Published in final edited form as:

J Child Psychol Psychiatry. 2020 October ; 61(10): 1070–1079. doi:10.1111/jcpp.13328.

Children's sleep, impulsivity, and anger: shared genetic etiology and implications for developmental psychopathology

Samantha A. Miadich^{1,2}, Amanda M. Shrewsbury¹, Leah D. Doane¹, Mary C. Davis¹, Sierra Clifford¹, Kathryn Lemery-Chalfant¹

¹Department of Psychology, Arizona State University, Tempe, AZ, USA

²Department of Psychology, University of Massachusetts Lowell, Lowell, MA, USA

Abstract

Background: Prior research has established links between poor sleep and problems in emotion regulation. Impulsivity and anger/frustration are core features of child psychopathology. Further, sleep problems are commonly associated with psychopathology. This study examined shared and unique genetic and environmental influences on sleep, impulsivity, and anger/frustration in the middle childhood period with potential ramifications for psychopathology.

Methods: Families (29.9% monozygotic, 38.6% same-sex dizygotic, 31.5% opposite-sex dizygotic) from a longitudinal twin study participated ($N = 613$ twins). Twins ($M_{\text{age}} = 8.37$, $SD = 0.66$; 49% female; 58% non-Latinx European American, 30% Latinx) wore actigraph watches for seven days to assess sleep. Primary caregivers (95.3% mothers) completed standardized questionnaires to assess twins' temperament (impulsivity, anger/frustration).

Results: Univariate ACE twin structural equation models indicated strong genetic influences (76%) on impulsivity, whereas the largest proportion of variance in anger/frustration was attributed to the shared environment (56%). Bivariate model fitting indicated that sleep—impulsivity and sleep—anger/frustration associations in children are genetic; thus, a mutual underlying genetic factor likely contributes to the commonality in these associations.

Conclusions: Given evidence that sleep problems, impulsivity, and anger/frustration are mechanisms associated with psychopathology, our findings suggest a genetic commonality and the need to focus on shared and unique risk factors when understanding etiology. Early intervention and prevention efforts should target both sleep problems and high levels of impulsivity and anger/frustration in children, which may have implications for later psychopathology.

Keywords

Sleep; impulsivity; anger

Correspondence: Samantha A. Miadich, Department of Psychology, University of Massachusetts Lowell, 850 Broadway Street, Lowell, MA 01854-3059, USA; Samantha_Miadich@uml.edu.

Conflict of interest statement: No Conflicts declared.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1. Twin intraclass correlations and cross-twin cross-trait correlations.

Table S2. Parameter estimates for full and best-fitting bivariate Cholesky decompositions.

Introduction

Over 30% of school-aged children do not meet the recommended hours of sleep per night, and this percentage increases as children age into middle and high school (National Sleep Foundation, 2014; Wheaton, Jones, Cooper, & Croft, 2018). Childhood sleep problems are common, often persistent across development if unaddressed, and linked to emotional and behavioral problems and psychopathology (Barclay & Gregory, 2014; Gregory & Sadeh, 2016). One factor related to sleep is temperament, defined as biologically based individual differences in reactivity and self-regulation (Rothbart & Bates, 2006). Difficult temperament is both predictive of and predicted by sleep problems (Bastien, Tétreault, & Bernier, 2019), and both temperamental difficulties and sleep problems (e.g., shortened durations, poor efficiency) in childhood are implicated in future psychopathology (Gregory & Sadeh, 2016; Nigg, 2006). In particular, impulsivity and anger/frustration are core features of childhood externalizing problems and have been linked to internalizing outcomes, with predictive utility for psychiatric symptomatology and diagnosis, including attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (CD; Beauchaine, Zisner, & Sauder, 2017; Carver, Johnson, & Joormann, 2008; Eisenberg et al., 2009; Leibenluft & Stoddard, 2013; Zeman, Shipman, & Suveg, 2002).

Understanding the etiology of these developmental precursors may shed light on risk prior to the onset of more severe psychopathology during adolescence (Costello, Copeland, & Angold, 2011). Little is known about the extent to which relations between childhood temperament and sleep are explained by genetic or environmental factors. We used a twin design to elucidate genetic and environmental contributions to the relations between sleep (duration and efficiency) and temperament (impulsivity and anger/frustration) in childhood, with implications for future psychopathology.

Sleep and temperament—precursors for psychopathology

Sleep difficulties and disruptions in childhood are associated with and predictive of psychopathology, including ADHD, depression, anxiety, bipolar, CD, oppositional-defiant disorder, and substance use, and these associations are likely bidirectional (Barclay & Gregory, 2014; Gregory & Sadeh, 2016). This research relies primarily on parent report of sleep, but prospective reciprocal associations between objectively assessed sleep and internalizing and externalizing symptoms have been found among a community sample of children assessed across middle childhood through early adolescence (Kelly & El-Sheikh, 2014). Specifically, decreased sleep duration and quality (sleep percentage, efficiency, sleep–wake episodes) predicted anxiety, depressive, and externalizing symptoms (Kelly & El-Sheikh, 2014). Such objective assessment of sleep is more accurate and unbiased than parent report of children's sleep, and enables assessment of a wider range of parameters (e.g., efficiency), offering a more well-rounded portrayal of sleep.

