = 82) included those whose data was missing due to a watch malfunction and those who had data for less than three nights of their study week. Sensitivity analyses excluding children who had only three of four nights of sleep (n = 27) by treating their objective sleep data as missing were conducted to determine whether results differed from those who had five or more nights of sleep, and whether all available objective sleep data (i.e., three or more nights of sleep) should be included in analyses (Acebo et al., 1999).

Family Stress

A principal components analysis was conducted to form the family stress composite. Additional information on the measures in the family stress composite, including samples items and reliability of each scale, is in Table 1. The first principal component explained 46.47% of the variance (scale loadings .62-.75) and regression values were retained as the family stress composite, which is composed of the following primary caregiver- report measures: Parental Stress Scale (Berry & Jones, 1995); Confusion, Hubbub, and Order Scale (Matheny et al., 1995); Parenting Styles and Dimensions- Authoritarianism (Robinson et al., 1995); Interpersonal Support Evaluation List (reverse scored; Cohen et al., 1985); Spouse/Partner Strain Scale (adapted from Schuster et al., 1990 and Whalen & Lachman, 2000); Perceived Stress Scale (Cohen et

al., 1983); and Family Conflict Survey (Porter & O'Leary, 1980). Similar family stress composites were used at previous study waves (Lecarie et al., 2022b; Miadich et al., 2019). As child internalizing symptoms was the outcome of the current study analyses, parent anxiety and depressive symptoms were not included in the 10 year family stress composite. Due to standardization, this composite variable has a mean of zero.

Peer Stress

The MacArthur Health and Behavior Questionnaire (HBQ; Armstrong & Goldstein, 2003) parent-report of their child's peer relations over the past six months was collected at the 10 year assessment. The peer relations scale consists of 11 items total, 8 of which examined peer acceptance/rejection (e.g., "Has lots of friends at school,"; α = .89) and 3 of which examined peer victimization (e.g., "Is picked on by other children,"; α = .78). Items were scored on a 1-4 Likert scale (1 = Not at all like, 2 = Very little like, 3 = Somewhat like, 4 = Very much like). A negative peer relations composite was formed by ensuring that all acceptance/rejection items were reverse-scored such that higher scores indicated worse peer relations. Then all acceptance/rejection and victimization items were averaged to create a mean score. Therefore, higher scores on the peer relations composite indicated lower peer acceptance and higher peer victimization (α = .91).

Zygosity

Primary caregivers completed the Zygosity Questionnaire for Young Twins (Goldsmith, 1991), a 32-item questionnaire about the birth and observed physical differences between the twins, which has been found to be over 95% consistent with zygosity determined by genotyping (Price et al., 2000). The questionnaire was

supplemented with physical similarity assessments at the home visits, as well as hospital birth records.

Covariates

For phenotypic analyses, age, race/ethnicity (1 = White, 0 = non-White), and family socioeconomic status (SES) at age 10 were included as covariates throughout analyses. SES was the standardized composite of the family-income-to-needs ratio, primary caregiver education level, and spouse/partner education level (Doane et al., 2019). BPI internalizing symptoms at age 10 was used as a baseline and controlled for throughout analyses when age 11 internalizing symptoms is the outcome measure. A COVID indicator was included as a covariate in all analyses (0 = participation occurred prior to 3/25/2020, 1 = participation occurred after 3/25/20), as the pandemic took place during the 11 year study wave. Body mass index (BMI) and a variable indicating whether the twins were in school or on summer/holiday break during participation (1 = on vacation, 0 = school year participation) were included as covariates in analyses examining sleep. Height and weight measures from the home visit at the 10 year wave were used to calculate BMI scores using the child BMI formula: weight (kg)/ $[\text{height(m)}]^2$ (Centers for Disease Control, 2015). Height (in inches) was measured using a tape measure and rounding to the nearest half inch. Weight (in pounds) was measured using the Tanita Child Scale, an FDA-approved full body composition scale (Tanita, 2016). Weight measurements were taken three times for each twin and an average was taken to calculate a single weight estimate for each child. For genetic analyses, covariates included sex and age to allow for the examination of the overall proportion of variance in

one or more phenotypes associated with broad genetic or environmental factors, while including meaningful variance from several variables (e.g., race and family SES).

Data Analysis

Prior to testing the study aims, descriptive statistics and bivariate correlations were examined to assess for normality. Distributions for variables were examined to determine outliers and to conduct necessary transformations or winsorizing of variables. The analyses for the first three aims were conducted in Mplus 7.0 (Muthén & Muthén, 2015) using full information maximum likelihood to account for missing data and the *type=complex* command to account for the nesting of twins within families. Analyses for all three phenotypic study aims were multigroup analyses such that they were conducted separately by child sex. The behavior genetic analyses for the fourth study aim were conducted in OpenMx within R (Neale et al., 2016).

For the first aim (*c* path, Figure 1), structural equation modeling was used to test associations between pubertal development and subsequent internalizing symptoms. For aim 2 (W_1 and W_2 on *c* path, Figure 1), structural equation modeling was used to test pubertal development, stress (i.e., family, peer), and their interaction in the prediction of internalizing symptoms. Interaction terms between pubertal development and each type of stress were created and tested in separate models, resulting in a total of 2 models.

For the third aim (a*b*, Figure 1), mediation models were run to test the potential mediating role of sleep between pubertal development and internalizing symptoms. Separate mediation models were run such that pubertal development, the sleep mediator (i.e., self-reported sleep insomnia, sleep hygiene, daytime sleepiness, or objectively

measured sleep duration, efficiency, midpoint, or midpoint variability), and internalizing symptoms were all at the individual, or twin, level, though the model adjusted for the nesting of twins within families. To test moderated mediation, conditional indirect effects were probed at conditional levels of the moderator (mean, +1 SD, -1 SD; Preacher et al., 2007) to test how level of stress (i.e., family, peer) related to differences in how pubertal development affects sleep (W_3 and W_4 on *a* path, Figure 1) and how sleep affects internalizing outcomes (W_5 and W_6 on *b* path, Figure 1). Total and direct effects were tested by examining the statistical significance of each path using 95% confidence intervals (Preacher et al., 2011). Indirect effects were tested using RMediation (Tofighi & MacKinnon, 2011).

The fourth aim was the behavioral genetics aim that utilized the twin design (Neale & Maes, 2004). First, univariate (ACE) twin models were fit to decompose the variance of peer stress and internalizing symptoms into additive genetic (A), shared environmental (C), and non-shared environmental factors. As monozygotic (MZ) twins share 100% of their segregating DNA and dizygotic (DZ) twins share 50%, the latent A factor was set to 1.00 correlation for MZ twins and .50 for DZ twins. For all cotwins, the correlation between latent C factors was set to 1.0 because C encompasses shared environmental factors, and E was uncorrelated. A full univariate model was compared to reduced models in which parameters were systematically dropped. Model fit was examined using the -2 log likelihood chi-square difference test of fit, such that a significant loss of fit indicates a parameter that is needed to represent the observed data.

A moderation of heritability model (Figure 2a) was used to test how the latent genetic, shared, and nonshared environmental factors of a trait varied as a function of a family-level moderator (Purcell, 2002). In this way, the first phenotype, or moderator (i.e., family stress), moderates the genetic and environmental components of the second phenotype (i.e., internalizing symptoms). Figure 2a represents this moderated model with the moderator (M) moderating the ACE components of the second phenotype (T, the trait). The path coefficients were expressed as linear functions of the moderator, as they represent the magnitude of the effect. A significant non-zero βx parameter indicates an interaction between the path coefficient and the moderator (Purcell, 2002). This model controlled for gene-environment correlation by parsing the shared genetic and environmental effects between the trait and moderator into a main effect. A significant loss of fit as a result of dropping that path indicates that this path should be retained in the model because there is a significant correlation between the moderator and the trait (Purcell, 2002).

CHAPTER 3

RESULTS

Descriptive Statistics and Preliminary Analyses

Tables 2 and 3 show participant demographics and descriptive statistics. Table 4 includes correlations between study variables. BPI internalizing symptoms (age 11) was positively correlated with many of the sleep indicators, both family stress and peer stress, internalizing symptoms at age 10, and the COVID indicator (i.e., twins participating after the start of COVID reported higher internalizing symptoms), for boys and girls. For all

twins, family stress and peer stress were positively correlated. Family stress and peer stress were correlated with many sleep indicators for boys, while, for girls, family stress was not correlated with sleep variables and peer stress was negatively correlated with sleep duration and positively correlated with daytime sleepiness.

Aim 1: Puberty to Internalizing

For girls, the association between pubertal development and internalizing symptoms one year later was not significant. For boys, there was a significant association between pubertal development and internalizing symptoms, such that boys with greater pubertal development reported higher internalizing symptoms (b = 0.13, p = .03; Table 5).

Aim 2: Stress Moderating the Association Between Pubertal Development and Internalizing Symptoms

Table 6 shows the moderation models for family stress (Model 1) and peer stress (Model 2). In Model 1, the main effects of pubertal development, family stress, and their interaction were all non-significant for girls. For boys, there was a significant main effect of pubertal development on internalizing symptoms (b = 0.12, p = .04), but the main effect of family stress and the interaction between pubertal development and family stress were not statistically significant. Model 2 demonstrated similar results to model 1 for boys, such that there was a significant main effect of pubertal development (b = 0.13, p = .03), while the main effect of peer stress, and the interaction between peer stress and pubertal development were not significant. For girls, there was significant interaction between peer stress and pubertal development on internalizing symptoms (b = 0.12, p =

.04). Simple slopes analyses indicated that the link between pubertal development and internalizing symptoms was not significant for girls with low (-1 SD below mean; b = -0.11, p = .05), average (b = -0.03, p = .58) or high (+1 SD above mean; b = 0.06, p = .41), peer stress levels, but the association between greater pubertal development and higher internalizing symptoms is stronger for girls with higher peer stress (Figure 3).

Aim 3: Sleep as a Mediating Process Between Puberty and Internalizing Symptoms

Results with children with fewer than five nights of sleep data treated as missing were consistent with results including all children with available sleep data, therefore, all available data was used included in analyses. Exploratory mediation analyses demonstrated a significant direct effect of pubertal development on internalizing symptoms for boys in all mediation models (Figure 4). For CRSP sleep hygiene and daytime sleepiness, boys demonstrated a positive association between sleep to internalizing symptoms such that greater problems with sleep hygiene and sleepiness predicted higher internalizing symptoms. There were no significant indirect paths of sleep variables between puberty and internalizing symptoms for boys. For girls, there were no significant paths for the sleep duration, midpoint, insomnia, or sleepiness models. Girls demonstrated significant positive associations between pubertal development to sleep for sleep efficiency, sleep hygiene, and sleep midpoint variability, such that greater pubertal development predicted higher sleep efficiency, greater sleep hygiene problems, and greater sleep midpoint variability.

Aim 3: Moderating Effects of Stress on Puberty to Sleep and Sleep to Internalizing Symptoms

Exploratory moderated mediation models were conducted with family stress and peer stress as moderators and sleep duration, sleep insomnia, and daytime sleepiness as mediators (Table 7). For the sleep insomnia mediation models (Table 7a, 7b), there was no significant moderation of family stress or peer stress for boys or girls, but peer stress was positively associated with insomnia symptoms for boys and girls. For models with sleep duration as the mediator (Table 7c, 7d), peer stress, but not family stress, interacted with sleep duration in the prediction of internalizing symptoms for boys and girls, but simple slopes of sleep duration at conditional levels (-1 SD, mean, +1 SD) of peer stress were not significant (Figure 5a, 5b). Lastly, the daytime sleepiness mediator models indicated no significant moderated paths (Table 7e, 7f). Daytime sleepiness directly predicted internalizing symptoms for boys, but not girls, such that boys with greater daytime sleepiness reported higher internalizing symptoms.

Aim 4: Moderated Heritability of Family Stress on Internalizing Symptoms

Twin intra-class correlations indicated greater MZ than DZ twin similarity on peer stress, supporting genetic influences, but not for internalizing symptoms (Table 8). Table 9 contains fit statistics and parameter estimates for full and reduced univariate ACE models for internalizing symptoms and peer stress. The reduced CE model fit best for internalizing symptoms, with moderate shared environmental variance. Peer stress was moderately heritable, with the full ACE model providing the best fit.

The family-level moderated heritability model did not converge when estimating the path from family stress to internalizing symptoms in the model, likely due to attempting to estimate too many parameters for the sample size. Instead, this path was regressed out and residualized scores were used instead. When the moderator is family-level, it has been demonstrated that there is not an elevated false positive rate when the association between the moderator and phenotype is not included in the model (van der Sluis et al., 2012). It should be noted, though, that a limitation of this model is that it cannot rule out passive gene-environment correlation (van der Sluis et al., 2012). Model fit statistics for the full and reduced moderated heritability models are provided in Table 10. Fit statistics demonstrated that the model without moderation was the best-fitting reduced model, such that family stress did not significantly moderate the ACE components of internalizing symptoms and ACE estimates remained constant across levels of family stress.

Sensitivity Analyses

Sensitivity analyses were conducted for the phenotypic aims in which the 10 year internalizing variable was removed from all models and not included as a covariate. For aim 1, associations were consistent with the results including the 10 year internalizing symptoms composite (Table 11). For aim 2 (Table 12), the family stress moderation model results were highly consistent with the initial analyses, with one exception; for girls, there was a significant main effect of family stress on internalizing symptoms such that greater family stress predicter higher internalizing symptoms. For the peer stress moderation model, results changed such that, for boys, there were significant main effects

of pubertal development and peer stress on internalizing symptoms such that greater pubertal development and higher peer stress predicted higher internalizing symptoms. Similar to boys, girls also demonstrated a significant main effect of peer stress. The interaction between peer stress and pubertal development was significant for girls (Figure 6).

Mediation models conducted for sensitivity analyses were largely similar to initial mediation models. These models continued to demonstrate a significant direct effect from pubertal development to internalizing symptoms for boys. There were also significant positive associations from sleep insomnia, sleep efficiency, sleep hygiene, and sleepiness to internalizing symptoms for boys. For girls, the majority of the initial findings remained the same. Differences from initial findings include significant associations between sleep insomnia and sleep midpoint to internalizing, as well as a significant association between sleepiness and internalizing symptoms. Sleep hygiene to internalizing symptoms was also significant for girls, which resulted in a significant indirect effect of pubertal development to internalizing symptoms via sleep hygiene for girls (Figure 7).

