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Depression: relationships to sleep paralysis and other sleep disturbances in a community sample

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SUMMARY

Sleep disturbances are important correlates of depression, with epidemiologic research heretofore focused on insomnia and sleepiness. This epidemiologic study's aim was to investigate, in a community sample, depression's relationships to other sleep disturbances: sleep paralysis (SP), hypnagogic/hypnopompic hallucinations (HH), cataplexy – considered rapid eye movement-related disturbances – and automatic behavior (AB). Although typical of narcolepsy, these disturbances are prevalent, albeit under-studied, in the population. Cross-sectional analyses (1998–2002), based on Wisconsin Sleep Cohort Study population-based data from 866 participants (mean age 54, 53% male), examined: depression (Zung Self-Rating Depression Scale), trait anxiety (Spielberger State-Trait Anxiety Inventory, STAI-T ≥ 75th percentile), and self-reported sleep disturbances. Descriptive sleep data were obtained by overnight polysomnography. Adjusted logistic regression models estimated depression's associations with each (>few times ever) outcome – SP, HH, AB, and cataplexy. Depression's associations with self-reported SP and cataplexy were not explained by anxiety. After anxiety adjustment, severe depression (Zung ≥55), vis-à-vis Zung <50, increased SP odds ~500% ($P = 0.0008$). Depression (Zung ≥50), after stratification by anxiety given an interaction ($P = 0.02$), increased self-reported cataplexy odds in non-anxious (OR 8.9, $P = 0.0008$) but not anxious (OR 1.1, $P = 0.82$) participants. Insomnia and sleepiness seemed only partial mediators or confounders for depression's associations with self-reported cataplexy and SP. Anxiety (OR 1.9, $P = 0.04$) partially explained depression's (Zung ≥55) association with HH (OR 2.2, $P = 0.08$). Anxiety (OR 1.6, $P = 0.02$) was also more related than depression to AB. Recognizing depression's relationships to oft-neglected sleep disturbances, most notably SP, might assist in better characterizing depression and the full range of its associated sleep problems in the population. Longitudinal studies are warranted to elucidate mediators and causality.

Keywords

depression; epidemiology; hypnagogic hallucinations; sleep disturbances; sleep paralysis

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INTRODUCTION

Sleep disturbances are well-established correlates of depression (Breslau *et al.*, 1996; Ford and Kamerow, 1989) and the most prevalent persisting residual symptoms among antidepressant responders (Nierenberg *et al.*, 1999). In clinical polysomnographic studies, depression has been predicted (Perlis *et al.*, 1997) and descriptively characterized by sleep disturbances, including poor sleep (e.g. increased awakenings) and rapid eye movement (REM) sleep stage abnormalities such as shortened REM sleep latency (Akiskal *et al.*, 1982; Kupfer and Foster, 1972; Reynolds *et al.*, 1983; Thase *et al.*, 1984). In epidemiologic studies, depression has been associated with increased insomnia and hypersomnia (Breslau *et al.*, 1996; Ford and Kamerow, 1989) or excessive daytime sleepiness (Hublin *et al.*, 1996; Ohayon *et al.*, 1997). Depression's relationships to other sleep-related disturbances have seldom been investigated in the population. Therefore, in the present study, we hypothesized that depression would be more prevalent in individuals with sleep paralysis (SP), hypnagogic/hypnopompic hallucinations (HH) and cataplexy – considered dissociated manifestations of REM sleep (Hishikawa and Shimizu, 1995; Roth *et al.*, 1968a), and also in automatic behavior (AB) – typified by semi-ABs and mishaps, and linked clinically to drowsiness periods and micro-sleeps (Ganado, 1958; Guilleminault *et al.*, 1975). These sleep-related disturbances, along with sleepiness, are core clinical symptoms of narcolepsy (Ganado, 1958; Yoss and Daly, 1957) and usually studied in this context. However, they often occur in the general population and non-clinical groups (Billiard *et al.*, 1987; Hublin *et al.*, 1994; Ohayon *et al.*, 1999; Partinen, 1982; Szklo-Coxe *et al.*, 2007), and can be triggered in non-narcoleptics by nocturnal disruptions (Takeuchi *et al.*, 1992), sleep debt (Mikulincer *et al.*, 1989), and sleepiness (Billiard *et al.*, 1987). Despite prevalence estimates as high as 15–43% for lifetime sleep paralysis in undergraduate and medical students (Cheyne *et al.*, 1999; Everett, 1963; ; Wing *et al.*, 1994), epidemiologic research on these disturbances' psychiatric correlates has heretofore been scant (Ohayon and Shapiro, 2000; Ohayon *et al.*, 1996, 1999).

Present analyses were based on population-based data from the Wisconsin Sleep Cohort Study (WSCS), a longitudinal study of the natural history of sleep disorders (Young *et al.*, 1993). In prior WSCS investigations, self-reported cataplexy, SP, HH, and AB were not found to be related significantly to HLA DQB1*0602, a genetic marker for narcolepsy (Mignot *et al.*, 1997), or consistently to several of narcolepsy's key diagnostic criteria (nocturnal sleep, sleep onset REM sleep periods) (Mignot *et al.*, 2006; Szklo-Coxe *et al.*, 2007), suggesting that they are not definite manifestations of or exclusive to narcolepsy in the population and that other correlates warrant consideration. We presently focus on depression and its relationships to these disturbances while considering anxiety. Regarded as clinically useful from prognostic and therapeutic viewpoints (Clayton *et al.*, 1991; Fava *et al.*, 1997; Paykel, 1972), depression accompanied by anxiety has been associated with increased psychiatric impairment and symtomatology (Brown *et al.*, 1996; Fava *et al.*, 2004; Paykel, 1972; VanValkenburg *et al.*, 1984), including insomnia (Clayton *et al.*, 1991; Coryell *et al.*, 1998). Insomnia and sleepiness were presently considered as potential confounders and/or mediators. They are outcomes (Roberts *et al.*, 1999) and correlates (Hublin *et al.*, 1996; Ohayon *et al.*, 1997) of depression and mood disturbances and have been linked to self-reported cataplexy, HH and SP (Billiard *et al.*, 1987; Ohayon *et al.*, 1996, [1999]; Szklo-Coxe *et al.*, 2007; Wing *et al.*, 1994).

METHODS

The sample comprised men and women enrolled in the WSCS, begun in 1989. WSCS's design has been described previously (Young *et al.*, 1993). Briefly, its overall sampling frame was an employee payroll listing of five state agencies in south central Wisconsin, ages 30–60 years at baseline. A two-stage random-stratified sampling procedure was used. Participation in the WSCS includes an overnight sleep study protocol with full polysomnography (PSG),

completion of the Zung Self-Rating Depression Scale (Zung, 1965) and self-administered Spielberger State-Trait Anxiety Inventory (STAI) (Spielberger *et al.*, 1983), anthropometric measurements, collection of blood, and a questionnaire on sleep variables. The protocol is repeated at 4-year intervals, and the sample is periodically surveyed by mail. The present sample was limited to 866 participants studied December 1998–2002 on whom complete data from the overnight protocol, a mailed survey conducted in 2000, and genotyping on HLA DQB1*0602 as a marker for narcolepsy susceptibility were available. Depression (measured on the overnight protocol) and outcomes (measured on the mailed survey) were thus assessed within ±2 years (December 1998 to December 2002) of one another.

