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Genetic and Environmental Influences on Sleep Problems: A Study of Preadolescent and Adolescent Twins

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Abstract

Background/ Aim—The aim of this study was to examine the extent to which additive genetic, shared environmental, and non-shared environmental factors contribute to adolescent and preadolescent sleep problems.

Methods—The sample consisted of a cohort of 270 monozygotic and 246 dizygotic twins from a university-based twin registry.

Results—Results demonstrated that genetic and environmental influences each appear to be important to adolescent sleep problems.

Conclusions—While the magnitude of genetic influence on sleep problems was consistent with findings from the adult literature, it was smaller than in studies with younger children, suggesting genetic effects may be less influential in adolescence and adulthood.

Keywords

ADOLESCENCE; SLEEP; GENETICS; ENVIRONMENTAL INFLUENCES

Introduction

Adolescence is widely recognized as a period of development associated with less than optimal sleep duration and increased daytime sleepiness. Though the combination of behavioral and environmental factors, normative developmental changes, intrinsic sleep processes and/or sleep disorders have been shown to affect adolescent sleep, genetics may also be a contributing factor.

While studies on the heritability of sleep problems in adolescents are lacking, the literature provides evidence for genetic influences on sleep in preschool and school-aged children, as well as adults. For example, in 3-year-old twins, van den Oord, Verhulst, and Boomsma (1996) found that 69% of the variance in sleep problems was due to genetic factors, while 31% was due to nonshared environmental influences. Additionally, Gregory, (2008) found in a study of 8-year-old twins that the majority of the variance in parasomnias and dyssomnias was due to genetic factors (51% and 71%, respectively) with the remaining variance accounted for primarily by non-shared environment. With regard to specific sleep

disorders, Nguyen et. al (2008) found that sleep terrors at 18 months and 30 months were explained by genetic (44% and 42%, respectively) and nonshared environmental factors (56% and 58%, respectively). Finally, a recent longitudinal study by Gregory, Fruhling, Lau, Dahl, and Eley (2009) found that sleep problems remained stable from 8–10 years of age and that most of the variance could be explained by genetic and nonshared environmental influences at both 8-years (63% genetic factors, 32% nonshared environment) and at 10-years (66% genetic factors, 7% shared environment, 27% nonshared environment). In combination, the evidence suggests that genetic factors play a substantial role in the sleep problems of young children.

Similarly at least one study of adult 17–88 year-old twin pairs (Heath, Kendler, Eaves, & Martin, 1990) estimated that genetic factors accounted for 33% of the variance in sleep quality and disturbance and 40% of the variance in sleep patterns, while there was no effect of shared family environment. Additionally, this study found no decline in the magnitude of genetic contribution with age.

For other behavioral conditions, such as eating disorders and anxiety, there are developmental differences in genetic and environmental effects that provide important clues about key etiologic factors and possible points of intervention. Thus, the purpose of the current study was to use a twin sample to investigate the magnitude of genetic and environmental influences on sleep problems in adolescents.

Methods

Participants

Participants included 270 monozygotic (MZ) and 246 dizygotic (DZ) twins and their mothers from the Michigan State University Twin Registry (MSUTR; Klump & Burt, 2006). The sample ranged in age from 10–17 with a mean age of 12.68 ± 1.51 years. Most of the subjects were female (61.1%), white (86%), and had a family income of more than \$60,000 (61.8%).

Procedure

This study involved a secondary analysis of data collected via the MSUTR. Twins were initially recruited for the adolescent MSUTR using birth records. This study was approved by the Institutional Review Board at Michigan State University, and written informed consent/assent was obtained from a parent/guardian, as well as the adolescents.

Measures

Demographics—Demographic information was collected from a parent questionnaire, which included information about each twin's age, ethnicity, birth weight, and gender, as well as parent income and education.

Zygosity—Zygosity was determined using a physical similarity questionnaire, which has demonstrated 95% accuracy (Plomin, DeFries, McClearn, & McGuffin, 2008). Indeterminate zygosity was resolved by one of the project's principle investigators, who reviewed photographs of each dyad and questionnaire results.