The associations among sleep, emotional and behavioral problems, and clinical symptomatology across development highlight the importance of understanding the genetic and environmental etiology of sleep problems in childhood, including relations with psychopathology risk factors such as temperament. However, only limited research in twin children has evaluated actigraphy-based sleep assessments (i.e., two studies) or their

associations with other predictors of psychopathology. In one study of 66 twelve-year-old twin pairs, objective sleep duration and efficiency showed moderate-to-high heritability ($h^2 = .57-.65$; Sletten et al., 2013). Inderkum and Tarokh (2018) found that among 26 adolescent twin pairs, objective efficiency was highly heritable ($>60\%$) and duration was heritable on free days (68%), but not on school days (15%). Due to the small sample sizes in these studies, estimates may not be reliable (Posthuma & Boomsma, 2000). Other findings from our sample suggest high heritability of actigraphy-based duration and efficiency in middle childhood ($h^2 = .79-.81$; Breitenstein, Doane, & Lemery-Chalfant, 2020). Although studies suggest common genetic influences between parent-reported sleep problems and depressive symptoms in childhood (8–10 years old; Gregory, Rijdsdijk, Lau, Dahl, & Eley, 2009), no twin studies to our knowledge have examined genetic and environmental associations between objective sleep and temperament as precursors for psychopathology.

The temperamental dimensions of impulsivity and anger/frustration are related to psychopathology (Moffitt et al., 2011; Rothbart & Bates, 2006). Impulsivity is the speed of response initiation; children who are impulsive behave quickly without processing consequences (Rothbart, Ahadi, Hershey, & Fisher, 2001). Impulsivity is thought to be a core feature of childhood psychopathology, particularly ADHD and other externalizing disorders, is implicated in the liability to developing externalizing disorders in early childhood through adolescence, and has been proposed as one developmental trajectory to psychopathology (Beauchaine et al., 2017; Martel, Levinson, Lee, & Smith, 2017). Anger/frustration is the frequency and intensity of displays of the primary emotion anger (e.g., an approach emotion elicited by blocked goals) and has been linked to externalizing and internalizing symptoms (Eisenberg et al., 2009; Leibenluft & Stoddard, 2013; Rothbart et al., 2001). Specifically, anger/frustration in middle childhood is associated with self-regulation and conduct problems, and persistent anger/frustration is related to adolescent psychopathology including anxiety, depression, and CD (Leibenluft & Stoddard, 2013).

Impulsivity is thought to be influenced by both genetic and environmental factors, and is moderately to highly heritable across the lifespan, with the highest estimates among children (Bezdjian, Baker, & Tuvblad, 2011). For example, parent and self-reports of impulsivity among children and adolescents show high heritability ($h^2 = .74-.82$; Brooker et al., 2020; Vierikko, Pulkkinen, Kaprio, & Rose, 2004). Heritability estimates of anger range from moderate to high, whereas shared environmental contributions range from negligible to almost 30% of the variance in anger, depending on child age and method of assessment (Clifford, Lemery-Chalfant, & Goldsmith, 2015; Deater-Deckard, Petrill, & Thompson, 2007; Goldsmith, Buss, & Lemery, 1997). Given the salience of impulsivity and anger/frustration for future psychopathology, it is important to understand their etiology and associations with sleep problems.

Sleep, impulsivity, and anger/frustration

Although sleep problems, impulsivity, and anger/frustration in childhood are individually associated with later psychopathology, associations among these factors in childhood are not fully understood. Knowledge of the genetic etiology of sleep and temperament would be helpful for understanding their combined influence on psychopathology. Phenotypic

associations between sleep and both impulsivity and anger/frustration have been noted in the childhood/adolescent literature (Gruber, Cassoff, Frenette, Wiebe, & Carrier, 2012; Paavonen et al., 2009; Van Dyk et al., 2017). For example, Gruber et al. (2012) found that among typically developing children in middle childhood, extension of the sleep period by as little as 30 min was associated with improvements in emotional lability and restless-impulsive behavior, while reduction by just one hour was associated with poorer outcomes, including more restless-impulsive behaviors. Additionally, in a study of healthy children, El-Sheikh and Buckhalt (2005) found that high negative emotionality was associated with problems in sleep duration and quality. Later in development, self-reported sleep quality is related to ADHD (including impulsivity symptoms), and these associations may be due to genetic and nonshared environmental influences (Gregory, Agnew-Blais, Matthews, Moffitt, & Arseneault, 2017).

Similarly, sleep extension may have positive ramifications for anger. Sleep extension on adolescents' school nights is associated with improved self-reports of anger (Van Dyk et al., 2017), whereas shorter durations are associated with greater reports of anger and impulsivity (Bauducco, Flink, Jansson-Fröjmark, & Linton, 2016; Itani et al., 2016). Less is known about associations between sleep and anger in middle childhood. Parent-reported sleep problems are related to difficulties in emotion regulation (e.g., Williams, Berthelsen, Walker, & Nicholson, 2017), although it is unclear if and how objective indicators of sleep quality relate to the two facets of temperament. One recent study found delayed sleep time to interact with negative emotionality (encompassing anger/frustration) in the prediction of internalizing and externalizing behaviors among young children (Cremone et al., 2018). Taken together, the prior literature suggests poor sleep may be implicated in difficulties with emotion regulation (e.g., more anger, impulsivity; Bauducco et al., 2016), and together poor sleep and emotion dysregulation may pose an increased risk for psychopathology. However, the underlying connections between sleep and temperament have yet to be fully explicated using a genetically informed design.

Current study

Although the importance of sleep and temperament for psychopathology is evident, the mechanisms linking these outcomes are less clear. Understanding the shared genetic and environmental influences of sleep, impulsivity, and anger/frustration in childhood can inform understanding of the development of psychopathology. Among adults, shared genes explain the relation between self-reported sleep disturbances and externalizing symptoms (Barclay, Eley, Maughan, Rowe, & Gregory, 2011). However, the present study is the first to our knowledge to examine the genetic and environmental underpinnings of objectively assessed sleep and dimensions of temperament (impulsivity and anger/frustration) in childhood.

