Persistence and Change in Symptoms of Insomnia among Adolescents

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Objective: To estimate the incidence, chronicity, and remission of symptoms of insomnia and to examine factors associated with the incidence and chronicity of insomnia among adolescents.

Methods: Data were collected using diagnostic interviews and questionnaires from 4175 youths aged 11 to 17 years at baseline, and 3134 of these youths followed-up a year later. Subjects were sampled from large managed care populations in a metropolitan area of over 4.7 million. Insomnia was assessed by youth-reported DSM-IV symptom criteria.

Results: One year incidence was 13.9% for 1 or more symptoms, 5.5% for 1 or more symptoms plus daytime fatigue or sleepiness, and 5.3% for insomnia caseness. Rates of chronicity were 45.8% for 1 or more symptoms, 34.7% with daytime fatigue or sleepiness, and 22.8% for insomnia caseness. There were no effects of age, sex, or family income in predicting incidence or chronicity of insomnia. There was a weak association of both somatic and psychological dysfunction with risk of future sleep outcomes, with stronger prediction for psychological dysfunction.

Conclusions: These results document further the public health burden of insomnia among adolescents. Prevalence of insomnia is comparable to that of other major psychiatric disorders such as mood, anxiety, disruptive, and substance use disorders. Incidence over one year also is high. Insomnia represents a chronic condition, further enhancing burden.

Keywords: Adolescents, insomnia, incidence, risk factors, chronicity

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THERE REMAINS A DEARTH OF DATA ON THE EPIDEMIOLOGY OF SLEEP DISORDERS AMONG ADOLESCENTS. BY WAY OF ILLUSTRATION, OHAYON AND GUILLEMINAULT[1] reviewed epidemiologic studies published over a 20-year period that had focused on insomnia, excessive sleepiness, sleep-disordered breathing, and parasomnia. Not a single one focused on adolescents.

Little has changed since that review. Four papers have estimated the prevalence of sleep disorders using diagnostic criteria[2-5], and all have focused on insomnia. These studies report prevalences ranging from 4.4% for point prevalence to 13.4% for past year for insomnia, to 10.7% for lifetime insomnia among adolescents.

Some evidence exists concerning the natural history of disordered sleep. The overwhelming focus has been on symptoms of insomnia or other types of sleep difficulties and not on sleep disorders per se. These studies report low to moderate stability of sleep problems from early childhood into adolescence and early adulthood for sleep measures such as duration, bedtime, wake time, difficulty initiating or maintaining sleep, napping, nonrestorative sleep, and daytime sleepiness or fatigue.[6-12]

Stability of sleep problems, in particular symptoms of insomnia, have been reported as high as 48% to 52% over 2 to 4 years[8, 13] to as low as 14.5% over 11 years, from early childhood to early adulthood,[7] to nonexistent between 4 and 16 years of age.[6]

Little has emerged from this admittedly small body of evidence concerning factors that are related to increased or decreased persistence. There is even less evidence regarding incidence. Although authors have noted the need for such studies,[14, 4-5] only 2 studies provide such data. Patten et al[13] estimated annual prevalence over a 4-year follow-up of 7% for any sleep problem and 2.3% for frequent sleep problems. Roberts, Lewinsohn, and Seeley[15] estimated the 1-year incidence for insomnia symptoms among adolescents as 12.5%. No other study has estimated incidence.

We have evidence that disturbed sleep is associated with both behavioral and emotional problems among adolescents, including depression, anxiety, conduct problems, suicidal thoughts and attempts, poor academic performance, and somatic problems such as fatigue, pain, and poor perceived health.[16-20] However, virtually all of these findings emanate from cross-sectional or case-control studies and, thus, do not establish whether sleep disturbances lead to deficits in functioning or deficits in functioning lead to sleep disturbance.

There also is some evidence that sleep problems are associated with negative outcomes over time among children and adolescents. Fredriksen et al[11] demonstrated that students who obtained less sleep over time reported more depressive symptoms and decreased self-esteem. Roberts, Roberts, and Chen[21] reported that, for adolescents with symptoms of insomnia at baseline, the odds of having a range of psychosocial deficits a year later averaged 2.5 across 11 different outcomes assessing interpersonal, psychological, and somatic functioning. Gregory and O'Connor[9] reported that sleep problems at age 4 predicted behavioral/emotional problems in midadolescence, and the association between sleep problems and depression/anxiety increased significantly during this time period. On the other hand,

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Strauch and Meier[7] found that sleep symptoms did not predict future scores on extraversion or neuroticism scales.