Sleep composite—The sleep composite score was derived from the four sleep related questions on the Child Behavior Checklist (CBCL; Achenbach 1991a), which was completed by the parent, and the Youth Self Report (YSR; Achenbach 1991b), completed by the adolescent. The CBCL and YSR are widely used screening measures designed to identify child and adolescent behavior and emotional problems, and the reliability and

validity of both have been well documented. Items used in this study each indicated a sleep problem including “overtired” (item 54), “sleeps less than most kids” (item 76), “sleeps more than most kids during the day and/or night” (item 77), and “trouble sleeping” (item 100), which have been used in other studies of sleep problems in youth and have been compared to both subjective and objective measures of sleep (Gregory, Cousins, Forbes et. al, 2009). Because sleeping less than most children and sleeping more than most children are independent symptoms (similar to eating behavior on a depression inventory), no items were reverse scored. Each item was given a score of 0 (not true), 1 (somewhat or sometimes true), or 2 (very true or often true), and the higher score (parent or child) for each item was used as the score for that item. The sum of these scores comprised the composite sleep variable, which ranged from 0–8 with higher scores indicating worse outcomes. Cronbach’s alpha for the sleep composite was 0.70.

Analyses

Twin intraclass correlations were calculated to provide initial indications of genetic versus environmental (i.e., shared and nonshared) influences on sleep problems. Given that MZ twins share all of their genes, and DZ twins share, on average, only half of their segregating genes, greater MZ relative to DZ twin correlations suggest that genetic factors are important for the trait in question. By contrast, similar MZ and DZ twin correlations suggest that shared environmental factors account for associations. Finally, nonshared environmental influences are implicated if the MZ twin correlation is less than 1.00. It must be noted, however, that an MZ twin correlation of less than 1.00 indicates the presence of both nonshared environmental factors and/or measurement error.

Univariate twin models were fit to raw data using the maximum likelihood option in Mx to directly examine additive genetic (A; the effect of individual genes summed over loci), shared environmental (C; environment common to siblings that acts to make them similar to each other), and non-shared environmental (E; environmental factors [and measurement error] differentiating twins within a pair) influences on sleep problems. Initially, the unrestricted ACE model was fit to the data. This model allows variances, covariances, and means to be estimated freely by minimizing the -2 times log likelihood of data ($-2\ln L$). Next, submodels of the ACE model (i.e., AE, CE, and E) were examined to determine if a reduced model provided a better fit to the data. The fit of the various submodels were examined using a likelihood-ratio chi-square difference test ($-2\ln L\Delta$) and Akaike’s Information Criteria ($AIC = \chi^2 - 2*df$). The likelihood-ratio chi-square difference test ($2\ln L\Delta$) examines differences in $-2\ln L$ values and degrees of freedom between nested models. If the chi-square difference test is nonsignificant, then the most parsimonious (i.e., reduced) model was preferred. The AIC also evaluates model fit, and does so placing some value on parsimony. Lower AICs indicate a better fit.

Results

Twin intraclass correlations demonstrated that all three factors (e.g., genetic, shared, and nonshared environmental factors) appeared to be important for sleep problems, as the MZ correlation was high (.70, $p < .05$), the MZ correlation was somewhat higher than the DZ correlation (.60, $p < .05$) though MZ/DZ twin differences in correlations were non-significant ($z = 1.26$, $p = .10$), and the MZ correlation was less than 1.00. As indicated in Table 1, the best-fitting model for sleep problems was the ACE model. Parameter estimates indicated that sleep problems are influenced predominately by shared environmental influences (42%) with genetic factors accounting for 30% of the variance and nonshared environmental effects accounting for 28% of the variance.

Discussion

Findings from this study were that shared environmental influences made particularly strong contributions to adolescent sleep problems. The heritability of sleep problems in this study (30%) is consistent with findings from the adult literature (Heath et. al, 1990), suggesting that the degree to which genetic effects influence sleep problems from adolescence to adulthood may be relatively consistent. While longitudinal studies are clearly needed, this study in comparison to other research (Heath et. al, 1990; van den Oord et. al 1996) suggests that the impact of genetics on sleep problems may be stronger during preschool and school age (63–69%). On the other hand, the effect of shared environment seems to follow an inverse U-shaped pattern, with research demonstrating a minimal effect (0–7%) during preschool, school-age, and again during adulthood (Gregory et. al, 2009; Heath et. al 1990; van den Oord et. al 1996). Interestingly, the current study suggests that adolescence may be the period of development where shared environmental factors play a significant role. It may be that the magnitude of environmental influence is greatest during adolescence where factors such as family routines, school start times, parental limits around sleep schedules and electronic use, and academic, extracurricular, and social demands play such an overwhelming role in determining sleep patterns.

