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 ScienceDirect

Journal of Adolescence 32 (2009) 1045–1057

Journal of
Adolescence

www.elsevier.com/locate/jado

Sleepless in adolescence: Prospective data on sleep deprivation, health and functioning

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Abstract

We estimate prevalence, incidence and persistence of short sleep or sleep deprivation in a two wave cohort study of 4175 youths 11–17 years old at baseline and 3134 of these a year later. Data were collected using computer interviews and questionnaires. Sleep deprivation was defined as 6 h or less per night during the past 4 weeks. Weighted logistic regression procedures were employed to calculate prevalence, incidence, persistence/chronicity, and odds ratios. Prevalence rates and rates of persistence suggest sleep deprivation is highly prevalent and chronic. Multivariate analyses indicate that short sleep increases risk across multiple domains of dysfunction, suggesting pervasive deleterious effects. The broad impact of sleep deprivation and its pervasiveness suggests interventions will need to focus on multilevel changes to increase sleep time and reduce the negative impact of sleep deprivation among adolescents.

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Keywords: Sleep deprivation; Adolescents; Somatic; Interpersonal and psychological functioning; Chronicity; Academic performance

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Sleep deprivation, or short sleep, is sleep time less than the average basal level of 7–8 h per night for adults (Colten & Altevogt, 2006) and about 9 h per night for adolescents (Carskadon, Acebo, & Jenni, 2004). Studies indicate that many adolescents do not obtain adequate nocturnal sleep (Carskadon, 1990a, 1990b; Gau & Soong, 1995; Johnson, Roth, Schultz, & Breslau, 2006; Liu, Uchiyama, Okawa, & Kurita, 2000; Morrison, McGee, & Stanton, 1992; Ohayon, Roberts, Zully, Smirne, & Priest, 2000; Roberts, Roberts, & Chan, 2006; Roberts, Roberts, & Chen, 2002; Terman & Hocking, 1913; Tynjala, Kannas, & Valimaa, 1993; Wolfson & Carskadon, 1998). As many as one-fourth of adolescents report sleeping 6 h or less per night (Wolfson & Carskadon, 1998). Recent studies of adults find that almost one-fifth report insufficient sleep (Kapur et al., 2002; Strine & Chapman, 2005).

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 (Carskadon, 2002; Colten & Altevogt, 2006; Wolfson, 1996, 2002; Wolfson & Carskadon, 1998). Laboratory studies using longitudinal designs have documented consistent changes in sleep/wake architecture in adolescents (Carskadon, 1982; Carskadon et al., 1980; Carskadon, Orav, & Dement, 1983). These changes become particularly pronounced from early puberty to late puberty (see Dahl & Lewin, 2002; Millman, 2005).

A good deal of research has been conducted on the correlates of sleep disturbance and sleep deprivation among adolescents, in particular insomnia (for reviews, see Carskadon, 2002; Colten & Altevogt, 2006; Fredriksen, Rhodes, Reddy, & Way, 2004; Roberts, Roberts, & Chen, 2001; Roberts et al., 2002). These data have come from community surveys and school surveys as well as laboratory studies. The available evidence suggests that disturbed sleep, such as insomnia and sleep deprivation, is 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, stomachaches and backaches. Laboratory studies in particular have documented impaired cognitive function, daytime sleepiness and fatigue as a consequence of sleep deprivation (for reviews, see Carskadon, 2002; Dahl & Lewin, 2002; Millman, 2005).

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. (2002) examined the effects of insomnia at baseline on subsequent functioning of adolescents a year later. The odds of dysfunction at followup for those with insomnia averaged 2.5 across 11 indicators of somatic, psychological and interpersonal functioning. For 9 of the 11 indicators, there was a clear, linear dose–response effect for increasing levels of insomnia at baseline. Other studies also have found that sleep problems, including sleep deprivation, increased the odds of subsequent mental health problems (Gregory & O'Connor, 2002; Morrison et al., 1992).

Perusal of the published literature finds almost no community-based, epidemiologic studies which have focused on consequences of sleep deprivation or, as it is often termed, short sleep. We found only one. Fredriksen et al. (2004) reported that while short sleep increased risk of future depression and lower self-esteem, the latter measures did not predict future short sleep.

