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Alice M. Gregory, Jessica C. Agnew-Blais, Timothy Matthews, Terrie E. Moffitt & Louise Arseneault

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ADHD and Sleep Quality: Longitudinal Analyses From Childhood to Early Adulthood in a Twin Cohort

Alice M. Gregory

Department of Psychology, Goldsmiths, University of London

Jessica C. Agnew-Blais and Timothy Matthews

MRC Social, Genetic, and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College London

Terrie E. Moffitt

*MRC Social, Genetic, and Developmental Psychiatry Centre, King's College London,
Department of Psychology & Neuroscience, Duke University and
Department of Psychiatry & Behavioral Sciences, Duke University Medical Center*

Louise Arseneault

MRC Social, Genetic, and Developmental Psychiatry Centre, King's College London

Attention-deficit/hyperactivity disorder (ADHD) is associated with poor sleep quality, but there is more to learn about the longitudinal association and aetiology of this association. We investigated the following: (a) Is there an association between childhood ADHD and poor sleep quality in young adulthood? (b) Is this driven by the long-term effects of childhood ADHD or concurrent associations with ADHD in young adulthood? (c) To what extent do genetic and environmental influences explain the overlap between symptoms of ADHD and poor sleep quality? Participants were from the Environmental Risk Longitudinal Twin Study of 2,232 twin children born in the United Kingdom in 1994–1995. We ascertained ADHD diagnoses at ages 5, 7, 10, 12, and 18. We assessed sleep quality using the Pittsburgh Sleep Quality Index at age 18. We used regression models to examine longitudinal associations and bivariate twin modelling to test genetic and environmental influences. Children with ADHD had poorer sleep quality in young adulthood, but only if their ADHD persisted. Adults with ADHD had more sleep problems than those without ADHD, over and above psychiatric comorbidity and maternal insomnia. ADHD and sleep problems in young adulthood were associated because of genetic (55%) and nonshared environmental influences (45%). Should ADHD remit, children with ADHD do not appear to have an increased risk of later sleep problems. Good quality sleep is important for multiple areas of functioning, and a better understanding of why adults with ADHD have poorer sleep quality will further the goal of improving treatments.

INTRODUCTION

ADHD and Sleep Quality

Children with attention-deficit/hyperactivity disorder (ADHD) experience poorer sleep quality compared to those without this disorder (Sung, Hiscock, Sciberras, & Efron, 2008; Yoon, Jain, & Shapiro, 2012). Parents of children with ADHD can find sleep problems a particularly challenging feature of looking

Correspondence should be addressed to Louise Arseneault, MRC Social, Genetic, and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, SE5 8Af, UK. E-mail: louise.arseneault@kcl.ac.uk

Alice M. Gregory and Jessica C. Agnew-Blais contributed equally and should be considered joint first authors.

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after their offspring (Cortese, 2015), and sleep problems in children with ADHD may be associated with poorer family functioning (Sung et al., 2008). Although historically sleep disturbances have often been overlooked and considered an unimportant secondary consequence of mental and physical health problems, this view is now outdated. Sleep is important, and poor sleep quality assessed at certain stages over the life course has been associated with multiple areas of psychological functioning including psychiatric disorders (Gregory & Sadeh, 2016) and poor academic performance (Curcio, Ferrara, & DeGennaro, 2006; Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010). Atypical sleep length or poor sleep quality has also been associated with markers of physical health problems including obesity (e.g. Patel & Hu, 2008), metabolic syndrome (Jennings, Muldoon, Hall, Buysse, & Manuck, 2007), and even mortality (Cappuccio, D'Elia, Strazzullo, & Miller, 2010).

Although concurrent associations between childhood ADHD and sleep disturbances are well-established (Yoon et al., 2012), less is known about the longitudinal associations between these problems (Cortese, 2015). What little longitudinal research there is has proved informative (Gregory, Van den Ende, Willis, & Verhulst, 2008; Hansen, Skirbekk, Oerbeck, Wentzel-Larsen, & Kristensen, 2013; Scott et al., 2013; Touchette et al., 2007). For example, one study demonstrated that ADHD severity predicted persistent sleep problems in children (Lycett, Mensah, Hiscock, & Sciberras, 2014). Another revealed that severe sleep problems in infancy predicted ADHD at 5 years of age (Thunstrom, 2002). Our own work in this area showed bidirectional associations between ADHD symptoms and sleep disturbance at age 4 and preadolescence (Gregory & O'Connor, 2002). In another study, we found that adults 38 years of age with insomnia did not appear to have a developmental history of ADHD but were instead more likely to have suffered from anxiety and depression during their youth (Goldman-Mellor et al., 2014). Results focusing on different developmental periods produce mixed results, and there is scarce evidence of the longitudinal association between sleep quality and ADHD from childhood to adulthood, perhaps partially because ADHD has been extensively believed to be a childhood disorder.