Findings from this study should be interpreted in the context of several limitations. First, our measure of sleep problems was broad, and the composite score may have limited reliability and validity. Additionally, our use of general screening measures (i.e., CBCL and YSR) may have limited our ability to detect significant effects, and this could be remedied with the use of a sleep specific measure. Finally, because our measure of sleep problems was imprecise, the associations between actual sleep and genetic and environmental factors may have been underestimated.

Conclusion

Overall, genetic influences were found to account for a small proportion of the variance in adolescent sleep problems, while shared environmental factors made a large contribution. Results provide preliminary evidence that the influence of genetic effects on sleep problems may decline with development and that shared environmental factors may be particularly important during adolescence.

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References

- Achenbach, TM. Manual for the Child Behavior Checklist/ 4–18 and 1991 Profile. Burlington, VT: University of Vermont; 1991a.
- Achenbach, TM. Manual for the Teacher's Report Form and 1991 Profile. Burlington, VT: University of Vermont; 1991b.
- Gregory A. A genetic decomposition of the association between parasomnias and dyssomnias in 8-year-old twins. *Archives of Pediatric and Adolescent Medicine*. 2008; 162:299–304.

- Gregory A, Cousins J, Forbes E, et al. Sleep items in the Child Behavior Checklist: A comparison with sleep diaries, actigraphy and polysomnography in a sample of anxious, depressed, and control youth. *Sleep*. 2009; 32:A98.
- Gregory A, Fruhling R, Lau J, Dahl R, Eley T. The direction of longitudinal associations between sleep problems and depression symptoms: A study of twins aged 8 and 10 years. *Sleep*. 2009; 32:189–199. [PubMed: 19238806]
- Heath A, Kendler K, Eaves L, Martin N. Evidence for genetic influences on sleep disturbance and sleep pattern in twins. *Sleep*. 2009; 13:318–335. [PubMed: 2267475]
- Klump K, Burt S. The Michigan State University Twin Registry (MSUTR): Genetic, environmental, and neurobiological influences on behavior across development. *Twin Research and Human Genetics*. 2006; 9:971–977. [PubMed: 17254439]
- Nguyen B, et al. Sleep terrors in children: A prospective study of twins. *Pediatrics*. 2008; 122:e1164–e1167. [PubMed: 19047218]
- Plomin, R.; DeFries, J.; McClearn, G.; McGuffin, P. *Behavioral Genetics*. 5th ed.. New York, NY: Worth Publishers; 2008.
- van den Oord E, Verhulst F, Boomsma D. A genetic study of maternal and paternal ratings of problem behaviors in 3-year-old twins. *Journal of Abnormal Psychology*. 1996; 105:349–357. [PubMed: 8772005]

Table 1
Univariate Twin Models Examining Genetic and Environmental Influences on Sleep Problems

Model	Standardized Parameter Estimates			Test Statistics		
	a^2	c^2	e^2	$-2\ln L$	df	p
ACE	.30 (.05, .58)	.42 (.16, .58)	.28 (.22, .38)	-51.33	479	--
AE	.73 (.65, .79)	--	.27 (.21, .35)	-42.53	480	8.80 (1)
CE	--	.64 (.56, .71)	.36 (.29, .44)	-42.82	480	8.51 (1)
E	--	--	1.0 (1.0, 1.0)	78.62	481	129.95 (2)
						<.001
						-883.38

Note. a^2 = additive genetic effects; c^2 = shared environmental effects; e^2 = nonshared environmental effects; $-2\ln L$ = -2 times log likelihood of data; df = degrees of freedom; AIC = Akaike's information criterion; $-2\ln L_A$ = differences in $-2\ln L$ values between the ACE fully unconstrained model and submodels. Ninety-five percent confidence intervals are in parentheses.