Understanding the epidemiology of a health outcome requires information on not only its etiology but also its consequences. Prospective assessment is key in regard to both antecedents and consequences. Only prospective studies that examine putative risk and protective factors at baseline to predict future sleep problems, with and without sleep problems at baseline, can address questions of antecedent factors. This is critical, since it is generally assumed that the factors producing morbidity are different before and after sleep disturbance occurs. In other words, factors that precede the problem may differ from those that sustain it or result from it (Eaton, 1995; Roberts, 1990; Roberts, Roberts, & Chen, 1998). Similarly, only prospective studies that assess sleep problems at baseline and putative risk and protective factors at baseline and subsequently can hope to identify and distinguish among factors that are antecedents, consequences, or both.

Given the paucity of epidemiologic research on the natural history of sleep deprivation, our purpose here is to examine its consequences among adolescents. We do this by estimating incidence and persistence. We then examine the impact of sleep deprivation on a range of indicators drawn from three broad domains of functioning: interpersonal relations, somatic health and psychological health. From the literature on sleep and its correlates, our overall hypothesis is that sleep deprivation will be prospectively associated with increased risk of dysfunction indicative of impairment in somatic health, interpersonal relations and psychological well-being. More specifically, based on the results of Fredriksen et al. (2004), we hypothesize that sleep deprivation will increase the risk of depression and of lower self-esteem but will not increase the risk of poor academic performance.

To our knowledge, this is the first study to examine the consequences of short sleep using both a prospective design and a broad range of functional consequences of sleep deprivation.

Methods

Subjects

The data are taken from Teen Health 2000 (TH2K). The sample was selected from households in the Houston metropolitan area enrolled in local health maintenance organizations (HMOs). One youth, aged 11–17 years, was sampled from each eligible household, oversampling for ethnic minority households. Every household with a child 11–17 years of age was eligible. Because there were proportionately fewer minority subscriber households, we developed sample weights which were adjusted by poststratification to reflect the age, ethnic and gender distribution of the five-county Houston metropolitan area in 2000. The precision of estimates is thereby improved and sample selection bias reduced to the extent that it is related to demographic composition (Andrews & Morgan, 1973). After the weighted procedure, no difference was identified between the two distributions with respect to the three demographic factors of age, gender, and ethnic group ($p = 0.99$, $p = 0.93$, $p = 0.99$).

Data were collected at baseline 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), demographic data on the youths and the household as well as queries about stress exposures. The interviews were conducted by trained, lay interviewers and took on average 1–2 h, depending on the number of psychiatric problems present. The questionnaires contained questions on symptoms of disturbed sleep and items assessing different dimensions of the ethnic experience. These took about 30 min to complete. Interviews and questionnaires were completed with 4175 youths. Interviews were completed in 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 Wave 2 cohort consisted of 3134 youths plus their caregivers (75% who were assessed in Wave 1 and Wave 2). All youths and parents gave written informed consent prior to participation. All study forms and procedures were approved by the University of Texas Health Sciences Center Committee for Protection of Human Subjects.

Measures

We used the National Institute of Mental Health (NIMH) Diagnostic Interview Schedule for Children, Version IV (DISC-IV) as the diagnostic instrument (Shaffer, Fisher, Lucas, Dulcan & Schwab-Stone, 2000). 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 (Fredriksen et al., 2004). The sleep items were taken from a variety of validated sleep questionnaires, including SleepEVAL (see Ohayon & Roberts, 2001; Ohayon et al., 2000). The items on symptoms of insomnia were operationalized using DSM-IV diagnostic criteria (see Roberts, Roberts, & Chan, 2008; Roberts, Roberts, & Duong, 2008).