Given the great significance of sleep quality for functioning in multiple domains in adulthood, one important question in need of further investigation is whether children with ADHD are likely to have poor sleep quality later in life. On one hand, we note that in many cases ADHD does not persist over time (Agnew-Blais et al., 2016; Faraone, Biederman, & Mick, 2006; Moffitt et al., 2015), and it is possible that once ADHD has remitted, sleep disturbances do as well. On the other hand, it is possible that sleep disturbances develop, or persist, independent of the course of ADHD. For example, children with sleep disturbances may develop negative associations with their bedrooms and bedtime that result in long-term sleep problems, regardless of change in ADHD status. Studies to date have not fully addressed this question due to typically short follow-up periods, and there have been calls to

further examine associations between ADHD and sleep across developmental stages (Cortese, 2015). Although concurrent associations between ADHD and sleep quality have been reported in both children and adults (Yoon et al., 2012), the adult period is relatively underresearched and the question of whether children with ADHD have sleep disturbances in young adulthood has not been thoroughly tested.

Mechanisms Underlying Associations Between Sleep Quality and ADHD

Explanations for the associations between sleep quality and ADHD are plentiful (for a general discussion of comorbidity, see Angold, Costello, & Erkanli, 1999). For example, it is possible that ADHD could lead to poor sleep quality; indeed, stimulant medication prescribed for ADHD can lead to sleep difficulties (Cortese et al., 2013). Another explanation focuses on common pathways influencing both ADHD and sleep quality, including common neurobiological pathways involved in arousal (Yoon et al., 2012). Although shared genetic mechanisms between ADHD and sleep have been proposed, there is relatively little behavioural genetic research exploring this association (for a review of twin studies focusing on sleep and associated difficulties, see Barclay & Gregory, 2013). What little research there is has focused on childhood, with one study suggesting that early links between sleep disturbance and ADHD symptoms could be largely due to environmental influences (Van den Oord, Boomsma, & Verhulst, 2000). Another study suggested that a functional polymorphism of the catechol-O-methyltransferase gene may be involved in risk of poor sleep continuity (e.g., reduced sleep efficiency) in those with ADHD (Gruber et al., 2006).

Research Questions

The aim of this study was to examine the longitudinal association between ADHD and sleep quality and to explore the genetic and environmental underpinnings of this association. Specifically, we tested (a) whether childhood ADHD is associated with poor sleep quality in young adulthood, (b) if the association between childhood ADHD and poorer sleep quality at age 18 is explained by ADHD in adulthood, and (c) the extent to which genetic and environmental influences explain the overlap between ADHD and sleep quality.

METHODS

Study Cohort

Participants were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which tracks the development of a birth cohort of 2,232 British children. The sample was drawn from a larger birth register of twins born in England and Wales in 1994–1995 (Trouton, Spinath, &

Plomin, 2002). The E-Risk sample was constructed in 1999–2000, when 1,116 families (93% of those eligible) with same-sex 5-year-old twins participated in home-visit assessments. This sample comprised 55% monozygotic and 45% dizygotic twin pairs; sex was evenly distributed within zygosity (49% male). Families were recruited to represent the UK population with newborns in the 1990s, on the basis of residential location throughout England and Wales and mother's age. Teenage mothers with twins were overselected to replace high-risk families who were selectively lost to the register through nonresponse. Older mothers having twins via assisted reproduction were underselected to avoid an excess of well-educated older mothers. Full details about the sample are reported elsewhere (Moffitt & Erisk Study Team, 2002).

Follow-up home visits were conducted when the children were ages 7 (98% participation), 10 (96%), 12 (96%), and 18 years (93%). The families were contacted by phone to arrange visits and received vouchers to acknowledge the time they spent for the interview. With parents' permission, questionnaires were mailed to the children's teachers, who returned questionnaires for 94% of children at age 5, 93% of those followed up at age 7, 90% at age 10, and 83% at age 12. We interviewed 2,066 participants when they were age 18. There were no differences between those who did and did not take part at age 18 in socioeconomic status when the cohort was initially defined ($\chi^2 = .86, p = .65$), or age-5 IQ ($t = .98, p = .33$), internalizing or externalizing problems ($t = .40, p = .69$ and $t = .41, p = .68$, respectively). Home visits at ages 5, 7, 10, and 12 years included assessments with participants and their mothers; home visits at age 18 included interviews only with participants. We asked participants to identify individuals who know them well to act as coinformants; 99.3% had coinformant data. The Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee approved each phase of the study. Parents gave informed consent and twins gave assent between 5 and 12 years and then informed consent at age 18.