Most, but not all,[3,8] studies have found sleep problems to be more common among girls than boys.[4, 13, 22-24] The association with age also has been inconsistent, with most studies reporting no age effect in adolescence.[3, 5, 13, 23] The role of socioeconomic status has been little explored in research on adolescent insomnia. Roberts, Roberts, and Chan[5] found no effect of family income on prevalence of Diagnostic and Statistical Manual of Mental Disorders, Version IV (DSM-IV) insomnia among adolescents, nor did Johnson et al.[4] The latter did find a slightly lower risk for higher parental education. Ohayon[14] concluded that there is little evidence that income and education represent independent risk factors for insomnia among adults.

Three papers have examined prospective predictors of disturbed sleep among_adolescents. Fredericksen et al[11] reported that, although sleep problems increased future depression and lower self-esteem, the latter measures did not predict future sleep problems. Knutson[12] found that pubertal status predicted future sleep disturbance (insomnia, nonrestorative sleep, lack of sleep) among females but not among males. Patten et al[13] found that rebelliousness, depression, and smoking at baseline predicted insomnia symptoms 4 years later among adolescents.

Understanding the epidemiology of a health outcome requires measures of prevalence and natural history, including incidence, duration, remission, and relapse. Assessment of incidence is key in regard to questions of etiology. 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 etiology. This is critical, since it is generally assumed that the causal structure producing morbidity is different before and after sleep disturbance or sleep disorders occur. In other words, factors that cause the problem may differ from those that sustain it or result from it.[25-27]

Our purpose here is to examine the natural history of insomnia among adolescents. We do this by estimating prevalence, incidence, chronicity, and remission. We then examine selected putative risk and protective factors and their ability to prospectively predict incidence of insomnia over 1 year, specifically age, sex, family income, ethnicity, and physical and mental health problems. To our knowledge, this is the first study to estimate the incidence of DSM-IV insomnia and associated risk and protective factors using such a prospective design.

METHODS

Subjects

The data are taken from Teen Health 2000. The sample was selected from households in the Houston metropolitan area enrolled in 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, we developed sample weights that were adjusted by poststratification 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.[28] Thus, the weighted estimates generalize to the population 11 to 17 years of age in a metropolitan area of 4.7 million people. Chi-square tests were used to compare ethnicity, sex, and age distributions between census data for the 5-county area and sample data. After the weighted procedure, no difference was identified between the 2 distributions with respect to the 3 demographic factors of age, sex, and ethnic group ($P = 0.99$, $P = 0.93$, $P = 0.99$).

Data were collected at baseline on sample youths and 1 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, as well as queries about stress exposures. The interviews were conducted by trained lay interviewers and took on average 1 to 2 hours, 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 minutes 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. We were able to obtain complete assessments from both youth and parent in Wave 2 from 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

There have been 4 studies of disturbed sleep using DSM-IV diagnostic criteria for adolescents,[3, 29] and 2 of these have been in the United States.[4,5] None of the existing psychiatric diagnostic interviews designed for epidemiologic research with children and adolescents have included modules eliciting symptoms of DSM sleep disorders. We used the National Institute of Mental Health Diagnostic Interview Schedule for Children, Version IV (DISC-IV).

The DISC-IV does not inquire about symptoms of disturbed sleep 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.

Our measures attempt to operationalize DSM-IV symptom criteria for a diagnosis of insomnia. That is, we collected data on the symptoms of insomnia specified in the DSM-IV. The insomnia items are trouble falling asleep (DIS), waking up in the middle of the night and finding it hard to go back to sleep (DSM1), waking up frequently but able to go back to sleep (DMS2), waking up very early (EMA), and nonrestor-

ative sleep (NRS). The time referent is the past 4 weeks. The subject could respond "rarely or never, sometimes, often, or almost everyday." The DSM-IV symptom criteria for insomnia include all of these symptoms, and the symptoms should cause significant distress or impairment. To qualify for a diagnosis of primary insomnia, the symptoms must not occur exclusively during another sleep disorder, occur during another psychiatric disorder, or be due to the effects of alcohol, drugs, or medication. The measures operationalize DSM-IV symptom criteria and thus have content validity. Our rates of insomnia are highly comparable to those reported by Ohayon et al (2000), demonstrating external validity, and are correlated with other factors consistent with the literature, providing evidence of construct validity.