CHAPTER 4

DISCUSSION

The results of this study support the importance of taking a biopsychosocial approach in understanding the puberty-internalizing relation and examining this relation separately for boys and girls. Results suggest that, in this sample, boys with greater pubertal development demonstrated higher internalizing symptoms one year later, but this pattern of results was not evident in girls. Further supporting sex differences, for boys,

neither peer stress nor family stress moderated the association between puberty and internalizing symptoms. Whereas, for girls, peer stress interacted with puberty to predict internalizing symptoms, while family stress did not. Mediation models examining sleep as a mediator between pubertal development and internalizing symptoms were largely non-significant, as were the moderated mediation models examining sleep as a mediator and family and peer stress as moderators. Although there was no significant mediation, girls demonstrated significant puberty-sleep pathways, while boys showed significant sleep-internalizing pathways. Behavior genetic analyses indicated that peer stress was moderately heritable, internalizing symptoms were environmentally influenced, and family stress did not moderate the heritability of internalizing symptoms. Overall, these study findings support the continued examination of the effects of pubertal development and other critical health factors (e.g., stress and sleep) on mental health during the transition to adolescence.

Puberty-Internalizing Association

The first aim of this study was guided by the gendered deviation hypothesis (Brooks-Gunn et al., 1994). Interestingly, results from this first study aim were not in line with the gendered deviation hypothesis as the association between pubertal development and internalizing symptoms was not significant in girls. While the gendered deviation hypothesis would support a negative association between puberty and internalizing symptoms for boys, results of the current study demonstrated a significant *positive* puberty-internalizing association for boys. The lack of significant results for girls opposes extant research that has consistently linked greater pubertal development with risk for

internalizing in girls, but many of these studies have focused on pubertal timing (e.g., early, on-time, and late maturation) rather than pubertal status and examined internalizing symptoms in later adolescence instead of early adolescence (Alloy et al., 2016; Ge et al., 2003; Pfeifer & Allen, 2021). A recent study examining pubertal synchrony (i.e., a measure of variability in pubertal development, or whether various pubertal indicators are developing synchronously within an individual) found sex differences in depression such that pubertal asynchrony was a risk factor for girls, but was a protective factor for boys (Stumper et al., 2020). In this way, for girls, it may be that the social deviance hypothesis (Petersen et al., 1988) is a more accurate representation of their puberty-internalizing association during early adolescence (i.e., age 10) and for pubertal status specifically. It is also possible that girls' negative perceptions surrounding puberty are starting to change in recent cohorts of early adolescent and adolescent youth via puberty education programs that promote positives attitudes and healthy practices related to pubertal development (Crockett et al., 2019). Prior literature has noted that perception of puberty is influenced by the extent to which girls are informed and knowledgeable on the course of pubertal development, and that girls who feel more prepared are more likely to experience an initial positive puberty experience (i.e., feeling happy, proud relieved, and excited; Short & Rosenthal., 2008). In this way, girls may have less worry regarding their physical body changes and, instead, more acceptance regarding different body types that come along with the course of pubertal development, so long as effective puberty health psychoeducation is provided (Barkhordari-Sharifabad et al., 2020). Future research should examine the link between girls' perceptions of their body development during

puberty and the amount of psychoeducation on pubertal processes they have received, as well as whether these associations differ across girls and their same-age boy counterparts.

The puberty-internalizing association for boys was opposite of my hypothesis and did not align with the gendered deviation hypothesis because boys with more advanced pubertal development demonstrated higher internalizing symptoms. This result may also point to the social deviance hypothesis, as boys may have difficulties when developing ahead of many of their peers, similar to what is known and widely consistent in the literature for girls (Petersen et al., 1988). Greater pubertal development has been linked to psychosocial advantages due to physical growth in height and muscle composition leading to athletic benefits, but this perspective is rooted in tradition and the idea of masculinity (Deardorff et al., 2019; Petersen et al., 1991). More recently, research has suggested that boys feel pressured to maintain a thin and muscular body image according to masculine gender norms as early as 8 years of age and persisting into adolescence (Tatangelo et al., 2018), and this body dissatisfaction may be linked to their increase in internalizing psychopathology. As the current study focused on age 10, the boys in this sample who were already undergoing puberty were likely developing well ahead of their same-age peers. Aside from growth in height, 52-71% of boys reported they had not yet experienced growth in any of the other pubertal indicators including growth of body hair, skin changes, deepening voice, and growth of facial hair. Therefore, the minority of boys in the sample who had started undergoing these types of pubertal growth may have been concerned that they were developing ahead of many of their peers. It is also important to recall that prior literature on pubertal development in boys is quite mixed, in part due to

the lack of an observable indicator equitable to menarche in girls, supporting the idea that pubertal development in boys may be going unnoticed as compared to girls (Deardorff et al., 2019). Despite this, some prior research has supported significant effects in boys in the direction found in the current study. Previous studies have demonstrated that boys with earlier pubertal timing are at risk for issues with psychosocial development, including higher anxiety and depressive symptoms (Huddleston & Ge, 2003; Mendle & Ferrero, 2012). Although a large portion of puberty research focuses on girls, these results suggest that boys too are socioemotionally impacted by pubertal development, even at the earlier pubertal stages, and that further research is needed to explore the effects of puberty on boys' overall health and development.

It may also be important to note that the age 11 study wave was conducted partly during the COVID-19 pandemic, so typical levels of internalizing symptoms and the relations between internalizing symptoms and other health factors may have been altered during this time, though analyses did control for study participation timing in relation to the start of the pandemic. Further, about half of the children who participated in the age 11 study wave completed virtual home visits and were currently or had recently been attending school virtually, so it is important to consider how social distancing and the use of virtual platforms may have impacted their study participation, particularly the internalizing symptoms outcome. One possible underlying mechanism that may be related to differential effects of pubertal development on internalizing symptoms in boys and girls during the pandemic is executive control. A recent study found that higher executive control may serve as a protective factor against increases in internalizing

symptoms for early-maturing youth who may be experiencing heightened uncertainty or stress (e.g., during a global pandemic; Chahal et al., 2021). As boys are known to have lower executive functioning skills than girls (Hasson & Fine, 2012), this points to a potential explanation of how the pandemic may have been experienced differently for boys and girls and why boys with greater pubertal development might have experienced heightened internalizing symptoms during this time, while girls' internalizing symptoms may not have been as highly affected by the pandemic. Future analyses with this sample might consider including executive control as a covariate or examining the effects of the COVID indicator as a moderator of the puberty-internalizing relation.

The Role of Family Stress

Regarding the second study aim, contrary to hypotheses, family stress was not found to be a significant moderator of the associations between pubertal development and internalizing symptoms for boys or girls. There are a couple of potential explanations for the lack of significant effects. First, at age 10, having just entered early adolescence, children's peer relationships often become the center of their time and attention as compared to their family relationships (Rubin et al., 2006). Therefore, family stress may not be as relevant or impactful for youth in the current study sample. It is important to note, though, that parents still have a significant impact on youth during early adolescence and low levels of parental behavioral control (i.e., firm and consistent discipline) has been linked with higher internalizing symptoms (Galambos et al., 2003). While parenting behaviors may be of particular importance during the early adolescent stage, parenting was not the sole focus of family stress captured in the current study.

Rather, our measure was a composite of many indicators related to family stress, including household stressors, parenting styles, and parental social support, all of which have been identified in previous literature as robust predictors of the development of internalizing symptoms. Indicators such as chaos in the home, family conflict, and authoritarian parenting are likely relevant to children's experiences at home, but other measures included in the composite such as interpersonal support and spouse/partner strain may not have as much of a directly salient influence on a child's home environment. Further, our family stress measure was primary caregiver-report, rather than youth-report. It is possible that family stress would have been a significant moderator of such associations if twin-report of family stress was gathered, but extant literature vastly focuses on parent-report. For example, a prior study examined parent-reported quality of the marital relationship and quality of the parent-child relationship in a multiple risk factor model and found prospective associations to child internalizing symptoms (Hammen et al., 2004). Another study used objectively coded stress via the Youth Life Stress Interview (Rudolph & Flynn, 2007), and composited youth and parent-report of various stressful events, such as parent-child, marital, and sibling relationships (Rudolph & Troop-Gordon, 2010). Therefore, it may be important for future studies to take both youth and caregiver perspectives into account and to consider the use of objective stress measures.

Peer Stress as a Moderator for Girls

While peer stress was not a significant moderator of the puberty-internalizing association in boys, it was significant for girls. This was not consistent with hypotheses,

but is in line with literature on sex differences in peer relations at this developmental stage indicating that girls are likely to place higher value on their peer relationships, and more emphasis on the salience of peer stress during early adolescence than their boy counterparts (Rose & Rudolph, 2006; Rudolph, 2002). Even though neither pubertal development nor peer stress were individually significant, puberty and stress interacted to predict higher internalizing symptoms, demonstrating that it is these factors in combination that are linked with mental health one year later for girls in the current study sample. This interaction between puberty and peer stress aligns with the diathesis-stress framework (Monroe & Simons, 1991), and compliments a prior study which found that early maturation is a vulnerability factor that interacts with peer stress to predict depressive symptoms during early adolescence (Sontag et al., 2011). Further, early pubertal timing and peer stress can both be classified as social stressors and prior research has shown that girls have not yet developed adequate resources for navigating these concurrent stressors, resulting in higher internalizing symptoms (Ge & Natsuaki, 2009). Specifically, prior studies have indicated that girls, as compared to boys, have deficits in emotional clarity, or the ability to understand their own emotions, and are more prone to rejection sensitivity (Hamilton et al., 2016; McGuire et al., 2019), and these traits may be the mechanisms at play in the significant interaction between puberty and peer stress to internalizing symptoms. Attainment of higher emotional intelligence (i.e., development of their own emotional clarity, as well as how to respond to others' emotions) and skills to cope with rejection may alleviate the association between puberty and internalizing symptoms for girls with high peer stress (Hamilton et al., 2016). In addition, similar to

the rationale for family stress, the peer stress variable was also a primary caregiver-reported measure, such that it was not able to capture youth perceptions of peer stress. As girls tend to outwardly have more difficulty with emotional expression than boys (Ramos et al., 2007), caregivers may be more prone to detect peer stress in girls than in boys. Inversely, it is also possible that parents may be more prone to observe emotional difficulties in girls due to parents socializing their children to express emotions consistent with traditional gender roles (Chaplin et al., 2005), and, therefore, boys' emotions may be more likely to go undetected.

Objective and Self-Reported Sleep as Mediators

For boys and girls, there were no significant mediation pathways from puberty to internalizing symptoms through various sleep health indicators, but there were some significant direct paths worth noting and warranting discussion. The lack of mediation was surprising, given prior studies that found evidence of significant paths from puberty to sleep (Diao et al., 2020; Hoyt et al., 2018), and sleep to internalizing symptoms (Nunes et al., 2020; Pieters et al., 2015; Shimizu et al., 2021). In the current study mediation models, boys continued to demonstrate a significant direct path from pubertal development to internalizing symptoms, in line with the results of the first study aim. The lack of significant puberty-sleep pathways for boys may be due to many boys in the sample being at the earlier stages of pubertal development during which they undergo greatest height acceleration necessitating more sleep. This explanation is in line with findings from the current study sample at age 8, which indicated that greater pubertal development resulted in better sleep outcomes for youth (Lecarie et al., 2022a). Boys also

demonstrated a few significant paths from sleep to internalizing symptoms, specifically for sleep hygiene and daytime sleepiness. While the objective sleep indicators did not seem to have an impact, it does seem like boys who reported poorer sleep habits and experienced greater sleepiness during the day showed higher internalizing symptoms. These results provide evidence that sleep is not a mediator between pubertal development and internalizing symptoms for boys at this age, but that both puberty and sleep seem to be individually associated with internalizing symptoms one year later. For girls, I identified significant links in the path from puberty to sleep, such that greater pubertal development resulted in higher sleep efficiency, greater midpoint variability, and more sleep hygiene problems. Also, of note is the sensitivity analysis which identified a significant mediation path from puberty to internalizing through sleep hygiene. Although no prior studies have found this exact association, one recent study demonstrated that sleep hygiene was a significant mediator between peer victimization and depressive and social anxiety symptoms in youth ages 10-12 (Barlett et al., 2023). Therefore, future studies may want to focus on implementing high quality sleep hygiene habits as a method of intervention during early adolescence to prevent the onset of anxiety and depressive symptoms.

Although the majority of models did not result in significant mediation, the various significant paths from puberty to sleep and sleep to internalizing symptoms suggest that sleep, particularly sleep hygiene, may be a potential malleable factor for youth undergoing pubertal development. Sleep hygiene encompasses sleep consistency, as well as other factors that may help to improve sleep quality and lower sleep variability,

promoting healthier sleep. As the biological shift in circadian rhythm typically occurs between the ages of 10 and 12 (Tarokh et al., 2011), the shift may have happened for some youth in our sample, but not all. Therefore, these sleep mediation models may be more significant as youth progress further into pubertal stages and later into adolescence. In addition, the examination of puberty as a moderator of the association between sleep and internalizing may be warranted. In this way, puberty may be the context in which we understand the relations between sleep and internalizing. It is possible that the links between sleep and internalizing symptoms become stronger later in pubertal development. Rather than examining the linear associations between sleep and internalizing, more recent research suggests the need to explore non-linear associations (i.e., short and long sleep durations are both related to higher internalizing symptoms; James & Hale, et al., 2017) in future studies, as this may provide us with information beyond what the linear sleep-internalizing associations can provide.