At follow-up, the study sample represented the full range of socioeconomic conditions in the United Kingdom, as reflected in the families' distribution on a neighborhood-level socioeconomic index (ACORN [A Classification of Residential Neighbourhoods], developed by CACI Inc. for commercial use in Great Britain; Odgers, Caspi, Russell, et al., 2012). ACORN uses census and other survey-based geodemographic discriminators to classify enumeration districts (~150 households) into socioeconomic groups ranging from "wealthy achievers" (Category 1) with high incomes, large single-family houses, and access to many amenities, to "hard-pressed" neighborhoods (Category 5) dominated by government-subsidized housing estates, low incomes, high unemployment, and single parents. ACORN classifications were geocoded to match the location of each E-Risk Study family's home (Odgers, Caspi, Bates, Sampson, & Moffitt,

2012). E-Risk families' ACORN distribution closely matches that of households nationwide: 25.6% of E-Risk families live in "wealthy achiever" neighborhoods compared to 25.3% nationwide, 5.3% versus 11.6% live in "urban prosperity" neighborhoods, 29.6% versus 26.9% live in "comfortably off" neighborhoods, 13.4% versus 13.9% live in "moderate means" neighborhoods, and 26.1% versus 20.7% live in "hard-pressed" neighborhoods. E-Risk underrepresents "urban prosperity" neighborhoods because such households are likely to be childless. The majority of E-Risk Study participants consider themselves White (90.4%).

ADHD

We ascertained ADHD diagnosis in childhood on the basis of mother and teacher reports of 18 symptoms of inattention and hyperactivity-impulsivity according to the criteria outlined in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV]; American Psychiatric Association, 1994; Caspi et al., 2008; Kuntsi et al., 2004; Polanczyk et al., 2010). We considered participants to have a diagnosis of childhood ADHD if they met criteria at age 5, 7, 10, or 12. Participants who had ADHD information on at least two childhood assessments and did not meet diagnostic criteria at any available assessment were classified as not having childhood ADHD. In total, 247 participants (12.1%) met criteria for ADHD in childhood.

We ascertained ADHD diagnosis at age 18 based on private structured interviews with participants regarding 18 symptoms of inattention and hyperactivity-impulsivity according to DSM-5 criteria (American Psychiatric Association, 2013). Symptoms were reported for the preceding 12 months. Participants had to endorse five or more inattentive and/or five or more hyperactivity-impulsivity symptoms to be diagnosed. We also required that symptoms interfered with individual's "life at home, or with family and friends" and "life at school or work" as rated 3 or higher on a scale from 1 (*mild interference*) to 5 (*severe*), thereby meeting criteria for impairment and pervasiveness. The DSM-5 requirement of symptom onset prior to age 12 was met if parents or teachers reported more than 2 ADHD symptoms at ages 5, 7, 10, or 12. We identified 166 (8.1 %) participants with ADHD at age 18. We also collected information from coinformants (most frequently from participants' mothers and co-twins) who rated participants on 8 ADHD symptoms at age 18.

Combining information from childhood and adult ADHD, we identified three mutually exclusive groups (Agnew-Blais et al., 2016): persistent ADHD (ADHD in childhood and at age 18, $n = 54$), remitted ADHD (ADHD in childhood, but not at age 18, $n = 193$), and late-onset ADHD (ADHD at age 18 only, $n = 112$). A total of 1,681 (82.4%) participants did not meet criteria for ADHD in childhood or adulthood.

Sleep Quality

At 18 years of age, sleep disturbance over the past month was assessed using the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupger, 1989), which is a questionnaire measure containing 18 items. Items include both open-ended questions (e.g., “During the past month, when have you usually gone to bed at night?”) and fixed-choice questions (“During the past month, how would you rate your sleep quality overall? ‘Very good; Fairly good; Fairly bad or Very bad?’”). Questions tap a range of aspects of sleep quality and can be used to derive seven component scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction), as well as a global score. Higher scores on this measure reflect poorer sleep quality. The PSQI is reported to have internal consistency and test–retest reliability in the .8 range (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Buysse et al., 1989; Carpenter & Andrykowski, 1998). In the current sample, the mean was 5.39 ($SD = 3.18$), and the Cronbach’s alpha was .69. A substantial proportion (39.9%) of the participants scored higher than 5 on the PSQI, which has been proposed as a clinical cutoff (Buysse et al., 1989). The PSQI score correlates highly with other measures of sleep such as sleep diary data (Backhaus et al., 2002).