Analyses

We present analyses defining insomnia several ways. First, we present data on the prevalence of each indicator symptom of insomnia. We then present prevalences of insomnia following DSM-IV criteria as closely as our data permitted. We do this in 2 ways. First, we estimate prevalence of at least 1 symptom of disturbed sleep with either daytime fatigue or daytime sleepiness (as indicators of impairment). Second, we then adjust that prevalence rate by excluding any subject who met the first 2 criteria who also met DSM-IV diagnostic criteria for a mood disorder, an anxiety disorder, or a substance use disorder in the past year. We refer to these as prevalences P_1, P_2, and P_3. We should note that P_3 is not equivalent to a full DSM-IV diagnosis of primary (or secondary) insomnia but approximates such a diagnosis as our measures permit.

Incidence refers to the proportion of youths who were not cases as defined by P_1, P_2, or P_3 at baseline who were cases a year later (in Wave 2). Likewise, chronicity is defined as youths who met criteria for P_1, P_2, or P_3 in both Wave 1 and Wave 2.

A number of risk factors and sequelae of insomnia among adolescents have been examined (for reviews, see Roberts, Roberts, and Chen[20, 21]). Covariates included here are age and sex of youths, family income, ethnic status, physical health functioning, mental health functioning, and life stress.[14] Age was treated as a continuous variable. Family income was treated as a categorical variable (less than $35,000, $35,000 to 64,999, $65,000 and above). Ethnic contrasts at baseline are limited to European (n = 1479), African (n = 1476) and Mexican Americans (n = 857). For analyses of ethnic status, the Wave 1 sample is 3812 rather than 4175 and the Wave1-Wave2 cohort is 2855.

Psychiatric disorders among youths were assessed with the DISC-IV, a highly structured instrument[30] designed to be administered by lay interviewers. In Teen Health 2000, we included anxiety disorders (agoraphobia, generalized anxiety, panic, social phobia, posttraumatic stress disorder), mood disorders (major depression, dysthymia, mania, hypomania), behavioral disorders (conduct, oppositional defiant, attention-deficit/hyperactivity disorders), eating disorders (bulimia, anorexia nervosa), and substance use (alcohol, marijuana, and other substance disorders).

Our definition of mood disorder was meeting diagnostic criteria for at least 1 disorder in the past 12 months (major depression, dysthymia, mania, and hypomania). A similar strategy was employed for anxiety disorders (agoraphobia, generalized anxiety, panic, social phobia, posttraumatic stress disorders), alcohol use disorders (abuse or dependence), and other substance use disorders (abuse of or dependence on marijuana and other substances).

Somatic functioning, or physical health problems, was measured with 3 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 9 activities of daily living. Responses were not difficult, a little difficult, somewhat difficult, or very difficult. The score ranged from 0 to 27. A third measure assessed how often in the past 4 weeks health problems impacted 6 types of family activities. Responses were very often, fairly often, sometimes, almost never, and never. Scores ranged from 0 to 24.

Psychological functioning, or emotional well-being, also was measured with 3 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 whether a youth had symptoms of depressed mood, anhedonia, or irritability (disturbed mood) at baseline.