Building upon prior work showing high heritability of objective sleep (Breitenstein et al., 2020), the current study aimed to examine (a) the genetic, shared environmental, and nonshared environmental influences on impulsivity and anger/frustration, and (b) whether sleep duration and efficiency are connected to impulsivity and anger/frustration through genetic and/or environmental influences. Based on prior research, we hypothesized that impulsivity would be highly heritable, and anger/frustration would be influenced by genetics

and the environment. Given strong genetic influences on sleep duration and efficiency, we hypothesized that their associations with impulsivity and anger/frustration would be genetic.

Methods

Participants

Participants are part of the Arizona Twin Project, an ongoing, longitudinal twin study (Lemery-Chalfant et al., 2019). The current study includes 613 children (309 families; 29.9% monozygotic [MZ] twins, 38.6% same-sex dizygotic [DZ] twins, 31.5% opposite-sex DZ twins). Five twins were excluded due to severe mental or physical disability. Families that lived out-of-state were unable to complete objective assessments and thus excluded. Children (49% female, 58% non-Latinx European American, 30% Latinx) were assessed at eight years ($M = 8.37$, $SD = 0.66$). Most primary caregivers were the twins' mothers (95.3%) and reported having gone to college, but no degree (27.9%), a college degree (36.7%), or a graduate degree (22.2%). Based on an income-to-needs ratio using household income, family size, and poverty thresholds, 7.3% of families lived below the poverty line, 23.1% were at or near the poverty line, 16.2% were lower-middle class, and 53.4% were middle to upper class.

Ethical considerations

Institutional Review Board approval was obtained prior to data collection. Caregiver informed consent and child assent were obtained at the beginning of the assessment.

Procedures

Families were contacted as twins approached Eight years of age to participate in an intensive assessment focused on children's sleep and health. The eight-year assessment included online surveys, two home visits, and a week of daily assessments including actigraphy. During this week, children wore actigraph watches for seven consecutive days.

Measures

Demographics.—Primary caregivers completed demographic forms including information on the children's age, sex, and race/ethnicity. Socioeconomic information included primary and secondary caregivers' educational attainment, gross household income, and household size.

Actigraphy.—Children wore Micro MotionLogger Watches, which are wrist-based accelerometers designed to distinguish between periods of sleep and activity (Ambulatory Monitoring, Inc, Ardsley, NY USA), on their nondominant wrist for seven consecutive days/nights. The Sadeh algorithm in Action W-2 software version 2.7.1 (Ambulatory Monitoring) program was used to analyze the data (Sadeh, Sharkey, & Carskadon, 1994). Prior studies have detailed the actigraphy assessment procedures (see Doane et al., 2019; Miadich et al., 2019). This study used two sleep parameters obtained from actigraphy: sleep duration (total minutes of sleep between night sleep onset and offset) and sleep efficiency (proportion of minutes asleep between sleep onset and sleep offset). Ninety percent of eligible children completed the actigraphy assessment with excellent compliance ($M_{nights} = 6.79$, $SD = 0.71$).

Ten percent of children had complete missing data for various reasons (e.g., noncompliance, watch malfunctions).

Temperament.—Primary caregivers completed the Temperament in Middle Childhood Questionnaire (Simonds, Kieras, Rueda, & Rothbart, 2007), which assesses dimensions of temperament including impulsivity (13 items; e.g., ‘grabs what s/he wants’; $\alpha = 0.87$) and anger/frustration (7 items; e.g., ‘gets mad when provoked by other children’; $\alpha = 0.82$) on a 1 (almost always untrue of your child) to 5 (almost always true of your child) scale. For both scales, a mean composite of the items was formed, and higher scores indicate more impulsivity and anger/frustration.

Data analysis

OpenMx in R (Neale et al., 2016) was used to fit biometrical structural equation ACE models to parse additive genetic (A), shared (common) environment (C; increases cotwin similarity), and unique environment (E; distinguishes cotwins, plus measurement error) contributions to variance. In this model, A is correlated at 1.00 for MZ twins (who share 100% of their genes) and 0.50 for DZ twins (who share half of their segregating genes). After fitting the full ACE model, A and C parameters are dropped in turn to determine their significance and arrive at the most parsimonious model. Univariate models were fit for each temperament dimension. Four bivariate Cholesky decompositions were conducted to examine genetic and environmental covariance between each sleep parameter and temperament. Similar to univariate models, paths were dropped sequentially, and fit statistics including the $-2LL$ and AIC were examined to determine the best-fitting model. Children’s sex and age were regressed out of sleep and temperament prior to model fitting, and sleep scores were reversed in order to have a positive correlation between sleep and impulsivity or anger/frustration. The current sample was well powered to partition estimates of variance in univariate models and adequately powered to detect expected moderate effects in bivariate models (Posthuma & Boomsma, 2000).