The University of Wisconsin Health Sciences Institutional Review Board approved the study protocol and consent procedures. Participants provided signed informed consent. The evening of overnight study, participants completed the Zung Scale and STAI. Eighteen-channel PSG (Grass Heritage PSG Digital Sleep System with Model 15A54 amplifiers) included electro-oculography, electroencephalography (EEG), and electromyography. Sleep stage for each 30-s epoch was scored blindly by technicians, according to conventional criteria (Rechtschaffen and Kales, 1968). Sleep latency, REM sleep latency, percent REM sleep, total sleep time (TST), wake after sleep onset (WASO), and sleep efficiency measures were obtained from PSG data. Whole blood was collected the morning following overnight PSG. DNA was extracted and samples typed at Stanford University for HLA DQB1*0602's presence, as previously described (Mignot *et al.*, 1999). On the mailed questionnaire, participants completed the Epworth Sleepiness Scale (Johns, 1991) and rated, on Likert scales, frequency of insomnia, and the outcomes – SP, HH, AB, and cataplexy items. Possible response categories for outcomes were (1) never, (2) only a few times ever, (3) rarely: <1/month, (4) sometimes: ≥1/month but <1/week, (5) often: ≥1/week. Presence of each outcome was defined as ever experiencing any individual item describing that outcome >few times ever – or responses of 3, 4, or 5 above. Analyses of individual items are shown in Table 1a. Broadly, increased percentages of depression were found across increasing severity levels (Table A1b). SP referred to 'ever awakening and found you were unable to move your whole body and felt paralyzed' either upon awakening either 'in the morning' and/or 'during your night's sleep'. HH, occurring during wake–sleep (hypnagogic) or sleep–wake (hypnopompic) transitions, referred to 'ever imagined that you hear or see strange and frightening things or people' when 'falling asleep at night' and/or 'wake up in the morning' and/or 'are drowsy'. Positive response to any of these items constituted presence. AB referred to 'ever had times when you suddenly felt like you "went blank" with no memory of that period of time' either 'when driving' and/or 'working at a desk or sitting quietly'. Self-reported cataplexy was defined as 'ever had episodes of muscle weakness in your legs or buckling of your knees' with ≥1 item(s), each representing a typical triggering emotion(s): 'when you laugh' and/or 'are angry' and/or 'tell or hear a joke' (Anic-Labat *et al.*, 1999).

The Zung self-rating depression scale (scaled score range 25–100) measured depression. Zung score ≥50 was considered indicative of clinical depression (Zung, 1965). Of those with depressive symptomatology (Zung ≥50), the median (score 54) was the severity cutpoint, with lesser severity depression defined as Zung 50–54; greater severity as Zung ≥55. Antidepressants were not included in depression's definition because they are prescribed for SP, HH, and cataplexy (Takahashi, 1976) and our cross-sectional data precluded determining depression's temporal relationship to antidepressants. Thus, models: (1) included antidepressants as independent variables/confounders; and (2) excluded antidepressant users.

The self-reported trait-anxiety scale of the State-Trait Anxiety Inventory (STAI-T), scored by summing ratings for 20 items (score range 20–80), measured 'relatively stable individual differences in anxiety-proneness (Spielberger *et al.*, 1983)'. The 75th percentile, used previously in cohort studies (Dayan *et al.*, 2002; Frasure-Smith and Lesperance, 2003), was

obtained from the overall sample (n = 1488) as a cutpoint to define anxiety (STAI-T ≥ 39). Sensitivity analyses using the ≥90th percentile (STAI-T ≥ 46) yielded similar results (Tables A2a and A2b) to the ≥75th percentile regarding depression's associations with outcomes.

Medications were categorized according to National Institute of Mental Health classifications (NIMH, 2002). Antidepressant classes included selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, monoamine oxidase inhibitors, and others. Anxiolytics included alprazolam, buspirone, clonazepam, diazepam, lorazepam, and oxazepam. Daytime sleepiness was defined as Epworth Sleepiness Score (ESS) > 11. Four insomnia variables examined (≥5 times/month) were: 'difficulty getting to sleep', 'wake up repeatedly during the night', 'wake up too early in the morning and can't get back to sleep', and 'wake up during the night and have a hard time getting back to sleep'. Other adjustment variables examined in logistic regression models included age (years), male, body mass index [BMI, weight (kg)/ eight (m^2), and DQB1 positivity – or an HLA DQB1*0602 allele, present in 95% of individuals with narcoleptic cataplexy (Mignot *et al.*, 1997). EEG parameters were: REM sleep latency, defined as time in minutes (min) from sleep onset to first REM sleep epoch; percent REM sleep (%), as time in REM sleep/TST; sleep latency, time (min) from lights off to first occurrence of stage 2 or REM sleep; TST, as total sleep epochs (30 s each); WASO, as time (min) awake after first sleep onset; and sleep efficiency (SE) (%), as TST/time in bed from lights off.

Statistical analysis

To consider their potential inclusion in logistic regression models, variables were analyzed by depression status (Table 1). Significance ($P \leq 0.05$, two sided) was assessed by Pearson's chi-square for categorical, and pairwise *t*-tests for continuous predictors. Pooled or sattertwait *t*-tests (two sided) for equal or unequal variances assessed significant differences. Means of EEG sleep measures (excluding participants on antidepressants and/or anxiolytics) by depression status are presented exclusively descriptively to describe sleep patterns by depressive symptoms. Covariates with significant odds ratios were entered into multivariable logistic regression models. Using SAS (SAS Institute Inc., Cary, NC, USA), logistic regression modeling assessed depression's unadjusted (Table 2) and adjusted (Table 4–Table 7) associations with outcomes. The interaction 'depression × anxiety' was evaluated in models and not found significant for SP (P = 0.66), HH (P = 0.64), or AB (P = 0.37). Only for self-reported cataplexy was there a significant interaction; thus, depression was stratified (rather than adjusted) by anxiety in these models. For all other models, depression and anxiety were adjusted for one another. Odds ratios (ORs) and their 95% confidence intervals (95% CI) were calculated from beta coefficient estimates obtained from these models. Associations were considered statistically significant at $P \leq 0.05$ level (two sided). Principal components analyses (Varimax rotated solution) suggested outcomes be modeled separately. Depression was categorized by severity for most models, except when stratified by anxiety for self-reported cataplexy (given small sample size). The logistic regression models included/excluded the following additional variables: (A) age, gender, BMI, antidepressant use; (B) model A variables plus (adjustment for or stratification by) anxiety; (C) model B variables excluding antidepressant users; (D) model B variables (adjustment or stratification for/by anxiety) plus sleepiness and insomnia – hypothesized to be potential confounders/mediators for self-reported SP and cataplexy, the outcomes most strongly related to depression.

RESULTS

Of the 866 study sample participants, 118 (13.6%) had depressive symptoms (Zung score ≥50) (Table 1). Average age was 54 years and did not differ between Zung categories. Male gender represented over one-half of the sample, but fewer men were in the depressed (Zung ≥50)

group. Of those with depressive symptoms, 43.3% had trait anxiety, 41% were on antidepressants, and 11% on anxiolytics.

The most prevalent outcome was AB (25.1%), and the least, cataplexy (2.9%). Individuals with Zung ≥50 had higher prevalences of all sleep disturbances – especially self-reported SP and cataplexy – when compared with those with Zung <50 (Table 1). Of the 156 participants on antidepressants in the sample (18%), most were on SSRIs (10.0%) and 31% had concurrent depressive symptomatology. Participants' average BMI was fairly high (29.4 kg/m^2) and similar in depression and no depression (Zung < 50) groups. Antidepressant and anxiolytic use, sleepiness and insomnia symptoms (difficulty falling asleep and repeated walkings) were more prevalent in participants with depressive symptoms than those with Zung < 50, but the prevalence of DBQ1*0602 status did not differ by depression status. Relative to Zung < 50, longer sleep latencies and trends of increased WASO, decreased average TST, and lower SE were observed in depression; while, perhaps unexpectedly, RL was increased and percent REM sleep similar in depression.

