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The Prospective Association between Sleep Deprivation and Depression among Adolescents

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Study Objectives: To examine the prospective, reciprocal association between sleep deprivation and depression among adolescents.
Design: A community-based two-wave cohort study.
Setting: A metropolitan area with a population of over 4 million.
Participants: 4,175 youths 11-17 at baseline, and 3,134 of these followed up a year later.
Measurements: Depression is measured using both symptoms of depression and DSM-IV major depression. Sleep deprivation is defined as ≤ 6 h of sleep per night.
Results: Sleep deprivation at baseline predicted both measures of depression at follow-up, controlling for depression at baseline. Examining the reciprocal association, major depression at baseline, but not symptoms predicted sleep deprivation at follow-up.
Conclusion: These results are the first to document reciprocal effects for major depression and sleep deprivation among adolescents using prospective data. The data suggest reduced quantity of sleep increases risk for major depression, which in turn increases risk for decreased sleep.
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INTRODUCTION

Sleep deprivation, or short sleep, is sleep time less that the average basal level of about 9 hours per night for adolescents.[1] Studies indicate that many adolescents do not obtain adequate nocturnal sleep.[2-12] As many as one-fourth of adolescents report sleeping 6 hours or less per night.[12,13]

There is consensus concerning changes in the transition from childhood to adolescence that result in increased sleep deprivation in adolescence. Transition to an earlier school start time, along with pubertal phase delay, significantly affect the quality of sleep, sleep-wake schedule, and daytime behavior. The combination of the phase delay, late-night activities or jobs, and early morning school demands can significantly constrict hours available to sleep.[12,14-17] Laboratory studies using longitudinal designs have documented consistent changes in sleep/wake architecture in adolescents.[18-20] These changes become particularly pronounced from early to late puberty.[21,22]

Research has been conducted on the correlates of sleep disturbance and sleep deprivation among adolescents.[13-15,23-25] The available evidence suggests that disturbed sleep and sleep deprivation are associated with deficits in functioning across a wide range of indicators of psychological, interpersonal, and somatic well-being. For example, adolescents with disturbed sleep report more depression, anxiety, anger, inattention and conduct problems, drug and alcohol use, impaired academic performance, and suicidal thoughts and behaviors. They also have been reported to have more fatigue, less energy, worse perceived health, and symptoms such as headaches, stomach aches, and backaches. Laboratory studies in particular have documented impaired cognitive function, daytime sleepiness, and fatigue as a consequence of sleep deprivation.[14,21,22]

However, almost all of the epidemiologic data on these associations emanate from prevalence or cross-sectional surveys. Thus, the question of whether, for example, sleep deprivation increases the risk of functional impairment among adolescents, or emotional, behavioral, and interpersonal problems increase the risk of sleep deprivation, remains unclear.

Roberts et al. examined the effects of deep deprivation among adolescents and found that short sleep (≤ 6 h) increased subsequent risk for school problems, low life satisfaction, poor perceive health, depressed mood, drug use, and poor grades.[24] Other studies also have found that sleep problems, including sleep deprivation, increased the odds of subsequent mental health problems,[6,26-29] including depressed mood.

Clearly, there appears to be an association between short sleep duration and symptoms of depressed mood. Again, while much of this evidence is from cross-sectional or prevalence studies, prospective or longitudinal studies also find short sleep increases risk for disturbed mood. However, to our knowledge, there are few data relating to the converse, i.e., that depressed mood increases risk for short sleep. Roberts et al. report that depressed mood (symptoms of depression) at baseline did not increase risk of restricted sleep (≤ 6 h) a year later.[13]

But what about the association between short sleep duration and clinical depression (e.g., major depression)? There is an extensive literature on the association between insomnia and major depression, particularly among adults,[30-34] as well as some data on adolescents.[35-37] The evidence indicates that insomnia, particularly chronic insomnia, increases subsequent risk of major depression. The evidence thus far, albeit limited, suggests that major depression confers little risk for developing

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insomnia. In a previous study, we found insomnia increased risk of major depression, but the reverse was not true.[38] But, to our knowledge, no study to date has examined the prospective association between sleep deprivation and major depression among adolescents.

