Epidemiology of Insomnia, Depression, and Anxiety

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Study Objectives: This study used empirically validated insomnia diagnostic criteria to compare depression and anxiety in people with insomnia and people not having insomnia. We also explored which specific sleep variables were significantly related to depression and anxiety. Finally, we compared depression and anxiety in (1) different insomnia types, (2) Caucasians and African Americans, and (3) genders. All analyses controlled for health variables, demographics, organic sleep disorders, and symptoms of organic sleep disorders.

Design: Cross-sectional and retrospective.

Participants: Community-based sample (N=772) of at least 50 men and 50 women in each 10-year age bracket from 20 to more than 89 years old.

Measurements: Self-report measures of health, sleep, depression, and anxiety.

Results: People with insomnia had greater depression and anxiety levels than people not having insomnia and were 9.82 and 17.35 times as likely to have clinically significant depression and anxiety, respectively. Increased insomnia frequency was related to increased depression and anxiety, and increased number of awakenings was also related to increased depression. These were the only 2 sleep variables significantly related to depression and anxiety. People with combined insomnia (ie, both onset and maintenance insomnia) had greater depression than did people with onset, maintenance, or mixed insomnia. There were no differences between other insomnia types. African Americans were 3.43 and 4.8 times more likely to have clinically significant depression and anxiety than Caucasians, respectively. Women had higher levels of depression than men.

Conclusion: These results reaffirm the close relationship of insomnia, depression, and anxiety, after rigorously controlling for other potential explanations for the relationship.

Keywords: Insomnia, depression, anxiety, epidemiology, gender, ethnicity.

Citation: Taylor DJ; Lichstein KL; Durrence HH et al. Epidemiology of insomnia, depression, and anxiety. *SLEEP* 2005;28(11): 1457-1464.

INTRODUCTION

CHRONIC INSOMNIA AFFECTS APPROXIMATELY 9% TO 12% OF THE POPULATION[1-3] AND IS MORE PREVALENT THAN HEART DISEASE, CANCER, AIDS, neurologic disease, breathing problems, urinary problems, diabetes, and gastrointestinal problems.[4] Researchers estimate the total annual cost of insomnia is $30 to $35 billion.[5] Although insomnia is highly prevalent, it is not commonly viewed as a significant threat to health. However, research has shown that a strong relationship exists between insomnia, depression, and anxiety, where insomnia may be a risk factor.[6] Unfortunately, the exact nature of this relationship remains unclear because previous studies have had varied definitions of insomnia and sometimes did not control for confounds. The current study defined insomnia using empirically validated criteria and controlled for possible confounds to obtain unbiased estimates of the relationship between insomnia, depression, and anxiety. We further elucidated this relationship by examining depression and anxiety associations with insomnia severity and insomnia type.

Many previous insomnia epidemiologic studies were reanalyzes of data from prominent epidemiologic studies (e.g., Alameda County, Epidemiologic Catchment Area, etc.) whose primary interests were not sleep. Thus, the definitions of insomnia were not as precise as the researchers likely would have preferred and varied considerably between studies. The most frequent operational definition of insomnia in these studies was a self-report of 2 or more weeks of insomnia at anytime within the past 6 months, 12 months, or lifetime.[1,3,7-10] These definitions of insomnia would allow people with transient insomnia (i.e., only 2 weeks in lifetime) to be considered as having current chronic insomnia, possibly making the prevalence rates of depression and anxiety inaccurate for people with current chronic insomnia.

Some studies have required a current complaint of insomnia. However, these studies focused on a restricted age range (e.g., > 50 years old)[9,11,12] or had broad definitions of insomnia (e.g., problems with sleep, feeling tired, sleep disturbance) and psychopathology (e.g., felt the need for help in the last year for tension or depression).[13,14]

Further, none of the studies discussed above attempted to rule out underlying sleep disorders and many did not attempt to rule out medical causes of the insomnia, depression, or anxiety.[1,3,7-11,13,14] This is important considering that depression, anxiety, and insomnia can all be caused or at least exacerbated by other medical or psychiatric disorders.[15,16] By not excluding or controlling for underlying organic sleep disorders and medical conditions, it is highly likely participants with comorbid insomnia were included in the analyses, again possibly making the prevalence rates inaccurate for people with primary insomnia.

In addition, no studies have attempted to determine what role severity of insomnia plays in the relationship with depression and anxiety. Insomnia is a very heterogeneous disorder with some individuals having difficulty a few nights a week just at bedtime,

Disclosure Statement

This was not an industry supported study. Dr. Taylor has served as a contract manuscript writer for Sepracor on four papers in the past 6 months. Dr. Lichstein has received research equipment from Mini Meter Corp. Dr. Durrence is an employee of Somaxon Pharmaceuticals, Inc. Drs. Riedel and Bush have indicated no financial conflicts of interest.

Submitted for publication December 2004
Accepted for publication July 2005

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and others having difficulty every night at both bedtime and in the middle of the night. Someone with less severe insomnia could be expected to incur fewer consequences. However, most treatment studies typically only report a few sleep variables (e.g., sleep-onset latency [SOL], total sleep time [TST], sleep efficiency [SE]). If other sleep variables were more closely related to depression and anxiety, it would be important to report them in outcome studies as well.