Covariates

At age 12, participants’ mothers were asked about insomnia. Mothers reported on their symptoms of insomnia in a private standardized interview. A diagnosis of insomnia was derived based largely on the criteria outlined by the *DSM-IV* (American Psychiatric Association, 1994). Specifically, mothers were asked if they themselves experienced difficulty falling asleep, difficulty staying asleep, or problems waking too early. Answers were provided on a 5-point scale: 0 (*none*), 1 (*mild*), 2 (*moderate*), 3 (*severe*), and 4 (*very severe*). Mothers were also asked, “How much do sleep problems interfere with your daily functioning?” from 1 (*not at all*) to 5 (*very much*). If mothers reported a sleep difficulty that they considered to be “severe” or “very severe” and reported an interference score at or greater than 3, they were considered to have insomnia. Based on these criteria, 9.6% of mothers reported themselves to have insomnia.

Participants were evaluated at the age-18 interview for the presence of depressive disorder, generalized anxiety disorder, alcohol dependence, cannabis dependence, and conduct disorder (at least moderate) over the previous 12 months according to *DSM-IV* criteria (American Psychiatric Association, 1994). For the generalized anxiety disorder diagnosis, we did not require the 6-month symptom duration criterion because of the youth of our study sample. Assessments were conducted in face-to-face interviews using the Diagnostic Interview Schedule (Robins, Cottler,

Bucholz, & Compton, 1995). The assessment of conduct disorder was conducted as part of a computer-assisted module. In addition, at age 18, participants were asked whether they had young children ($N = 41$) and were queried about ADHD medication use ($N = 13$).

Statistical Analyses

To determine whether childhood ADHD was associated with poorer sleep quality at age 18, we used linear regression with childhood ADHD diagnosis predicting age-18 PSQI total score, adjusting for the participant’s sex and childhood social class. To test whether the association between childhood ADHD and age-18 sleep quality was accounted for by young adult ADHD, we additionally adjusted for age-18 ADHD diagnosis in the prior model. To further illustrate these findings, we compared sleep quality among individuals with no ADHD, persistent, remitted, and late-onset ADHD at age 18. To investigate whether psychiatric comorbidities at age 18 explain the association between ADHD and poorer sleep quality, we additionally adjusted for age-18 mental health problems. Furthermore, we controlled for potential genetic confounding by adjusting for maternal insomnia. Finally, we performed sensitivity analyses excluding participants currently taking ADHD medication and those with young children, and conducted analyses using co-informant ratings of ADHD symptoms at age 18 in place of self-report. We did not find significant interactions between sex and either childhood or young adult ADHD in predicting sleep quality at age 18; therefore we present findings across both genders. Analyses were conducted in STATA (StataCorp, 2009) and accounted for nonindependence of the clustered twin observations using the Huber/White variance estimator (Williams, 2000).

We used twin modelling to examine the nature of the association between ADHD and sleep quality (Rijsdijk & Sham, 2002). Twin studies compare the similarity of MZ twin pairs to the similarity of DZ twin pairs to estimate genetic and environmental influences on traits. Because MZ twins share 100% of their genes and DZ twins share on average half of their segregating genes, the difference in the MZ versus DZ twin correlation is used to estimate the relative contribution of additive genetic influences (A; where alleles “add up” to influence behaviour), shared environmental influences (C; environmental influences that act to make individuals within a family alike), and nonshared environmental influences (E; environmental influences that act to make individuals within a family different; this source of variance also includes measurement error). If the correlation between MZ pairs is greater than that of DZ pairs, genetic influences are playing a role. DZ twin correlations greater than half the size of the MZ correlations suggest that shared environment is important, whereas MZ correlations below 1 indicate the influence of nonshared environment (see Plomin, DeFries, Knopik, & Neiderhiser, 2013).

Model-fitting was conducted in the program OpenMx, a widely used programme for analysing behavioural genetic

data (Boker et al., 2011). For our twin analyses, we focused on a *symptom* count for both variables. This is considered to be the optimal method when running twin analyses in order to maximise power and for computational simplicity. A Cholesky decomposition was fitted to estimate the effects of A, C, and E on ADHD symptoms and sleep quality. This was interpreted as a correlated factors solution (Loehlin, 1996), which allows the genetic and environmental influences on these phenotypes to correlate. The fit of more parsimonious submodels can be tested by dropping parameters and comparing the fit statistic of the submodel, provided as minus twice the log-likelihood ($-2LL$), against that of the full ACE model. We tested a model containing genetic and nonshared environmental influences only (an AE model) to determine whether we could drop the C parameters without a significant deterioration in fit. Information about the precision of parameter estimates (and their explained variance) was obtained by likelihood-based 95% confidence intervals (CIs).