Family and Peer Stress as Moderators on Mediation Pathways

The moderated mediation models were largely non-significant. There were no significant interactions in any of the models examining family stress as the moderator. This was consistent with results from aim 2 in which family stress was not a significant moderator of the association between puberty to internalizing symptoms. With peer stress as the moderator, the sleep insomnia and daytime sleepiness models did not have significant interactions, but there was a significant interaction between sleep duration and peer stress on internalizing symptoms for boys and girls. Simple slopes were not significant, suggesting that the association between sleep duration and internalizing

symptoms was not stronger at any particular level of sleep duration. Nonetheless, the significant interaction between sleep duration and peer stress for boys and girls indicates that the slopes of the association between sleep duration and internalizing symptoms varied at different levels of peer stress. For boys and girls, internalizing symptoms decreased as sleep duration increased for adolescents with low peer stress, while adolescents with high peer stress demonstrated a positive association between sleep duration and internalizing symptoms. While prior research has not identified significant links for youth with low peer stress (Burani et al., 2021), the current study demonstrated that youth with low peer stress and high sleep duration had the lowest levels of internalizing symptoms. The finding for high peer stress adolescents is counterintuitive, such that youth in this group had positive associations between sleep duration and internalizing symptoms (i.e., higher sleep duration was linked with greater internalizing symptoms) for youth experiencing higher peer stress. Prior research has indicated that the interaction of high stress and greater sleep problems to be associated with the highest levels of internalizing symptoms (Burani et al., 2021). In addition, extant literature has provided evidence that both oversleeping and under sleeping may be linked with depression in adults (for review, see Zhai et al., 2015). The "Goldilocks hypothesis" has been supported in a recent study such that too little and too much sleep were both associated with risk behaviors, such as delinquency (Mears et al., 2022), and it is likely that curvilinear effects may also exist for the internalizing behavior outcome of the current study. It is possible that current study findings may be capturing youth who use sleep to cope with stress (known as the escape-to-sleep coping strategy; Goosby,

Cheadle, Strong-Bak, et al., 2018; Yip, 2015), and these youth may be more susceptible to internalizing symptoms. These youth may be sleeping above the recommended amount of sleep to compensate for being in high stress environments.

Heritability of Internalizing Symptoms

Taking a twin modeling approach allowed for a further understanding of the development of internalizing symptoms as well as the relations between stress and internalizing symptoms. At this early adolescent stage, internalizing symptoms were primarily environmentally influenced, while peer stress was moderately heritable. The lack of genetic influences on internalizing symptoms is inconsistent with a review of the extant literature examining the heritability of child and adolescent anxiety and depression which concluded internalizing symptoms to have a strong and constant genetic influence across childhood and adolescence, suggesting temporal stability (Franić et al., 2010). Of note, this review highlighted the importance of informant effects. Specifically, longitudinal studies have typically used parent or teacher-report for young children and then switched to self-report in older children and adolescents, while the current study relied on only self-report of internalizing symptoms due to the age of the participants. Therefore, it is possible that the inconsistency in findings from previous literature and the current study may be due to informant effects.

However, the current study finding aligns with additional research suggesting that environmental influences on internalizing symptoms vary with age and shared environmental effects decrease across development (Patterson et al., 2018), as current study findings demonstrated higher non-shared environmental influences (E = .63) as

compared to shared environmental influences (C = .37). Early adolescence is likely an age during which non-shared environmental influences become more developmentally important, as adolescents start to form their own identity independent of their family and as they experience increasing autonomy from their parents (Zimmer-Gembeck & Collins, 2006). Even though one study found no evidence of gender differences in the heritability of internalizing symptoms (Franić et al., 2010), it will be important to conduct analyses separately in boys and girls across development to see whether this finding might be replicated. The onset of internalizing symptoms is twice as likely to occur in girls as compared to boys by mid adolescence and, therefore, examination of boys and girls separately may allow for further understanding of these temporal differences in symptoms (Hankin, 2008).

The finding that peer stress is moderately heritable supports the early adolescent developmental stage of the current study sample. Early adolescence is a stage in which relationships with peers become more salient (Bukowski et al., 2006). This finding suggests that stress due to peer relations experienced by youth may be, in part, due to genetic influences. It is important to note again, though, that our peer stress variable was parent-report and did not directly measure youth perceived peer stress. Nonetheless, prior research supports genetic influences of various aspects of peer relations, including friendship relations and deviant peer affiliations (Brendgen, 2012). In this way, youth may be genetically predisposed to specifically seek out supportive or stressful peer experiences, known as active gene-environment correlation in which children's heritable traits influence their choice of environment (Plomin et al., 2008). As phenotypic aims of

the current study suggested sex differences in the effects of peer stress, it will be important for future studies to examine the heritability of peer stress in boys and girls separately, with a larger sample size than the current study. As with internalizing symptoms, it may be helpful to continue longitudinal examinations of the heritability of peer stress in late childhood and in later adolescence.

Family stress was not a significant moderator of the heritability of internalizing symptoms in the current study sample. This finding does not support my hypothesis and a prior study of 17-year-old adolescents, which showed that family stress (i.e., mother–child and father–child relationship problems) moderated nonshared environmental influences on internalizing symptoms, such that environmental influences were stronger in higher stress contexts, while additive genetic and shared environmental influences remained stable across stress contexts (Hicks et al., 2009). Therefore, it is critical to provide potential explanations as to why moderated heritability was not found in the current study. First, like the rationale for lack of moderation of family stress on the association between puberty and internalizing symptoms in the phenotypic analyses of the current study, the measurement of family stress should be considered. Our family stress composite did not directly measure the level of family stress perceived by the adolescent, which may partially explain lack of significant results. Second, we were not able to conduct this behavioral genetic aim separately for boys and girls due to our sample size. Conducting this aim with all twins together may have washed out significant associations that may have emerged in boys and girls separately. Third, the current study was only able to examine family stress, as opposed to peer stress, as a moderator of the

heritability of internalizing symptoms due to sample size limitations (i.e., robust estimation of twin-level moderated heritability model typically requires a sample size of 1,000 twin pairs; van der Sluis et al., 2012), but it is possible that a peer stress moderated heritability model would have resulted in significant findings. Continuing to examine the effects of stress on the heritability of internalizing symptoms across adolescent development will be beneficial in clarifying developmental changes, as previous research points to increases in nonshared environmental stressors (e.g., negative life events, peer victimization) as significant moderators on the relation between pubertal stage and depression (Stumper & Alloy, 2021). Fourth, although this study focused on stress as a moderator of the heritability of internalizing symptoms, it may be important to look at puberty as the moderator, given prior research which found that early pubertal status moderated internalizing symptoms and was associated with increased genetic influences of internalizing symptoms in girls, but not boys (Corley et al., 2015).

Limitations and future directions

There are several limitations to this study that should be taken into consideration. Examining only one measure of pubertal status at a single timepoint does not allow for a full understanding of pubertal development, as other important pubertal indicators (i.e., timing, tempo, synchrony) were not included. This limited our study to examining pubertal development at its onset in early adolescence, especially for boys, who typically enter puberty about 1-2 years after their same-age girls. Second, although teacher-report of peer stress was collected, it was only for a smaller subset of this sample and was not highly correlated with parent-report of peer stress, so teacher-report was not used in the

current study analyses. It is possible that primary caregivers may not be as accurate reporters of what their child is experiencing at school as compared to teachers. Third, family stress and peer stress were both parent-report and, therefore, may not reflect children's perceptions of their own stress. The inclusion of children's self-report of their own firsthand stress experiences can lead to more accurate ratings, as caregivers may under-report their children's stress, particularly in non-clinical settings (Martin et al., 2004). Fourth, our sample size limited our ability to conduct an individual-level moderated heritability model of peer stress on internalizing symptoms and did not allow us to examine boys and girls separately (van der Sluis et al., 2012), as was done for the phenotypic study aims. Last, we controlled for whether twins participated before or after the start of the COVID-19 pandemic, but we did not account for length of time between onset of the pandemic and study participation. This may be important, especially when examining internalizing symptoms, as some research has demonstrated that mental health may not have been impacted immediately following the start of the pandemic, but that there were long-term mental health effects for adolescents (Golberstein et al., 2020; Janssen et al., 2020).

Future research can build on the findings of the current study in multiple ways. Examining other indicators of puberty (e.g., Tanner stages, hormones) in addition to self-reported puberty to engage in multi-method assessment of puberty is warranted, as certain pubertal indicators may be only concurrently or prospectively associated with internalizing symptoms (Barendse et al., 2022). Further, longitudinal analyses to examine the entirety of pubertal development would allow for understanding of not just current

stage of pubertal development, but also pubertal tempo, or how quickly children progress through pubertal stages. Longitudinal examination of pubertal processes would also allow for assessment of subgroup differences, such as early-onset and late-onset groups (Dorn & Susman, 2019). In addition to multiple indicators of puberty, it may be important to examine additional models with psychopathology outcomes including externalizing symptoms and a general symptom composite of both internalizing and externalizing symptoms. Recent studies suggest that earlier pubertal development is a transdiagnostic risk factor for psychopathology more broadly, such that future studies might want to examine externalizing symptoms along with internalizing symptoms, as well as take a bifactor (p factor) approach (Hamlat et al., 2019; Mendle et al., 2020). As the various sleep indicators did not significantly mediate the path from puberty to internalizing symptoms, it may be important to explore other potential mediators in the puberty-internalizing relation. Other studies have identified a few other significant mediators particularly for girls (i.e., rejection, rumination, sexual harassment; Mendle et al., 2020; Skoog et al., 2016) that warrant further investigation. Continuing to examine sex differences, especially in behavior genetic analyses, as well as examining racial/ethnic differences is a necessary next step, as ethnic/racial differences have been found in pubertal development, sleep, and depression (Bailey et al., 2019; Hoyt et al., 2018; Susman et al., 2010). Further, cultural and contextual variables (i.e., social norms, behaviors, and values) that impact health disparities are also relevant to understanding puberty and informing prevention and intervention efforts (Deardorff et al., 2019; Dorn & Susman, 2019).

This study extends previous research in several ways. While most existing research has examined later stages of the pubertal process, this study focused on the relations between health factors at pubertal onset. Rather than relying solely on youth-report or parent-report of puberty, we averaged the values from these two reporters in an effort to obtain a more accurate overall report of pubertal development. For sleep, this study used both objective and subjective reports of sleep, which allowed for a broader understanding of both sleep patterns and children's perceptions of their sleep. Lastly, the longitudinal nature of this study is a strength, as we were able to examine the influence of puberty, stress, and sleep on internalizing symptoms one year later.

Overall, this study makes important contributions to our understanding of the relations between critical health factors during the onset of adolescence. Study findings point to potential targets of intervention for boys and girls undergoing puberty. For boys, intervention efforts moving forward might best be focused on psychoeducation regarding puberty, and also on sleep hygiene tips, to prevent the onset of internalizing symptoms. If such interventions are developed there should be simultaneous rigorous evaluation of puberty education programs to ensure program efficacy (Crockett et al., 2019). Such programs should include parental preparation and positive attitude, as well as health care providers conveying perspective to parents and youth that puberty is a natural and health-affirming process (Short & Rosenthal, 2008). For girls undergoing puberty, more attention towards the effects of peer stress on internalizing symptoms is needed. Cognitive behavioral and stress management strategies may be beneficial in alleviating girls' peer stress in an effort to prevent the onset of internalizing symptoms.

In line with the biopsychosocial approach, the current study extended existing literature by simultaneously examining the effects of puberty, stress, and sleep on subsequent mental health during the critical developmental stage of early adolescence, as well as the inclusion of behavioral genetic approaches to examine the heritability of such health factors. Overall, study findings call for the continued examination of sex differences in the association between these health factors, additional moderated heritability models with larger samples sizes, and longitudinal analyses taking the full pubertal process into account. As puberty has been deemed a "window of opportunity" and is known for shaping lifespan health (Dorn et al., 2019), it is critical for parents, teachers, and health providers to provide guidance to youth on the physical and psychological changes that occur during puberty at the onset (i.e., early adolescence) to lessen health issues and risks that might emerge during adolescence and later in life.

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Table 1. *Measures included in the family stress composite (10 year assessment).*

Construct	Measure	Sample Items	α
Parental stress	Parental Stress Scale (reverse scored; Berry & Jones, 1985)	18 items; It is difficult to balance different responsibilities because of my child(ren); The behavior of my child(ren) is often embarrassing or stressful to me	.86
Chaos in the home	Confusion, Hubbub, and Order Scale (Matheny, Wachs, Ludwig, & Phillips, 1995)	15 items; No matter how hard we try we always seem to be running late; It's a real "zoo" at our home	.74
Authoritarian parenting	Parenting Styles and Dimensions-Authoritarianism (Robinson et al., 1995)	12 items; I use physical punishment as a way of disciplining Twin A; I yell or shout when Twin A misbehaves	.79
Interpersonal support	Interpersonal Support Evaluation List (reverse scored; Cohen et al., 1985)	12 items; There is someone I can turn to for advice about handling problems with my family; If I were sick, I could easily find someone to help me with daily chores	.88
Spouse/ partner strain	Spouse/Partner Strain Scale (adapted from Schuster et al., 1990 and Whalen & Lachman, 2000)	6 items; How often does your spouse or partner make too many demands on you?; How often is he or she critical of your behavior?	.85
Perceived stress	Perceived Stress Scale (Cohen et al., 1983)	4 items; How often have you felt that you were unable to control the important things in your life?; How often have you felt that things were going your way?	.72
Family conflict	Family Conflict Survey (Porter & O'Leary, 1980)	10 items; How often has Twin A/B heard you arguing about your and your spouse/partner's roles in the family? (Homemaker, working, etc.)	.78

Table 2. *Participant Demographics*

	n	*%*
Sex		
Male	396	49.0
Female	422	51.0
Ethnicity		
Non-Hispanic White/Euro American	417	56.5
Hispanic/Latino		
Asian/Asian American	190	25.7
Black/African American	19	2.6
Native American	24	3.3
Native Hawaiian	22	3.0
Bi-/Multi Racial	4	0.5
Other	50	6.8
	12	1.6
Vacation		
Summer/Vacation Participation	229	29.8
Not Summer/ Vacation	539	70.2
Income-to needs Ratio		
Living in Poverty	38	6.6
Near the Poverty Line	124	21.6
Lower Middle Class	130	22.7
Middle to Upper Class	282	49.1
Primary Caregiver Education		
Less than high school	8	1.1
High school or equivalent	54	7.3
Some college	208	28.2
College degree	279	37.8
Two or more years of graduate school	31	4.2
Graduate or professional degree	158	21.4
COVID		
Participated before 3/25/2020	294	55.5
Participated after 3/25/2020	236	44.5

Note. All variables measured at 10 year study wave with the exception of COVID measured at 11 year study wave.