As shown in Table A3, anxiety was related to all outcomes ($P \leq 0.02$) except SP; sleepiness was significantly related to all but HH; insomnia symptoms (difficulty falling asleep and repeated walkings) to all outcomes ($P \leq 0.05$); and genotype HLA DQB1*0602 to none. Anxiolytics (OR 3.8) and antidepressants (OR 3.2) were associated ($P \leq 0.02$) with self-reported cataplexy; and antidepressants related ($P \leq 0.02$) to AB (OR 1.8). As DQBI*0602 and anxiolytics were not confounders for, or independently related to, any outcomes in adjusted models, they were not included in final models presented but are shown in Table A4. By contrast, in adjusted models, antidepressants were confounders for and related to self-reported cataplexy and AB, and thus were included.

With antidepressants, age, sex, and BMI adjustments (Table 2), depressive symptomatology combining lesser and greater severity (versus Zung < 50), was significantly related to all outcomes, with the strongest association seen for cataplexy (OR 3.43, $P = 0.006$). The Zung depression scale incorporates two items ('I get tired for no reason' and 'I have trouble sleeping through the night') related to tiredness and insomnia which might create built-in associations and obfuscate relationships. In sensitivity analyses, similar relationships (not shown in Tables) were found when a modified Zung scale without these items was used. Upon adjustment for age, gender, BMI, and depression, antidepressants were also associated independently with cataplexy (albeit non-significantly) and AB ($P = 0.03$).

Of participants with Zung ≥50, 58.5% had lesser, and 41.5% greater severity depression, representing 8.0% and 5.7% of the sample respectively (Table 3). Prevalences of sleep-related disturbances, particularly SP, were higher in those with greater severity of depression symptoms. All outcomes were more prevalent in anxiety, suggesting anxiety warranted consideration alongside depression.

Main adjusted models

Table 4–Table 7 show results of models describing, mostly, the relationships of depression severity to sleep-related outcomes, with further adjustment or stratification for/by anxiety (B–D models), exclusions of antidepressant users (C models), and additional adjustment for sleepiness and insomnia (D models) – potential confounders or mediators only for self-reported SP and cataplexy. Depression of lesser severity (Zung 50–54) was associated only with self-reported cataplexy. Thus, the following description of results focuses on depression of greater severity (Zung ≥55).

Sleep paralysis

Depression of greater severity was strongly associated with SP (OR 5.0) upon simultaneous adjustment for antidepressant use, age, sex and BMI (model A) (Table 4). This relationship became even stronger with additional adjustment for anxiety (model B) (OR 5.8, $P = 0.0008$) or excluding persons on antidepressants (model C) (OR 6.2, $P = 0.01$), and persisted after further adjustment for daytime sleepiness and insomnia (model D) (OR 4.5, $P = 0.006$), even when a modified Zung scale was used (OR 4.8, $P = 0.006$). Insomnia – but not antidepressant use, anxiety, or daytime sleepiness – was also found to be independently related to SP (model D, OR 2.5, $P = 0.004$).

Hypnagogic/hypnopompic hallucinations

Compared with non-depressed participants, depression of greater severity was related to a 3.2-fold increase in the odds of HH after adjustment for antidepressant use, age, sex and BMI (Table 5a, model A). This positive association became weaker (OR 2.2) with the addition of anxiety (model B), also independently related to HH (OR 1.9, $P = 0.04$); and disappeared with the exclusion of antidepressant users (model C). Further analyses (Table 5b) suggested that, in antidepressant users, percentages of HH increased with depression symptom severity, but that, in non-antidepressant users, the HH prevalence in the highest severity group resembled that in no depression.

Automatic behavior

Compared to individuals without depression, greater severity of depression symptoms was associated with two-fold increased odds of AB upon adjustment for antidepressants, age, gender and BMI (Table 6). This association became weaker and non-significant after adjustment for anxiety (model B), suggesting anxiety explained, at least partially, depression's relationship to AB. Excluding participants on antidepressants (model C) did not alter results more than adjusting for anxiety. Anxiety was found to be independently and significantly related to AB (models B and C). Adjusting for age, sex, BMI, anxiety, antidepressant use was also related, although only with borderline significance ($P = 0.07$), to AB (model B).

Self-reported cataplexy

Both lesser and greater severity depression were strongly associated with cataplexy, after adjustments for antidepressants, age, gender, and BMI (Table 7a, model A). The only sleep-related outcome for which the interaction 'depression × anxiety' was significant was self-reported cataplexy ($P = 0.02$) (Table A6), thus making it inappropriate to adjust for anxiety when assessing depression's relationship to cataplexy (see Table A7 for rates). When depression was stratified by anxiety (Table 7b, model B), power was not sufficient to categorize depression by severity. Overall depression (Zung ≥50) in non-anxious participants was highly significantly related to an almost ninefold increase in self-reported cataplexy odds (model B). This association became stronger after excluding persons on antidepressants (model C). However, upon simultaneous adjustment for model B variables plus sleepiness and insomnia, the odds ratio decreased to 4.9 ($P = 0.02$). Nevertheless, the relationship was still strong, even when a modified Zung scale was used (OR 5.7, $P = 0.01$). By contrast, no relationship was seen between depression and self-reported cataplexy in anxious participants in any of the models. Anxiety without depression was also related to cataplexy (OR 3.2, $P = 0.03$) (Table A6, estimated from interaction model B).

Secondary analyses

We conducted a broad, limited (as outcomes were not necessarily concurrent) test of discriminant validity to examine depression's relationships to SP, without and with HH, and of HH, without and with SP (Tables A5a and A5b). Even in the subgroup without HH, SP was

strongly related to depression (OR 3.0, 95% CI 1.4–6.5), as in the overall sample (OR 3.1). In the subgroup with HH, SP was less strongly (and non-significantly), related to depression (OR 2.2). Similarly, in the subgroup without SP, depression was related to HH (OR 2.3), which is similar to that in the overall sample (OR 2.4). In the subgroup with SP, depression was related more weakly to HH, perhaps due to lower precision (OR 1.7).

DISCUSSION

In our population-based investigation, depressive symptomatology was strongly related to SP and to self-reported cataplexy, and these associations were not explained by anxiety in depression. Depression, however, was related less strongly to HH, with anxiety (independently related to HH) partly explaining this relationship. Moreover, anxiety was more strongly related to AB than depression.

Present findings reveal the magnitude of depression's association with SP and are broadly consistent with the increased percentage of depressive disorders found in participants with SP (although non-significant with adjustments) and bipolar disorder's relationship (adjusted) to SP (Ohayon *et al.*, 1999). Our findings are also generally consistent with case reports of SP's links to reactive depression (Gangdev and Ramjee, 1996) and depressive states in familial SP (Roth *et al.*, 1968b). Present findings might also suggest that SP shares pathophysiologic mechanisms with depression. A panic disorder-SP link has been previously reported (Bell *et al.*, 1986; Hinton *et al.*, 2005; Paradis *et al.*, 1997). Also, as with depression (Kendler *et al.*, 1999), a familial occurrence has been reported for SP outside of narcolepsy (Bell *et al.*, 1986; Roth *et al.*, 1968b).

Anxiety did not change depression's associations with SP and was not related to SP, unlike other reports (Hinton *et al.*, 2005; Ohayon and Shapiro, 2000). This may be due to our investigation of general trait anxiety, rather than a specific disorder like post-traumatic stress disorder, and/or our predominantly Caucasian sample, e.g. SP more prevalent in African Americans with panic disorder (Bell *et al.*, 1986).

In participants without anxiety, depression was associated with an increased prevalence odds of self-reported cataplexy. In participants with anxiety, however, depression and self-reported cataplexy were not associated. This finding suggests that depression without anxiety (non-anxious depression) may represent a distinct depression subtype (Fava *et al.*, 1997), and individuals with 'pure' depression (or 'pure' anxiety) may be more susceptible to cataplexy episodes than individuals with comorbid disorders. Pure depression, depression with anxiety, and pure anxiety have often been differentiated, e.g. by cognitive specificity (Duzois and Dobson, 2001) or EEG profiles (Akiskal *et al.*, 1984). In our study, depression's association with cataplexy was not explained by anxiety in depression.