Finally, the relationship between different types of insomnia (ie, onset, maintenance, terminal), and depression and anxiety remains unclear. Although most studies in this area have found no differences between insomnia types in terms of depression or anxiety,[17-21] some researchers have shown that anxiety is more common in sleep-onset insomnia.[13,22] These discrepancies are again a likely consequence of varying insomnia definitions and limited control of confounding variables.

The present study examined depression and anxiety levels in a community sample, comparing groups regarding insomnia status, insomnia severity, and insomnia type. It was hypothesized that (1) people with insomnia would have significantly higher depression and anxiety levels and (2) as insomnia severity worsened, levels of depression and anxiety would increase. Due to the discrepancies in the insomnia-type literature, it was difficult to hypothesize a priori what results would be found in comparisons of these groups.

METHODS

Procedure

Participants were recruited using a random digit dialing protocol in Shelby County, Tennessee. Volunteers were then mailed an informed-consent form with a questionnaire packet comprising 14 sleep diaries, a general information form, an anxiety questionnaire, a depression questionnaire, and other daytime-functioning measures. Participants were to complete the questionnaires after completing the 14 sleep diaries. A more detailed description of the procedures used in this study are given in a previously published book.[4] The University of Memphis institutional review board on human research approved all methods used, and appropriate informed consent was obtained from all participants.

Measures

General Information Form

A general information form[4] was used to collect data on age, ethnicity, sleep-disorder presence, sleep-disorder symptoms, health status, height, weight, cigarette use, alcohol use, and caffeine use. This form was developed by our research group and does not have reliability and validity data. Participants were asked if they had a sleep problem and then to define it. Participants were excluded from these analyses if they reported anything other than no sleep problem or insomnia (e.g., apnea).

A health problem was operationally defined as an affirmative answer to 1 of the following global disease categories: heart disease, cancer, AIDS, high blood pressure, neurologic disease (e.g., seizures, Parkinson disease), breathing problems (e.g., asthma, emphysema), urinary problems (e.g., kidney disease, prostate problems), diabetes, chronic pain (e.g., arthritis, back pain, migraines), and gastrointestinal problems (e.g., stomach, irritable bowel syndrome, ulcers).

Symptoms of an occult sleep disorder were operationally defined as an affirmative answer to 1 of the following questions: Are you a heavy snorer? (snoring); Do you have difficulty breathing or gasp for breath during sleep? (apnea); Do your legs jerk frequently during sleep or do they feel restless before sleep onset? (periodic limb movement disorder or restless legs syndrome). Affirmative answers to these items were controlled for in the following analyses.

Beck Depression Inventory

Daytime mood was assessed with the Beck Depression Inventory (BDI),[23] which is a 21-item measure of depression with scores ranging from 0 to 63. Higher scores indicate greater depression. It is among the most widely used depression measures and has extensive reliability and validity data.[24] Participants were further categorized as having minimal depression (BDI < 10) or clinically significant depression (moderate to severe) (BDI > 18). These cutoff scores were based on values derived by independent raters of psychiatric patients in an inpatient environment.[24]

State-Trait Anxiety Inventory-Form Y Trait Scale

Daytime trait levels of anxiety were assessed with the State-Trait Anxiety Inventory (STAI),[25] which is a 20-item inventory, with scores ranging from 20 to 80. Higher scores indicate greater anxiety. The STAI is one of the most commonly used self-report anxiety measures and has adequate reliability and validity.[26] Participants were further categorized as having minimal anxiety (T-score of < 50) or clinically significant anxiety (T-score of > 70). These cutoff scores represent the mean and 2 SD above the mean for working adults, respectively.[26]

Sleep Diaries

Participants completed sleep diaries[4] upon arising each morning for a 2-week period. The diaries asked participants to give an estimate of their sleep the night before (e.g., bedtime, sleep onset). Typically, overnight sleep studies are considered the gold standard for assessment of sleep disorders.[27,28] Although researchers have found that people with insomnia consistently underestimate TST and overestimate SOLs in comparison with polysomnography, correlations between sleep diaries and polysomnography remain high (r = .63-.87) and sleep diaries are better than single-point retrospective estimates of typical sleep.[28] Further, the Standards of Practice Committee of the American Sleep Disorders Association[29] has stated that polysomnography is only "indicated when a sleep-related breathing disorder or periodic limb movement disorder is suspected, initial diagnosis is uncertain, treatment fails, or precipitous arousals occur with violent or injurious behavior," and that it is "not indicated for the routine evaluation of …chronic insomnia, or insomnia associated with psychiatric disorders." Although sleep diaries are not optimum for examining the relationship between insomnia, depression, and anxiety, they do closely approximate what occurs in clinical practice, making our results highly generalizable.