Table 3. *Descriptive Statistics*

		Girls				Boys			
	n	*M*	*SD*	*Skew*	*Kurtosis*	*M*	*SD*	*Skew*	*Kurtosis*
Age	764	10.94	1.18	0.65	1.98	10.83	1.11	0.48	0.12
BPI Internalizing (10 year)	601	2.50	0.67	0.15	-0.19	2.49	0.69	0.27	-0.17
Body mass index	506	18.71	3.96	2.08	8.46	18.54	4.47	1.52	3.04
BPI Internalizing (11 year)	525	2.56	0.67	0.09	-0.32	2.46	0.68	0.22	-0.64
Pubertal development	761	1.91	0.57	0.87	0.25	1.65	0.44	0.83	0.84
Sleep duration	516	7.92	0.73	-0.51	0.31	7.84	0.69	-0.31	0.14
Sleep efficiency	516	91.57	5.76	-1.24	1.15	90.80	5.41	-0.88	0.39
Sleep midpoint	516	2.50	0.93	0.70	0.88	2.61	0.89	0.81	0.76
Sleep midpoint variability	516	0.69	0.41	1.06	0.58	0.63	0.36	1.32	2.09
CRSP sleep hygiene	598	1.99	0.39	0.39	-0.34	2.07	0.38	0.84	1.76
CRSP sleep disturbances-insomnia	598	2.39	0.73	0.44	-0.10	2.54	0.70	0.32	-0.34
CRSP daytime sleepiness	598	1.72	0.68	1.26	1.37	1.81	0.82	1.39	1.99
Family stress	605	-0.04	1.04	0.74	0.44	0.05	0.96	0.80	0.83
Peer stress	730	1.35	0.46	1.77	2.63	1.42	0.51	1.48	1.53

Note. 10 year $N = 785$. 11 year $N = 628$. Participation in both waves $N = 594$. All variables measured at 10 year study wave with the exception of BPI internalizing. Peer stress winsorized to ±3 SD from the mean

Table 4. *Intercorrelations Among Study Variables for Girls (above the diagonal) and Boys (below the diagonal)*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Internalizing (age 11)	**1**	.04	-.08	-.01	.15*	-.01	.15*	.16*	.21**	.18*	.14*	.01	-.07	.59**	-.04	-.07	.20**	.23**
2. Pubertal development	.11	**1**	-.23**	.14*	.24**	.29**	.11	.07	.02	-.06	.08	.60**	-.17*	.10	-.16**	.38**	-.05	-.01
3. Sleep duration	.00	-.15*	**1**	.46**	-.15*	-.24**	-.11	-.08	-.14*	-.09	-.17**	-.32**	.15*	-.10	.11	-.21**	.00	-.01
4. Sleep efficiency	.15*	-.02	.53**	**1**	.03	.12	-.04	-.08	-.05	-.12	-.08	.13*	-.14*	-.03	.04	-.15*	-.07	.05
5. Sleep midpt	.09	.07	-.08	.02	**1**	.48**	.27**	-.02	-.01	.13	.09	.36**	-.08	.10	-.10	.02	.29**	-.09
6. Sleep midpt var	.07	.10	-.18**	.06	.33**	**1**	.14*	-.04	.07	.08	.10	.35**	-.21**	.02	-.18**	.10	-.07	-.05
7. CRSP sleep hygiene	.30**	.02	-.12	.00	.24**	.22**	**1**	.29**	.33**	.07	.04	.02	-.13*	.20**	-.20**	.02	.15*	-.16*
8. CRSP disturbances	.27**	.05	.10	.04	.14*	.07	.20**	**1**	.27**	.02	.09	.06	-.08	.41**	-.04	.01	.00	-.16*
9. CRSP sleepiness	.31**	.05	-.01	.08	.13*	.16*	.37**	.38**	**1**	.08	.14*	.03	-.09	.28**	-.13*	.04	.00	-.00
10. Family stress	.14*	.03	-.06	.01	-.00	.16*	.19**	.07	.11	**1**	.29**	-.02	.06	.12	-.05	.31**	.06	.11
11. Peer stress	.19**	.14**	-.06	-.02	.04	.13*	.13*	.16*	.15*	.25**	**1**	.09	-.04	.22**	-.00	.11	.09	-.04
12. Age	-.13*	.39**	-.25**	.05	.25**	.06	-.13*	-.02	-.06	-.01	.10	**1**	-.06	.04	-.04	.18**	.02	-.15*
13. Ethnicity	-.06	-.04	.18**	-.09	-.05	-.18**	-.07	.10	-.06	.02	-.03	-.07	**1**	-.02	.22**	-.07	.06	.09
14. Internalizing (age 10)	.63**	.02	.08	.12	.07	.14*	.19*	.35**	.20**	.06	.24**	-.07	-.04	**1**	-.05	.04	.06	.07
15. Family SES	-.07	-.06	.13*	.03	-.28**	-.22**	-.16**	-.14*	-.08	-.09	-.14**	-.05	.21**	-.08	**1**	-.09	.11*	.00
16. Body mass index	.12	.25**	-.34**	-.11	-.06	.02	-.03	.02	.05	.21**	.28**	.19**	-.15*	.09	-.24**	**1**	-.18**	-.05
17. Vacation	.06	-.00	.08	.05	.35**	.02	.07	.01	.03	-.04	.07	.06	-.02	-.02	-.01	-.07	**1**	-.10
18. COVID	.22**	-.04	-.06	-.14	.08	-.09	.14*	-.02	-.05	.15*	.00	-.12	.15*	.17**	.06	-.02	-.06	**1**

Note. * $p < .05$. ** $p < .01$. Internalizing = internalizing composite of the Berkeley Puppet Interview. Peer stress winsorized to ±3 SD from the mean. Ethnicity (0 = Non-white, 1 = White). Family SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. Vacation (1 = on vacation, 0 = school year participation). COVID (0 = Participated before 3/25/2020, 1 = Participated after 3/25/2020).

Table 5. *Effect of Pubertal Development (age 10) on Internalizing Symptoms (age 11)*

	β	SE	*p*	*95% CI*
Girls				
Pubertal development	-0.04	0.08	0.64	-0.19, 0.12
BPI Internalizing (10 year)	**0.57**	**0.05**	**0.00**	**0.47, 0.67**
Age	0.02	0.08	0.76	-0.13, 0.18
Ethnicity	-0.10	0.06	0.10	-0.22, 0.02
SES	-0.02	0.06	0.76	-0.14, 0.11
COVID	**0.18**	**0.05**	**0.00**	**0.08, 0.28**
Boys				
Pubertal development	**0.13**	**0.06**	**0.03**	**0.01, 0.25**
BPI Internalizing (10 year)	**0.58**	**0.05**	**0.00**	**0.49, 0.68**
Age	-0.11	0.06	0.09	-0.23, 0.02
Ethnicity	-0.07	0.06	0.20	-0.18, 0.04
SES	-0.03	0.05	0.59	-0.13, 0.07
COVID	**0.13**	**0.06**	**0.02**	**0.03, 0.24**

Note. Results are from standardized model and significant effects are bolded. BPI Internalizing = internalizing composite (depression, separation anxiety, over-anxiousness) of Berkeley Puppet Interview. Ethnicity (0 = Non-white, 1 = White). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. COVID (0 = Participated before 3/25/2020, 1 = Participated after 3/25/2020).

Table 6. *Family Stress (Model 1) and Peer Stress (Model 2) as Moderators of the Association between Pubertal Development and Internalizing Symptoms*

	Model 1				Model 2			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	-0.02	0.07	0.75	-0.17, 0.12	-0.03	0.08	0.68	-0.18, 0.12
Family stress	0.07	0.07	0.33	-0.07, 0.21	--	--	--	--
Peer stress	--	--	--	--	-0.01	0.07	0.93	-0.14, 0.13
Family stress X Pubertal development	0.10	0.08	0.21	-0.06, 0.25	--	--	--	--
Peer stress X Pubertal development	--	--	--	--	**0.12**	**0.06**	**0.04**	**0.01, 0.24**
BPI Internalizing (10 year)	**0.57**	**0.05**	**0.00**	**0.47, 0.67**	**0.56**	**0.05**	**0.00**	**0.45, 0.67**
Age	0.02	0.07	0.78	-0.12, 0.16	0.04	0.07	0.63	-0.11, 0.18
Ethnicity	-0.10	0.06	0.10	-0.21, 0.02	-0.09	0.06	0.11	-0.21, 0.02
SES	-0.01	0.06	0.89	-0.13, 0.12	-0.02	0.06	0.73	-0.14, 0.10
COVID	**0.17**	**0.05**	**0.00**	**0.07, 0.27**	**0.18**	**0.05**	**0.00**	**0.08, 0.27**
Boys								
Pubertal development	**0.12**	**0.06**	**0.04**	**0.01, 0.24**	**0.13**	**0.06**	**0.03**	**0.01, 0.25**
Family stress	0.05	0.05	0.34	-0.05, 0.16	--	--	--	--
Peer stress	--	--	--	--	0.03	0.05	0.63	-0.08, 0.13
Family stress X Pubertal development	-0.03	0.07	0.72	-0.17, 0.12	--	--	--	--
Peer stress X Pubertal development					-0.04	0.05	0.38	-0.13, 0.05
BPI Internalizing (10 year)	**0.59**	**0.05**	**0.00**	**0.49, 0.69**	**0.58**	**0.05**	**0.00**	**0.48, 0.68**
Age	-0.11	0.06	0.08	-0.23, 0.01	**-0.12**	**0.06**	**0.04**	**-0.23, -0.00**
Ethnicity	-0.07	0.05	0.17	-0.18, 0.03	-0.07	0.05	0.18	-0.18, 0.03
SES	-0.02	0.05	0.66	-0.12, 0.08	-0.02	0.05	0.65	-0.12, 0.08
COVID	**0.12**	**0.05**	**0.02**	**0.02, 0.23**	**0.13**	**0.05**	**0.02**	**0.02, 0.24**

Note. Results are from standardized model and significant effects are bolded. BPI Internalizing = internalizing composite (depression, separation anxiety, over-anxiousness) of the Berkeley Puppet Interview. Ethnicity (0 = Non-white, 1 = White). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. COVID (0 = Participated before 3/25/2020, 1 = Participated after 3/25/2020)

Table 7. *Moderated mediation models in which family stress and peer stress moderate the direct and indirect paths from pubertal development to internalizing symptoms*

(a) Family stress moderator, sleep insomnia mediator

Predictor	Sleep insomnia				BPI Internalizing (11 year)			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	0.06	0.11	0.59	-0.16, 0.28	0.07	0.05	0.18	-0.03, 0.18
Family stress	0.04	0.07	0.54	-0.09, 0.17	-0.01	0.14	0.92	-0.29, 0.26
Family stress X Pubertal development	0.09	0.06	0.13	-0.03, 0.22	0.06	0.05	0.24	-0.04, 0.15
Sleep insomnia	--	--	--	--	-0.07	0.07	0.38	-0.21, 0.08
Family stress X sleep insomnia	--	--	--	--	0.02	0.15	0.90	-0.28, 0.32
BPI Internalizing (10 year)	--	--	--	--	**0.62**	**0.06**	**0.000**	**0.51, 0.74**
COVID	--	--	--	--	**0.14**	**0.04**	**0.001**	**0.06, 0.23**
Age	-0.001	0.07	0.99	-0.13, 0.13	0.01	0.04	0.73	-0.07, 0.10
Ethnicity	0.02	0.10	0.85	-0.17, 0.21	-0.07	0.07	0.30	-0.21, 0.06
SES	-0.04	0.10	0.70	-0.25, 0.17	-0.01	0.08	0.85	-0.16, 0.13
Body mass index	0.02	0.09	0.85	-0.17, 0.20	--	--	--	--
Vacation	0.02	0.07	0.75	-0.11, 0.15	--	--	--	--
Boys								
Pubertal development	0.09	0.08	0.27	-0.07, 0.25	0.07	0.05	0.19	-0.03, 0.17
Family stress	0.04	0.07	0.59	-0.09, 0.16	-0.01	0.14	0.93	-0.29, 0.26
Family stress X Pubertal development	0.07	0.06	0.21	-0.04, 0.18	0.04	0.04	0.34	-0.04, 0.12
Sleep insomnia	--	--	--	--	0.04	0.06	0.55	-0.09, 0.17
Family stress X sleep insomnia	--	--	--	--	0.02	0.15	0.90	-0.28, 0.31
BPI Internalizing (10 year)	--	--	--	--	**0.59**	**0.06**	**0.000**	**0.47, 0.72**
COVID	--	--	--	--	**0.13**	**0.04**	**0.003**	**0.04, 0.22**
Age	0.02	0.07	0.78	-0.12, 0.16	-0.02	0.04	0.70	-0.11, 0.07
Ethnicity	0.09	0.09	0.31	-0.09, 0.28	-0.07	0.06	0.29	-0.20, 0.06
SES	-0.06	0.09	0.50	-0.23, 0.11	-0.07	0.05	0.20	-0.18, 0.04
Body mass index	-0.01	0.08	0.93	-0.16, 0.14	--	--	--	--
Vacation	0.02	0.07	0.75	-0.11, 0.15	--	--	--	--

(b) Peer stress moderator, sleep insomnia mediator

Predictor	Sleep insomnia				BPI Internalizing (11 year)			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	-0.03	0.10	0.79	-0.22, 0.17	0.07	0.04	0.14	-0.02, 0.15
Peer stress	**0.17**	**0.06**	**0.01**	**0.05, 0.29**	-0.06	0.18	0.74	-0.42, 0.30
Peer stress X Pubertal development	0.01	0.07	0.86	-0.12, 0.14	0.03	0.04	0.50	-0.05, 0.11
Sleep insomnia	--	--	--	--	-0.07	0.07	0.33	-0.20, 0.07
Peer stress X sleep insomnia	--	--	--	--	0.05	0.19	0.78	-0.31, 0.41
BPI Internalizing (10 year)	--	--	--	--	**0.60**	**0.05**	**0.00**	**0.50, 0.71**
COVID	--	--	--	--	**0.15**	**0.04**	**0.00**	**0.07, 0.22**
Age	0.00	0.06	0.99	-0.13, 0.13	0.00	0.04	0.99	-0.09, 0.09
Ethnicity	-0.09	0.08	0.28	-0.25, 0.07	-0.08	0.07	0.23	-0.21, 0.05
SES	0.001	0.09	0.99	-0.17, 0.18	-0.056	0.07	0.43	-0.19, 0.08
Body mass index	0.01	0.08	0.87	-0.14, 0.17	--	--	--	--
Vacation	0.003	0.06	0.96	-0.11, .11	--	--	--	--
Boys								
Pubertal development	0.04	0.07	0.58	-0.10, 0.18	0.07	0.05	0.14	-0.02, 0.16
Peer stress	**0.21**	**0.06**	**0.00**	**0.09, 0.32**	-0.07	0.19	0.70	-0.44, 0.29
Peer stress X Pubertal development	0.01	0.07	0.84	-0.12, 0.15	0.03	0.04	0.42	-0.05, 0.12
Sleep insomnia	--	--	--	--	0.03	0.06	0.60	-0.09, 0.15
Peer stress X sleep insomnia	--	--	--	--	0.06	0.19	0.74	-0.30, 0.43
BPI Internalizing (10 year)	--	--	--	--	**0.58**	**0.06**	**0.00**	**0.46, 0.70**
COVID	--	--	--	--	**0.14**	**0.04**	**0.00**	**0.06, 0.22**
Age	0.03	0.06	0.66	-0.09, 0.15	-0.02	0.06	0.63	-0.11, 0.07
Ethnicity	0.13	0.08	0.09	-0.02, 0.28	**-0.13**	**0.06**	**0.03**	**-0.25, -0.01**
SES	-0.13	0.08	0.11	-0.29, 0.03	-0.04	0.05	0.50	-0.14, 0.07
Body mass index	-0.06	0.07	0.39	-0.20, 0.08	--	--	--	--
Vacation	0.003	0.06	0.97	-0.15, 0.12	--	--	--	--