Although isolated cataplexy has been previously reported in non-narcoleptics (Hartse *et al.* 1988), and in sleepy undergraduates and army draftees (Billiard *et al.*, 1987; Wing *et al.*, 1994), its relationship to depression has not been studied to date. Our findings on cataplexy may support the affective spectrum disorder proposal: cataplexy has been hypothesized by Hudson and Pope (1990) to share a common pathophysiology with depression, including response to many antidepressant classes (Hudson and Pope, 1990; Hudson *et al.*, 2003), although the aforementioned study (Hudson *et al.*, 2003) had insufficient data to test the hypothesis regarding cataplexy and its coaggregation with depression.

Findings that anxiety and depression, although not consistently in all models, were related to HH generally agree with prior Canadian and European findings of HH linked to mood and anxiety disorders (Ohayon *et al.*, 1996, 1999). Our findings for HH may extend findings of depression with anxiety related to worsened symptomatology and outcomes including

increased insomnia, depersonalization/derealization (Clayton *et al.*, 1991; Coryell *et al.*, 1998; Paykel, 1972; VanValkenburg *et al.*, 1984), hypochondriasis (VanValkenburg *et al.*, 1984), autonomic (Lenze *et al.*, 2000) and somatic symptoms (Clayton *et al.*, 1991). Derealization might also reflect AB, described as trance-like or 'fugue-like', 'twilight' state episodes, and characterized by amnesia (Ganado, 1958; Guilleminault *et al.*, 1975).

Epidemiologic studies have previously found depression to be strongly related to self-reported insomnia and/or sleepiness (Ford and Kamerow, 1989; Hublin *et al.*, 1996; Ohayon *et al.*, 1997), consistent also with our findings, although this was not a specific hypothesis. EEG sleep measures pertaining to difficulty initiating and maintaining sleep (longer sleep latencies, poorer sleep continuity) revealed trends (albeit not statistically significant) in our depressed participants consistent with reports in other depressed patients (Kupfer *et al.*, 1982; Reynolds *et al.*, 1983) and with increased self-reported insomnia in our participants. Decreased sleep efficiency, an abnormal sleep profile typifying depression (Thase *et al.*, 1997), was also found in our participants. Most sleep parameters did not significantly differ by Zung depression status, consistent with prior findings that as many as 55% of depressed outpatients have 'normal' EEG sleep profiles resembling those of healthy controls (Thase *et al.*, 1997). Certain expected differences in polysomnographic results (e.g. REM sleep latency or percent REM sleep) (Akiskal *et al.*, 1982, 1984; Kupfer and Foster, 1972; Reynolds *et al.*, 1983; Thase *et al.*, 1984) were not presently observed, perhaps due to our examining community-based participants with predominantly minimal-mild depressive symptomatology. Of our sample, 12% had minimal-mild depressive symptomatology (Zung 50–59), representing 87% of those with Zung-defined depression.

The weakening of depression's relationships to self-reported SP and, especially, cataplexy upon addition of insomnia and sleepiness to the models suggest that they are confounders or mediators for these associations (model D, Table 4; model C, Table 7b). SP has been previously linked to interrupted and non-restorative sleep (Ohayon *et al.*, 1999;Takeuchi *et al.*, 1992;Wing *et al.*, 1994). Depression may underlie poor sleep and insomnia, which may then lead to SP. Cataplexy-like episodes have been linked to sleepiness in non-clinical populations (Billiard *et al.*, 1987;Ohayon *et al.*, 1996;Szklo-Coxe *et al.*, 2007;Wing *et al.*, 1994). Notwithstanding our data's cross-sectional nature, marked decreases of depression's estimates with insomnia and sleepiness suggest that these might be mediators. However, associations remained strong, suggesting other explanations are also likely and warrant investigation.

Cross-sectional data precluded ascertaining whether depression improved with antidepressants. Thus, our definition of depression (Zung ≥50) did not include antidepressants. Findings may therefore even underestimate depression's relationships to SP and self-reported cataplexy. Antidepressants were found to be related to, and confounders for, depression's relationships to AB and cataplexy, consistent with cataplexy's favorable clinical response to antidepressants (Takahashi, 1976). Depression's relationships to all outcomes but HH were similar whether antidepressants were adjusted for or antidepressant users excluded, suggesting these relationships were not due to antidepressants. Depression's association with HH only in antidepressant users (Table 5a) is difficult to interpret; the timeline regarding its appearance pre- or post-anti-depressant use cannot be established cross-sectionally. A possible interpretation, especially as antidepressants were not related to HH, is that removing antidepressants users removed more severely depressed participants.

Although the Zung has limitations, such as lack of correspondence to DSM-IV depression, it is correlated with clinical patient evaluation (Zung, 1965). Active depressive symptomatology was our primary interest given our community-based sample and predominantly mild depression characterizing participants. STAI, designed to measure anxiety symptoms in community-based samples such as ours, corresponds to clinical domains for Generalized

Anxiety Disorder by DSM-IV criteria, supporting its current applicability (Okun *et al.*, 1996). Nevertheless, its trait scale may not differentiate between anxiety and depression constructs and may instead measure depression and negative effect (Bieling *et al.*, 1998; Endler *et al.*, 1992). However, if STAI-T were also measuring depression, one would not expect the association of depression with cataplexy to be present only in participants without anxiety (Table 7b).

Our study's strengths overall include use of accepted (reliable and valid) self-report instruments in a large community- based sample and thorough examination of depression and anxiety after multiple adjustments. Although the Zung scale is considered a good clinical depression measure, questionnaire assessments may decrease the clinical implications of findings. Automatic behavior, in particular, is difficult to assess via questionnaire, especially if related to sleep disorders or other medical problems, although physicians may also experience difficulties diagnosing these disturbances in patients. A key limitation of this study is its cross-sectional design, precluding assessment of temporal relationships and untangling whether sleepiness and insomnia were mediators or confounders. Our study could not elucidate causal relationships and uncover underlying mechanisms, such as whether outcomes were specific to REM sleep. Nevertheless, presently reported associations are meaningful irrespective of whether outcomes are REM sleep specific, as they extend research on depression's relationships to sleep-related disturbances seldom examined, especially outside narcolepsy. Findings are therefore more than theoretically relevant and generally consistent with previous limited work on these disturbances and their psychiatric correlates.

Hypnagogic/hypnopompic hallucinations (HH) were assessed when falling asleep at night and waking up in the morning, but not in the middle of the night. The questions on HH incorporated 'strange or frightening' in their descriptions; therefore, participants who experienced HH without this affective component may have been excluded. Our definition was also limited to visual, auditory, and affective modalities. HH occurring with smell (Maury, 1848), motor (Hollingworth, 1911), tactile (Ohayon and Shapiro, 2000), or movement sensations (e.g. falling), or feeling of sensed presence (Cheyne *et al.*, 1999) were not assessed. Measuring these sensations may have led to a more complete assessment of these disturbances in our study.

Sleep paralysis and HH often co-occur (Cheyne *et al.*, 1999). HH without SP may have been more likely to increase reporting of entoptic hallucinations unrelated to REM sleep, although the affective dimension of the question may have reduced reporting of these. Our broad test of discriminant validity suggested HH in SP was not responsible for depression's strong relationship to SP, and SP in HH did not explain depression's weaker association with HH. Moreover, depression was related differently (more strongly) to SP than to HH, indirectly suggesting discriminant validity.