The following variables were calculated using data from the sleep diaries: mean SOL, mean number of awakenings during the night, mean wake time after sleep onset (WASO), mean TST, mean SE (the ratio of TST to total time spent in bed x 100), mean sleep quality rating (1 = very poor to 5 = excellent), mean total

Table 1—Frequency Analysis of Demographic, Sleep, Depression, and Anxiety Features in Full Sample*

Variables	No.	%
Sex		
Men	381	49.4
Women	391	50.6
Ethnicity		
African American	223	28.9
Caucasian	539	69.8
Hispanic	1	0.1
Asian	7	0.9
Missing	2	0.3
Sleep		
People not having insomnia	393	50.9
People with insomnia	151	19.6
False negatives†	106	13.7
False positives‡	84	10.9
Sleep Apnea	17	2.2
Periodic limb movement/restless legs	6	0.8
Insomnia, not chronic	5	0.6
Hypersomnia	4	0.5
Narcolepsy	2	0.3
Other	2	0.3
Unclassifiable	2	0.3
Psychiatric		
Clinically significant depression	74	9.6
Clinically significant anxiety	63	8.2

*Sample size = 772
†People complaining of insomnia but without the insomnia sleep pattern
‡People not complaining of insomnia but having the insomnia sleep pattern

nap time, and insomnia frequency (number of episodes of insomnia per 2 weeks).

People with insomnia were operationally defined as individuals with a self-report of insomnia for at least 6 months; a daytime complaint; and at least 3 nights a week of (1) SOL ≥ 31 minutes, (2) WASO ≥ 31 minutes, or (3) a combination of the 2. These criteria have been empirically validated and represent the most common practice in insomnia treatment research.[30]

Insomnia types were operationally defined as Onset (SOL criteria ≥ 3 times a week), Maintenance (WASO criteria ≥ 3 times a week), Mixed (if the neither SOL or WASO criteria were ≥ 3 times a week, when SOL and WASO criteria were combined, they occurred ≥ 3 times a week), and Combined (both SOL and WASO criteria were met ≥ 3 times a week). Participants in the combined group were not included in the onset or maintenance groups. Documenting insomnia types is common clinical practice, but the differences between the types have not been widely explored.

All of the following statistical analyses were performed using SPSS 12.0.1 for Windows (SPSS, Inc. Chicago, IL).

RESULTS

Participants

A total of 1,769 volunteers were recruited, with an adjusted response rate of 37.7%.[4] Table 1 shows the demographic, sleep-disorder, and psychiatric variable distributions of the final sample of 772 participants. A series of simple multivariate logistic regressions were performed on this sample of 772 participants, with people with insomnia and people not having insomnia as dependent variables, and all demographics (age, ethnicity, sex) entered simultaneously as the independent variables. These results show that women were 1.8 times more likely than men to have insomnia (95% confidence interval [CI] = 1.23-2.67, P < .01). People with insomnia were also older (61.5 vs 50.4 years) than people not having insomnia (P < .001).

Table 2—Results of Confounding Variables Analyses in the Samples* of People With Insomnia Versus People Not Having Insomnia

Independent variables	Dependent variables	Confounds†
Insomnia Status	BDI	BMI, cigarettes, diabetes, pain, PLMD/RLS
	STAI	None
	Dichotomous BDI	Cigarettes, neurological problems, PLMD/RLS
	Dichotomous STAI	None
Insomnia Types	BDI	BMI, cancer, diabetes, respiratory problems
	STAI	Age, diabetes, ethnicity, snoring
Insomnia Severity	BDI	Age, BMI, cigarettes, diabetes
	STAI	Age, BMI, ethnicity, diabetes, heart problems, snoring, urinary problems

*Sample size = 534.
†Confounds are listed alphabetically.
BDI refers to Beck Depression Inventory; BMI, body mass index; PLMD/RLS, periodic limb movement or restless legs syndrome symptoms; STAI, State-Trait Anxiety Inventory-Form Y trait scale.

Participants were excluded from the following analyses if they met any of the following criteria: no ethnicity data given, Asian or Hispanic descent, any reported sleep disorder other than insomnia, transient insomnia, insomnia sleep pattern but no insomnia complaint, or insomnia complaint without insomnia sleep pattern. Asians, Hispanics, and those with no ethnicity data were excluded from the following analyses because there were so few of them and so that we could examine the differences between African Americans and Caucasians. We included only those individuals defined as people with insomnia or people not having insomnia in order to have a "cleaner" comparison of the 2 groups. Individuals not complaining of insomnia but having the insomnia sleep pattern were excluded because standard clinical diagnostic systems specifically require a complaint of insomnia for a diagnosis of insomnia.[15, 31]

After these exclusions, the final sample, which will be used in all further analyses, numbered 534 participants, with 150 people with insomnia and 384 people not having insomnia (19.7% and 50.4%, respectively).

Confounding Variable Analysis

As described in the introduction, past epidemiologic research often has not controlled for potential confounding variables (e.g., demographics, sleep disorders, or medical disorders). Because of the large number of possible confounders, we decided to use a combination of approaches recommended by Mickey and Greenland[32] for determining which variables represented significant

Table 3—Beck Depression Inventory for Insomnia Status, Ethnicity, and Sex in the Samples* of People with Insomnia Versus People Not Having Insomnia

	No.†	Unadjusted Mean (SD)	Adjusted‡ Mean (SD)
Insomnia			
Not Having Insomnia	379	5.72 (5.37)	6.63*** (7.19)
With Insomnia	148	13.08 (8.93)	12.42*** (6.79)
Ethnicity			
Caucasian	393	7.24 (6.68)	8.59** (6.93)
African American	134	9.40 (8.85)	10.45** (6.46)
Sex			
Men	267	6.89 (6.51)	8.95* (7.34)
Women	260	8.72 (8.02)	10.09* (6.92)