(c) Family stress moderator, sleep duration mediator

Predictor	Sleep duration				BPI Internalizing (11 year)			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	0.01	0.11	0.96	-0.20, 0.21	**0.11**	**0.06**	**0.04**	**0.004, 0.22**
Family stress	-0.02	0.06	0.74	-0.14, 0.10	-0.03	0.62	0.97	-1.25, 1.20
Family stress X Pubertal development	0.13	0.07	0.06	-0.01, 0.27	0.04	0.06	0.43	-0.07, 0.15
Sleep duration	--	--	--	--	0.03	0.07	0.69	-0.10, 0.16
Family stress X sleep duration	--	--	--	--	0.04	0.62	0.95	-1.18, 1.25
BPI Internalizing (10 year)	--	--	--	--	**0.58**	**0.06**	**0.000**	**0.45, 0.71**
COVID	--	--	--	--	**0.18**	**0.05**	**0.000**	**0.09, 0.27**
Age	**-0.23**	**0.08**	**0.004**	**-0.39, 0.07**	-0.03	0.06	0.57	-0.15, 0.08
Ethnicity	0.13	0.08	0.11	-0.03, 0.30	-0.05	0.08	0.49	-0.21, 0.10
SES	0.001	0.09	0.99	-0.17, 0.17	-0.02	0.08	0.81	-0.18, 0.14
Body mass index	**-0.20**	**0.09**	**0.03**	**-0.38, -0.02**	--	--	--	--
Vacation	0.06	0.06	0.30	-0.05, 0.18	--	--	--	--
Boys								
Pubertal development	0.01	0.09	0.90	-0.16, 0.18	**0.10**	**0.05**	**0.049**	**0.000, 0.21**
Family stress	-0.02	0.06	0.77	-0.14, 0.11	-0.02	0.60	0.97	-1.20, 1.15
Family stress X Pubertal development	0.11	0.07	0.12	-0.03, 0.24	0.03	0.05	0.53	-0.06, 0.13
Sleep duration	--	--	--	--	0.001	0.06	0.98	-0.11, 0.11
Family stress X sleep duration	--	--	--	--	0.03	0.59	0.96	-1.13, 1.20
BPI Internalizing (10 year)	--	--	--	--	**0.60**	**0.06**	**0.000**	**0.47, 0.72**
COVID	--	--	--	--	**0.15**	**0.04**	**0.000**	**0.07, 0.24**
Age	**-0.20**	**0.08**	**0.01**	**-0.36, -0.05**	-0.02	0.05	0.62	-0.12, 0.07
Ethnicity	-0.05	0.09	0.58	-0.23, 0.13	-0.10	0.06	0.11	-0.23, 0.02
SES	0.10	0.08	0.20	-0.06, 0.27	-0.05	0.06	0.36	-0.17, 0.06
Body mass index	**-0.27**	**0.08**	**0.001**	**-0.43, -0.11**	--	--	--	--
Vacation	0.06	0.06	0.30	-0.06, 0.19	--	--	--	--

(d) Peer stress moderator, sleep duration mediator

Predictor	Sleep duration				BPI Internalizing (11 year)			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	-0.03	0.10	0.79	-0.21, 0.16	**0.10**	**0.04**	**0.03**	**0.01, 0.18**
Peer stress	-0.004	0.06	0.95	-0.12, 0.12	**-1.40**	**0.52**	**0.01**	**-2.42, -0.37**
Peer stress X Pubertal development	-0.03	0.05	0.57	-0.14, 0.08	0.07	0.04	0.10	-0.01, 0.15
Sleep duration	--	--	--	--	0.03	0.06	0.57	-0.08, 0.15
Peer stress X sleep duration	--	--	--	--	**1.43**	**0.50**	**0.005**	**0.44, 2.42**
BPI Internalizing (10 year)	--	--	--	--	**0.54**	**0.06**	**0.000**	**0.42, 0.66**
COVID	--	--	--	--	**0.16**	**0.04**	**0.000**	**0.08, 0.25**
Age	**-0.22**	**0.07**	**0.001**	**-0.34, -0.09**	-0.03	0.06	0.58	-0.14, 0.08
Ethnicity	0.10	0.09	0.27	-0.12, 0.20	-0.05	0.07	0.47	-0.19, 0.09
SES	0.04	0.08	0.63	-0.34, 0.03	-0.05	0.07	0.47	-0.20, 0.09
Body mass index	-0.16	0.09	0.09	-0.34, 0.03	--	--	--	--
Vacation	0.07	0.05	0.17	-0.03, 0.18	--	--	--	--
Boys								
Pubertal development	0.03	0.08	0.73	-0.13, 0.18	**0.10**	**0.04**	**0.03**	**0.01, 0.19**
Peer stress	-0.01	0.07	0.94	-0.13, 0.12	**-1.51**	**0.52**	**0.004**	**-2.53, -0.48**
Peer stress X Pubertal development	-0.04	0.06	0.55	-0.15, 0.08	0.07	0.04	0.10	-0.01, 0.15
Sleep duration	--	--	--	--	-0.001	0.06	0.99	-0.11, 0.11
Peer stress X sleep duration	--	--	--	--	**1.50**	**0.50**	**0.003**	**0.51, 2.49**
BPI Internalizing (10 year)	--	--	--	--	**0.58**	**0.06**	**0.000**	**0.46, 0.70**
COVID	--	--	--	--	**0.16**	**0.04**	**0.000**	**0.07, 0.24**
Age	**-0.21**	**0.06**	**0.002**	**-0.33, -0.08**	-0.02	0.05	0.74	-0.11, 0.08
Ethnicity	0.02	0.08	0.83	-0.14, 0.17	-0.10	0.06	0.11	-0.21, 0.02
SES	0.10	0.07	0.18	-0.04, -0.12	-0.07	0.06	0.23	-0.18, 0.04
Body mass index	**-0.26**	**0.07**	**0.000**	**-0.41, -0.21**	--	--	--	--
Vacation	0.08	0.06	0.18	-0.03, 0.08	--	--	--	--

(e) Family stress moderator, daytime sleepiness mediator

Predictor	Daytime Sleepiness				BPI Internalizing (11 year)			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	0.04	0.10	0.70	-0.15, 0.23	0.07	0.05	0.17	-0.03, 0.17
Family stress	0.06	0.07	0.40	-0.08, 0.19	-0.23	0.13	0.08	-0.49, 0.03
Family stress X pubertal development	0.03	0.05	0.57	-0.07, 0.13	0.04	0.04	0.35	-0.04, 0.12
Daytime sleepiness	--	--	--	--	0.005	0.08	0.95	-0.16, 0.17
Family stress X daytime sleepiness	--	--	--	--	0.23	0.13	0.09	-0.03, 0.49
BPI Internalizing (10 year)	--	--	--	--	**0.61**	**0.06**	**0.000**	**0.49, 0.73**
COVID	--	--	--	--	**0.15**	**0.04**	**0.001**	**0.06, 0.23**
Age	0.02	0.07	0.82	-0.12, 0.16	0.01	0.04	0.78	-0.07, 0.09
Ethnicity	-0.004	0.10	0.97	-0.20, 0.19	-0.08	0.07	0.24	-0.21, 0.05
SES	-0.09	0.09	0.33	-0.28, 0.09	-0.01	0.07	0.87	-0.16, 0.13
Body mass index	-0.05	0.10	0.63	-0.25, 0.15	--	--	--	--
Vacation	0.05	0.08	0.52	-0.10, 0.20	--	--	--	--
Boys								
Pubertal development	0.06	0.07	0.41	-0.08, 0.20	0.07	0.05	0.19	-0.03, 0.17
Family stress	0.04	0.06	0.45	-0.07, 0.15	-0.20	0.12	0.10	-0.43, 0.04
Family stress X Pubertal development	0.02	0.04	0.66	-0.06, 0.10	0.03	0.04	0.46	-0.05, 0.11
Daytime sleepiness	--	--	--	--	**0.17**	**0.07**	**0.01**	**0.04, 0.30**
Family stress X daytime sleepiness	--	--	--	--	0.23	0.13	0.08	-0.03, 0.48
BPI Internalizing (10 year)	--	--	--	--	**0.57**	**0.07**	**0.000**	**0.44, 0.70**
COVID	--	--	--	--	**0.13**	**0.05**	**0.004**	**0.04, 0.23**
Age	-0.004	0.06	0.95	-0.12, 0.11	-0.02	0.04	0.62	-0.11, 0.06
Ethnicity	-0.01	0.10	0.89	-0.21, 0.18	-0.06	0.07	0.35	-0.19, 0.07
SES	-0.04	0.09	0.69	-0.22, 0.14	-0.06	0.05	0.23	-0.17, 0.04
Body mass index	0.04	0.09	0.70	-0.15, 0.22	--	--	--	--
Vacation	0.04	0.06	0.53	-0.08, 0.16	--	--	--	--

(f) Peer stress moderator, daytime sleepiness mediator

Predictor	Daytime Sleepiness				BPI Internalizing (11 year)			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	-0.03	0.09	0.71	-0.20, 0.19	0.07	0.04	0.11	-0.02, 0.16
Peer stress	0.10	0.08	0.19	-0.05, 0.26	-0.03	0.12	0.79	-0.27, 0.20
Peer stress X Pubertal development	0.07	0.06	0.28	-0.05, 0.19	0.02	0.04	0.69	-0.06, 0.10
Daytime sleepiness	--	--	--	--	0.01	0.07	0.92	-0.13, 0.15
Peer stress X daytime sleepiness	--	--	--	--	0.01	0.13	0.91	-0.24, 0.27
BPI Internalizing (10 year)	--	--	--	--	**0.58**	**0.06**	**0.000**	**0.47, 0.69**
COVID	--	--	--	--	**0.16**	**0.04**	**0.000**	**0.08, 0.24**
Age	0.04	0.06	0.46	-0.07, 0.16	0.000	0.04	0.999	-0.08, 0.08
Ethnicity	-0.05	0.09	0.60	-0.22, 0.13	-0.08	0.07	0.25	-0.21, 0.05
SES	-0.10	0.08	0.23	-0.26, 0.06	-0.05	0.07	0.45	-0.19, 0.08
Body mass index	-0.06	0.10	0.56	-0.25, 0.14	--	--	--	--
Vacation	0.01	0.06	0.90	-0.12, 0.13	--	--	--	--
Boys								
Pubertal development	0.01	0.06	0.91	-0.11, 0.13	0.07	0.05	0.11	-0.02, 0.17
Peer stress	0.10	0.06	0.11	-0.02, 0.23	-0.04	0.12	0.76	-0.28, 0.21
Peer stress X Pubertal development	0.07	0.05	0.20	-0.04, 0.17	0.02	0.04	0.64	-0.06, 0.10
Daytime sleepiness	--	--	--	--	**0.20**	**0.06**	**0.002**	**0.07, 0.33**
Peer stress X daytime sleepiness	--	--	--	--	0.02	0.13	0.88	-0.24, 0.28
BPI Internalizing (10 year)	--	--	--	--	**0.55**	**0.06**	**0.000**	**0.43, 0.67**
COVID	--	--	--	--	**0.16**	**0.04**	**0.000**	**0.08, 0.24**
Age	0.02	0.05	0.65	-0.08, 0.12	-0.03	0.05	0.58	-0.11, 0.06
Ethnicity	-0.06	0.08	0.50	-0.22, 0.11	**-0.12**	**0.06**	**0.049**	**-0.25, -0.001**
SES	-0.06	0.08	0.42	-0.21, 0.09	-0.03	0.05	0.58	-0.13, 0.07
Body mass index	0.02	0.08	0.83	-0.15, 0.18	--	--	--	--
Vacation	0.01	0.05	0.90	-0.10, 0.11	--	--	--	--

Table 8. *Twin Intraclass Correlations for Peer Stress (age 10) and Internalizing Symptoms (age 11)*

	MZ	**DZ**
Peer Stress	.85	.56
BPI Internalizing	.41	.36

Note. BPI Internalizing = internalizing composite (depression, separation anxiety, over-anxiousness) of the Berkeley Puppet Interview.

Table 9. *Univariate ACE Model Fit and Parameter Estimates*

	Model	-2LL	*df*	Δ-2LL	Δ*df*	*p*	AIC	A	C	E
BPI Internalizing	ACE	1035.46	521	--	--	--	-6.54	.11	.29	.59
	AE	1039.09	522	3.63	1	0.06	-4.91			
	CE	**1035.74**	**522**	**0.27**	**1**	**0.6**	**-8.26**	--	.37	.63
	E	1073.67	523	38.20	2	< .001	27.67			
Peer Stress	**ACE**	**775.19**	**724**	**--**	**--**	**--**	**-672.81**	.63.	.24	.13
	AE	782.21	725	7.03	1	0.01	-667.79			
	CE	822.38	725	47.19	1	< .001	-627.62			
	E	1010.78	726	235.59	2	< .001	-441.22			

Note. BPI Internalizing = internalizing composite (depression, separation anxiety, over-anxiousness) of the Berkeley Puppet Interview. Bolded models denote the best-fitting models for each variable. A, C, and E are standardized squared parameter estimates for additive genetic (A), common environment (C), and nonshared environment (E) factors. −2LL=-2 log likelihood; Δ= change; AIC= Akaike's Information Criterion.