Results

Preliminary findings

On average, children slept 8.12 hr ($SD = 0.71$) a night and had a 90.07% ($SD = 5.47$) sleep efficiency across the assessment week. Both impulsivity ($M = 3.03$, $SD = 0.68$) and anger/frustration ($M = 2.91$, $SD = 0.80$) ranged from one to five. Phenotypic correlations were moderate among sleep duration and efficiency ($r = .65$, $p < .001$), and impulsivity and anger/frustration ($r = .54$, $p < .001$). Sleep duration was correlated with impulsivity ($r = -.17$, $p < .001$) and anger/frustration ($r = -.16$, $p < .001$), as was sleep efficiency (impulsivity: $r = -.15$, $p = .001$; anger/frustration: $r = -.11$, $p = .01$). Twin intraclass correlations for impulsivity revealed that MZ twins had a greater correlation (.76) than same-sex and opposite-sex DZ twins (.34–.57; Table S1), suggesting a genetic influence. For anger/frustration, correlations between MZ twins (.81) were closer in magnitude to same-sex and opposite-sex DZ twin correlations (.63–.75), suggesting modest genetic influences and larger shared environmental influences. Cross-twin cross-trait correlations for both impulsivity and

anger/frustration were stronger for MZ than DZ twins for both sleep parameters, suggesting common genetic influences (Table S1).

Univariate ACE models

Fit statistics and parameter estimates of the full and reduced univariate ACE models for impulsivity and anger/frustration are reported in Table 1. The reduced AE model fits best for impulsivity, and indicated a high genetic contribution (76%). For anger/frustration, the full ACE model fits best, with the majority of variance attributable to shared environmental influences (56%), and additive genetic influences explaining approximately a fourth of the variance.

Bivariate ACE models

Table 2 includes the fit statistics for the full and reduced models for the four bivariate models. Figure 1 includes the standardized variance estimates of the full bivariate ACE models (dashed paths are dropped in best-fitting models). For sleep duration and impulsivity, the AE-A-AE model fits best, meaning that duration was influenced by A (.76) and E (.24) factors and associated with impulsivity ($A=.76$, $E=.24$) solely for additive genetic reasons (see Table S2 for all estimates of full and best-fitting models; note that full model estimates are presented in figures and best-fitting model estimates are described in text and Table S2). Both sleep duration and impulsivity were shown to be highly heritable, consistent with univariate models. The correlation between the latent genetic influences on duration and the latent genetic influences on impulsivity was modest ($rA = .18$), meaning that although common genetic influences fully explained the phenotypic correlation, genetic influences on each variable were still largely independent. The model with the best fit for the sleep efficiency and impulsivity association was also the AE-A-AE model, suggesting the commonality between efficiency and impulsivity at this age is due to additive genetic influences. Also consistent with univariate models, sleep efficiency (.71) and impulsivity (.76) were highly heritable in the best-fitting model. The genetic correlation between efficiency and impulsivity was $rA = .13$.

Findings were similar for models examining anger/frustration in relation to sleep. The bivariate decomposition for sleep duration and anger/frustration revealed that the AE-A-AE model fits best and that the additive genetic correlation between duration and anger/frustration was $rA = .28$, meaning that in the best-fitting model, duration was influenced by A (.76) and E (.24) factors and associated with anger/frustration ($A = .26$, $C = .55$, $E = .19$) solely for additive genetic reasons (*full model estimates are presented in figures, and best-fitting model estimates are described in text and Table S2*). Unlike sleep duration, anger/frustration had a large shared environmental influence and a smaller additive genetic influence. For efficiency and anger/frustration, the model with the best fit was also the AE-A-AE model. Consistent with prior models, in the reduced best-fitting model, sleep efficiency was highly heritable (.71) and anger/frustration was largely influenced by the shared environment (.56) and additive genetics (.25). The genetic correlation between efficiency and anger/frustration was $rA = .29$.

Discussion

The current study examined the associations of eight-year-old children's objectively assessed sleep duration and efficiency with their impulsivity and anger/frustration. Impulsivity was highly heritable, whereas the greatest proportion of variance in anger/frustration was attributed to shared environmental influences. Bivariate analyses revealed that objective sleep duration and efficiency were largely independent of impulsivity and anger/frustration at this age. However, to the extent that these two risk factors for psychopathology are related, this link is driven by common genetic influences. Notably, sleep was assessed daily for a week via actigraphy and the temperament questionnaire covered the last six months, potentially explaining the small effects.

Consistent with past literature suggesting moderate-to-high heritability, with higher estimates in childhood samples (Bezdjian et al., 2011; Brooker et al., 2020), impulsivity was highly heritable ($h^2 = .76$), with no shared environmental influences. Child anger/frustration was less heritable ($h^2 = .25$), with large shared environmental influences (56%). Prior research is mixed regarding the heritability of anger/frustration, and current findings are consistent with research finding shared environmental influences on anger in childhood (Clifford et al., 2015; Deater-Deckard et al., 2007; Goldsmith et al., 1997). Shared environmental influences were stronger in this study, potentially due to greater socioeconomic and cultural diversity in our sample relative to many other twin studies, leading to greater variation in trait-relevant cultural factors or stressors.

Consistent with previous phenotypic findings, shorter sleep durations and poorer efficiencies were associated with greater reported impulsivity (Gruber et al., 2012; Paavonen et al., 2009), with these associations due to additive genetics. This may indicate a genetic vulnerability underlying both poor sleep and impulsivity. For example, prior research has implicated serotonergic dysregulation in poor sleep and impulsivity (Carver et al., 2008; Harvey, Murray, Chandler, & Soehner, 2011). Research with rats found that problematic and restricted sleep led to changes in serotonin-receptor sensitivity, suggesting a link between poor sleep and dysregulation of the serotonergic system (Kamphuis, Meerlo, Koolhaas, & Lancel, 2012). Further, dysregulation in serotonergic function has been linked to mental health problems including depression, suicide, anxiety disorders, and eating disorders (Harvey et al., 2011). The current findings provide further support for an underlying genetic connection between sleep duration, efficiency, and impulsivity, which may be shared with future psychopathology.