*Sample size of 527
†Seven subjects excluded from these analyses of covariance due to missing data in 1 of the covariates.
‡Means with the following covariates in the model: PLMD/RLS, diabetes, pain, body mass index, cigarette use.
***P < .001
**P < .01
*P < .05

confounds. Univariate analyses were first performed with possible confounders as the independent variables, and depression and anxiety scores as the dependent variables. Those independent variables significant at the P < .20 level were added as covariates to a base model containing insomnia status, insomnia type, or insomnia severity measures as the independent variables and depression or anxiety scores as the dependent variables. If the unadjusted estimate of any independent variables changed by more than 15% when the possible confounding variables were added, then the base model was abandoned in favor of this new adjusted model. Covariates were then taken out one at a time until the adjusted coefficient of the independent variables changed appreciably (> 15%), at which time that variable was placed back into the model and this model was considered the final model. This approach should ensure an unbiased estimate of the relationship between insomnia, depression, and anxiety, while adequately controlling for confounding variables. Table 2 shows those variables that were found to be confounders in each analysis using the above procedures.

Sex and Ethnicity

Ethnicity and sex were also included in the insomnia-status analyses of covariance and logistic regressions. This was done because (1) limited data are available comparing ethnicities on depression and anxiety, some of which are conflicting[33-35] and (2) previous research from our laboratory has shown significant differences regarding depression, anxiety, and sleep problems between ethnic groups and sexes.[4] When race, sex, or their interaction with insomnia were nonsignificant, these variables were dropped from the final model, but their frequency counts or means were reported in the accompanying tables.

Insomnia Presence and BDI and STAI Scores

First, to determine if people with insomnia had higher levels of depression and anxiety levels than people not having insomnia, we performed 2 (insomnia status) x 2 (sex) x 2 (ethnicity) analyses of covariance, with either BDI or STAI mean scores as the dependent variables and the confounds listed in Table 2 as covariates. It is important to note that analysis of covariance performs means testing on adjusted means.

Table 4—Unadjusted Means on the State-Trait Anxiety Inventory for Insomnia Status, Ethnicity, and Sex

	No.	Mean (SD)
Insomnia		
Not having insomnia	384***	33.14 (9.33)
With insomnia	150***	42.26 (11.70)
Ethnicity		
African American	138*	38.52 (12.12)
Caucasian	396*	34.72 (10.20)
Sex		
Men	270	34.62 (10.45)
Women	264	36.82 (11.15)
Insomnia × Ethnicity		
Not having insomnia		
African American	96	34.33 (10.56)
Caucasian	288	32.75 (8.87)
With insomnia		
African American	42**	48.12 (9.85)
Caucasian	108**	39.98 (11.61)

***P < .001
**P < .01
*P < .05

As can be seen in Table 3, after controlling for confounding variables (Table 2), people with insomnia had significantly higher BDI scores than people not having insomnia ($F_{1,518}$ = 75.38, P < .001, partial η^2 = 0.13), African Americans had higher BDI scores than Caucasians ($F_{1,518}$ = 8.65, P < .01, partial η^2= 0.02), and women had higher BDI scores than men (P = 4.31, P < .05, partial η^2= 0.01). The interaction terms were not significant.

As can be seen in Table 4, after controlling for confounding variables (Table 2), people with insomnia had significantly higher STAI scores than people not having insomnia ($F_{1,530}$ = 96.78, P < .001, partial η^2= 0.15), and African Americans had higher STAI scores than Caucasians ($F_{1,530}$ = 20.67, P < .001, partial η^2= 0.04). There was also a significant insomnia status-by-ethnicity interaction ($F_{1,530}$ = 9.44, P < .01, partial η^2= 0.02), in which African American people with insomnia had higher STAI scores than Caucasian people with insomnia ($F_{1,529}$ = 20.60, P < .001), but no differences were seen between ethnicity groups in people not having insomnia. There were no differences between sexes on STAI scores.

Insomnia Presence and Clinically Significant Depression and Anxiety

Next, to determine if people with insomnia had more clinically significant depression or anxiety than people not having insomnia, we dichotomized the BDI and STAI scores (described in measures). We then used simple multivariate logistic regression, with insomnia status (people with insomnia vs people not having insomnia) entered simultaneously with sex, ethnicity, and confounds (Table 2) as the independent variables and clinically significant depression or anxiety as the dependent variables.

A total of 7.9% of the final sample had clinically significant depression as measured by the BDI, which is comparable to National Institute of Mental Health data showing that 9.5% of the

Table 5—Unadjusted Prevalence Rates of Beck Depression Inventory Scores Indicative of Minimal Depression or Clinically Significant Depression for Insomnia Status, Ethnicity, and Sex

Variable	Group	Depression Levels	
		Minimal/None % (no.)	Clinically Significant % (no.)
Insomnia			
	With insomnia	37.3 (56)	20*** (30)
	Not having insomnia	84.6 (325)	3.1*** (12)
Ethnicity			
	African American	62.3 (86)	14.5** (20)
	Caucasian	74.5 (295)	5.6** (22)
Sex			
	Men	77 (208)	5.6 (15)
	Women	65.5 (173)	10.2 (27)

Percentage refers to the percentage of people within the group with minimal (Beck Depression Inventory [BDI] < 10) or clinically significant depression (BDI > 18). Percentages do not total to 100% because those individuals with BDI scores > 10 and < 18 were not included in the table.
***P < .001
**P < .01.

population suffers from major depressive disorder.[36] Further, (see Table 5) people with insomnia were 9.82 times more likely than people not having insomnia to have clinically significant depression (95% CI= 4.41-21.86, P < .001), and African Americans were 3.43 times more likely than Caucasians to have clinically significant depression (95% CI= 1.54-7.67, P < .01) after controlling for confounds (Table 2). There were no sex differences, and none of the interaction terms were significant.