Stressors measured are neighborhood stress, school stress, and family stress. Subjective appraisal of community stress is assessed by asking adolescents how much of a problem (very serious, somewhat serious, minor, not really a problem) each of 8 conditions are in their neighborhood: crime, gangs, traffic, noise, trash and litter, lighting, public transportation, and recreation ($\alpha = 0.88$). We also inquired about perceived stressfulness of the social environment for those who still attend school by asking adolescents how much a problem (very serious, somewhat serious, minor, not really a problem) each of 11 conditions are at their school: violence, gangs, weapons, drugs, noise in classrooms, trash and litter, crowded classrooms, how teachers treat students, lack of supplies and equipment, prejudice and discrimination, and stealing ($\alpha = 0.80$). Our measure of family stress is taken from the work of Wickrama, Lorenz, and Conger.[31] The scale inquires about 10 behaviors of parents toward children. There is an adolescent scale relating to each parent ($\alpha > 0.85$). Intuitively, it would be expected that, for youths in school (and most of our sample were), activities related to school such as homework and extracurricular activities as well as demands of a job might affect sleep patterns. Evidence cited above provides support for this. Accordingly, we included 3 items related to these domains. Each inquired whether, during the past 4 weeks, the respondent slept less than usual (1) due to studying or homework; (2) because of school activities such as sports, clubs, etc.; and (3) because of having a job.

For generation of the confidence interval for the prevalence and odds ratio, survey mean (svymean) and survey logistic regression (svylogit) procedures in STATA V8.2 (StataCorp, LP, College Station, Texas) were employed. This procedure uses Taylor series approximation to compute the standard error of the odds ratio. Lepowski and Bowles[32] have indicated that the difference in computing standard error between this method and other repeated replication methods such as the jackknife is very small. For each variable Xi that had missing values, we

Table 1—Unweighted Sample Characteristics, Teen Health 2000

Characteristics	Wave 1 sample n = 4175 %	Wave 1 cohort n = 3134 %	Wave 2 cohort n = 3134 %
Sex of youth			
Male	51.14	50.77	50.77
Female	48.86	49.23	49.23
Age of youth			
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
Others	4.65	4.75	4.75
Family income			
$65,000 +	35.29	37.81	40.73
$ 35,000 - $ 64,999	40.71	39.86	39.16
< $35,000	24.00	22.33	20.11
Parental marital status			
Married	75.71	76.26	76.10
Others	24.29	23.74	23.90

No significant differences were found comparing distribution for each characteristic of W1 sample and W1 cohort.
Age (W1 cohort vs W2 cohort): $P < 0.0001$.
Income (W1 cohort vs W2 cohort): $P = 0.03$.

conducted multivariate logistic regression on the complete data set by using the rest of the selected variables. The probability of Xi = 1 for each subject were calculated from parameter estimates and was further used to generate the missing value. In case of experiencing predictors having missing values, their medians were filled in the calculation of probability**.** This process was repeated sequentially for the next variable with missing values. Then, we iterated the whole procedure 500 times to get less biased parameter estimates and standard errors. The average odds ratio and 95% confidence intervals are reported in Tables 3 to 5.

RESULTS

Table 1 presents characteristics of the Wave 1 sample and the Wave 1 – Wave 2 cohort. The only differences in Wave 1 were the lower income of the 2 minority groups. The only difference in the Wave 1 – Wave 2 cohort at Wave 2 was that youths were older and family income increased slightly.

Table 2 presents prevalence rates, incidence rates, and proportion of youths who had the sleep indicator in both waves (chronic) and of youths who had the indicator in Wave 1 but did not in Wave 2 (remitted). First, in terms of prevalence, there were no differences between the Wave 1 sample and the cohort at Wave 1. The most prevalent symptom was NRS, with 18% reporting this symptom, followed by DIS (7%). One in 4 youths reported 1 or more symptoms of insomnia almost every day (P_1) in the past month. Our estimated prevalence of DSM-IV caseness (P_3) was 4.7% and 4.9%, in the baseline sample and the cohort at baseline (NS).

In terms of incidence, 15% of those not reporting NRS at baseline did so in Wave 2. Incidence of 1 or more symptoms was 19%. For our proxy measure of DSM-IV insomnia, incidence was 5.6%. Table 2 clearly demonstrates the chronic nature of insomnia. For youths who had a sleep problem in Wave 1, 41% reported still having NRS a year later. A third reported DIS in Wave 1 and Wave 2, whereas 46% reported 1 or more