Extending the adolescent literature to middle childhood, shorter sleep durations and poor efficiencies were associated with anger/frustration, with these relations explained by additive genetic influences (Bauducco et al., 2016; Van Dyk et al., 2017). In prior work, adolescents reported greater anger/hostility and irritability under sleep restriction (6.5 hours/night) versus a healthy sleeping condition (Baum et al., 2014). Additionally, during the sleep restriction phase, adolescents and parents reported more problems with adolescents' emotional reactions and increased outbursts. Anger and self-regulation may be linked to externalizing behaviors, specifically conduct problems, due to genetic influences (Deater-Deckard et al., 2007). Anger has been associated with other forms of psychopathology

across development, including internalizing and externalizing symptoms, borderline personality disorder, bipolar depression, ADHD, and substance use problems (Eisenberg et al., 2009; Lubke, Ouwers, de Moor, Trull, & Boomsma, 2015). Therefore, it is plausible that there is a common heritable dysregulation factor underlying poor sleep and anger/frustration and that this dysregulation may also influence psychopathology, accounting for the co-occurrence of these constructs. Further, distinct but related research has investigated a general factor underlying liability to psychopathology, termed the p factor, and has demonstrated that increases in this common and heritable factor influences risk for and are predictive of psychopathology (Caspi & Moffitt, 2018).

Limitations

In addition to the strengths of the study, including the diverse community-based sample of children and objectively assessed sleep, this study had several limitations. Primary caregivers reported on the temperament of both twins, potentially leading to overestimation of shared environmental influences. However, although shared environmental influences were substantial for anger/frustration, they were negligible for impulsivity, demonstrating caregivers' abilities to distinguish between twins. Another consideration is the generalizability of findings to singleton families. Both the objective sleep and temperament descriptive statistics are similar to studies of singletons (El-Sheikh et al., 2013; Kotelnikova, Olino, Klein, Mackrell, & Hayden, 2017). Finally, the models did not account for gene-environment interactions or correlations. For example, genetic associations between sleep, impulsivity, and anger/frustration may be explained by gene-environment interaction with family-level factors, which register as additive genetic influences in ACE models.

Clinical implications and future directions

We took correlated known precursors to psychopathology and fit a twin model to determine the extent to which the risk for psychopathology is shared or unique. These factors shared genetic covariance; therefore, the risk is not fully independent and future studies should examine whether some underlying factor such as dysregulation accounts for this shared risk. To note, only two dimensions of temperament and two sleep parameters were examined in the current study, and we suggest that future studies investigate other dimensions of temperament (e.g., inhibitory control) and/or other facets of sleep (e.g., sleep onset latency, timing of the sleep period). With multivariate twin models, future studies should examine whether genetic factors underlying relations between poor sleep, impulsivity, and anger/frustration also account for associations with psychopathology, or if these factors are separate risk factors. In middle childhood, within a community sample, we found little overlap between sleep and temperament, but sleep, impulsivity, and anger/frustration may become more correlated across puberty and the transition to adolescence, and longitudinal research across this period of increasing risk may prove valuable. Additionally, larger twin studies should consider incorporating objective assessments of sleep to investigate whether these findings can be replicated. Finally, future directions should include studying high-risk subgroups, such as children with clinically significant sleep problems. Poor sleep may mediate relations between temperament and psychopathology. Although current findings suggest an underlying genetic connection between poor sleep, impulsivity, and anger/frustration, these associations were relatively small at this age; therefore, it may be

important to target sleep problems and increased levels of impulsivity and anger/frustration separately in middle childhood to potentially improve and prevent future psychopathology.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

This study was supported by two grants from the National Institute of Child Health and Human Development (NICHD R01HD079520; PIs K.L.-C. & L.D.D. and NICHD R01HD086085; PIs M.C.D. & K.L.-C.). The authors have declared that they have no competing or potential conflicts of interest.

References

- Barclay NL, Eley TC, Maughan B, Rowe R, & Gregory AM (2011). Associations between diurnal preference, sleep quality and externalizing behaviours: A behavioural genetic analysis. *Psychological Medicine*, 41, 1029–1040. [PubMed: 20836908]
- Barclay NL, & Gregory AM (2014). Sleep in childhood and adolescence: Age-specific sleep characteristics, common sleep disturbances and associated difficulties. *Current Topics in Behavioral Neurosciences*, 16, 337–365. [PubMed: 24170426]
- Bastien L, Tétreault É, & Bernier A (2019). Disentangling the direction of associations between sleep and temperament in toddlers. *Behavioral Sleep Medicine*, 18, 523–536. [PubMed: 31233348]
- Bauducco SV, Flink IK, Jansson-Fröjmark M, & Linton SJ (2016). Sleep duration and patterns in adolescents: correlates and the role of daily stressors. *Sleep Health*, 2, 211–218. [PubMed: 29073425]
- Baum KT, Desai A, Field J, Miller LE, Rausch J, & Beebe DW (2014). Sleep restriction worsens mood and emotion regulation in adolescents. *Journal of Child Psychology and Psychiatry*, 55, 180–190. [PubMed: 24889207]
- Beauchaine TP, Zisner AR, & Sauder CL (2017). Trait impulsivity and the externalizing spectrum. *Annual Review of Clinical Psychology*, 13, 343–368.
- Bezdjian S, Baker LA, & Tuvblad C (2011). Genetic and environmental influences on impulsivity: A meta-analysis of twin, family and adoption studies. *Clinical Psychology Review*, 31, 1209–1223. [PubMed: 21889436]
- Breitenstein RS, Doane LD, & Lemery-Chalfant K (2020). Children's objective sleep assessed with wrist-based accelerometers: Strong heritability of objective quantity and quality unique from parent-reported sleep. *Sleep*. 10.1093/sleep/zsaa142
- Brooker RJ, Moore MN, Van Hulle CA, Beekman CR, Begnoche JP, Lemery-Chalfant K, & Goldsmith HH (2020). Attentional control explains covariation between symptoms of Attention-Deficit/Hyperactivity Disorder and anxiety during adolescence. *Journal of Research on Adolescence*, 30, 126–141. [PubMed: 31095814]
- Carver CS, Johnson SL, & Joormann J (2008). Serotonergic function, two-mode models of self-regulation, and vulnerability to depression: what depression has in common with impulsive aggression. *Psychological Bulletin*, 134, 912–943. [PubMed: 18954161]
- Caspi A, & Moffitt TE (2018). All for one and one for all: Mental disorders in one dimension. *American Journal of Psychiatry*, 175, 831–844.
- Clifford S, Lemery-Chalfant K, & Goldsmith HH (2015). The unique and shared genetic and environmental contributions to fear, anger, and sadness in childhood. *Child Development*, 86, 1538–1556. [PubMed: 26182850]
- Costello EJ, Copeland W, & Angold A (2011). Trends in psychopathology across the adolescent years: What changes when children become adolescents, and when adolescents become adults? *Journal of Child Psychology and Psychiatry*, 52, 1015–1025. [PubMed: 21815892]