Some researchers have suggested that race is simply a proxy for socioeconomic status. Indeed, studies that assess both ethnicity and social and economic factors routinely find that ethnic discrepancies are either removed or minimized when variables such as socioeconomic and health status are factored out.[37-39] To answer this question, we performed a simple logistic regression with race and highest level of education (a frequent measure of socioeconomic status) entered simultaneously as independent variables and clinically significant depression as the dependent variables. This model revealed that African Americans were still 2.3 times (P < .05) more likely to have clinically significant depression after controlling for the fact that people with clinically significant depression had significantly lower levels of education than people with no depression (12.77 vs 14.79; P < .01).

A total of 7.5% of the final sample had clinically significant anxiety as measured by the STAI. As can be seen in Table 6, people with insomnia were 17.35 times more likely than people not having insomnia to have clinically significant anxiety (95% CI= 7.62-39.49, P < .001), and African Americans were 4.80 times more likely than Caucasians to have clinically significant anxiety (95% CI= 2.10-10.94, P < .001) after controlling for confounds (Table 2). There were no sex differences, and the interactions were nonsignificant.

To again answer the question of race as a proxy for socioeconomic status, we performed another simple logistic regression with race and highest level of education entered simultaneously as independent variables and clinically significant anxiety as the dependent variable. This model revealed that African Americans

Table 6—Unadjusted Prevalence Rates of State-Trait Anxiety Inventory Scores Indicative of Minimal Anxiety or Clinically Significant Anxiety for Insomnia Status, Ethnicity and Sex

Variable	Group	Anxiety Levels	
		Minimal/ None % (no.)	Clinically Significant % (no.)
Insomnia			
	People with insomnia	31.3 (47)	19.3*** (29)
	People not having insomnia	65.6 (252)	2.9*** (11)
Ethnicity			
	African American	46.4 (64)	13.8*** (19)
	Caucasian	59.3 (235)	5.3*** (21)
Sex			
	Men	60 (162)	6.3 (17)
	Women	51.9 (137)	8.7 (23)

Percentage refers to the percentage of people within the group with minimal (T-score < 50) or clinically significant (T-score >70) anxiety. Percentages do not total to 100% because those individuals with State-Trait Anxiety Inventory T-scores < 50 and > 70 were not included in the table.
***P < .001.

were still 2.8 times (P < .05) more likely to have clinically significant anxiety after controlling for the fact that people with clinically significant anxiety had significantly lower levels of education than people with no anxiety (12.87 vs 14.77; P < .01).

Insomnia Severity and BDI and STAI scores

Hierarchical linear multiple regression analyses were performed within the people with insomnia group to determine if plausible insomnia severity variables (ie, duration of insomnia, SOL, WASO, number of awakenings during the night, TST, SE, and insomnia frequency) were significantly related to depression and anxiety scores after controlling for confound variables (Table 2). Confound variables were entered simultaneously into the first block, and then insomnia severity variables were entered into the second block using a stepwise method, with either BDI or STAI scores as the dependent variables.

Overall, the regression equation was a good fit for BDI scores, accounting for 36.7% of the variation in scores ($F_{6,143}$ = 13.05, P < .001). When all other variables were held constant, BDI scores increased by 0.17 SD for every 1 SD increase in insomnia frequency (t_{143} = 2.28, P < .05), and BDI scores increased by 0.16 SD for every 1 SD increase in the mean number of awakenings per night (t_{143} = 2.10, P < .05).

The regression equation was also a good fit for the STAI, accounting for 35.7% of the variation in scores ($F_{6,139}$ = 11.22, P < .001). When all other variables were held constant, STAI scores increased by 0.20 SD for every 1 SD increase in insomnia frequency, (t_{139} = 2.85, P < .01). TST and WASO were no longer significantly related to STAI scores after controlling for confounds.

Insomnia Types and BDI and STAI Scores

The most common type of insomnia was maintenance, (34% of people with insomnia), followed by combined (ie, both SOL

Table 7—Unadjusted and Adjusted Means on the Beck Depression Inventory for Insomnia Status, Ethnicity, and Sex

	No.	Unadjusted Mean (SD)	Adjusted** Mean (SD)
Onset Insomnia	35	12.11 (9.59)	11.93 (7.52)
Maintenance Insomnia	51	11.90 (8.43)	12.73 (7.50)
Mixed Insomnia	24	10.50 (4.89)	10.64 (7.53)
Combined Insomnia	39	17.36 (9.79)	16.35* (7.58)

**Means with the following covariates in the model: apnea, cancer, diabetes, age, body mass index, and cigarette use.
*P < .05

and WASO criteria were met) (26.7%), onset (23.3%), and mixed (16%). Insomnia types differed only on ethnicity, (χ^2_3 = 13.37 ([N=150], P < .01), with a larger proportion of African Americans in the onset group and combined groups than in the mixed or maintenance groups (all P values < .05).