A number of risk factors for and sequelae of insomnia among adolescents have been examined (for reviews, see Johnson et al., 2006; Roberts, Roberts, & Chen, 2000, 2002). Four classes of outcomes are examined: psychological, interpersonal relations, somatic dysfunction, and academic performance. Psychological functioning, or mental health problems, was measured with six indicators. One item asked youths to rate their life satisfaction as very satisfied, pretty satisfied, about equal, pretty dissatisfied and very dissatisfied. A second item asked youths to rate their emotional or mental health as excellent, good, fair, poor and very poor. A third measure was an eight item version of Rosenberg's self-esteem scale ($\alpha = 0.77$) (Rosenberg, 1965). Low self-esteem was scored as 29 or less (out of 32 possible). Three measures were derived from the DISC-IV. Because diagnostic criteria for depression include indicators of disturbed sleep, we measured disturbed mood (whether youths had experienced depressed mood, anhedonia, or irritable mood) for a period of at least 2 weeks in the past year. Alcohol use was measured by reports of consuming any alcohol in the past year. Drug use similarly was assessed by use of marijuana and any other substances in the past year. We also examined the effects of substance use using a more stringent definition of six or more times in the past year. The results were essentially unchanged in terms of odds ratios. The less stringent definition increased substantially the number of cases for

analyses (for description of measures, see Roberts, Roberts, & Chan, 2008; Roberts et al., 2002; Roberts, Roberts, & Duong, 2008).

Interpersonal functioning was measured by three items that inquired about the extent of problems (a lot, some, only a few, no problems) experienced with friends or peers, at home with family members, and at school. We have used these measures in previous research (Roberts et al., 2000).

Somatic functioning, or physical health problems, was measured with three indicators. One item asked youths to rate their health as excellent, very good, good, fair or poor. A second measure consisted of a scale asking how difficult in the past 4 weeks had physical health problems made it to perform nine activities of daily living. Responses were not difficult, a little difficult, somewhat difficult, or very difficult. The score ranged from 0 to 27 ($\alpha = 0.88$). A third measure assessed how often in the past 4 weeks health problems impacted six types of family activities. Responses were very often, fairly often, sometimes, almost never, and never. Scores ranged from 0 to 24 ($\alpha = 0.87$).

As noted earlier, studies have reported that there is an association between sleep problems and lower academic achievement (see Dahl & Lewin, 2002), although the recent study by Fredriksen et al. (2004) did not find this association. We included an item on academic performance: During the past 6 months, how well have you been doing in your school work? Better than average, average, somewhat below average, and below average. The latter two categories were scored as poor academic performance or low grades.

Analysis

We present data on prevalence, incidence and persistence of short sleep using two measures. First, we estimate prevalence, incidence and persistence of short sleep on weeknights and weekends (Short Sleep WN/WE). Second, we then estimate the same rates for those who had short sleep only on weeknights (Short Sleep WN).

Incidence refers to the proportion of youths who were not cases at baseline who were cases a year later (in Wave 2). Persistence (chronicity) is defined as youths who met criteria for Short Sleep WN/WE or WN in Wave 1 and again in Wave 2. Here, we focus on the impact on subsequent functioning of youths who reported either 6 h of sleep or less on weeknights and weekends (Short Sleep WN/WE) or 6 h of sleep or less only on weeknights (Short Sleep WN) at baseline.

For generation of the confidence interval for prevalence, incidence and odds ratio involving short sleep, survey mean (svymean) and survey logistic regression (svylogit) procedures in STATA v9.0 were employed. This procedure uses Taylor series approximation to compute the standard error of the odds ratio. Lepowski and Bowles (2007) have indicated that the difference in computing standard error between this method and other repeated replication methods such as jackknife is very small.

Covariates included here are age, gender of youths, family income, ethnic status and puberty status. Age was treated as a continuous variable as was family income and puberty status. Pubertal status was measured following the work of Peterson, Crockett, Richards, and Boxer (1988). Three items each for girls and boys inquire about levels of development of secondary sexual characteristics. The items were summed, with scores ranging from 0 to 9 ($\alpha = 0.75$ for girls

and 0.74 for boys). Ethnic contrasts at baseline are limited to European ($n = 1479$), African ($n = 1476$) and Mexican Americans ($n = 857$). For these analyses the Wave 1 sample is 3812 rather than 4175 and the Wave 1-Wave 2 cohort is 2855.

Results

Table 1 indicates no significant difference between the Wave 1 sample and cohort. The only difference between the cohort at Wave 1 and Wave 2 is that, of course, youths were older. Family income also increased modestly. The proportion of youths reporting 6 h or less of sleep on weeknights increased from 20% to 25% while those sleeping 9 or more hours declined from about 28% to 22%. The proportion sleeping 7–8 h remained unchanged.