That was the focus of our research. We examined the prospective association between short sleep or sleep deprivation and major depression in adolescence. Using data from a two-wave cohort study of youths 11-17 years at baseline, Teen Health 2000 (TH2K), we examined the reciprocal association between short sleep and major depression, e.g., whether short sleep increases the risk for major depression, whether major depression increases the risk for short sleep, whether the association is asymmetric, or whether there is an association at all. The answers to this question have both etiologic and clinical implications. If there are reciprocal effects, then each partially accounts for the other in a causal way. Understanding the epidemiology of one requires understanding the other. On the other hand, if there is bi-directionality, clinically the presence of one suggests assessment and intervention for the other. We may need to focus on both for optimal effect.

METHODS

Sample

The sample was selected from households in the Houston metropolitan area enrolled in two local health maintenance organizations. One youth, aged 11 to 17 years, was sampled from each eligible household, oversampling for ethnic minority households. Initial recruitment was by telephone contact with parents. A brief screener was administered on ethnic status of the sample youths and to confirm data on age and sex of youths. Every household with a child 11 to 17 years of age was eligible. Because there were proportionately fewer minority subscriber households, sample weights were developed and adjusted by post-stratification to reflect the age, ethnic, and sex distribution of the 5-county Houston metropolitan area in 2000. The precision of estimates are thereby improved and sample selection bias reduced to the extent that it is related to demographic composition.[39] Thus, the weighted estimates generalize to the population 11 to 17 years of age in a metropolitan area of 4.7 million people.

Data were collected on sample youths and one adult caregiver using computer-assisted personal interviews and self-administered questionnaires. The computerized interview contained the structured psychiatric interview (see below) and demographic data on the youths and the household. Height and weight measures were conducted after the completion of the interviews. The interviews and measurements were conducted by trained lay interviewers. The interviews took on average 1 to 2 h, depending on the number of psychiatric problems present. Interviews, questionnaires, and measurements were completed with 4,175 youths at baseline, representing 66% of the eligible households. There were no significant differences among ethnic groups in completion rates. Youths and caregivers were followed up approximately 12 months later, using the same assessment battery used at baseline. The cohort consisted of 3,134 youths plus their caregivers in Wave 2 (75% of Wave 1 dyads). All youths and parents gave written informed consent prior to participation. All study forms and procedures were approved by the University of Texas Health Science Center Committee for Protection of Human Subjects.

Measures

Depression

Depression was measured using two alternate strategies. We examined major depression, defined as a major depressive episode in the previous 12 months (prevalence was 1.7%) using DSM-IV criteria and the National Institute of Mental Health (NIMH) Diagnostic Interview Schedule for Children, Version IV (DISC-IV) as the diagnostic instrument.[44] Then, given that much of the literature has focused on symptoms of depression, we examine disturbed mood in the past 12 months, defined as depressed mood, irritable mood, or anhedonia (baseline prevalence was 57.6%).

Data on psychiatric disorders were collected using the youth version of the DISC-IV, a highly structured instrument with demonstrated reliability and validity.[44] Interviews were conducted by college-educated lay interviewers who had been extensively trained using protocols provided by Columbia University.[41,42] Interviews with the DISC-IV were administered using laptop computers.