To determine if people with different insomnia types had differing levels of depression and anxiety, we performed analyses of covariance, with insomnia types as the independent variables, either BDI or STAI mean scores as the dependent variables, and the confounds listed in Table 2 as covariates. Main effects and interactions of ethnicity and sex were not examined in these analyses due to inadequate cell sizes.

Table 7 shows significant differences between insomnia types on the BDI ($F_{3,139}$ = 3.40, P < .05, partial η^2= 0.07) after controlling for confounds (Table 2). Posthoc testing on adjusted means, using the Student-Newman-Keuls test, found that people with combined insomnia had higher BDI scores than any other group (all P values < .05).

A one-way analysis of covariance with STAI as the dependent variable showed a nonsignificant trend (P = .06) similar to that of the BDI data, after controlling for confounds (Table 2).

DISCUSSION

After controlling for confounding variables, people with insomnia had significantly higher levels of depression and anxiety than people not having insomnia. When those scores were dichotomized, people with insomnia were 9.82 times more likely than people not having insomnia to have clinically significant depression and 17.35 times more likely to have clinically significant anxiety. These results are not surprising considering that many previous studies have found significant relationships between insomnia, depression, and anxiety.[1,3,6-11,13,14] However, this is the first study to exclude individuals with a report of an organic sleep disorder and to evaluate and control for possible confounds (e.g., medical disorders, ethnicity, sex) to maximize the likelihood of an unbiased estimate of the relationship between insomnia, depression, and anxiety.

Next, after controlling for significant confounds, we found that, as insomnia frequency increased, so too did both BDI and STAI scores. In addition, as the mean number of awakenings per night increased, so too did BDI scores. It was surprising that the measures traditionally considered when studying insomnia (ie, SOL, WASO, TST, SE) were not significantly related to depression and anxiety. It appears that insomnia frequency is more closely related to depression and anxiety severity than is the portion of night during which the insomnia occurs or how much sleep the individual is getting. This finding strongly suggests that future treatment studies should report these variables as outcomes, as well as those more traditionally reported.

People with combined insomnia had higher depression scores than all other groups, but no differences were found between the other groups. No differences were found on anxiety scores between insomnia types. These results are in agreement with what the majority of other researchers in this field have found,[17-21] in that traditional insomnia-type groups (ie, onset and maintenance) were not different on measures of depression and anxiety. Our study did show that individuals with combined insomnia (ie, satisfied multiple definitions) had more depression than did individuals with other insomnia types. But to be fair, those individuals with combined insomnia simply have more-severe insomnia (ie, more episodes) and would be expected to have more daytime consequences, as displayed by the severity analyses above.

African Americans had higher levels of anxiety and depression than Caucasians. African Americans were 3.4 times more likely to have clinically significant depression and 4.8 times more likely to have clinically significant anxiety. These results support those that have been found by other researchers.[33,35,40] One explanation for these results could be that African Americans are more likely than Caucasians to be exposed to a variety of psychosocial stressors—such as discrimination, socioeconomic hardship, and increased caregiver burden—that may contribute to increased distress, resulting in higher levels of depression and anxiety.[41,42]

It is also important to note that our ethnicity results are in disagreement with those of some other researchers.[43,44] There are some possible explanations for this disagreement. One is that studies have shown that African Americans universally underreport health problems.[45-47] Thus, it is plausible that African Americans were more likely to endorse symptoms on our questionnaires than they were to endorse symptoms during an interview, as has been done in previous studies. Another explanation might be that race is simply a proxy for socioeconomic status and that the often unmeasured socioeconomic factors are the real culprit behind racial disparities. However, after we controlled for socioeconomic status in our study, African Americans still had 2.4 and 2.8 times more clinically significant depression and anxiety, respectively.

Women were more likely than men to have insomnia and had higher depression levels but not higher anxiety levels. No differences were seen between the sexes on clinically significant depression or anxiety. With the exception of finding no sex differences on anxiety, the sex results were essentially what was expected and were in agreement with what the majority of past research has found.[13,18,22,44,48] It is still unclear why women have more depression and insomnia than men. One theory is that fluctuating hormone levels may play a role.[49] A second theory relates to different coping styles, with women being more likely to ruminate on their problems, whereas men are more apt to distract themselves or use drugs and alcohol to cope.[50] An American Psychological Association panel attributed the differences to women being exposed to higher levels of stressful events.[51]

Unfortunately, this study was unable to provide differential diagnoses of depression, anxiety and medical disorders. Even though BDI and STAI scores are accurate measures of depression and anxiety, only clinical interviewing is effective in diagnosing these disorders. With regard to medical disorders, individuals often have health problems that they are unaware of until they undergo appropriate physical and laboratory testing (e.g., diabe-

tes, cancer). However, this is one of the first studies to take into account known medical problems.

The current study also did not obtain detailed sleep, medical, and psychiatric histories, making it impossible to determine if the symptoms of depression and anxiety or insomnia emerged first. Even if detailed histories had been obtained, due to the nature of epidemiology, a causative relationship could not have been established. However, we believe that it is plausible that a reciprocal relationship exists between insomnia and these disorders.