RESULTS

Longitudinal Associations Between ADHD and Sleep Quality

Individuals with a diagnosis of ADHD in childhood had poorer sleep quality in young adulthood ($\beta = .07$, 95% CIs

[.02, .12], $p = .006$), (Table 1). After adjusting for ADHD diagnosis in adulthood, we found that childhood ADHD was no longer associated with age-18 sleep quality ($\beta = .03$, 95% CIs [-.01, .08], $p = .143$), whereas young adult ADHD was significantly associated with poorer sleep quality ($\beta = .19$, 95% CIs [.14, .25], $p < .001$). This indicates that the persistence of ADHD explains the association between childhood ADHD and later sleep quality: Children with ADHD have poor quality sleep when they reach young adulthood only if they have persistent ADHD. This finding is illustrated in Figure 1, which compares sleep quality among individuals with no ADHD, persistent, remitted, and late-onset ADHD. Compared to the group without ADHD, the persistent and late-onset groups had poorer sleep quality in adulthood; however, among those with remitted ADHD, sleep quality was not significantly different from individuals who never had ADHD.

The association between young adult ADHD and poor sleep quality remained after simultaneous adjustment for a wide range of comorbid mental health problems (Table 1). This is particularly noteworthy, as each of the mental health problems examined, with the exception of cannabis dependence, was associated with poor sleep quality (Table 1). Furthermore, we found the association remained when adjusting for maternal insomnia (Table 1). To explore the association between young adult ADHD and sleep quality further, we distinguished hyperactive/impulsive and inattentive symptoms to test whether sleep quality was associated with a

TABLE 1
Results of Regression Analyses Predicting Sleep Quality

	<i>Sleep Quality</i>			
	<i>Model 1</i> (<i>Childhood ADHD</i>)	<i>Model 2</i> (+ <i>Adult ADHD</i>)	<i>Model 3</i> (+ <i>Psychiatric Comorbidity</i>)	<i>Model 4</i> (+ <i>Maternal Insomnia</i>)
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
Childhood ADHD Diagnosis	0.07** [0.02, 0.12]	0.03 [-0.01, 0.08]	0.01 [-0.03, 0.05]	0.01 [-0.03, 0.06]
Young Adult ADHD Diagnosis		0.19*** [0.14, 0.25]	0.12*** [0.06, 0.17]	0.12*** [0.06, 0.17]
Depression			0.23*** [0.18, 0.28]	0.22*** [0.17, 0.27]
Generalized Anxiety Disorder			0.10*** [0.05, 0.15]	0.11*** [0.06, 0.16]
Cannabis Dependence			0.04 [†] [-0.01, 0.09]	0.04 [†] [-0.01, 0.09]
Alcohol Abuse			0.06** [0.02, 0.10]	0.06** [0.02, 0.10]
Conduct Disorder			0.10*** [0.05, 0.15]	0.10*** [0.05, 0.16]
Maternal Insomnia				0.07** [0.03, 0.12]

Note: All models are adjusted for sex and social class; each regression model builds upon the last, adding additional variables. Childhood attention-deficit/hyperactivity disorder (ADHD) was assessed at ages 5, 7, 10, and 12 years. Sleep quality: higher score = poorer sleep. Adulthood ADHD and psychiatric comorbidity were assessed at 18 years of age. CI = confidence interval.

[†] $p < .10$. ** $p < .01$. *** $p < .001$.

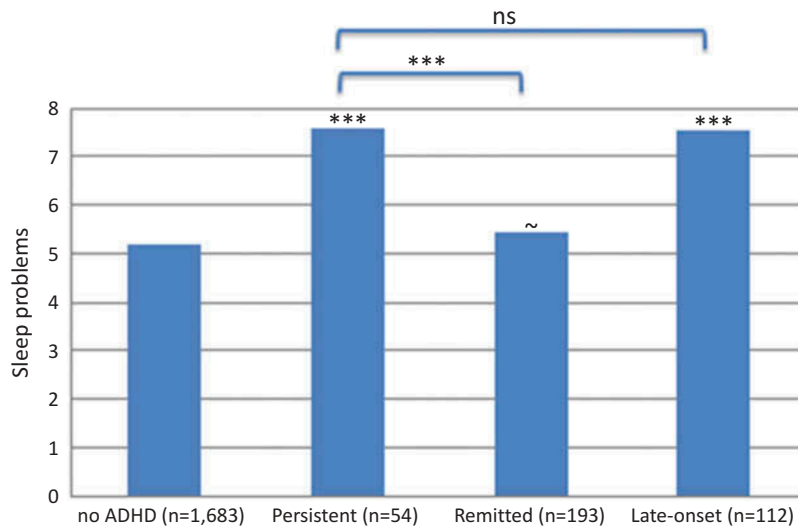


FIGURE 1 Sleep quality score among individuals without attention-deficit/hyperactivity disorder (ADHD) and with persistent, remitted, and late-onset ADHD. *Note.* Asterisks directly above the bars indicate the significance of tests comparing each ADHD group to individuals with no ADHD; asterisks above the brackets indicate significance of statistical comparisons between individuals with persistent and late-onset ADHD, and persistent and remitted ADHD. Sleep problems (higher score = poorer sleep quality). $\sim p < 0.10$; $***p < 0.001$.