Another potential problem in our study is that depressed subjects may lack a positive self-schema (Duzois and Dobson, 2001) or have cognitive biases favoring negative self-related information or interpretations of ambiguous situations (Nunn *et al.*, 1997). This interconnected negative self-representational system, leading to biased information processing (Duzois and Dobson, 2001), may have resulted in depressed participants' overestimating the investigated sleep disturbances. However, it is not clear whether this bias would have affected sleep disturbance reporting, as this outcome has not been specifically studied to date with respect to cognitive interference effects. Given depressed (including with anxiety) participants' tendencies to recall less positive information (Duzois and Dobson, 2001) and process more negative information (Nunn *et al.*, 1997), it is possible HH – defined as 'strange and frightening' – might have been over-recalled. If depressed participants had positive emotions less frequently, they might have underreported cataplexy, for cataplexy was defined as muscle weakness triggered by emotions, and positive emotion is the most sensitive trigger of cataplexy

(Anic-Labat *et al.*, 1999; Krahn *et al.*, 2005). However, findings including and excluding the anger (negative emotion) item were similar (Table A8). Depressed participants' recalling of less positive information might be interpreted as 'automatic behavior', resulting in its overestimate, a lesser concern, however, given depression's weaker relationship to this outcome. In any event, without a sleep expert confirming participants' claims, the outcomes could have been overestimated given depressed patients' propensity for negative processing bias. Nevertheless, similar clinical and questionnaire-assessed estimates of these symptoms have been reported (Bassetti and Aldrich, 1997).

Depression (assessed on the overnight protocol) and outcomes (from mailed survey) were evaluated within ±2 years of one another, a limitation of our study. Also, outcome item questions asked about ever experiencing a disturbance and did not differentiate between outcomes occurring (infrequently to frequently) presently versus in the past. In narcolepsy's natural history, the sleep disturbances examined are considered intermittent – occurring briefly, vanishing, and recurring later (Billiard *et al.*, 1983; Passouant and Billiard, 1976). Although we did not study narcoleptics, similar problems may apply as we did not specify a time frame. Frequent SP, for example, could refer to the present or some (unspecified) time in the past. Thus, SP's association with depressive symptoms (generally, day-to-day) within a 2-year time frame refers to when outcome items were completed rather than when they occurred.

We extend epidemiologic findings on depression-sleep disturbances beyond insomnia and sleepiness to SP and self-reported cataplexy, which may assist in better recognizing and describing depression in the community. Relationships of anxiety to HH and AB, and of depression, albeit less strong, to HH also suggest psychiatric correlates of these sleep disturbances, particularly anxiety, warrant attention.

Depression's heavy burden includes its chronic nature (Judd *et al.*, 1997), considerable symptom severity, role impairment (Andrade *et al.*, 2003; Kessler *et al.*, 1997), and hidden costs like lost productive time (Stewart *et al.*, 2003). Ford and Kamerow (1989) suggest that inquiry regarding sleep disturbances might enhance recognition of depression by health providers, a recommendation that might be extended, in particular, to SP. Future longitudinal epidemiologic studies on these and other rarely studied sleep problems may be warranted to better characterize the full range of sleep disturbances with which depression – alone or comorbid with anxiety – may be associated in the population. Evolution of the sleep disturbances investigated with respect to recurrences and changes in depression and anxiety merits further inquiry in the population.

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APPENDICES

Table A1a Frequency (percentage of total sample) of each self-reported sleep disturbance item

	(1) Never, n (%)	(2) Only few times ever, n (%)	(3) Rarely (<1/month), n (%)	(4) Sometimes (≥1/month but <1/week), n (%)	(5) Often (≥1/week), n (%)
Sleep paralysis					
In the morning	770 (88.9)	81 (9.4)	11 (1.3)	3 (0.35)	1 (0.12)
During night's sleep	643 (74.3)	182 (21.0)	26 (3.0)	12 (1.4)	3 (0.35)
Hypnagogic/hypnopompic hallucinations					
When falling asleep at night	682 (78.8)	126 (14.6)	38 (4.4)	17 (2.0)	3 (0.35)
When awakening in morning	786 (90.8)	58 (6.7)	14 (1.6)	7 (0.81)	1 (0.12)
When drowsy	771 (89.1)	70 (8.1)	13 (1.5)	10 (1.2)	1 (0.12)
Automatic behavior					
When driving	366 (42.4)	312 (36.1)	104 (12.0)	61 (7.1)	21 (2.4)
When working desk/sitting quietly	485 (56.1)	485 (30.2)	67 (7.8)	39 (4.5)	13 (1.5)
Cataplexy					
With laughter	795 (91.8)	57 (6.6)	8 (0.92)	4 (0.46)	2 (0.23)
With anger	801 (92.5)	49 (5.7)	10 (1.2)	4 (0.46)	2 (0.23)
With joking	811 (93.9)	41 (4.8)	7 (0.81)	3 (0.35)	2 (0.23)

Table A1b Frequency (percentage) of depression (Zung score ≥ 50) by frequency of each self-reported sleep disturbance item

	(1) Never, *n* (%)	(2) Only few Times ever, *n* (%)	(3) Rarely (<1/month), *n* (%)	(4) Sometimes (≥1/month but <1/week), *n* (%)	(5) Often (≥1/week), *n* (%)
Sleep paralysis					
In the morning	96 (12.5)	14 (17.3)	6 (54.6)	1 (33.3)	1 (100.0)
During night's sleep	81 (12.6)	26 (14.3)	4 (15.4)	6 (50.0)	1 (33.3)
Hypnagogic/hypnopompic hallucinations					
When falling asleep at night	85 (12.5)	18 (14.3)	12 (31.6)	1 (5.9)	2 (66.7)
When awakening in morning	100 (12.7)	12 (20.7)	4 (28.6)	1 (14.3)	1 (100)
When drowsy	98 (12.7)	15 (21.4)	3 (23.1)	1 (10.0)	1 (100)
Automatic behavior					
When driving	47 (12.8)	33 (10.6)	17 (16.4)	12 (19.7)	8 (38.1)
When working at desk/sitting quietly	55 (11.3)	35 (13.4)	14 (20.9)	9 (23.1)	4 (30.8)
Cataplexy					
With laughter	100 (12.6)	57 (19.3)	8 (19.3)	4 (62.5)	2 (50.0)
With anger	98 (12.2)	14 (28.6)	5 (50.0)	0 (0.0)	1 (50.0)
When joking	106 (13.1)	7 (17.1)	3 (42.9)	1 (33.3)	1 (50.0)

Table A2a Adjusted* associations of depression, categorized by severity, and trait anxiety (≥90th percentile), with self-reported sleep paralysis, hypnagogic/hypnopompic hallucinations and automatic behavior

	Sleep paralysis (SP)		Hypnagogic/hypnopompic hallucinations (HH)		Automatic behavior (AB)	
	Odds ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value
Depression						
Zung score < 50 (reference)	1.00		1.00		1.00	
Lower severity (Zung ≥55)	1.62 (0.56–4.69)	0.37	1.17 (0.49–2.77)	0.72	1.28 (0.71–2.29)	0.42
Higher severity (Zung 50–54)	4.97 (1.79–13.79)	0.002	1.90 (0.75–4.82)	0.18	1.87 (0.93–3.74)	0.08
Anxiety (STAI-T ≥90th)† vs. <90th	0.99 (0.37–2.65)	0.99	2.51 (1.19–5.32)	0.02	1.25 (0.70–2.22)	0.45
Antidepressant use vs. none	1.33 (0.63–2.81)	0.46	0.87 (0.46–1.65)	0.67	1.50 (1.01–2.23)	0.05
Age	0.94 (0.89–0.98)	0.003	0.94 (0.91–0.98)	0.0009	0.97 (0.95–0.99)	0.002
Female vs. male	0.82 (0.43–1.55)	0.54	1.38 (0.83–2.27)	0.21	0.94 (0.68–1.30)	0.69
Body mass index (kg/m^2)	1.02 (0.98–1.07)	0.32	1.01 (0.98–1.05)	0.46	1.03 (1.01–1.05)	0.04