Sleep Deprivation

The DISC-IV does not inquire about symptoms of insomnia other than in the context of other DSM-IV disorders (such as mood or anxiety disorders). To supplement the DISC-IV, we inquired about symptoms of disturbed sleep, focusing primarily on symptoms of insomnia, their frequency and duration. Two questions inquired about hours of sleep on average the subject experienced on weeknights during the past 4 weeks and also on weekend nights. Six hours or less was defined as short sleep duration or deprivation,[3,23,41] following the lead of earlier studies. That is, short sleep was defined 2 ways: ≤ 6 h only on weeknights and ≤ 6 h on both weeknights and weekend nights. The sleep items were taken from a variety of validated sleep questionnaires, including SleepEVAL.[42,45]

Covariates

We include as covariates known correlates of both depression and sleep: age, gender, and family income. Family income was assessed using total household income in the past year: < $35,000, $35,000 - $64,999, and $65,000 or more. Age was assessed by age at most recent birthdate: 12 or less, 13-15, and 16 or older. Table 1 presents characteristics of the sample and cohort. As can be seen, the sample is diverse. In terms of hours of sleep, about 20% slept ≤ 6 h on weeknights. About 9% slept ≤ 6 h every night.

Analyses

First, the relationship between sleep deprivation on weeknights or both weeknights and weekends and depression (yes, no) at Wave 1 was examined, calculating crude odds ratios and then adjusted odds ratios controlling for age, gender, and family income. Second, sleep deprivation at Wave 1 was used to predict depression at Wave 2, first examining crude odds ratios and then adjusted odds ratios controlling for the same

covariates. We also controlled for depression at baseline, either major depression or symptoms, depending on the focus. We then repeated this strategy using depression at Wave 1 to predict sleep deprivation at Wave 2, controlling for sleep at Wave 1.

The estimated odds ratios and their 95% confidence limits were calculated using survey logistic regression (Proc Surveylogistic) procedures in SAS V9.1[46] and Taylor series approximation to compute the standard error of the odds ratio. Lepkowski and Bowles[47] have indicated that the difference in computing standard error between this method and other repeated replication methods such as the jackknife is very small.

RESULTS

Table 2 presents results for the association between sleep deprivation and depression at baseline. Short sleep consistently was associated with depression, both major depression and depressive symptoms, although the effect was less robust for symptoms.

In Table 3, the same prospective association was observed for sleep deprivation and both measures of depression. Adjustment for covariates affects the associations, but all were still significant ($P < 0.05$). Again, the effect is stronger for major depression than for symptoms.

When we examined the reverse prospective association in Table 4, there was increased risk for short sleep among those depressed at baseline. However, after controlling for covariates, only one association remained significant—major depression at baseline increased risk of sleep deprivation only on weeknights.

DISCUSSION

We found that sleep deprivation, defined as 6 hours or less on weeknights and every night (including weekends) and symptoms of depression covary at baseline. In this regard, our results are consistent with the literature, which largely is based on cross-sectional research. When we examined whether sleep deprivation at baseline increased risk of depressive symptoms

Table 1—Unweighted sample characteristics, Teen Health 2000 sample and cohort

Characteristics	Wave 1 N = 4,175 %	Wave 2 cohort N = 3,134 %
Gender of Youth		
Male	51.14	50.77
Female	48.86	49.23
Age of Youth		
≥ 16 years	24.91	40.36
13–15 years	48.05	48.63
≤ 12 years	27.04	11.01
Ethnicity of Youth		
European American	35.43	37.01
African American	35.35	34.59
Latino American	24.57	23.64
Other	4.65	4.75
Family Income		
≥ $65,000	35.29	40.73
$35,000–$64,999	40.71	39.16
< $35,000	24.00	20.11
Parental Marital Status		
Married	75.71	76.10
Others	24.29	23.90
Sleep Duration Weeknights, Past Month		
≤ 6 hours	19.83	24.85
7-8 hours	52.51	52.92
≥ 9 hours	27.66	22.23

Table 2—Odds ratios for the association between sleep deprivation and depression at Wave 1