specific type of ADHD symptoms. We found that both types of symptoms were independently associated (controlling for each other) with poorer sleep quality: hyperactive/impulsive ($\beta = .13$, 95% CIs [.07, .19], $p < .001$; inattentive ($\beta = .20$, 95% CIs [.14, .25], $p < .001$). Associations between young adult ADHD and sleep quality held in further analyses excluding participants taking ADHD medication at age 18 ($\beta = .12$, 95% CIs [.06, .17], $p < .001$), or who had young children by age 18 ($\beta = .12$, 95% CIs [.07, .17], $p < .001$). We also found that coinformant-reported ADHD symptoms at age 18 were significantly associated with PSQI score ($\beta = .16$, 95% CIs [.11, .22], $p < .001$) and that after including coinformant-rated ADHD symptoms in regression models, childhood ADHD diagnosis was again no longer significantly associated with PSQI score. This indicates that findings regarding ADHD at age 18 are not biased by participants reporting on their own symptoms.

Genetic and Environmental Influences on the Association Between ADHD and Sleep Quality

Given that the association between ADHD and poor sleep quality appeared to be driven by adulthood ADHD, we focused on the adulthood period exclusively in the behavioural genetic analyses. Twin correlations are presented in Table 2. The univariate correlations (e.g., correlation between ADHD symptoms for Twin 1 and Twin 2) for both ADHD and sleep quality are higher for MZ twins than DZ twins, suggesting genetic influence. For both ADHD symptoms and sleep quality, the MZ twin correlation was much less than unity, suggesting a substantial role for nonshared environment and/or measurement error. The

TABLE 2
Correlations by Twin Group

	T1 ADHD	T1 Sleep	T2 ADHD	T2 Sleep
MZ twins				
T1 ADHD	1			
T1 Sleep	.27***	1		
T2 ADHD	.37***	.14**	1	
T2 Sleep	.16***	.34***	.31***	1
DZ twins				
T1 ADHD	1			
T1 Sleep	.32***	1		
T2 ADHD	.09	.12**	1	
T2 Sleep	.14**	.13**	.24***	1

Note: T1 = Twin 1; T2 = Twin 2; ADHD = attention-deficit/hyperactivity disorder symptoms (sum of inattentive and hyperactivity symptoms); sleep = sleep quality (higher score = poorer sleep quality); MZ = monozygotic twins; DZ = dizygotic twins.

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

cross-twin cross-trait correlations (e.g., ADHD symptoms in Twin 1 and sleep quality in Twin 2) were all larger for MZ twins as compared to DZ (Table 2), suggesting genetic influence on this association.

For the behavioural genetic model, the shared environment (C) component could be dropped without a significant loss of fit ($\Delta -2LL = .02$; $\Delta df = 3$, $p = 1$) and the AE model is presented and the AE bivariate model is presented in Figure 2. First, we present the univariate estimates for ADHD and sleep quality from the bivariate model. Individual differences in ADHD symptoms could be explained by influences that were both genetic (.34), 95% CIs [.27, .41], and nonshared environmental (.66), 95% CIs

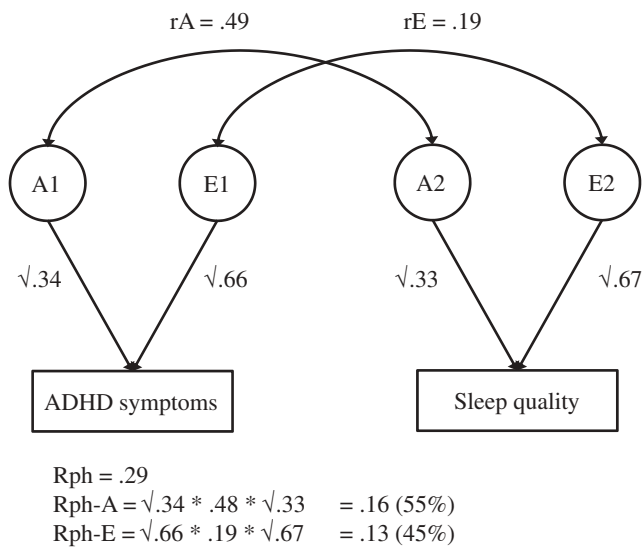


FIGURE 2 AE correlated factors model.

Note. A = genetic; E = nonshared environmental; rA = genetic correlation; rE = nonshared environmental correlation; R_{ph} = phenotypic correlation; ADHD = attention-deficit/hyperactivity symptoms (sum of inattentive and hyperactivity symptoms); sleep = sleep quality (higher score = poorer sleep quality).