Table 10. *Model fit statistics for moderated heritability model*

Model	-2LL	*df*	Δ-2LL	Δ*df*	*p*	Δ AIC
BPI Internalizing	782.49	391	--	--	--	0.49
No moderation	**786.23**	**394**	**3.74**	**3**	**0.29**	**-1.77**
No C or E moderation	783.39	393	0.9	2	0.64	-2.61
No A moderation	784.32	392	1.82	1	0.18	0.32
No C moderation	783.17	392	0.68	1	0.41	-0.83
No E moderation	783.18	392	0.68	1	0.41	-0.82
No A or C moderation	785.29	393	2.80	2	0.25	-0.71
No A or E moderation	784.71	393	2.22	2	0.33	-1.29

Note. BPI Internalizing = internalizing composite (depression, separation anxiety, over-anxiousness) of the Berkeley Puppet Interview. Bolded model denotes the best-fitting model. −2LL=-2 log likelihood; Δ= change; AIC= Akaike's Information Criterion.

Table 11. *Sensitivity Analysis of Effect of Pubertal Development (age 10) on Internalizing Symptoms (age 11) Without Controlling for Internalizing Symptoms (age 10)*

	β	SE	*p*	*95% CI*
Girls				
Pubertal development	0.01	0.09	0.90	-0.17, 0.20
Age	0.03	0.08	0.75	-0.14, 0.19
Ethnicity	-0.09	0.08	0.29	-0.25, 0.07
SES	-0.01	0.08	0.94	-0.17, 0.16
COVID	**0.25**	**0.06**	**0.00**	**0.12, 0.37**
Boys				
Pubertal development	**0.17**	**0.07**	**0.02**	**0.03, 0.32**
Age	**-0.16**	**0.08**	**0.03**	**-0.31, -0.01**
Ethnicity	-0.10	0.07	0.17	-0.23, 0.04
SES	-0.05	0.07	0.42	-0.19, 0.08
COVID	**0.23**	**0.07**	**0.00**	**0.10, 0.36**

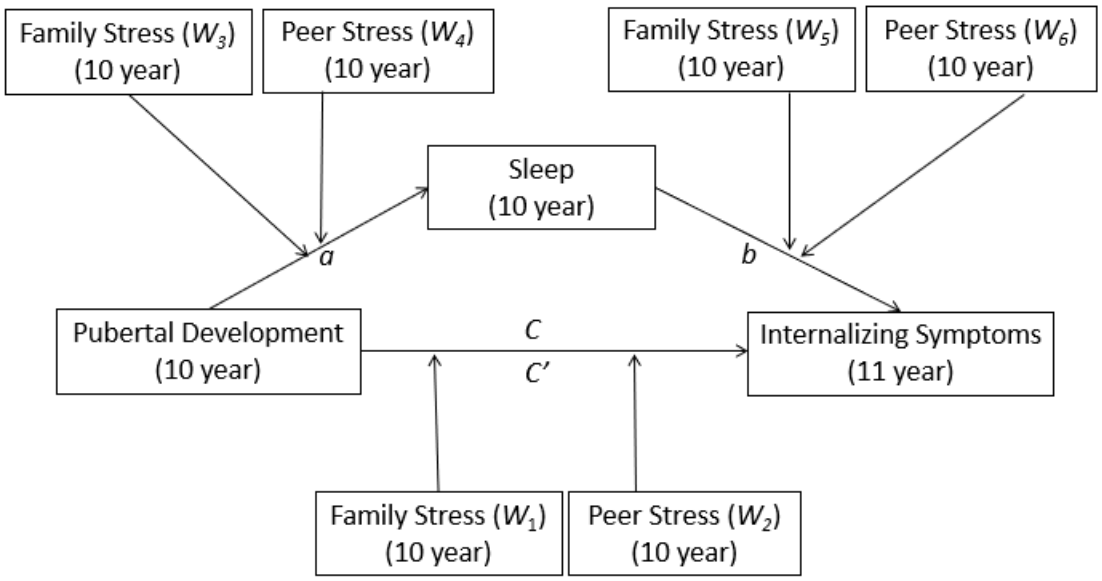
Note. Results are from standardized model and significant effects are bolded. Ethnicity (0 = Non-white, 1 = White). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. COVID (0 = Participated before 3/25/2020, 1 = Participated after 3/25/2020).

Table 12. *Sensitivity Analysis for Family Stress (Model 1) and Peer Stress (Model 2) as Moderators of the Association between Pubertal Development and Internalizing Symptoms Without Controlling for Internalizing Symptoms (age 10)*

	Model 1				Model 2			
	β	SE	*p*	95% CI	β	SE	*p*	*95% CI*
Girls								
Pubertal development	0.04	0.09	0.68	-0.13, 0.21	0.01	0.08	0.90	-0.18, 0.12
Family stress	**0.18**	**0.08**	**0.03**	**0.02, 0.33**	--	--	--	--
Peer stress	--	--	--	--	**0.17**	**0.08**	**0.04**	**-0.14, 0.13**
Family stress X Pubertal development	0.05	0.09	0.60	-0.13, 0.22	--	--	--	--
Peer stress X Pubertal development	--	--	--	--	**0.16**	**0.06**	**0.01**	**0.01, 0.24**
Age	0.00	0.08	0.96	-0.15, 0.16	0.02	0.08	0.77	-0.11, 0.18
Ethnicity	-0.10	0.08	0.21	-0.26, 0.06	-0.07	0.08	0.37	-0.21, 0.02
SES	0.02	0.08	0.82	-0.15, 0.18	-0.02	0.08	0.78	-0.14, 0.10
COVID	**0.22**	**0.06**	**0.00**	**0.10, 0.35**	**0.24**	**0.06**	**0.00**	**0.08, 0.27**
Boys								
Pubertal development	**0.17**	**0.07**	**0.02**	**0.03, 0.31**	**0.15**	**0.07**	**0.04**	**0.01, 0.25**
Family stress	0.10	0.07	0.16	-0.04, 0.25	--	--	--	--
Peer stress	--	--	--	--	**0.19**	**0.07**	**0.01**	**-0.08, 0.13**
Family stress X Pubertal development	0.09	0.08	0.30	-0.08, 0.25	--	--	--	--
Peer stress X Pubertal development					-0.07	0.07	0.32	-0.13, 0.05
Age	**-0.16**	**0.07**	**0.03**	**-0.30, 0.01**	**-0.19**	**0.07**	**0.01**	**-0.23, -0.00**
Ethnicity	-0.10	0.07	0.12	-0.24, 0.03	-0.09	0.07	0.17	-0.18, 0.03
SES	-0.04	0.07	0.59	-0.17, 0.09	-0.04	0.07	0.57	-0.12, 0.08
COVID	**0.21**	**0.07**	**0.00**	**0.08, 0.34**	**0.23**	**0.06**	**0.00**	**0.02, 0.24**

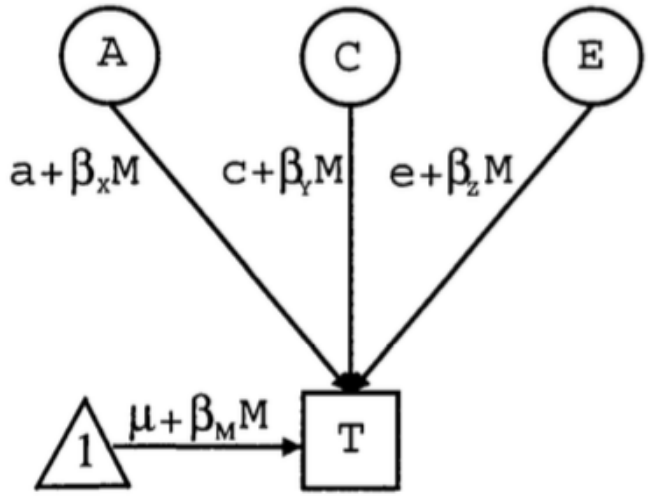
Note. Results are from standardized model and significant effects are bolded. BPI Internalizing = internalizing composite (depression, separation anxiety, over-anxiousness) of the Berkeley Puppet Interview. Ethnicity (0 = Non-white, 1 = White). SES (socioeconomic status) = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. COVID (0 = Participated before 3/25/2020, 1 = Participated after 3/25/2020).

Figure 1. Conceptual Model for the Current Study

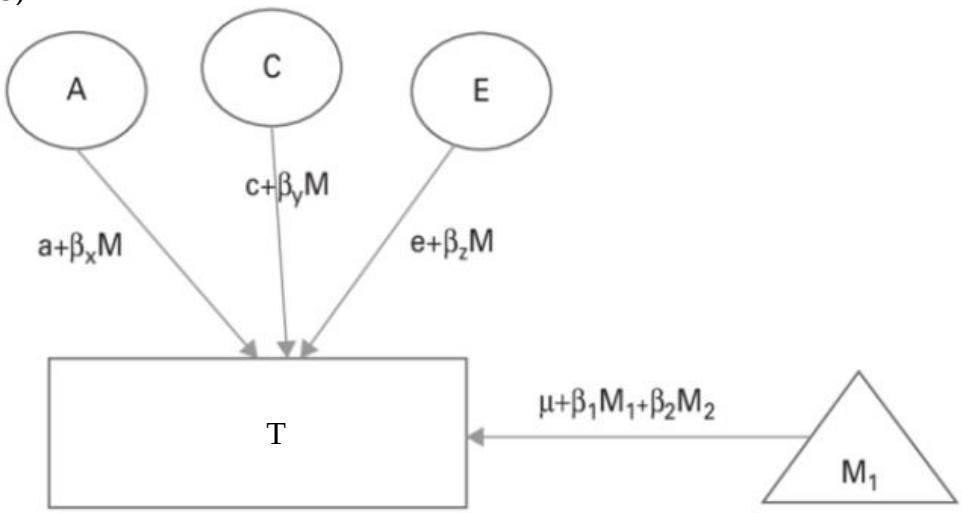


Note. Model depicts study aims 1, 2, and 3. (1) Testing total effect of pubertal development predicting internalizing symptoms (*c*). (2) Testing moderated effect of peer stress and family stress on the link between pubertal development and internalizing symptoms. (3) Testing the indirect effect of pubertal development predicting internalizing symptoms through objective sleep (*ab*).

Figure 2. *Moderated Heritability Models*
(a)



(b)



Note. (a) Moderated heritability model showing the moderation of one family-level phenotype (i.e., family stress) on one individual-level phenotype (i.e., internalizing symptoms). (b) Moderated heritability model showing the moderation of one individual-level phenotype (i.e., peer stress) on one individual-level phenotype (i.e., internalizing symptoms). These models show one twin for simplicity, but co-twin variables and paths are estimated in the models. A = additive genetic variance, C = shared environmental variance, E = nonshared environmental variance, M = moderator, T = the trait, or second phenotype. Equations next to each path represent the linear relationship between the path coefficient and the moderator. The non-linear moderators are not shown. If β_x is significantly non-zero, this demonstrates an interaction between the path coefficient and the moderator (Purcell, 2002; van der Sluis et al., 2012).

Figure 3. *Simple slopes plot for the association between pubertal development and internalizing symptoms at low, average, and high levels of peer stress, for girls*

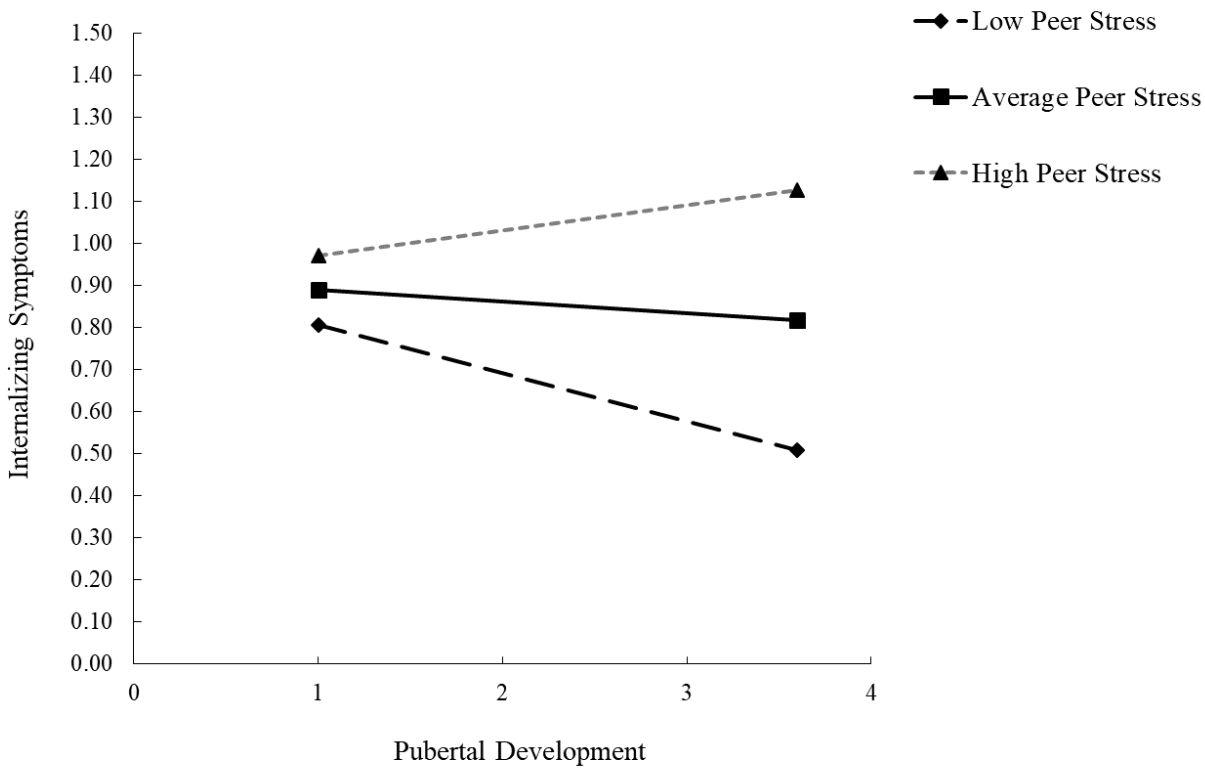
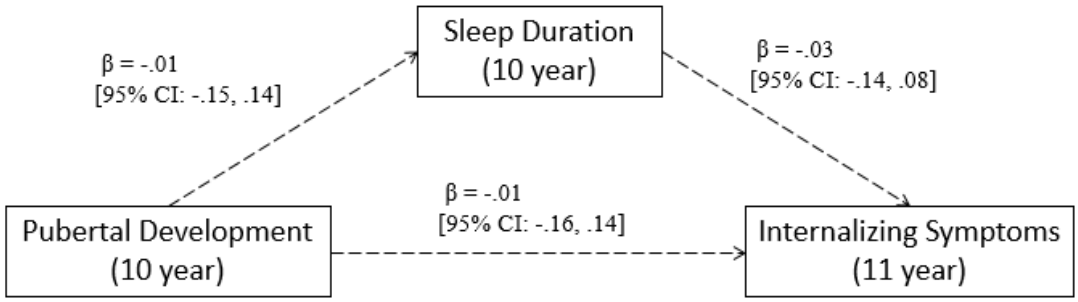
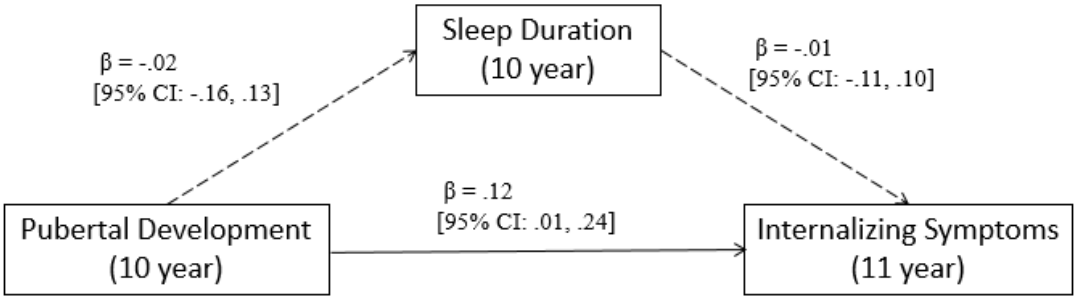