Table 2 indicates that prevalences of Short Sleep WN/WE and WN were not significantly different in Wave 1 and Wave 2 for the cohort. Incidence of Short Sleep WN/WE was 8.2%, about half the incidence of Short Sleep WN. About 1 in 6 reported an episode of WN in Wave 2 but did not do so in Wave 1. In other words, they were incident cases. Persistence of short sleep was substantial, one third of youths who met criteria for Short Sleep WN/WE in Wave 1 did so again in Wave 2. Over half did so for Short Sleep WN.

Table 3 presents analyses for short sleep at Wave 1 and outcomes at Wave 2, adjusting for functioning levels at baseline, adding controls for age, gender, ethnicity, family income and pubertal status. For Short Sleep WN/WE, only six predictors were significant (problems at school, low life satisfaction, poor perceived mental health, depressed mood, other drug use, and poor grades). For Short Sleep WN, five of the predictors were significant (problems at school, life satisfaction, other drug use, low self-esteem, and poor grades).

We then replicated the analyses in Table 3, adjusting for symptoms of insomnia. The latter was defined as one or more symptoms almost every day for the past month (difficulty initiating sleep, difficulty maintaining sleep, early morning waking, and nonrestorative sleep). These results are presented in Table 4. As can be seen, the results were slightly different. For short sleep every night, depressed mood and other drug use were no longer predicted by short sleep. For sleep deprivation only on weekdays, problems at school and life satisfaction were no longer significant. Poor grades and other drug use remained significant.

Discussion

The prevalence, 12-month incidence and chronicity of short sleep were high in this cohort of adolescents. Prevalence was almost 10% for Short Sleep WN/WE and 20% for Short Sleep WN. In terms of incidence, almost 17% were new cases of Short Sleep WN in Wave 2. Half of the cases of Short Sleep WN were cases in Wave 1 and again in Wave 2. Rates of Short Sleep WN/WE were lower for both incidence and persistence. To our knowledge, these are the first estimates of incidence and chronicity of short sleep among adolescents based on a large-scale epidemiologic survey. The data suggest that sleep deprivation is not only common, but also represents a chronic problem among youths.

In the only other community-based prospective study of the consequences of short sleep, Fredriksen et al. (2004) found that sleeping 6 h or less on school nights increased risk for

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

Characteristics	Wave 1 (<i>N</i> = 4175)	Wave 1 cohort (<i>N</i> = 3134)	Wave 2 cohort (<i>N</i> = 3134)
	%	%	%
Gender of youth			
Male	51.14	50.77	50.77
Female	48.86	49.23	49.23
Age of youth (years)			
16+	24.91	23.42	40.36
Between 13 and 15	48.05	49.74	48.63
12 or less	27.04	26.83	11.01
Ethnicity of youth			
European American	35.43	37.01	37.01
African American	35.35	34.59	34.59
Latino American	24.57	23.64	23.64
Other	4.65	4.75	4.75
Family income			
\$65,000+	35.29	22.83	40.73
\$35,000–\$64,999	40.71	39.86	39.16
<\$35,000	24.00	37.87	20.11
Parental marital status			
Married	75.71	76.26	76.10
Others	24.29	23.74	23.90
Hours of sleep			
Weeknights past month			
≤6	19.83	19.63	24.85
7–8	52.51	53.45	52.92
9+	27.66	26.92	22.23

Age (W1 cohort vs. W1 non-cohort): $p = 0.0001$.

Ethnicity (W1 cohort vs. W1 non-cohort): $p = 0.0015$.

Income (W1 cohort vs. W1 non-cohort): $p < 0.0001$.

symptoms of depression and lower self-esteem, but found no effect on academic performance. When we introduced controls for age, gender, ethnicity, family income, and pubertal status, depressed mood was not predicted by short sleep on weeknights, but low self-esteem and poor academic performance were.

Table 2
Weighted prevalence, incidence and persistence of short sleep, Wave 1–Wave 2.

	Prevalence, W1 (C.I.)	Prevalence, W2 (C.I.)	W1–W2 Incidence (%) (C.I.)	W1–W2 Persistence (%) (C.I.)
Short Sleep WN/WE	8.89 (7.93–9.85)	10.38 (9.25–11.52)	10.38 (9.25–11.52)	32.48 (26.54–38.41)
Short Sleep WN	19.82 (18.49–21.16)	24.38 (22.75–26.01)	16.92 (15.35–18.49)	53.87 (49.51–58.24)

Short Sleep WN/WN = Sleep 6 h or less on weeknights and weekends.