Table 2—Prevalence and Stability of Insomnia – W1, W2 Cohort

	Prevalence rates		W1, W2 cohort change		
	W1 sample %, 95% CI	W1 cohort %, 95% CI	Total incidence %, 95% CI	Chronic %, 95% CI	Remitted %, 95% CI
NRS	18.14 (16.85 – 19.43)	18.23 (16.75 – 19.71)	12.01 (10.77 – 13.25)	40.99 (36.48 – 45.49)	59.01 (54.51 – 63.52)
DIS	7.05 (6.18 – 7.92)	7.53 (6.50 – 8.56)	3.31 (2.62 - 3.99)	33.35 (26.65 – 40.05)	66.65 (59.95 – 73.35)
DMS1	3.11 (2.54 – 3.67)	2.93 (2.31 – 3.54)	2.24 (1.69 – 2.79)	19.37 (11.22 – 27.52)	80.63 (72.48 – 88.78)
DMS2	5.15 (4.41 – 5.89)	5.11 (4.26 – 5.97)	3.90 (3.17 - 4.64)	19.40 (12.02 – 26.07)	80.96 (73.93 – 87.98)
EMA	3.20 (2.61 – 3.78)	3.27 (2.60 – 3.93)	2.42 (1.84 – 3.01)	14.57 (6.86 – 22.27)	85.43 (77.73 – 93.14)
P_1	26.83 (25.36 – 28.30)	27.12 (25.42 – 28.82)	13.97 (12.65 , 15.29)	45.85 (42.15 – 49.54)	54.15 (50.46 – 57.85)
P_2	6.66 (5.84 – 7.49)	7.03 (6.05 – 8.01)	5.50 (4.63 – 6.37)	34.71 (27.73 – 41.69)	65.29 (58.31 – 72.27)
P_3	4.70 (4.02 – 5.39)	4.94 (4.14 – 5.75)	5.36 (4.50 – 6.22)	23.63 (16.39 – 30.88)	76.37 (69.12 – 83.61)

NRS refers to nonrestorative sleep; DIS, trouble falling asleep; DMS_1, nighttime waking, difficult to sleep again; DMS_2, nighttime waking, but return to sleep; EMA, early morning awakening; P_1, 1 or more insomnia symptoms; P_2, at least 1 symptom and daytime fatigue/sleepiness; P_3, P_2 after mood, anxiety, and substance use disorders are excluded. No significant differences were found comparing W1 sample and W1 cohort with respect to NRS, DIS, DMS1, DMS2, and EMA.

Table 3—Univariate and Multivariate Analyses of Risk and Protective Factors for P_1

	Univariate			Multvariate		
Effect	**OR**	**95%CI**		**OR**	**95%CI**	
Sex						
Female:Male	1.31[a]	1.11	1.56	1.30[a]	1.07	1.58
Age						
M:L	0.93	0.75	1.14	0.72[a]	0.57	0.91
H:L	1.25	0.99	1.59	0.90	0.68	1.19
Ethnicity						
AA:EA	1.07	0.88	1.29	0.93	0.75	1.15
MA:EA	0.98	0.78	1.24	0.90	0.70	1.15
Family Income						
M:L	0.91	0.72	1.15	0.93	0.71	1.21
H:L	0.81	0.64	1.02	0.91	0.69	1.20
Perceived Health						
Fair :Good	2.22[a]	1.70	2.91	1.29	0.94	1.77
Limitations						
1+ : None	1.82[a]	1.53	2.17	1.33[a]	1.09	1.63
Impact of Illness						
High : Low	1.65[a]	1.39	1.97	1.12	0.91	1.37
Life Satisfaction						
No: Yes	2.76[a]	2.25	3.40	1.73[a]	1.34	2.23
Perceived Mental Health						
Fair: Low	2.84[a]	2.30	3.50	1.46[a]	1.11	1.93
Depressed Mood						
Yes : No	1.69[a]	1.42	2.02	1.25[a]	1.02	1.53
School Stress						
M:L	1.30[a]	1.04	1.64	1.25	0.97	1.62
H:L	2.48[a]	1.99	3.08	1.84[a]	1.39	2.43
Neighborhood Stress						
M:L	1.21	0.97	1.51	0.91	0.70	1.17
H:L	1.56[a]	1.25	1.96	0.86	0.65	1.13
Father Stress						
M:L	1.59[a]	1.26	2.01	1.30	0.99	1.71
H:L	2.55[a]	2.05	3.18	1.58[a]	1.19	2.10
Mother stress						
M:L	1.50[a]	1.19	1.89	1.00	0.76	1.31
H:L	2.91[a]	2.31	3.68	1.37[a]	1.04	1.86

P_1 refers to 1 or more insomnia symptoms; CI, confidence interval; EA, European American; AA, African American; MA, Mexican American; L, low; M, middle; H, high.
[a]Statistically significant at level of 0.05.