	Depression at Wave 1			
	Crude OR, 95% CI		Adjusted* OR, 95% CI	
Sleep Deprivation at Wave 1	Major Depression	Depression Symptoms	Major Depression	Depression Symptoms
Short Sleep WN/WE	3.86† (2.28-6.54)	1.56† (1.31-1.85)	3.79† (2.21-6.50)	1.42† (1.17-1.71)
Short Sleep WN	2.84† (1.50-5.41)	1.69† (1.31-2.17)	2.80† (1.42-5.50)	1.55† (1.19-2.02)

Depression Symptoms = Depressed mood, anhedonia, or irritable mood. Short Sleep WN/WN = Sleep ≤ 6 h on weeknights and weekends. Short Sleep WN = Sleep ≤ 6 h on weeknights. *Adjusting for age, gender, and family income. †Odds ratios are statistically significant ($P < 0.05$).

Table 3—Odds ratios for the association between sleep deprivation at Wave 1 and depression at Wave 2

	Depression at Wave 2			
	Crude OR, 95% CI		Adjusted* OR, 95% CI	
Sleep Deprivation at Wave 1	Major Depression	Depression Symptoms	Major Depression	Depression Symptoms
Short Sleep WN/WE	4.58† (2.36-8.86)	1.39† (1.14-1.68)	3.12† (1.55-6.27)	1.25† (1.01-1.54)
Short Sleep WN	5.21† (2.48-10.93)	1.56† (1.19-2.05)	3.76† (1.65-8.58)	1.38† (1.02-1.85)

Depression Symptoms = depressed mood, anhedonia, or irritable mood. Short Sleep WN/WN = Sleep ≤ 6 h on weeknights and weekends. Short Sleep WN = Sleep ≤ 6 h on weeknights. *Adjusting for age, gender, family income, and depression (either major depression or symptoms at Wave 1. †Odds ratios are statistically significant ($P < 0.05$).

Table 4—Odds ratios for the association between depression at Wave 1 and sleep deprivation at Wave 2

	Sleep Deprivation at Wave 2			
	Crude OR, 95% CI		Adjusted* OR, 95% CI	
Depression at Wave 1	**Short Sleep WN/WE**	**Short Sleep WN**	**Short Sleep WN/WE**	**Short Sleep WN**
Major Depression	3.47† (1.91-6.32)	5.36† (2.91-9.89)	1.95 (0.94-4.04)	4.28† (2.21-8.32)
Depression Symptoms	1.34† (1.12-1.60)	1.41† (1.09-1.81)	1.14 (0.93-1.40)	1.24 (0.94-1.63)

Depression Symptoms = Depressed mood, anhedonia, or irritable mood. Short Sleep WN/WN = Sleep ≤ 6 h on weeknights and weekends. Short Sleep WN = Sleep ≤ 6 h or less on weeknights. *Adjusting for age, gender, family income, and sleep deprivation at Wave 1. †Odds ratios are statistically significant ($P < 0.05$).

at follow-up, we found a 25% to 38% increased risk. This is similar to previous studies.[23,24,27]

However, when we looked at reciprocal effects, depressive symptoms predicting sleep deprivation, we found no association. Similar results have been reported for insomnia among youths[35] as well as for depressed mood.[13] The latter paper was based on the same data used here, Teen Health 2000.

When we turned to the association between sleep deprivation and major depression, we found the two were correlated at baseline. We also found that baseline sleep deprivation increased risk for subsequent major depression, by a factor of more than 3. This association was much stronger than for depressive symptoms, as noted above. When we examined the reciprocal association, controlling for covariates, the odds were 1.95 (n.s.) for sleep deprivation every night, but 4.28 ($P < 0.05$) for sleep deprivation on weeknights. While not statistically significant, an odds of almost 2 is not trivial, but power is problematic given the low prevalence of major depression.