[.59, .73]. Individual differences in sleep quality were also explained by genetic (.33), 95% CIs [.26, .39], and nonshared environmental (.67), 95% CIs [.61, .74] influences. Second, we observed a moderate genetic overlap between ADHD symptoms and sleep quality ($rA = .49$), 95% CIs [.35, .64]. The nonshared environmental correlation was smaller ($rE = .19$), 95% CIs [.11, .26]. The phenotypic correlation was explained roughly equally by genetic influences (55%) and nonshared environmental influences (45%).

DISCUSSION

ADHD in Childhood and Sleep Quality in Adulthood

The importance of good quality sleep for multiple areas of functioning in adulthood is increasingly evident. Therefore, understanding more about the established association with ADHD is essential. Here we report longitudinal data from early childhood to young adulthood in a twin sample representative of the general U.K. population in terms of socioeconomic status. Children with ADHD have an increased risk for experiencing poor sleep quality in early adulthood, but only if their ADHD persists. If ADHD remits over time, individuals are not more likely than those who had never been diagnosed with ADHD to have poorer sleep quality in young adulthood. Together with the finding that ADHD remits in a substantial proportion of the population, this provides the positive message that when ADHD in childhood remits, these people do not show poorer sleep than

others as they move into adulthood. These findings have clinical significance, as they can reassure parents and children with ADHD.

Childhood ADHD was not associated with later sleep problems after taking into account the persistence of ADHD symptoms in young adulthood. This suggests that *concurrent* ADHD symptoms explain the longitudinal association between childhood ADHD and young adult sleep. Indeed, it is possible that certain characteristics of ADHD such as being easily distracted by extraneous stimuli reduce good quality sleep (e.g., by delaying sleep onset). A further possibility is that ADHD medication disrupts sleep (Cortese et al., 2013). However, medication use is unlikely to explain our results, as when we excluded those taking ADHD medication from our concurrent analyses, the association was substantively unchanged.

It is also possible that disturbed sleep leads to ADHD symptoms. It is clear that those experiencing poor sleep quality may display symptoms typical of ADHD (e.g. Owens, 2005). Furthermore, previous research has shown that when certain sleep-related problems are improved (e.g., an adenotonsillectomy is performed to reduce sleep-disordered breathing), ADHD symptoms may also decrease (Sedky, Bennett, & Carvalho, 2014). An explanation for the association between *persistent* ADHD and sleep difficulties is that underlying brain vulnerability may be driving this association. Future work aimed at explaining the association between ADHD and sleep difficulties may benefit from a greater focus on the key brain centres involved in the regulation of arousal, attention, and sleep (Owens et al., 2013). Our current results are consistent with studies on childhood and adolescence emotional difficulties that showed sleep disturbances predict later difficulties, but perhaps not the reverse (Gregory & O'Connor, 2002; for a review, see Alvaro, Roberts, & Harris, 2013).

The association between ADHD and poor sleep quality during adulthood was robust controlling for many potential confounders. We observed that ADHD was independently associated with poor sleep quality, over and above several mental health problems in young adulthood. Indeed, although not a central aim of our study, it is noteworthy that except for cannabis dependence, all other adult psychiatric disorders examined in this study were associated with adult sleep quality. Furthermore, depression showed the strongest association with sleep quality. These findings are consistent with a growing body of literature showing links between sleep quality and psychopathology (and particularly internalising problems; Goldman-Mellor et al., 2014; Riemann, 2007). It is unsurprising that sleep quality is associated with internalising problems, given that insomnia or hypersomnia is listed as a symptom of major depressive disorder in the *DSM-5* (American Psychiatric Association, 2013). Furthermore, the association we observed was not accounted for by the few parents in our sample with young children. These current analyses, however, do not tell us

about the direction of the association (young adult ADHD leading to poorer sleep vs. poorer sleep leading to ADHD symptoms). We were not able to examine the possibility that sleep disturbances in childhood predicted later ADHD, and it is well-established that inadequate sleep can result in symptoms that mimic those of ADHD in children (Dahl, 1996). This argument is applicable also to adults (e.g., poor sleep in adults could result in symptoms of ADHD such as difficulty sustaining attention or remembering daily tasks).