Table 4—Univariate and Multivariate Analyses of Risk and Protective Factors for P_2

	Univariate			Multivariate		
Effect	**OR**	**95%CI**		**OR**	**95%CI**	
Sex						
Female:Male	2.04[a]	1.50	2.79	1.91[a]	1.35	2.69
Age						
M:L	2.01[a]	1.32	3.06	0.78	0.50	1.22
H:L	1.32	0.90	1.95	0.95	0.58	1.56
Ethnicity						
AA:EA	1.00	0.72	1.40	0.83	0.56	1.22
MA:EA	1.02	0.68	1.52	0.92	0.60	1.42
Family Income						
M:L	0.91	0.61	1.37	0.94	0.58	1.51
H:L	0.97	0.64	1.46	1.02	0.63	1.65
Perceived Health						
Fair :Good	2.97[a]	2.01	4.39	1.21	0.74	1.97
Limitations						
1+ : None	3.01[a]	2.17	4.18	2.12[a]	1.45	3.10
Impact of Illness						
High : Low	2.13[a]	1.55	2.93	1.19	0.82	1.74
Life Satisfaction						
No: Yes	4.01[a]	3.89	4.13	1.65[a]	1.11	2.45
Perceived Mental Health						
Fair: Low	4.30[a]	3.15	5.89	1.82[a]	1.17	2.84
Depressed Mood						
Yes : No	1.69[a]	1.42	2.02	1.44	0.96	2.16
School Stress						
M:L	7.68[a]	4.76	12.40	2.93[a]	1.61	5.32
H:L	3.02[a]	1.80	5.07	5.01[a]	2.75	9.13
Neighborhood Stress						
M:L	1.26	0.81	1.95	0.81	0.49	1.34
H:L	2.63[a]	2.60	2.66	0.96	0.59	1.59
Father Stress						
M:L	3.82[a]	2.52	5.79	1.61	0.95	2.71
H:L	2.06[a]	1.31	3.26	1.68	0.99	2.83
Mother stress						
M:L	1.71[a]	1.06	2.75	0.71	0.42	1.21
H:L	4.43[a]	2.83	6.93	1.25	0.72	2.17

P_2 refers to at least 1 symptom and daytime fatigue/sleepiness; CI, confidence interval; EA, European American; AA, African American; MA, Mexican American; L, low; M, middle; H, high.
[a]Statistically significant at level of 0.05.

symptoms in both waves. More than 1 in 5 reported DSM-IV insomnia at follow-up. However, for every sleep outcome examined, a majority with the problem at baseline did not report the problem a year later.

Tables 3, 4, and 5 present results examining first the odds of each of the sleep outcomes (P_1, P_2, P_3) for each individual predictor, without controls for other factors (first column in each table). We then conducted multivariate logistic regression analyses (second column) in which predictors were selected based on those with an odds ratio significant at $P < 0.25$ for the multivariate model. Retention of variables retained in the full model was determined using the likelihood ratio test, until the optimal goodness-of-fit measure was achieved. These analyses are limited to predicting incident cases of P_1, P_2, and P_3, that is, subjects who were not cases in Wave 1 but were cases in Wave 2.

The results in Table 3 for univariate analyses indicate female sex, worse perceived health, somatic health problems, mental health problems, and stressors all increased the risk of any symptoms of insomnia. Multivariate analyses revealed female sex, younger age, somatic health (2 indicators), mental health (all 3 indicators), and high stressors (3 indicators) all independently predicted P_1.

Table 4 presents the analyses for P_2. Univariate risks were associated with female sex, lower income, worse somatic health (all 3 indicators), worse mental health (all 3 indicators), and stress (all 4 indicators). Multivariate analyses retained, as significant independent predictors, female sex, limitations due to health, life satisfaction and perceived mental health, school stress, neighborhood stress, and father stress.