Limitations

As noted earlier, our sleep items asked whether subjects had experienced symptoms almost every day for the past 4 weeks. Therefore, our results are limited, in that we could not date onset and thus were not able to partition our sample into those with acute versus long-term sleep deprivation. In a recent paper, Buysse et al.[48] found differential results related to duration of insomnia among young adults. In their epidemiologic study in the United Kingdom, Ohayon et al.[49] found that the median duration of insomnia symptoms was 24 months. We could not examine whether risk-factor profiles differed for those with sleep disturbance of shorter and longer duration (> 24 months), although it might be expected that the association with somatic, psychological, and interpersonal functioning would be pronounced for chronic sleep problems of longer duration.

Another limitation is that we did not have objective data on disturbed sleep. That is, we did not have physiologic studies. While such data would be useful to have, self-reports and interview-based measures remain the measures of choice in community surveys. Our study was no exception. We should note that there are data suggesting that subjective measures of sleep from children and adolescents are modestly correlated with objective measures of disturbed sleep.[50] We should note as well that the use of laboratory measures are impractical in large field studies such as TH2K.[5,7] We should note that studies have shown that youths with MDD report subjective sleep disturbance that is not accompanied by objective indicators of disturbed sleep.[51]

We also should note that although our sleep assessment inquired about symptoms experienced in the past 4 weeks, this involved retrospective recall. Use of sleep diaries would have provided daily data on sleep symptoms, but are difficult to employ in large, community-based epidemiologic surveys such as TH2K.

Questions might arise about our sample design. We did not employ an area probability design. To compensate for this design effect, we post-stratified our sample to approximate the age, gender, and ethnic composition of the population 11 to 17 years. Our weighted sample closely approximated the age, gender, and ethnic composition of the five-county metropolitan area. Our follow-up rate was 75%, which raises the issue of potential bias. However, in a previous paper,[43] data demonstrated our Wave 1 sample and the baseline data for the Wave 1 – Wave 2 cohort were highly comparable, indicating little bias in rates of psychiatric disorders including depression was introduced by attrition. There also was no bias evident in prevalence rates for short sleep in Wave 1 and 2.

We also did not interview parents about either sleep disturbance or depression in their adolescents. Although there is argument that data from multiple informants are desirable, many studies have demonstrated considerable discordance is parent-child reports of youth problems.[52,53] We used data only from youths.

CONCLUSIONS

We are the first to examine the prospective association between sleep deprivation and major depression in adolescents. We also are the first to directly investigate whether there is a reciprocal effect.

We find that sleep deprivation has a strong effect on risk for major depression, increasing risk 4- to 5-fold in crude analyses and 3-fold in multivariate analyses controlling for depression at baseline. Sleep deprivation also increased risk of depressive symptoms, but the effect, while significant, was greatly attenuated compared to major depression.

When we examined the reverse, the results were quite different. There was no increased risk of sleep deprivation among those with depressive symptoms. There was an effect for major depression but this was limited to sleep deprivation only on weeknights. It may be that since major depression impairs functioning in multiple life domains,[40] and the combination of phase delay, late night activities or jobs and early morning school demands during weekdays and weeknights[12,14,17] further restrict sleep, that this explains in part this association.

These results, particularly for major depression, suggest that quantity of sleep, following DSM-IV guidelines[40] increases risk for major depression, which in turn increases risk for decreased sleep. This is not surprising, given the phenomenology of both sleep disturbances and major depression. DSM-IV diagnostic criteria for major depression focus on insomnia (insufficient sleep) or hypersomnia (sleeping too much), hence enhancing the likelihood of major depression increasing subsequent risk of short sleep duration.

Our results extend the available data on sleep and depression and are the first prospective data on this question. Clearly more data are needed on the prospective reciprocal association between these major public health problems in adolescence. In particular, our results need to be replicated in different geographic and ethnocultural populations in the United States as well as cross-nationally. While our results for sleep and psychiatric disorder[8,24] appear comparable to other studies, the sample was drawn from one large metropolitan area in the Southwest, and generalizability or external validity is always a concern. What is needed as well are studies which examine developmental trajectories of sleep and depression from childhood through adolescence into adulthood.

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