- Cremone A, de Jong DM, Kurdziel LBF, Desrochers P, Sayer A, LeBourgeois MK, Spencer RMC, & McDermott JM (2018). Sleep tight, act right: Negative affect, sleep and behavior problems during early childhood. *Child Development*, 89, e42–e59. [PubMed: 28129449]
- Deater-Deckard K, Petrill SA, & Thompson LA (2007). Anger/frustration, task persistence, and conduct problems in childhood: A behavioral genetic analysis. *Journal of Child Psychology and Psychiatry*, 48, 80–87. [PubMed: 17244273]
- Doane LD, Breitenstein RS, Beekman C, Clifford S, Smith TJ, & Lemery-Chalfant K (2019). Early life socioeconomic disparities in children's sleep: The mediating role of the current home environment. *Journal of Youth and Adolescence*, 48, 56–70. [PubMed: 30121716]
- Eisenberg N, Valiente C, Spinrad TL, Cumberland A, Liew J, Reiser M, Zhou Q, & Losoya SH (2009). Longitudinal relations of children's effortful control, impulsivity, and negative emotionality to their externalizing, internalizing, and co-occurring behavior problems. *Developmental Psychology*, 45, 988–1008. [PubMed: 19586175]
- El-Sheikh M, Bagley EJ, Keiley M, Elmore-Staton L, Chen E, & Buckhalt JA (2013). Economic adversity and children's sleep problems: multiple indicators and moderation of effects. *Health Psychology*, 32, 849–859. [PubMed: 23148451]
- El-Sheikh M, & Buckhalt JA (2005). Vagal regulation and emotional intensity predict children's sleep problems. *Developmental Psychobiology*, 46, 307–317. [PubMed: 15832322]
- Goldsmith HH, Buss KA, & Lemery KS (1997). Toddler and childhood temperament: expanded content, stronger genetic evidence, new evidence for the importance of environment. *Developmental Psychology*, 33, 891–905. [PubMed: 9383612]
- Gregory AM, Agnew-Blais JC, Matthews T, Moffitt TE, & Arseneault L (2017). Associations between ADHD and sleep quality: Longitudinal analyses from a nationally-representative cohort of twins. *Journal of Clinical Child and Adolescent Psychology*, 46, 284–294. [PubMed: 27485465]
- Gregory AM, Rijdsdijk FV, Lau JYF, Dahl RE, & Eley TC (2009). The direction of longitudinal associations between sleep problems and depression symptoms: A study of twins aged 8 and 10 years. *Sleep*, 32, 189–199. [PubMed: 19238806]
- Gregory AM, & Sadeh A (2016). Annual Research Review: Sleep problems in childhood psychiatric disorders-A review of the latest science. *Journal of Child Psychology and Psychiatry*, 57, 296–317. [PubMed: 26412255]
- Gruber R, Cassoff J, Frenette S, Wiebe S, & Carrier J (2012). Impact of sleep extension and restriction on children's emotional lability and impulsivity. *Pediatrics*, 130, e1155–e1161. [PubMed: 23071214]
- Harvey AG, Murray G, Chandler RA, & Soehner A (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review*, 31, 225–235. [PubMed: 20471738]
- Inderkum AP, Tarokh L (2018). High heritability of adolescent sleep-wake behavior on free, but not school days: A long-term twin study. *Sleep*, 41(3). 10.1093/sleep/zsy004
- Itani O, Kaneita Y, Munezawa T, Ikeda M, Osaki Y, Higuchi S, Kanda H, Nakagome S, Suzuki K, & Ohida T (2016). Anger and impulsivity among Japanese adolescents: A nationwide representative survey. *Journal of Clinical Psychiatry*, 77, e860–e866.
- Kamphuis J, Meerlo P, Koolhaas JM, & Lancel M (2012). Poor sleep as a potential causal factor in aggression and violence. *Sleep Medicine*, 13, 327–334. [PubMed: 22305407]
- Kelly RJ, & El-Sheikh M (2014). Reciprocal relations between children's sleep and their adjustment over time. *Developmental Psychology*, 50, 1137–1147. [PubMed: 24188035]
- Kotelnikova Y, Olino TM, Klein DN, Mackrell SVM, & Hayden EP (2017). Higher and lower order factor analyses of the Temperament in Middle Childhood Questionnaire. *Assessment*, 24, 1050–1061. [PubMed: 27002124]
- Leibenluft E, & Stoddard J (2013). The developmental psychopathology of irritability. *Development and Psychopathology*, 25, 1473–1487. [PubMed: 24342851]
- Lemery-Chalfant K, Oro V, Rea-Sandin G, Miadich SA, Lecarie E, Clifford S, Doane LD, & Davis MC (2019). Arizona Twin Project: Specificity in risk and resilience for developmental psychopathology and health. *Twin Research and Human Genetics*, 22, 681–685. [PubMed: 31868161]