Figure 4. *Objective and Self-report Sleep Indicators as Mediators Between Pubertal Development and Internalizing Symptoms*

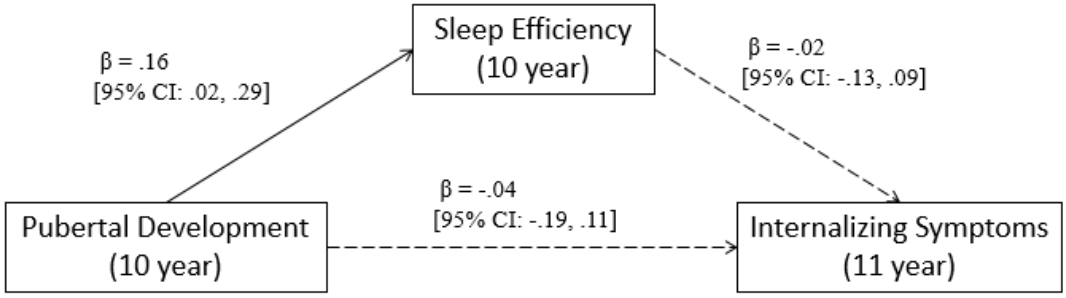
(a) Sleep Duration- Girls



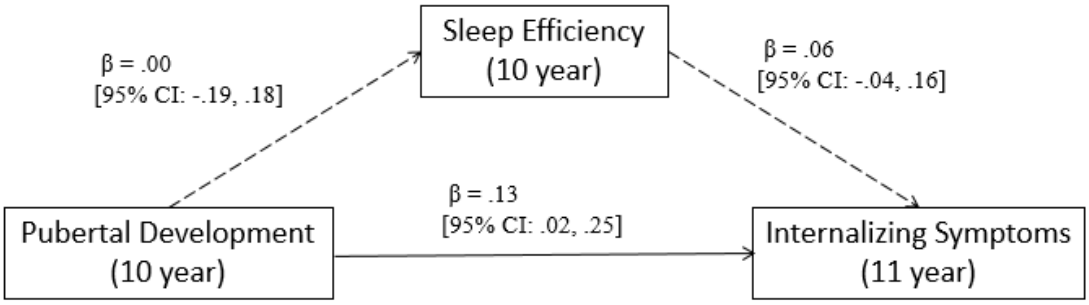
(b) Sleep Duration- Boys



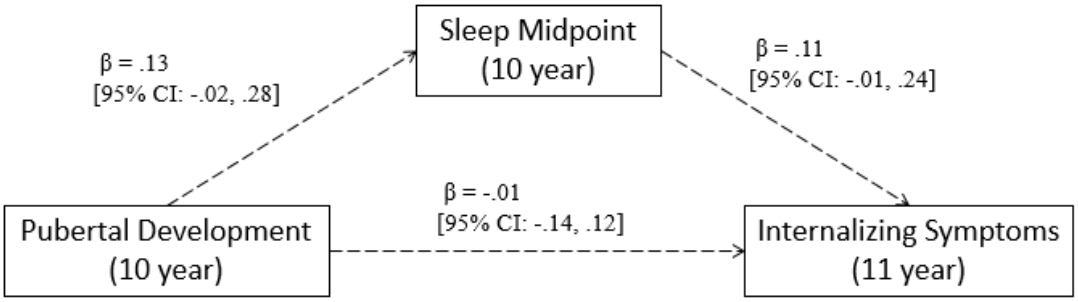
(c) Sleep Efficiency- Girls



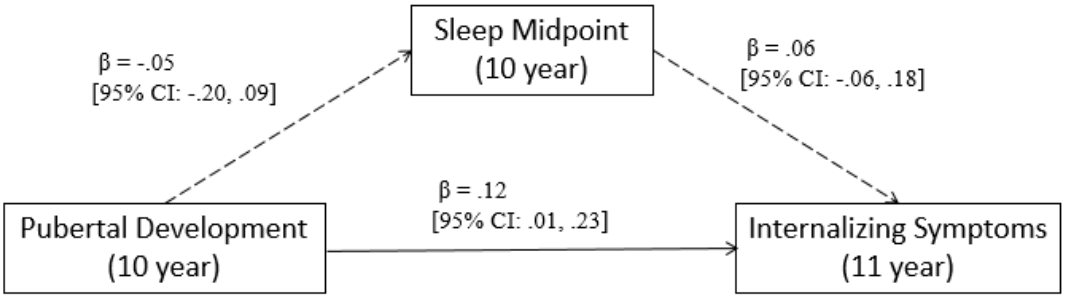
(d) Sleep Efficiency- Boys



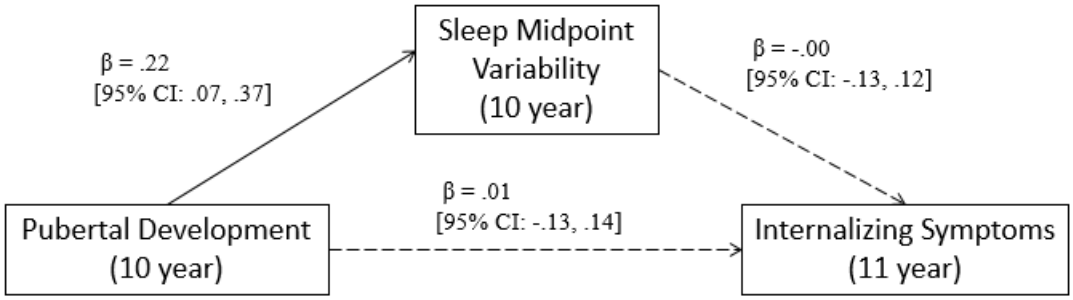
(e) Sleep Midpoint- Girls



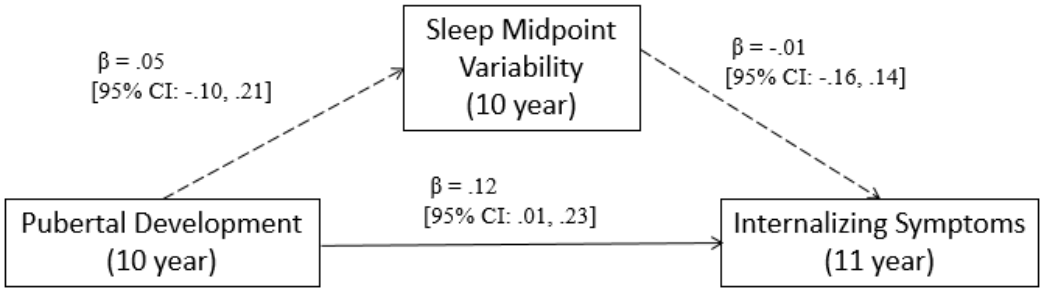
(f) Sleep Midpoint- Boys



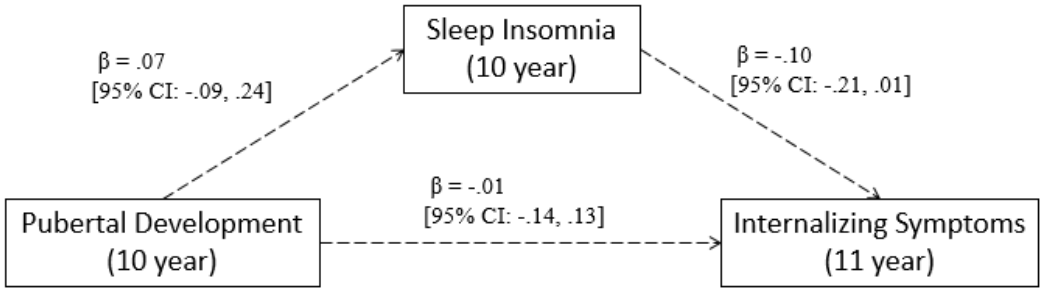
(g) Sleep Midpoint Variability- Girls



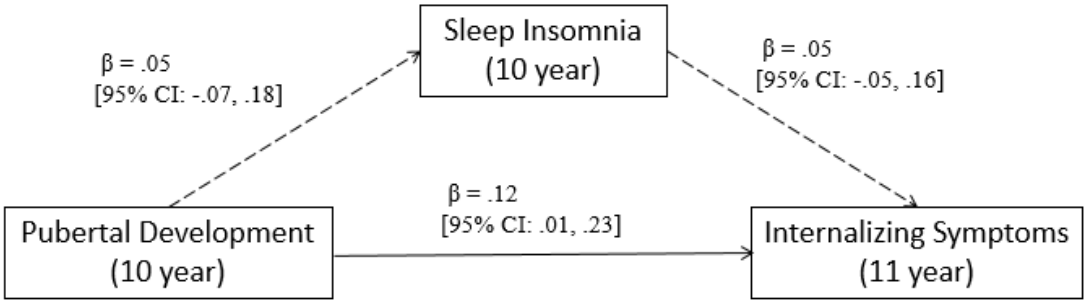
(h) Sleep Midpoint Variability- Boys



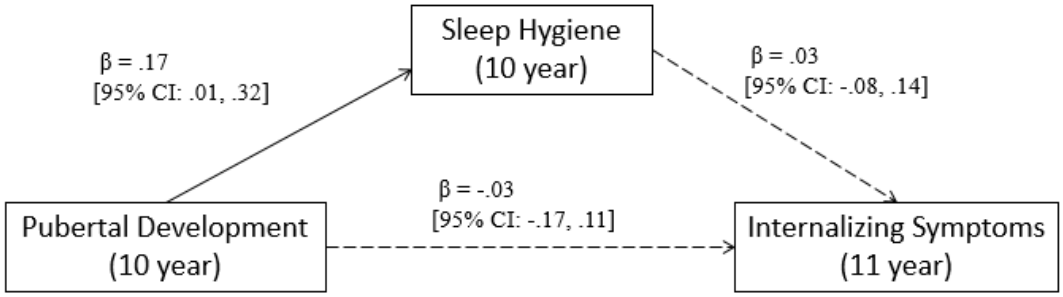
(i) Sleep Insomnia- Girls



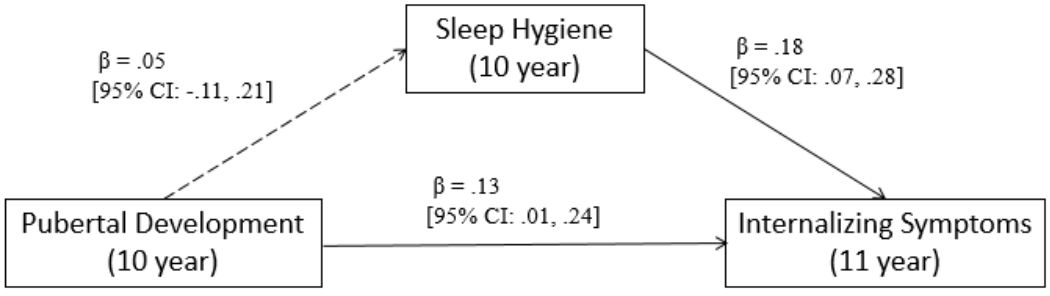
(j) Sleep Insomnia- Boys



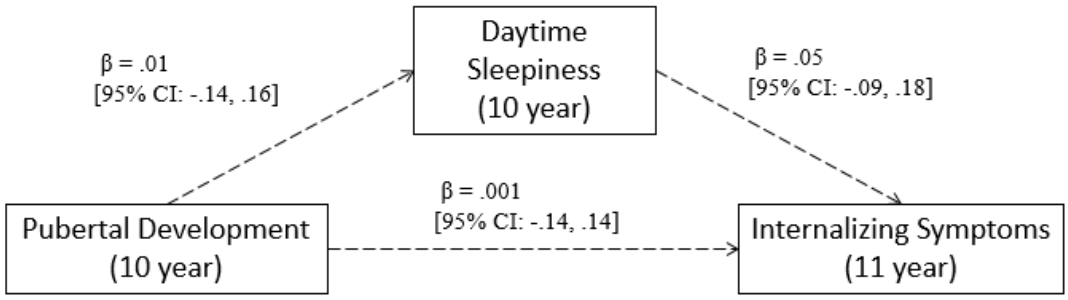
(k) Sleep Hygiene- Girls



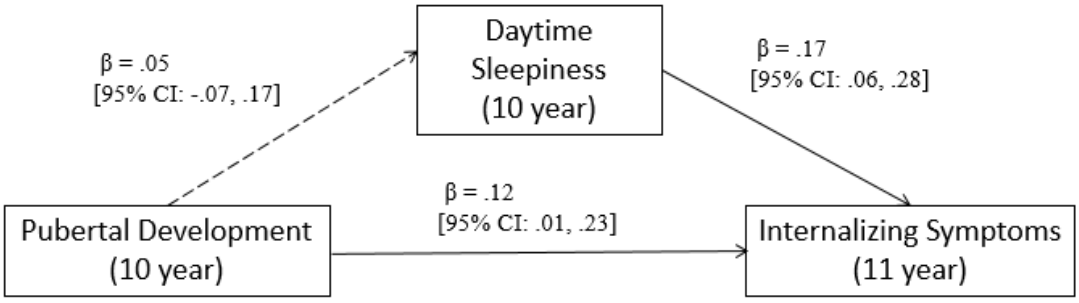
(l) Sleep Hygiene- Boys



(m) Daytime Sleepiness- Girls



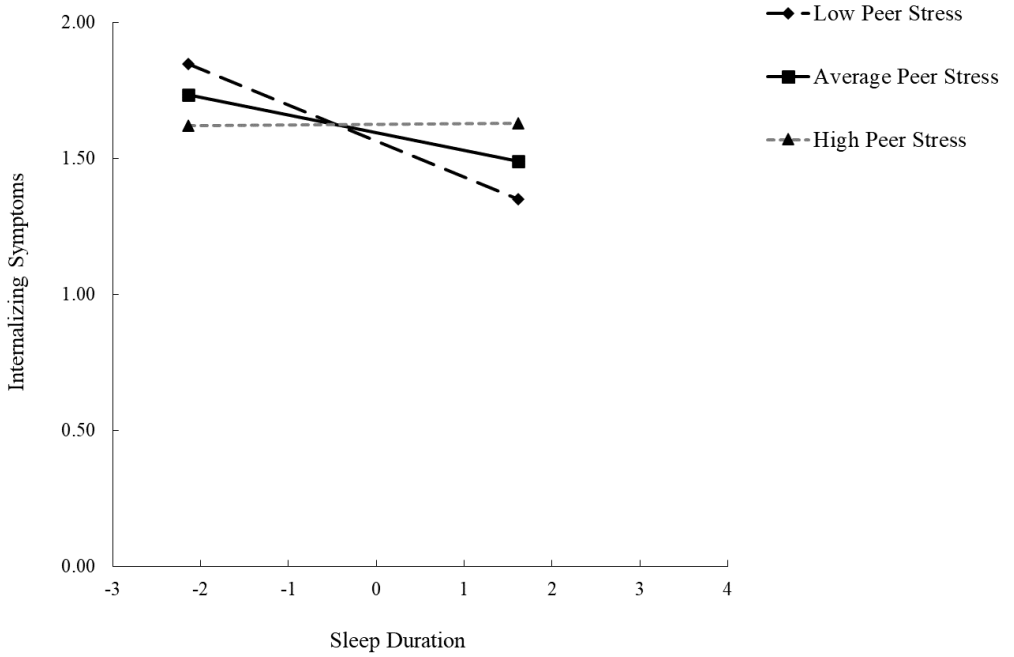
(n) Daytime Sleepiness- Boys



Note. Mediation model results for girls (top) and boys (bottom) for (a, b) sleep duration, (c, d) sleep efficiency, (e, f) sleep midpoint, (g, h) sleep midpoint variability, (i, j) sleep insomnia, (k, l) sleep hygiene, and (m, n) sleep daytime sleepiness. Standardized coefficients are provided. Statistically significant paths are solid lines and non-significant paths are dashed. * $p < 0.05$

Figure 5. *Simple slopes plot for the association between sleep duration (centered) and internalizing symptoms at low, average, and high levels of peer stress, for boys (a) and girls (b)*

(a) Boys



(b) Girls

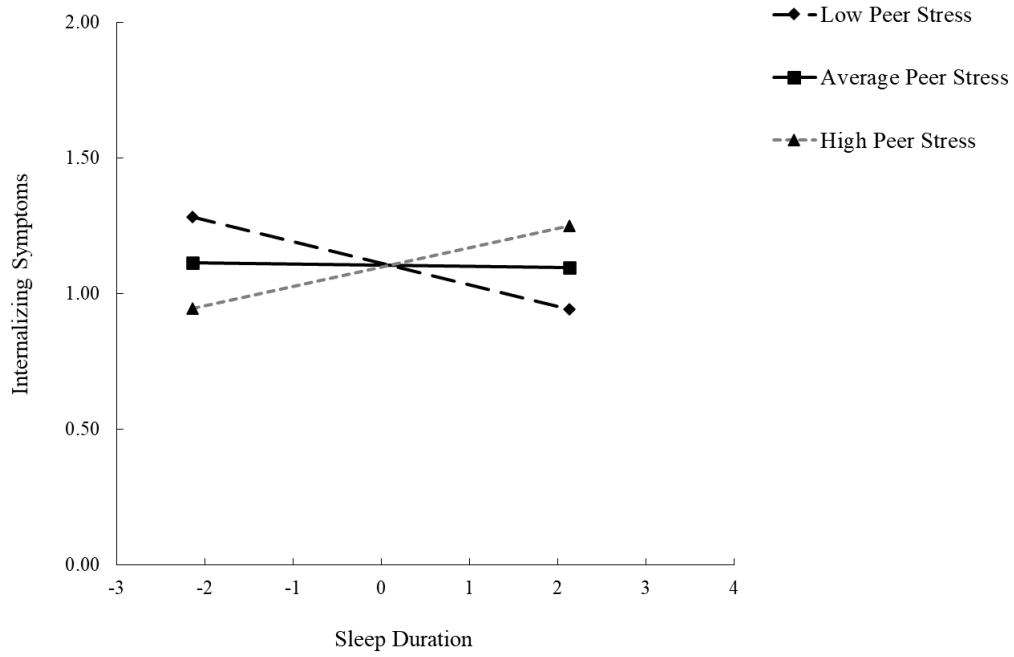


Figure 6. *Sensitivity analysis without controlling for internalizing symptoms (age 10). Simple slopes plot for the association between pubertal development and internalizing symptoms at low, average, and high levels of peer stress, for girls*

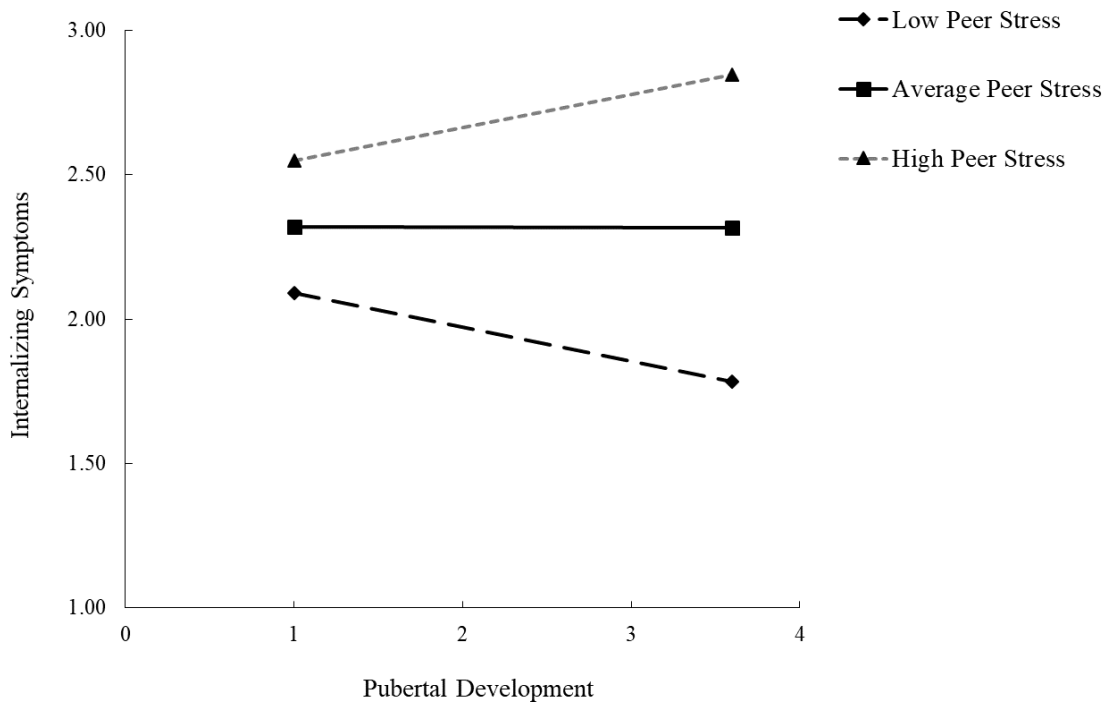
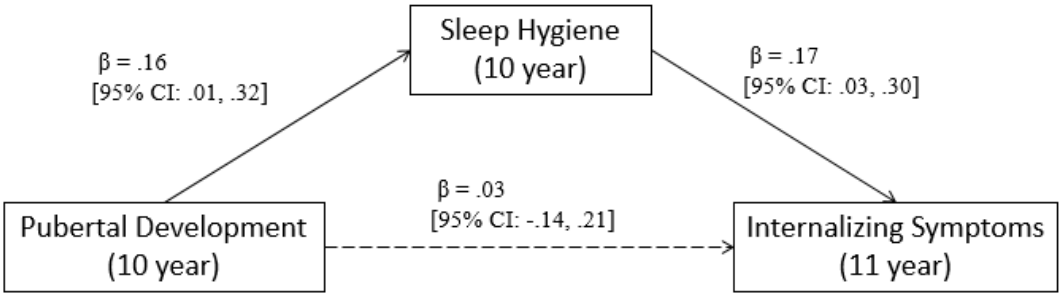


Figure 7. *Sensitivity Analysis of Sleep Hygiene as a Mediator Between Puberty and Internalizing Symptoms for Girls*



Note. The indirect path between family stress and internalizing symptoms through interpersonal stress was significant, $B = 0.027$, SE = 0.018, 95% CI= .000, .069. Standardized coefficients are provided. Statistically significant paths are solid lines and non-significant paths are dashed. * $p < 0.05$

APPENDIX A

IRB AND FUNDING ACKNOWLEDGEMENTS

Appendix A: IRB and Funding Acknowledgements

This work was supported by the US Eunice Kennedy Shriver National Institute of Child Health and Human Development: R01 HD079520 and R01 HD086085 to Kathryn Lemery-Chalfant and Leah Doane, and Mary Davis and Kathryn Lemery-Chalfant. Approval was obtained by the Institutional Review Boards at Arizona State University (IRB# STUDY00000637 and STUDY00004309).

ASU Knowledge Enterprise Development

APPROVAL: EXPEDITED REVIEW

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

Dear Kathryn Lemery:

On 2/10/2014 the ASU IRB reviewed the following protocol:

Type of Review:	Initial Study
Title:	Social and Genetic Contributions to Children's Sleep, Health and Functioning
Investigator:	Kathryn Lemery
IRB ID:	STUDY00000637
Category of review:	(7)(b) Social science methods, (7)(a) Behavioral research
Funding:	None
Grant Title:	None
Grant ID:	None