Table A2b Adjusted relationships* (from depression × anxiety interaction model) of depression stratified by trait-anxiety (≥90th percentile) to self-reported cataplexy (*n* = 866)

	Odds ratio‡
In participants without anxiety (STAI-T < 90th), depression (Zung ≥50) versus no†	4.59
In participants with anxiety (STAI-T < 90th), depression ≥50 versus no†	0.52

CI, confidence interval, STAI-T, Trait-Anxiety Scale of Spielberger State-Trait Anxiety Inventory. Interpretrations: analyses using ≥90th percentiles yielded similar results with respect to their effects on depression's associations with the outcomes above, as compared with ≥75th percentiles. Specifically, depression's relationships to SP were not explained by anxiety's presence and anxiety still partially explained depression's relationship to HH. Although anxiety lowered depression's estimate for AB to non-significance, the confidence intervals were similar to ≥75th percentile. Anxiety ≥90th percentile was not associated significantly with AB. Cross tabulations indicated that there were many anxious participants in the 75th–90th percentile (mildly anxious) and when these became part of the <90th reference, the difference with the ≥90th became negligible (not significant).

*Odds ratios from multiple logistic regression models.

†Trait anxiety measured as STAI-T ≥90th percentile (STAI-T score ≥46).

STAI-T, Trait-Anxiety Scale of Spielberger State-Trait Anxiety Inventory. Depression categories of severity (Zung score 50–54, and Zung score ≥55) used in other models were collapsed for this model into Zung score ≥50 given small sample size and similar relationships of lesser and greater severity depression to this outcome. Interpretations: as with the STAI-T ≥75th percentile, depression's relationship to cataplexy persisted in participants without anxiety (STAI-T ≥90th).

*Multivariable logistic regression model also adjusted for age, sex, body mass index, antidepressant use.

†No depression symptoms (Zung < 50) is the reference category (odds ratio is 1.00).

‡*P*-value for interaction term (from model) for depression × anxiety (trait) is 0.02.

Table A3

Percentages of self-reported sleep disturbance outcomes and their unadjusted odds ratios[*,†] (95% confidence intervals) for predictors other than depression in the Wisconsin Sleep Cohort Study (n = 866)

	Sleep paralysis		Hypnagogic/hypnopompic hallucinations		Automatic behavior		Cataplexy	
	%	OR (95% CI)	%	OR (95% CI)	%	OR (95% CI)	%	OR (95% CI)
Anxiety (STAI-T ≥75th percentile) vs. < 75th	7.46	1.71 (0.90–3.24)	15.42	2.57* (1.58–4.20)	35.82	2.00* (1.42–2.81)	5.97	2.18* (1.43–7.10)
Antidepressant use (n = 157) vs. none	8.28	1.91 (0.98–3.73)	10.83	1.36 (0.77–2.41)	34.39	1.76* (1.21–2.55)	6.37	3.15* (1.39–7.14)
Anxiolytic use (n = 32) vs. none	6.25	1.22 (0.28–5.29)	6.25	0.69 (0.16–2.96)	34.38	1.60 (0.76–3.38)	9.38	3.81† (1.08–13.45)
DQB1-0602 positive (n = 209) vs. negative	6.70	1.45 (0.75–2.77)	8.13	0.91 (0.52–1.60)	26.32	1.10 (0.77–1.56)	2.39	0.78 (0.28–2.10)
Frequent insomnia (≥5/month) vs. ≤4/month								
Difficulty falling asleep (n = 133)	10.53	2.65* (1.37–5.13)	17.29	2.73* (1.60–4.64)	35.34	1.80* (1.21–2.67)	6.02	2.68† (1.13–6.35)
Wake up repeatedly during night (n = 282)	8.90	2.74* (1.49–5.02)	11.39	1.65† (1.02–2.68)	32.03	1.70* (1.24–2.34)	4.63	2.30† (1.04–5.11)
Sleepiness: ESS > 11 vs. ≤11 (n = 228)	7.89	1.94† (1.05–3.59)	10.09	1.26 (0.76–2.12)	35.09	1.98* (1.42–2.75)	7.89	7.73* (3.18–18.76)

OR, odds ratios, CI, confidence intervals; STAI-T, Trait-Anxiety from Spielberger Trait-Anxiety Inventory; ESS, Epworth Sleepiness Scale.

*$P \leq 0.02$.

†$P \leq 0.05$.

Table A4

Fully adjusted associations[†] of depression, with self-reported sleep paralysis, hypnagogic/hypnopompic hallucinations, automatic behavior, and cataplexy in 866 Wisconsin Sleep Cohort Study participants

	Sleep paralysis		Hypnagogic/hypnopompic hallucinations		Automatic behavior		Cataplexy	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-v**
Depression[‡] vs. Zung < 50 (ref)	2.90 (1.43–5.87)	0.003	2.32 (1.27–4.2)	0.006	1.64 (1.06–2.53)	0.03	3.26 (1.33–8.01)	0.0
Antidepressant use vs. none	1.56 (0.44–1.55)	0.24	1.06 (0.57–1.99)	0.85	1.53 (1.02–2.30)	0.04	1.97 (0.76–5.09)	0.1
Age	0.94 (0.90–0.98)	0.005	0.94 (0.91–0.97)	0.0004	0.97 (0.95–0.99)	0.002	0.96 (0.91–1.01)	0.1
Female vs. male	0.82 (0.44–1.55)	0.55	1.36 (0.83–2.24)	0.22	0.94 (0.68–1.30)	0.71	0.98 (0.42–2.30)	0.9
BMI	1.03 (0.98–1.07)	0.26	1.01 (0.98–1.05)	0.46	1.03 (1.00–1.05)	0.03	1.04 (0.98–1.10)	0.2
Anxiolytic use vs. none	0.65 (0.14–3.09)	0.59	0.46 (0.10–2.12)	0.32	1.10 (0.49–2.44)	0.82	1.74 (0.43–7.14)	0.4
HLA DQB1*0602[§] vs. no alleles	1.47 (0.76–2.87)	0.25	0.89 (0.50–1.58)	0.68	1.09 (0.76–1.56)	0.65	0.83 (0.30–2.28)	0.7

OR, odds ratios; CI, confidence intervals; BMI, body mass index measured as weight (kg)/height (m^2); HLA, human leukocyte antigen.

[†] Odds ratios estimated from four multiple logistic regression models.

[‡] Depression defined as Zung score ≥50.

[§] HLA DQB1*0602 defined as presence of one or two alleles.

Table A5a Unadjusted associations* of depression with sleep paralysis in overall sample and in subgroups without and with hypnagogic/hypnopompic hallucinations (HH) in the Wisconsin Sleep Cohort Study

	Sleep paralysis					
	Total sample (without and with HH) (*n* = 866)		Subgroup without HH (*n* = 791)		Subgroup with HH (*n* = 75)	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Depression (Zung score ≥50) vs. Zung <50	3.11 (1.60–6.05)	0.0008	3.00 (1.39–6.45)	0.005	2.22 (0.55–8.93)	0.26

Table A5b Unadjusted associations* of depression with hypnagogic/hypnopompic hallucinations (HH) in overall sample and in subgroups without and with sleep paralysis in the Wisconsin Sleep Cohort Study

	Hypnagogic/hypnopompic hallucinations					
	Total sample (without and with SP) (*n* = 866)		Subgroup without sleep paralysis (*n* = 821)		Subgroup with sleep paralysis (*n* = 45)	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Depression (Zung score ≥50) vs. Zung < 50	2.37 (1.35–4.16)	0.003	2.25 (1.21–4.17)	0.01	1.67 (0.39–7.19)	0.49

OR, odds ratios; CI, confidence intervals. Sleep paralysis (self-reported) was defined as being unable to move your body and feeling paralyzed >few times ever either upon awakening in the morning (AM) and/or awakening during night sleep. Hypnagogic/hypnopompic hallucinations (self-reported) was defined as hearing or seeing strange and frightening things or people > few times ever either with sleep onset PM and/or awakening AM and/or when drowsy.