- Lubke GH, Ouwers KG, de Moor MHM, Trull TJ, & Boomsma DI (2015). Population heterogeneity of trait anger and differential associations of trait anger facets with borderline personality features, neuroticism, depression, Attention Deficit Hyperactivity Disorder (ADHD), and alcohol problems. *Psychiatry Research*, 230, 553–560. [PubMed: 26454404]
- Martel MM, Levinson CA, Lee CA, & Smith TE (2017). Impulsivity symptoms as core to the developmental externalizing spectrum. *Journal of Abnormal Child Psychology*, 45, 83–90. [PubMed: 27017822]
- Miadich SA, Doane LD, Davis MC, & Lemery-Chalfant K (2019). Early parental positive personality and stress: Longitudinal associations with children's sleep. *British Journal of Health Psychology*, 24, 629–650. [PubMed: 31004419]
- Moffitt TE, Arseneault L, Belsky D, Dickson N, Robert J, Harrington H, Houts R, Poulton R, Roberts BW, Ross S, Sears MR, Thomson WM, & Caspi A (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences of the United States of America*, 108, 2693–2698. [PubMed: 21262822]
- NationalSleepFoundation. (2014). SleepinAmericapoll: Sleepin the modern family. Available from <http://www.sleepfoundation.org/sleep-polls-data/sleep-in-america-poll/2014-sleep-in-the-modern-family>. Accessed October 10, 2019.
- Neale MC, Hunter MD, Pritkin J, Zahery M, Brick TR, Kirkpatrick RM, Estabrook R, Bates TC, Maes HH, & Boker SM (2016). OpenMx 2.0: Extended structural equation and statistical modeling. *Psychometrika*, 81, 535–549. [PubMed: 25622929]
- Nigg JT (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 47, 395–422. [PubMed: 16492265]
- Paavonen EJ, Räikkönen K, Lahti J, Komsu H, Heinonen K, Pesonen AK, Järvenpää AL, Strandberg T, Kajantie E, & Porkka-Heiskanen T (2009). Short sleep duration and behavioral symptoms of attention-deficit/hyperactivity disorder in healthy 7-to 8-year-old children. *Pediatrics*, 123, e857–e864. [PubMed: 19403479]
- Posthuma D, & Boomsma DI (2000). A note on the statistical power in extended twin studies. *Behavior Genetics*, 30, 147–158. [PubMed: 10979605]
- Rothbart MK, Ahadi SA, Hershey KL, & Fisher P (2001). Investigations of temperament at three to seven years: The children's behavior questionnaire. *Child Development*, 72, 1394–1408. [PubMed: 11699677]
- Rothbart M, & Bates J (2006). Temperament. In Eisenberg N, Damon W, & Lerner RM (Eds.), *Handbook of child psychology*, 6th edn, (pp. 99–166). Hoboken, NJ: John Wiley & Sons, Inc.
- Sadeh A, Sharkey KM, & Carskadon MA (1994). Activity-based sleep-wake identification: An empirical test of methodological issues. *Sleep*, 17, 201–207. [PubMed: 7939118]
- Simonds J, Kieras JE, Rueda MR, & Rothbart MK (2007). Effortful control, executive attention, and emotional regulation in 7–10-year-old children. *Cognitive Development*, 22, 474–488.
- Sletten TL, Rajaratnam SM, Wright MJ, Zhu G, Nai-smith S, Martin NG, & Hickie I (2013). Genetic and environmental contributions to sleep-wake behavior in 12-year-old twins. *Sleep*, 36, 1715–1722. [PubMed: 24179306]
- Van Dyk TR, Zhang N, Catlin PA, Cornist K, McAlister S, Whitacre C, & Beebe DW (2017). Feasibility and emotional impact of experimentally extending sleep in short-sleeping adolescents. *Sleep*, 40. 10.1093/sleep/zsx123
- Vierikko E, Pulkkinen L, Kaprio J, & Rose RJ (2004). Genetic and environmental influences on the relationship between aggression and hyperactivity-impulsivity as rated by teachers and parents. *Twin Research*, 7, 261–274. [PubMed: 15193171]
- Wheaton AG, Jones SE, Cooper AC, & Croft JB (2018). Short sleep duration among middle school and high school students — United States, 2015. *Morbidity and Mortality Weekly Report*, 67, 85–90. [PubMed: 29370154]
- Williams KE, Berthelsen D, Walker S, & Nicholson JM (2017). A developmental cascade model of behavioral sleep problems and emotional and attentional self-regulation across early childhood. *Behavioral Sleep Medicine*, 15, 1–21. [PubMed: 26619760]

Zeman J, Shipman K, & Suveg C (2002). Anger and sadness regulation: Predictions to internalizing and externalizing symptoms in children. *Journal of Clinical Child and Adolescent Psychology*, 31, 393–398. [PubMed: 12149977]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Key points

- Many children get inadequate sleep, and sleep deficiencies and problems are associated with psychopathology.
- Impulsivity and anger/frustration are core features of the development of psychopathology.
- Sleep duration and efficiency were negatively associated with impulsivity and anger/frustration.
- Associations between the sleep parameters and impulsivity and anger/frustration were driven by shared genetic influences.
- Findings suggest a genetic commonality and the need to focus on shared and unique risk factors when understanding etiology.