The effects of insomnia on depression and anxiety can be described by the diathesis-stress model.[52] This model states that individuals are predisposed to develop certain diseases (e.g., anxiety or depression), and stress serves as a catalyst for the development of the disease. In such a model, the stress created by an episode of insomnia might exacerbate or instigate the development or recurrence of depression or anxiety. Significant time spent awake in bed in the dark might serve as a blank palette on which to paint remembrances of past failures or worrying about future disasters and the next day's responsibilities, thus causing increases in depression and anxiety. Conversely, the daytime ruminations typically associated with depression and anxiety (e.g., ruminations of past failures, depressing relationships, the next day's responsibilities, worrying about future disasters) likely spill over into the bedroom at night, increasing mental and physiological arousal and, thus, inducing insomnia.

The results of this study show that there are clearly a large proportion of people with insomnia who show clinically significant depression (20%) and anxiety (19.3%). Due to the possible reciprocal relationship of these disorders, more effort needs to be focused on explicating the exact nature of the relationship and finding ways to intervene. One area of future research that is strongly needed is a prospective epidemiologic study of sleep, with adequate definitions of insomnia, as well as the documentation of psychological and medical disorders through structured psychological interviews, medical histories, physical examinations, and laboratory testing. Although several prospective studies have been performed in this area (for a review, see reference 6), they all suffered from the same limitations of the cross-sectional studies described in the introduction. In addition, further studies need to be designed to determine the best method of treating these individuals, since they are generally excluded from current treatment studies, but they obviously represent a large percentage of the sample of people with insomnia. Studies also should be undertaken to determine what benefit it would be to specifically treat insomnia in patients with major depressive disorder to determine if this might improve depression response and relapse rates.

REFERENCES

1. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? JAMA 1989;262:1479-84.
2. Gallup Organization. Sleep In America: 1995. Princeton, NJ: Gallup; 1995.
3. Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment. Prevalence and correlates. Arch Gen Psychiatry 1985;42:225-32.
4. Lichstein KL., Durrence, HH, Riedel, BW, Bush, AJ. Epidemiology of sleep: age, gender, and ethnicity. Mahwah, NJ: Lawrence Erlbaum.; 2004.
5. Walsh JK, Engelhardt CL. The direct economic costs of insomnia in the United States for 1995. Sleep 1999;22 Suppl 2:S386-93.
6. Taylor DJ, Lichstein KL, Durrence HH. Insomnia as a health risk factor. Behav Sleep Med 2003;1:227.
7. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry 1996;39:411-8.
8. Dryman A, Eaton WW. Affective symptoms associated with the onset of major depression in the community: findings from the US National Institute of Mental Health Epidemiologic Catchment Area Program. Acta Psychiatr Scand 1991;84:1-5.
9. Vollrath M, Wicki W, Angst J. The Zurich study. VIII. Insomnia: association with depression, anxiety, somatic syndromes, and course of insomnia. Eur Arch Psychiatry Neurol Sci 1989;239:113-24.
10. Weissman MM, Greenwald S, Nino-Murcia G, Dement WC. The morbidity of insomnia uncomplicated by psychiatric disorders. Gen Hosp Psychiatry 1997;19:245-50.
11. Roberts RE, Shema SJ, Kaplan GA, Strawbridge WJ. Sleep complaints and depression in an aging cohort: A prospective perspective. Am J Psychiatry 2000;157:81-8.
12. Livingston G, Blizard B, Mann A. Does sleep disturbance predict depression in elderly people? A study in inner London. Br J Gen Pract 1993;43:445-8.
13. Bixler EO, Kales A, Soldatos CR, Kales JD, Healey S. Prevalence of sleep disorders in the Los Angeles metropolitan area. Am J Psychiatry 1979;136:1257-62.
14. Kuppermann M, Lubeck DP, Mazonson PD, et al. Sleep problems and their correlates in a working population. J Gen Intern Med 1995;10:25-32.
15. American Psychiatric Association. Diagnostic and statistical manual of mental disorders-Text Revision. 4th ed. Washington: American Psychiatric Association; 2000.
16. Ohayon MM. Prevalence of DSM-independent variables diagnostic criteria of insomnia: distinguishing insomnia related to mental disorders from sleep disorders. J Psychiatr Res 1997;31:333-46.
17. Brabbins CJ, Dewey ME, Copeland JRM, et al. Insomnia in the elderly: Prevalence, gender differences and relationships with morbidity and mortality. Int J Geriatr Psychiatry 1993;8:473-80.
18. Karacan I, Thornby JI, Williams RL. Sleep disturbance: a community survey. In: Guilleminault C, Lugaresi E, editors. Sleep/Wake Disorders: Natural History, Epidemiology, and Long-term Evolution. New York: Raven Press; 1983:37-60.
19. Kim K, Uchiyama M, Okawa M, Liu X, Ogihara R. An epidemiological study of insomnia among the Japanese general population. Sleep 2000;23:41-7.
20. Newman AB, Enright PL, Manolio TA, Haponik EF, Wahl PW. Sleep disturbance, psychosocial correlates, and cardiovascular disease in 5201 older adults: the cardiovascular health study. J Am Geriatr Soc 1997;45:1-7.
21. Quera-Salva MA, Orluc A, Goldenberg F, Guilleminault C. Insomnia and use of hypnotics: study of a French population. Sleep 1991;14:386-91.
22. Gislason T, Reynisdottir H, Kristbjarnarson H, Benediktsdottir B. Sleep habits and sleep disturbances among the elderly—an epidemiological survey. J Intern Med 1993;234:31-9.
23. Beck AT, Steer RA. Beck Depression Inventory: Manual. San Antonio: The Psychological Corporation; 1993.
24. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. Clin Psychol Rev 1988;8:77-100.
25. Spielberger CD, Gorsuch RL, Lushene RE. State-Trait Anxiety Inventory. Palo Alto: Consulting Psychologists Press; 1970.
26. Spielberger CD, Gorsuch, RL, Lushene, PR, Jacobs, GA. Manual for the State-Trait Anxiety Inventory: STAI (form Y). Palo Alto: Consulting Psychologists Press, Inc; 1983.
27. Carskadon MA, Dement WC, Mitler MM, Guilleminault C, Zarcone VP, Spiegel R. Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. Am J Psychiatry 1976;133:1382-8.