* Odds ratios estimated from logistic regression models.

Table A6

Adjusted associations of depression × anxiety with self-reported cataplexy

	β coefficient estimate	Standard error	*P*-value
Interaction model 7B: adjusted for antidepressants (*n* = 866)			
Intercept	−3.3204	1.8897	0.08
Depression*	2.1840	0.6534	0.0008
Trait anxiety†	1.1638	0.5496	0.03
Depression × trait anxiety	−2.0475	0.8819	0.02
Age	−0.0387	0.0287	0.18
Female	−0.0160	0.4357	0.97
Antidepressant use	0.7091	0.4545	0.12
Body mass index	0.0339	0.0339	0.25
Interaction model 7C: excluding antidepressant users (*n* = 709)			
Intercept	−3.1594	2.3296	0.13
Depression*	2.2305	0.8532	0.009
Trait anxiety†	1.2136	0.6542	0.069
Depression × trait anxiety	−2.4600	1.2377	0.05
Age	−0.0365	0.0364	0.32
Female	−0.8619	0.5679	0.13
Body mass index	0.0489	0.0319	0.13

*Depression defined as Zung score ≥50.

†Trait anxiety defined as ≥75th percentile on trait anxiety scale of Spielberger State-Trait Anxiety Scale. Body mass index measured in kg/m^2.

Table A7

Rate of depression* in self-reported cataplexy stratified by anxiety†

	Prevalence of self-reported cataplexy	
	No anxiety	**Anxiety**
Depression (entire population, *n* = 866)		
No	9/634 (1.4%)	6/114 (5.3%)
Yes	4/31 (12.9%)	6/87 (6.9%)
P-value	< 0.0001	0.63
Depression (subset not on antidepressants, *n* = 709)		
No	1.3%	4.7%
Yes	9.5%	4.1%
P-value	0.0028	0.87

*Depression defined as Zung score ≥50. No depression as Zung score < 50.

†Anxiety defined as trait anxiety (STAI-T ≥ 75th percentile or a score of ≥39).

No anxiety defined as STAI-T < 75th percentile.

Table A8

Adjusted[†] relationship of depression to self-reported cataplexy with laughter and/or with joking but not with anger in 866 Wisconsin Sleep Cohort participants

	Self-reported cataplexy with laughter and/or joking[‡]	
	Odds ratio (95% confidence interval)	***P*-value**
Depression (Zung score ≥50) vs. Zung score <50 (reference)	4.03 (1.35–12.02)	0.01

[†] Adjusted for age, sex, body mass index, antidepressant use, anxiolytic use and HLA DQB1*0602.

[‡] Cataplexy with anger excluded from the definition of cataplexy, leaving 16 participants with cataplexy with joking and/or laughter but not anger. The corresponding odds ratio for the same logistic regression model when cataplexy was defined as with laugher and/or with joking and/or with anger was similar: OR 3.26, 95% CI 3.26–8.01, $P = 0.01$.

Table 1

Outcomes, covariates, and descriptive sleep characteristics for overall sample and by depression status† in Wisconsin Sleep Cohort Study participants (*n* = 866)

	Overall sample	No depressive symptoms (Zung score <50)	Depressive symptoms (Zung score ≥50)
Frequency	866	748	118
Prevalence (95% CI) (%)	100	86.4	13.6
Age (years) (mean ± SD)	53.6 ± 7.6	53.6 ± 7.6	52.0 ± 7.7
Male† (%)	52.9	55.1	39.0
Outcomes (self-reported), >few times ever			
Sleep paralysis† (%)	5.2	4.1	11.9
Hypnagogic/hypnopompic hallucinations† (%)	8.7	7.5	16.1
Automatic behavior† (%)	25.1	23.3	36.4
Cataplexy† (%)	2.9	2.0	8.5
Covariates			
Trait anxiety (≥75th percentile) † (%)	23.2	4.7	43.3
Antidepressant use† (%)	18.1	14.6	40.7
Anxiolytic use† (%)	3.7	2.5	11.0
Body mass index (kg/m^2) † (mean ± SD)	29.4 ± 6.2	29.2 ± 6.2	29.4 ± 6.7
Daytime sleepiness: Epworth Sleepiness Score > 11† (%)	26.3	23.4	44.9
Insomnia ≥5/month (%)			
Difficulty getting to sleep†	15.4	13.4	28.0
Wake up during the night & have a hard time getting back to sleep	21.1	20.1	27.4
Wake up repeatedly during the night†	32.6	30.6	45.3
Wake up too early in the morning & cannot get back to sleep	18.8	17.8	24.6
'Narcolepsy' allele(s), genotype HLA DQB1*0602 (%)	24.1	24.5	22.0
Descriptive sleep variables: EEG nocturnal measures‡ (mean ± SD)			
Sleep latency† (min)	12.0 ± 13.8	11.8 ± 13.9	13.4 ± 12.6
REM sleep latency† (min)	107.9 ± 56.1	106.7 ± 55.4	118.5 ± 61.2
Percent REM sleep (%)	18.5 ± 5.7	18.6 ± 5.6	18.2 ± 6.0
Total sleep time (min)	375.0 ± 56.2	375.1 ± 55.3	374.4 ± 63.9
Wake after sleep onset (min)	65.4 ± 38.4	65.0 ± 37.4	69.0 ± 47.1
Sleep efficiency (%)	82.1 ± 9.4	82.2 ± 9.2	80.9 ± 11.0

Zung, Zung Self-Rating Depression Scale; STAI-T, trait scale of Spielberger State-Trait Anxiety Inventory; ≥75th percentile, upper quartile or score ≥39; EEG, electroencephalographic sleep measured by polysomnography; REM, rapid eye movement; HLA, human leukocyte antigen. Means with additional exclusions – sedatives and/or stimulants, WASO ≥200 min, SE <0.5%, RL <0 min, RL >400 min, SL >60 min, were similar to findings shown.

† Significant differences (two-sided test) at $P \leq 0.05$ for Zung score ≥50 versus Zung score <50.

‡ For EEG sleep variables, participants on antidepressants and/or anxiolytics were excluded ($n = 599$).

Table 2

Adjusted associations* of depression and antidepressants with four self-reported sleep disturbances

	Sleep paralysis		Hypnagogic/hypnopompic hallucinations		Automatic behavior		Cataplexy	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
Depression (Zung ≥50) versus no depression (Zung <50)†	2.78 (1.37–5.63)	0.005	2.24 (1.24–4.08)	0.008	1.64 (1.07–2.53)	0.03	3.43 (1.42–8.28)	0.006
Antidepressant use versus none	1.45 (0.70–2.98)	0.32	0.98 (0.53–1.82)	0.95	1.55 (1.04–2.30)	0.03	2.19 (0.90–5.35)	0.09

*Also adjusted for age, sex, and body mass index. For models with further adjustments, see Table A4.

†No depression refers to no depression symptoms (reference group).

Table 3

Percentages of self-reported sleep disturbance outcomes by depression severity and anxiety status

	Sleep paralysis	Hypnagogic/hypnopompic hallucinations	Automatic behavior	Cataplexy
Depression				
Greater severity of symptoms (Zung score ≥55) (*n* = 49)	18.4	20.4	42.9	10.2
Lesser severity of symptoms (Zung score 50–54) (*n* = 69)	7.2	13.0	31.9	7.3
No depression (Zung score < 50, reference) (*n* = 748)	4.1	7.5	23.3	2.0
Trait anxiety† (STAI-T ≥ 75th percentile) (*n* = 665)	7.5	15.4	35.8	6.0
No trait anxiety (STAI-T < 75th percentile, reference) (*n* = 201)	4.5	6.6	21.8	2.0

All values are in percentages. STAI-T, Trait Scale of Spielberger State-Trait Anxiety Inventory.