Figure 1.

(A–D) The full bivariate Cholesky decompositions for sleep duration and efficiency with impulsivity and duration and efficiency with anger/frustration (paths that could be dropped without a significant worsening of fit are dashed). See Table S2 for all estimates of the full and best-fitting reduced models. Standardized variance estimates are presented in the figures. Lower bounds were set to zero, and several confidence intervals were approximating close to this bound in the full models and should be interpreted with caution. A = additive genetic influences, C = common/shared environmental influences, E = unique environmental

influences. The subscript $_{11}$ denotes the first phenotype (sleep parameter) and $_{22}$ denotes the second phenotype (anger/frustration)

Table 1

Fit statistics and parameter estimates for temperament univariate ACE models

	Model	-2LL	df	AIC	-2LL	df	p	A	C	E
	ACE	1057.81	567	-76.19				.66 (.40,.92)	.09 (-.15,.31)	.25 (.18,.35)
Impulsivity	AE	1058.44	568	-77.56	0.63	1	.43	.76 (.62,.82)	-	.24 (.18,.33)
	CE	1076.71	568	-59.29	18.90	1	<.001	-	.51 (.42,.59)	.49 (.41,.58)
	E	1162.65	569	24.66	104.85	2	<.001	-	-	1.00 (1.00,1.00)
Anger/Frustration	ACE	1151.95	567	17.95				.25 (.07,.42)	.56 (.40,.69)	.19 (.14,.27)
	AE	1186.17	568	50.17	34.22	1	<.001	.83 (.77,.87)	-	.17 (.13,.23)
	CE	1158.65	568	22.65	6.70	1	.01	-	.72 (.65,.77)	.28 (.23,.35)
	E	1362.09	569	224.09	210.13	2	<.001	-	-	1.00 (1.00,1.00)

The best-fitting model is bolded for each temperament factor. *p*-Values are in reference to the chi-square test for the change in -2LL from the full model to the submodel.

Table 2

Fit statistics for bivariate Cholesky decompositions

	Model	-2LL	df	AIC	-2LL	df	p
Sleep Duration and Impulsivity	ACE-ACE-ACE	2,054.90	1,095	-135.10			
	ACE-AE-ACE	2,055.14	1,096	-136.86	0.24	1	.62
	AE-AE-AE	2,056.27	1,098	-139.73	1.37	3	.71
	CE-CE-CE	2,091.77	1,098	-104.23	36.88	3	<.001
	AE-A-AE	2,056.27	1,099	-141.73	1.37	4	.85
Sleep Efficiency and Impulsivity	AE-E-AE	2,067.28	1,099	-130.72	12.39	4	.01
	E-E-E	2,252.90	1,101	50.90	198.00	6	<.001
	ACE-ACE-ACE	1,814.22	1,095	-375.78			
	ACE-AE-ACE	1,815.02	1,096	-376.98	0.80	1	.37
	AE-AE-AE	1,818.75	1,098	-377.25	4.53	3	.21
Sleep Duration and Anger/Frustration	CE-CE-CE	1,840.86	1,098	-355.14	26.64	3	<.001
	AE-A-AE	1,818.75	1,099	-379.25	4.53	4	.34
	AE-E-AE	1,827.35	1,099	-370.65	13.13	4	.01
	E-E-E	2,014.29	1,101	-187.71	200.07	6	<.001
	ACE-ACE-ACE	2,148.40	1,095	-41.60			
Sleep Efficiency and Anger/Frustration	ACE-AE-ACE	2,148.93	1,096	-43.07	0.53	1	.47
	AE-AE-ACE	2,149.44	1,097	-44.56	1.04	2	.59
	AE-A-AE	2,149.44	1,098	-46.56	1.04	3	.79
	AE-AE-AE	2,183.55	1,098	-12.45	35.15	3	<.001
	CE-CE-CE	2,173.32	1,098	-22.68	24.92	3	<.001
Sleep Duration and Anger/Frustration	AE-A-AE	2,183.55	1,099	-14.45	35.15	4	<.001
	AE-E-AE	2,195.01	1,099	-2.99	46.62	4	<.001
	E-E-E	2,451.20	1,101	249.20	302.81	6	<.001
	ACE-ACE-ACE	1,911.29	1,095	-278.71			
	ACE-AE-ACE	1,911.34	1,096	-280.66	0.05	1	.82
Sleep Efficiency and Anger/Frustration	AE-AE-ACE	1,915.07	1,097	-278.93	3.78	2	.15
	AE-A-AE	1,915.07	1,098	-280.93	3.78	3	.29
	AE-AE-AE	1,950.03	1,098	-245.97	38.75	3	<.001

Model	-2LL	df	AIC	-2LL	df	p
CE-CE-CE	1,927.19	1,098	-268.81	15.90	3	.001
AE-A-AE	1,950.03	1,099	-247.97	38.75	4	<.001
AE-E-AE	1,955.08	1,099	-242.92	43.79	4	<.001
E-E-E	2,217.28	1,101	15.28	305.99	6	<.001

The best-fitting model is bolded for each model. *p*-Values are in reference to the chi-square test for the change in -2LL from the full model to the submodel. For the impulsivity models, when C was dropped from both phenotypes, the model did not fit worse; therefore, we did not test dropping the C path from each phenotype separately.