Genetic and Environmental Influences

Our genetic estimate for ADHD is similar to those from other twin studies investigating ADHD in adulthood (Franke et al., 2012). Similarly, the genetic estimate for sleep quality reported here was similar to the ones in other samples using exactly the same measure reported by us (Gregory et al., 2011; Taylor, Gregory, Freeman, & Ronald, 2015) and by others (Genderson et al., 2013). More generally, this fits well with robust evidence that genes are important for all complex traits (Polderman et al., 2015). We observed substantial genetic overlap between ADHD and sleep quality, and overall, genes explained roughly half of the association between these variables. This is in line with the generalist genes hypothesis, which proposes that specific genes may confer a general risk but that the development of specific symptom clusters may be determined by the environment (Eley, 1997). Our finding here is unsurprising given a recent review of behavioural genetics (Plomin, DeFries, Knopik, & Neiderhiser, 2016). Indeed, the authors proposed that one of the top 10 replicated findings from the field is that correlations between traits are substantially influenced by genes. Although the estimate reported here differs from certain others focusing on younger populations (Van den Oord et al., 2000), it is important to note that heritability is a population statistic, so changes in estimates at different developmental stages are unsurprising (Plomin et al., 2013).

Our finding of substantial genetic and environmental influences on the overlap between sleep disturbances and ADHD opens the door to some hypotheses for future research. First, attention has been directed to elucidating whether sleep problems lead to mental health problems or vice versa. Our findings indicate that both sleep problems and ADHD have common origins, partly genetically influenced. Investigations into elucidating the co-occurrence of poor sleep quality and mental health problems may shift focus in the direction of influences predating both problems. Second, in the future, polygenic risk score analyses could be used to understand the genetic overlap of these two disorders. For example, it will be possible to examine whether ADHD polygenic risk score explains variation in young adult sleep problems. Such an approach would use genome-wide information to consider whether common risk alleles associated with childhood ADHD are also associated with sleep quality. This new approach may offer better chances of explaining genetic covariance for the phenotypes (Hamshere et al., 2013). As we

continue to identify genes involved in complex traits, we expect to find genetic variants associated with both ADHD and sleep disturbances. Third, our findings indicate that influences from the environment are not to be neglected for a better understanding of the association between ADHD and sleep problems in adulthood. The omnipresence of new technologies, communication devices and social media, and how people use these, may have an effect on the co-occurrence of both problems. The use of illicit substances, including alcohol and drugs, may also play a role in the development of ADHD and sleep problems in young adulthood. Indeed, it is known that ADHD is a risk for alcohol use (Molina & Pelham, 2003), and drinking alcohol can impact negatively upon sleep quality (Ebrahim, Shapiro, Williams, & Fenwick, 2013). We predict that these environmental influences may therefore help to account for some of the overlap between ADHD and sleep disturbance.

Limitations

Certain limitations must be acknowledged. First, we collected self-report of both ADHD and sleep quality, which may have artificially inflated the associations. However, we found very similar results when using co-informant rated ADHD symptoms at age 18, rather than self-report. Furthermore, the use of polysomnography has been considered the gold-standard method of assessing sleep. Although we acknowledge the clear value of polysomnography, we note that each method used to assess sleep provides a unique insight into aspects of sleep that other techniques may not capture and that assessing sleep using multiple methods provides the most comprehensive picture (Gregory & Sadeh, 2016). It is interesting that some studies in adulthood (but not all; see Philipson et al., 2005) have reported links between ADHD and objectively assessed sleep quality (e.g., Boonstra et al., 2007; Sobanski, Schredl, Kettler, & Alm, 2008).

Second, we collected information on sleep quality in young adulthood but not prior during childhood. As a consequence, we could not test the direction of the association between sleep quality and ADHD. Based on previous literature examining different time spans, it is possible that such an association would have been found (Gregory & O'Connor, 2002; Thunstrom, 2002). Relatedly, research suggests that attempts to improve sleep may have a positive impact upon ADHD symptoms within childhood (see Hiscock et al., 2015), and further research needs to extend this interesting work.

Third, we focused on sleep quality in the full range rather than sleep disorders. Given that ADHD has been associated with a whole host of sleep disorders, such as sleep-disordered breathing and periodic limb movement disorder (Yoon et al., 2012), it is possible that such disorders may have been underlying the association reported here.

Fourth, it is important to note limitations that have been levelled at the twin design. It has been proposed that twins may not be representative of the wider nontwin population. Here, however, we found that the prevalence rates of ADHD and mean PSQI scores were within the range of other

studies focusing on nontwin populations (Buysse et al., 2008; Centers for Disease Control and Prevention, 2010; Hayashino et al., 2010; Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015). Replications of our results using other genetically informative designs would be of value.

Conclusion

In conclusion, our results provide the positive message that ADHD does not predict later sleep quality over and above the persistence of ADHD into adulthood. The concurrent association between ADHD and poor sleep quality in the young adult years, which in comparison to the childhood years has received limited attention, was robust and influenced by genetic and nonshared environmental factors. Replication is now essential before the full implications of these results are clear. Taking this work further, we acknowledge that a next step is to elucidate the pathways by which shared genes and environmental factors may influence the association between ADHD and sleep disturbances with the longer term aim of reducing the likelihood of this association, which can cause such suffering and impairment.

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