Short Sleep WN = Sleep 6 h or less on weeknights.

Table 3

Multivariate relations^a between short sleep and functioning problems, without adjustment for insomnia in Wave 1.

Wave 1	Somatic problems, W2			Interpersonal problems, W2			Psychological problems, W2						Academic problems, W2
	OR, 95% C.I.			OR, 95% C.I.			OR, 95% C.I.						OR, 95% C.I.
	Perceived health	Limitations	Impact of illness	Problems at home	Problems with peers	Problems at school	Low life satisfaction	Poor perceived mental health	Depressed mood	Alcohol use	Other drug use	Low self-esteem	Poor grades
Short Sleep	1.25	1.34	1.27	1.29	1.19	1.80 ^b	2.01 ^b	1.88 ^b	1.41 ^b	0.93	1.66 ^b	1.35	2.10 ^b
WN/WE	(0.72–2.15)	(0.97–1.87)	(0.93–1.73)	(0.91–1.84)	(0.73–1.94)	(1.25–2.57)	(1.38–2.92)	(1.26–2.79)	(1.03–1.91)	(0.62–1.41)	(1.02–2.70)	(0.95–1.92)	(1.38–3.21)
Short	1.23	1.24	1.08	1.17	0.93	1.38 ^b	1.46 ^b	1.29	1.23	1.25	1.71 ^b	1.35 ^b	1.58 ^b
Sleep WN	(0.82–1.83)	(0.98–1.57)	(0.86–1.35)	(0.90–1.52)	(0.63–1.37)	(1.04–1.82)	(1.08–1.97)	(0.95–1.76)	(0.98–1.53)	(0.94–1.65)	(1.20–2.44)	(1.03–1.77)	(1.14–2.20)

Short Sleep WN/WE = Sleep 6 h or less on weeknights and weekends.

Short Sleep WN = Sleep 6 h or less on weekends.

^a Odds ratios were adjusted for Wave 1 values of the same measurement, age, gender, ethnicity, family income, and pubertal status.

^b Odds ratios are statistically significant ($p \leq 0.05$).

Table 4

Multivariate relations^a between short sleep and functioning problems, adjusting for insomnia in Wave 1.

Wave 1	Somatic problems, W2			Interpersonal problems, W2			Psychological problems, W2					Academic problems, W2	
	OR, 95% C.I.			OR, 95% C.I.			OR, 95% C.I.					OR, 95% C.I.	
	Perceived health	Limitations	Impact of illness	Problems at home	Problems with peers	Problems at school	Low life satisfaction	Poor perceived mental health	Depressed mood	Alcohol use	Other drug use	Low self-esteem	Poor grades
Short Sleep	1.26	1.23	1.12	1.16	1.12	1.65 ^b	1.73 ^b	1.62 ^b	1.28	0.91	1.51	1.17	2.01 ^b
WN/WE	(0.73–2.20)	(0.88–1.72)	(0.81–1.55)	(0.80–1.67)	(0.68–1.85)	(1.14–2.37)	(1.17–1.54)	(1.09–2.42)	(0.94–1.75)	(0.60–1.39)	(0.92–2.49)	(0.81–1.68)	(1.31–3.08)
Short Sleep	1.23	1.15	0.96	1.05	0.88	1.26	1.27	1.11	1.12	1.23	1.59 ^b	1.20	1.50 ^b
WN	(0.82–1.85)	(0.91–1.46)	(0.75–1.21)	(0.80–1.38)	(0.59–1.30)	(0.95–1.68)	(0.93–1.73)	(0.81–1.52)	(0.90–1.41)	(0.92–1.65)	(1.10–2.29)	(0.91–1.58)	(1.07–2.11)

Short Sleep WN/WE = Sleep 6 h or less on weeknights and weekends.

Short Sleep WN = Sleep 6 h or less on weeknights.

^a Odds ratios were adjusted for Wave 1 values of the same measurement, age, gender, ethnicity, family income, pubertal status and insomnia.

^b Odds ratios are statistically significant ($p < 0.05$).