28. Coursey RD, Frankel BL, Gaarder KR, Mott DE. A comparison of relaxation techniques with electrosleep therapy for chronic, sleep-onset insomnia a sleep-EEG study. Biofeedback Self Regul 1980;5:57-73.
29. Littner M, Hirshkowitz M, Kramer M, et al. Practice parameters for using polysomnography to evaluate insomnia: an update. Sleep 2003;26:754-60.
30. Lichstein KL, Durrence HH, Taylor DJ, Bush AJ, Riedel BW. Quantitative criteria for insomnia. Behav Res Ther 2003;41:427-45.
31. The International Classification of Sleep Disorders: Diagnostic and Coding Manual. Rochester, MN: American Sleep Disorders Association.; 1990.
32. Mickey RM, Greenland S. The impact of confounder selection criteria on effect estimation. Am J Epidemiol 1989;129:125-37.
33. Blazer D, George LK, Landerman R, Pennybacker M, Melville ML, Woodbury M, et al. Psychiatric disorders: A rural/urban comparison. Arch Gen Psychiatry 1984;42:651-6.
34. Horwath E, Johnson J, Hornig CD. Epidemiology of panic disorder in African-Americans. Am J Psychiatry 1993;150:465-9.
35. Robins LN, Helzer JE, Weissman MM, et al. Lifetime prevalence of specific psychiatric disorders in three sites. Arch Gen Psychiatry 1984;41:949-58.
36. Regier DA, Narrow WE, Rae DS, Manderscheid RW, et al. The de facto US mental and addictive disorders service system: Epidemiologic Catchment Area prospective 1-year prevalence rates of disorders and services. Arch Gen Psychiatry 1993;50:85-94.
37. Adler NE, Ostrove JM. Socioeconomic status and health: What we know and what we don't. 1999:3.
38. Kubik K, Blackwell L, Heit M. Does socioeconomic status explain racial differences in urinary incontinence knowledge? Am J Obstet Gynecol 2004;191:188-93.
39. Scharf SM, Seiden L, DeMore J, Carter-Pokras O. Racial differences in clinical presentation of patients with sleep-disordered breathing. Sleep Breath 2004;8:173-83.
40. Neal AM, Turner SM. Anxiety disorders research with African Americans: Current status. Psychol Bull 1991;109:400.
41. Anderson NA. Racial differences in stress-induced cardiovascular reactivity and hypertension: Current status and substantive issues. v1989;105:89-105.
42. Vitaliano PP, Russo J, Bailey SL, Young HM, McCann BS. Psychosocial factors associated with cardiovascular reactivity in older adults. Psychosom Med 1993;55:164-77.
43. Zhang AY, Snowden LR. Ethnic characteristics of mental disorders in five U.S. communities. Cultural Diversity Ethnic Minority Psychol 1999;5:134.
44. Blazer DG, Kessler RC, McGonagle KA, Swartz MS. The prevalence and distribution of major depression in a national community sample: the National Comorbidity Survey. Am J Psychiatry 1994;151:979.
45. Haley WE, Roth DL, Coleton MI, et al. Appraisal, coping, and social support as mediators of well-being in Black and White family caregivers of patients with Alzheimer's disease. J Consult Clin Psychol 1996;64:121.
46. Jean-Louis G, Magai CM, Cohen CI, Zizi F, von Gizycki H, DiPalma J, et al. Ethnic differences in self-reported sleep problems in older adults. Sleep 2001;24:926-33.
47. Knight BG, McCallum TJ. Heart rate reactivity and depression in African-American and white dementia caregivers: Reporting bias or positive coping? Aging Ment Health 1998;2:212.
48. Kessler RC, McGonagle KA, Swartz M, Blazer DG. Sex and depression in the National Comorbidity Survey: I. Lifetime prevalence, chronicity and recurrence. J Affect Disord 1993;29:85.
49. Seeman MV. Psychopathology in women and men: focus on female hormones. Am J Psychiatry 1997;154:1641-7.
50. Nolen-Hoeksema S. Sex differences in unipolar depression: evidence and theory. Psychol Bull 1987;101:259-82.
51. McGrath E, Keita GP, Strickland BR, Russo NF. Women and depression: risk factors and treatment issues: Final report of the American Psychological Association's National Task Force on Women and Depression; 1990.
52. Monroe SM, Simons AD. Diathesis-stress theories in the context of life stress research: implications for the depressive disorders. Psychol Bull 1991;110:406-25.