In Table 5, all of the univariate odds ratios were significant ($P < 0.05$) except age, ethnicity, and family income. However, the multivariate model identified only 4 significant independent

Table 5—Univariate and Multivariate Analyses of Risk and Protective Factors for P_3

	Univariate			Multivariate		
Effect	**OR**	**95%CI**		**OR**	**95%CI**	
Sex						
Female:Male	1.70[a]	1.20	2.42	1.61[a]	1.10	2.36
Age						
M:L	1.44	0.90	2.30	0.70	0.43	1.14
H:L	1.07	0.70	1.65	0.81	0.47	1.40
Ethnicity						
AA:EA	2.66[a]	1.83	3.85	0.93	0.61	1.41
MA:EA	0.77	0.47	1.25	0.69	0.41	1.15
Family Income						
M:L	0.95	0.60	1.51	0.84	0.49	1.43
H:L	0.95	0.59	1.52	0.91	0.53	1.54
Perceived Health						
Fair :Good	1.69[a]	1.03	2.76	0.76	0.42	1.39
Limitations						
1+ : None	2.39[a]	1.66	3.45	1.91[a]	1.24	2.93
Impact of Illness						
High : Low	1.96[a]	1.36	2.81	1.23	0.81	1.87
Life Satisfaction						
No: Yes	2.90[a]	2.01	4.19	1.41	0.88	2.26
Perceived Mental Health						
Fair: Low	2.94[a]	2.04	4.25	1.66	0.96	2.86
Depressed Mood						
Yes : No	1.69[a]	1.42	2.02	1.16	0.75	1.81
School Stress						
M:L	5.42[a]	3.18	9.25	3.23[a]	1.69	6.16
H:L	3.04[a]	1.74	5.32	4.34[a]	2.20	8.54
Neighborhood Stress						
M:L	1.02	0.63	1.66	0.72	0.42	1.22
H:L	1.82[a]	1.15	2.87	0.79	0.46	1.36
Father Stress						
M:L	2.74[a]	1.74	4.32	1.42	0.82	2.45
H:L	1.68[a]	1.02	2.77	1.32	0.76	2.32
Mother stress						
M:L	1.73[a]	1.02	2.91	0.92	0.53	1.60
H:L	3.68[a]	2.24	6.04	1.49	0.81	2.74

$P_3 - P_2$ after mood, anxiety and substance use disorders are excluded. CI refers to confidence interval; EA, European American; AA, African American; MA, Mexican American; L, low; M, middle; H, high.

[a]Statistically significant at level of 0.05.

predictors of our measure of DSM-IV insomnia caseness: female sex, limitation due to health, school stress, and mother stress.

DISCUSSION

Our prevalence rate for DSM-IV insomnia caseness is highly comparable to the rates reported by Ohayon et al[3] and Johnson et al.[4] Our incidence rate for insomnia is comparable to the annual prevalence over 4 years reported by Patten et al.[13] Our rate of chronicity for insomnia was highly comparable to the rates reported by Morrison et al[8] and by Patten et al.[13]

Although there are limited data on the prevalence, incidence, and chronicity of symptoms of DSM-IV insomnia, our results add to the growing evidence that the burden of insomnia among adolescents is comparable to that of other major psychiatric disorders such as mood disorders, anxiety disorders, disruptive disorders, and substance abuse.[33-36] Clearly, insomnia is not only a major public health problem, but also a major chronic disease affecting a substantial proportion of the adolescent population.

In terms of predictors, our results indicate consistent sex effects, some age effects and no effects for family income or ethnic status on incidence of insomnia over 12 months. In the only other study that has used prospective data, Patten et al[13] found that family income, age, and sex did not predict chronicity over 4 years. They also that found age and sex were not predictors. Johnson et al,[4] in the only study to examine lifetime rates of insomnia among adolescents, also found no sex differences overall and no difference before puberty between males and females. However, after puberty, girls were more likely than boys to have insomnia. Clearly, the role of sex in adolescent insomnia, as in most psychiatric disorders, involves a complex etiologic web.