When we examined the deleterious effects of short sleep on both weeknights and weekend nights, multivariate analyses revealed that short sleep all week increased risk for depression and academic functioning, but did not increase risk for lower self-esteem. Thus, we were not able to replicate the findings by Fredriksen et al. (2004) regarding the deleterious effects of short sleep for risk of depressed mood and lower self-esteem. Our results for academic performance were consistent. Short sleep on weeknights increased risk of poor academic performance, as did short sleep on weekends and weeknights. Thus, our results, using the same measure of short sleep, only partially replicate those by Fredriksen et al. (2004) for depression and self-esteem.

Almost by definition, insomnia implies insufficient sleep in terms of both quality and quantity (American Psychiatric Association, 2000). When we introduced controls for insomnia, Short Sleep WN/WE increased subsequent risk for problems at school, poor life satisfaction, poor perceived mental health, and poor grades. For Short Sleep WN, the only significant outcomes were other drug use and poor grades. Here, again, we could not replicate the findings by Fredriksen et al. (2004) for depression, self-esteem or academic performance.

As noted earlier, our sleep items asked whether subjects had experienced symptoms almost every day for the past 4 weeks. Thus, our results are limited in that we could not date onset and thus are not able to partition our sample into those with acute vs. long-term sleep deprivation. In a recent paper, Buysse, Angst, Gamma, Ajdacic, Eich, and Ressler (2007) found differential results related to duration of insomnia among young adults. In their epidemiologic study in the United Kingdom, Ohayon, Caulet, Priest, and Guilleminault (1997) 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 (longer than 24 months), although it might be expected that the impact on somatic, psychological, and interpersonal functioning would be pronounced for chronic sleep problems of long 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 correlated with objective measures of disturbed sleep (Sadeh et al., 1995). We should note as well that the use of laboratory measures is impractical in large field studies such as TH2K (Liu et al., 2000; Ohayon et al., 2000).

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 11–17. Our weighted sample closely approximated the age, gender and ethnic composition of the five-county metropolitan area. Our followup rate was 75%, which raises the issue of potential bias. However, Tables 1 and 2 present data showing our Wave 1 sample and the baseline data for the Wave 1–Wave 2 cohort were highly comparable, indicating little bias was introduced by attrition. Also, as can be seen in Table 2, there was no bias evident in prevalence rates for short sleep in Wave 1 and Wave 2.

The data presented here and elsewhere suggest one-fourth to one-fifth of adolescents get insufficient sleep. As noted earlier, both cross-sectional and longitudinal studies have identified a range of deficits in child and adolescent functioning associated with disturbed sleep. Virtually all of these studies have focused on insomnia (see Johnson et al., 2006; Ohayon et al., 2000; Roberts et al., 2000, 2002; Roberts, Roberts, & Duong, 2008). The study by Fredriksen et al. (2004)

examined the impact of one type of sleep disturbance, short sleep, and its consequences on depression, self-esteem and academic performance. Their results indicate short sleep increased risk for depression and low self-esteem. We reexamined this question, demonstrating that short sleep has in some cases much broader negative consequences for future functioning involving somatic health, interpersonal relations, life satisfaction, substance use, and poor academic performance. The most deleterious impact on functioning was observed among youths who were sleep deprived on both weeknights and weekend nights. Our results for depression and self-esteem were mixed.

Sleep deprivation among adolescents appears to be, in some respects, the norm rather than the exception in contemporary society. In TH2K, only 17.2% of youths reported sleeping an optimal 9 h of sleep and only 28.8% reported 8 h of sleep on weeknights (see Carskadon et al., 2004). Over 43% slept 7 or fewer hours on weeknights. The modal sleep duration was 8 h (29.3%) for Short Sleep WN in Wave 1 and also in Wave 2 (27.8%). The demands and diversions impacting sleep patterns are present at the individual, familial, community and societal level (see Colten & Altevogt, 2006). This implies that interventions will require multilevel strategies to increase sleep time and reduce the negative impact of sleep deprivation among adolescents, as suggested by Dahl and Lewin (2002). Such multilevel strategies might focus on what could be termed external factors (peer pressure, parent role in sleep schedules, work and school obligations) vs. individual level factors (mental health, substance use, internal circadian period, self-regulation).

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