Documents Reviewed:	• ATP_informedconsent_2.5_final.pdf, Category: Consent Form; • ATPIRB_HRP-503a.docx, Category: IRB Protocol; • ATP_IRBMeasureDoc.pdf, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions); • ATP_IRBDiaryAppendix.pdf, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions); • ATP_recruitmentletterNEW.pdf, Category: Recruitment Materials; • ATP_recruitmentletterRETURNERS.pdf, Category: Recruitment Materials; • ATP_responseletter.pdf, Category: Recruitment Materials;

The IRB approved the protocol from 2/10/2014 to 2/9/2015 inclusive. Three weeks before 2/9/2015 you are to submit a completed "FORM: Continuing Review (HRP-212)" and required attachments to request continuing approval or closure.

If continuing review approval is not granted before the expiration date of 2/9/2015 approval of this protocol expires on that date. When consent is appropriate, you must use final, watermarked versions available under the "Documents" tab in ERA-IRB.

In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

Sincerely,

IRB Administrator

cc:

Kathryn Lemery
Reagan Styles
Sierra Clifford
Nancy Gonzales
Leah Doane
Craig Enders
Carlos Valiente

ASU Knowledge Enterprise Development

APPROVAL: EXPEDITED REVIEW

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

Dear Kathryn Lemery:

On 5/2/2016 the ASU IRB reviewed the following protocol:

Type of Review:	Initial Study
Title:	Genetic and Environmental Origins of the Development of Pain in Children
Investigator:	Kathryn Lemery
IRB ID:	STUDY00004309
Category of review:	(6) Voice, video, digital, or image recordings, (3) Noninvasive biological specimens, (7)(b) Social science methods, (7)(a) Behavioral research
Funding:	Name: HHS-NIH: National Institute of Child Health & Human Development (NICHD), Grant Office ID: FP00004748
Grant Title:	FP00004748;
Grant ID:	FP00004748;
Documents Reviewed:	• ATP Child Verbal Assent Pain Transmission, Category: Consent Form; • Survey 1: parent report of child health, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions); • ATP Pain Transmission IRB April 2016, Category: IRB Protocol; • ATP Pilot Recruitment Flyer, Category: Recruitment materials/advertisements /verbal scripts/phone scripts; • Survey 3: child report of health, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions); • Pilot child verbal assent, Category: Consent Form; • R01 grant, Category: Sponsor Attachment;

	• Pilot parent informed consent, Category: Consent Form; • Response Card, Category: Recruitment materials/advertisements /verbal scripts/phone scripts; • Recruitment Letter, Category: Recruitment materials/advertisements /verbal scripts/phone scripts; • ATP Parent Main Consent Pain Project, Category: Consent Form; • Survey 2: parent health and family environment, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions); • Survey 4: demographics, Category: Measures (Survey questions/Interview questions /interview guides/focus group questions);

The IRB approved the protocol from 5/2/2016 to 5/1/2017 inclusive. Three weeks before 5/1/2017 you are to submit a completed Continuing Review application and required attachments to request continuing approval or closure.

If continuing review approval is not granted before the expiration date of 5/1/2017 approval of this protocol expires on that date. When consent is appropriate, you must use final, watermarked versions available under the "Documents" tab in ERA-IRB.

In conducting this protocol you are required to follow the requirements listed in the INVESTIGATOR MANUAL (HRP-103).

Sincerely,

IRB Administrator

cc:

Mary Davis
Kevin Grimm