In his comprehensive review, Ohayon[14] concluded that, among adults, higher risk for insomnia is related to female sex and older age. There is little evidence to suggest a strong independent effect for income or education. On the other hand, medical conditions or poor physical health are strongly associated with insomnia. Mental disorders, at least among adults, appear to be the strongest risk factor for insomnia, in particular mood disorders.

The data for adolescents also suggest that somatic and psychological problems are strong correlates of sleep problems, in particular insomnia.[20, 21] However, most of the evidence has come from cross-sectional or prevalence studies. We also found that problems with somatic and psychological functioning are strongly associated with the prevalence of sleep problems. However, we found that these initial associations were not robust using prospective data. In fact, there were few associations that were statistically significant when predicting incidence or chronicity.

Limitations

The nature of our study imposed certain limitations on our analyses and interpretations. First and foremost, our structured diagnostic interview did not include modules on DSM-IV insomnia or other sleep disorders. Thus, we were not able to distinguish between primary and secondary sleep disorders, nor were we able to examine the role of comorbid sleep disorders in relation to ethnicity. Although our measures permitted us to assess Criteria A, B (to a limited degree), D and E, we were not able to assess Criterion C (presence of other sleep disorders). Nonetheless, our symptom criteria capture most of the core research diagnostic criteria for insomnia disorders proposed by the American Academy of Sleep Medicine Work Group.[37] As noted by the Work Group, there is great diversity in definitions of insomnia in the literature. Furthermore, there are few data demonstrating the reliability and validity of DSM diagnostic criteria for insomnia, posing considerable problems in the interpretation of epidemiologic data.

This inability to assess comorbid sleep disorders, however, may have little effect on our overall prevalence estimate for insomnia. Based on their analyses of data on adolescents, Ohayon

and Roberts[29] concluded that multiple diagnoses of sleep disorders are a minor issue and affect rates of individual disorders vary little. In addition, our measures for B (impairment) were limited to daytime fatigue and daytime sleepiness. We had other indicators of impairment at home, at school, and with peers, as well as a measure of global functioning—the Child Global Assessment Scale—but none of these were specific to sleep.

As noted earlier, our sleep items asked whether subjects had experienced symptoms of insomnia almost every day for the past 4 weeks. Thus, our results are limited in that we were not able to partition our sample into those with acute versus chronic sleep problems. In their epidemiologic study in the United Kingdom, Ohayon et al[38] 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, although it might be expected that the impact on somatic, psychological, and interpersonal functioning would be more pronounced for chronic sleep problems.[39]

Another limitation is that we did not have objective data on disturbed sleep. That is, we did not have physiologic studies. Although 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.[40]

Questions might arise about our sample design. We did not employ an area probability design. To compensate for this design effect, we poststratified our sample to approximate the age, sex, and ethnic composition of the population[12-17] of the 5-county metropolitan area in which all of our study households were located. Our wave 2 completion rate was 75%. Data from Table 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.

CONCLUSIONS

The results presented here, as well as the results from the limited number of studies published, suggest that the risk of incident or chronic episodes of insomnia may not vary systematically across subgroups defined by age, sex, family income, or ethnic status. If these findings withstand the scrutiny of replication, then we should look elsewhere to better understand the epidemiology of insomnia.

Our findings on the roles of somatic and psychological dysfunction in the risk of subsequent insomnia provide some prospective confirmation of results emanating from cross-sectional or prevalence studies. That is, some sleep outcomes were predicted by our measures of somatic and psychological dysfunction, but the associations were fewer and weaker than those that we observed for prevalence data. Thus, our results provide limited data on etiologic factors for insomnia.

Given the explosion of results from research on behavioral genetics, this may be 1 promising line of inquiry. However, as noted by Ohayon,[14] there is yet little understanding of the genetics of most sleep disorders, including insomnia. There is some evidence that insomniacs have a family history of insomnia,[41, 42] with the biologic mother most frequently the relative with insomnia. There also is evidence that earlier onset of insomnia has stronger familial clustering.[43] However, as noted recently,[43] sleep disorders, including insomnia, are heterogeneous and their genetics undoubtedly will prove to be complex.

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