*P-values ≤0.005 for all four outcomes from Mantel–Haenszel chi-squared tests comparing greater severity depression, lesser severity depression, and no depression.

†P-values ≤0.003 for hypnagogic/hypnopompic hallucinations, automatic behavior, and cataplexy from Mantel–Haenszel chi-squared tests comparing anxiety to no anxiety.

Table 4

Adjusted associations* of depression and other covariates with self-reported sleep paralysis

	Basic model (A) (*n* = 866)		Basic model (B) adjusted for anxiety† (*n* = 866)		Basic model (C) adjusted for anxiety and excluding all antidepressant users (*n* = 709)		Basic model (D) adjusted for anxiety, sleepiness, insomnia (*n* = 866)	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
Depression								
No depression (Zung score < 50, reference)	1.00		1.00		1.00		1.00	
Lesser severity depression (Zung score 50–54)	1.62 (0.60–4.41)	0.35	1.85 (0.62–5.47)	0.27	2.52 (0.73–8.68)	0.14	1.54 (0.52–4.60)	0.44
Greater severity depression (Zung score ≥55)	4.95 (2.05–11.99)	0.0004	5.81 (2.08–16.18)	0.0008	6.16 (1.54–24.67)	0.01	4.48 (1.55–12.98)	0.006
Antidepressant use versus none	1.33 (0.90–0.98)	0.45	1.36 (0.64–2.87)	0.43	N/A		1.38 (0.64–2.96)	0.41
Trait anxiety (STAI-T ≥75th) versus STAI-T <75th			1.02 (0.98–1.07)	0.33	1.04 (0.99–1.09)	0.10	0.75 (0.33–1.72)	0.50
Daytime sleepiness (ESS score >11) versus ≤11							1.61 (0.83–3.10)	0.16
Insomnia‡ ≥5/month versus <5/month							2.54 (1.36–4.76)	0.004

OR, odds ratios; CI, confidence intervals; STAI-T ≥75, 75th percentile (score of ≥39) on Trait-Anxiety Scale of Spielberger State-Trait Anxiety Inventory; ESS, Epworth Sleepiness Score; NA, not applicable. Depression refers to depressive symptomatology. In models A, B, and D, there were 45 participants with sleep paralysis; in model C there were 32 participants with sleep paralysis.

*Odds ratios estimated from four logistic regression models and also adjusted for age, sex, body mass index (BMI) in kg/height (m^2).

†*P*-trend = 0.001 for relationship of depression severity to sleep paralysis.

‡Insomnia (self-reported) defined as waking up repeatedly during the night.

Table 5a Adjusted associations* of depression and other covariates with self-reported hypnagogic/hypnopompic hallucinations

	Basic model (A) (*n* = 866)		Basic model (B) adjusted for anxiety† (*n* = 866)		Basic model (C) adjusted for anxiety and excluding antidepressant users (*n* = 709)	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
Depression						
No depression (Zung < 50, reference)	1.00		1.00		1.00	
Lesser severity depression (Zung 50–54)	1.72 (0.79–3.72)	0.17	1.23 (0.53–2.83)	0.64	1.23 (0.34–3.33)	0.69
Greater severity depression (Zung ≥55)	3.20 (1.43–7.14)	0.005	2.19 (0.91–5.25)	0.08	0.78 (0.16–3.74)	0.75
Antidepressant use versus none	0.94 (0.50–1.75)	0.84	0.88 (0.46–1.65)	0.70	N/A	
Trait Anxiety‡ (STAI-T ≥75th) versus STAI-T <75th			1.87 (1.04–3.37)	0.04	1.76 (0.90–3.47)	0.10

Table 5b Percentages of hypnagogic/hypnopompic hallucinations by depression status stratified by antidepressant users

	Hypnagogic/hypnopompic hallucinations	
	No antidepressant users (*n* = 709), *n* (%)	**Only antidepressant users (*n* = 157), *n* (%)**
No depression (Zung score < 50, reference 54)	50 (7.82)	6 (5.50)
Lesser severity depression (Zung score 50–54)	6 (13.04)	3 (13.04)
Greater severity depression (Zung score ≥55)	2 (8.33)	8 (32.0)

OR, odds ratios; CI, confidence intervals; STAI-T, Trait-Anxiety Scale of Spielberger State-Trait Anxiety Inventory; NA, not applicable. In models A and B, there were 75 participants with hypnagogic/hypnopompic hallucinations (HH); in model C, there were 58 participants with HH.

*Odds ratios estimated from three logistic regression models and also adjusted for age, sex, body mass index (BMI) in kg/height (m^2).

†*P*-trend = 0.09 for relationship of depression severity to hypnagogic/hypnopompic hallucinations.

‡Trait-anxiety measured as STAI-T ≥75th percentile (score of ≥39) versus <75th percentile (score of <39).

Table 6

Adjusted associations* of depression and other covariates with self-reported automatic behavior

	Basic model (A) (*n* = 866)		Basic model (B) adjusted for anxiety† (*n* = 866)		Basic model (C) adjusted for anxiety excluding all antidepressant users (*n* = 709)	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
Depression						
No depression (Zung score < 50, reference)	1.00		1.00		1.00	
Lesser severity depression (Zung score 50–54)	1.38 (0.80–2.38)	0.25	1.08 (0.60–1.94)	0.81	1.22 (0.60–2.49)	0.58
Greater severity depression (Zung score ≥55)	2.10 (1.13–3.91)	0.02	1.58 (0.82–3.09)	0.18	1.67 (0.67–4.19)	0.27
Antidepressant use versus none	1.52 (1.02–2.26)	0.04	1.45 (0.97–2.16)	0.07	N/A	
Trait anxiety‡ (STAI-T ≥75th) versus STAI-T < 75th			1.60 (1.07–2.39)	0.02	1.81 (1.13–2.91)	0.01

OR, odds ratios; CI, confidence intervals; BMI, body mass index; STAI-T, Trait-Anxiety Scale of Spielberger State-Trait Anxiety Inventory; N/A, not applicable. In models A and B, there were 217 participants with automatic behavior (AB); in model C there were 163 participants with AB.

*Odds ratios estimated from three logistic regression models and also adjusted for age, sex, body mass index (BMI) in kg/height (m^2).

†*P*-trend = 0.40 (not statistically significant) for relationship of depression severity to automatic behavior.

‡Trait anxiety measured as STAI-T ≥75th percentile (score of ≥39) versus < 75th percentile (score of < 39).

Table 7a Adjusted associations* of depression and antidepressants with self-reported cataplexy† (model A)

	Odds ratio 95% CI	*P*-value
Depression‡		
No depression (Zung score < 50, reference)	1.00	
Lesser severity depression (Zung score 50–54)	3.07 (1.05–8.99)	0.04
Greater severity depression (Zung score ≥55)	4.00 (1.27–12.38)	0.02
Antidepressants versus none	2.14 (0.87–5.29)	0.10

Table 7b Adjusted associations* of depression†, stratified by trait anxiety status, with self-reported cataplexy

	From basic interaction model adjusted for antidepressants (B)‡ (*n* = 866)		From basic interaction model excluding antidepressant users (C)‡ (*n* = 709)		From basic interaction model adjusted for antidepressants, sleepiness and insomnia§ (D)‡ (*n* = 866)	
	OR* (95% CI)	*P*-value	OR* (95% CI)	*P*-value	OR* 95% CI	*P*-value
In participants without anxiety (STAI-T < 75th)						
Depression (Zung ≥50) versus no depression	8.85 (2.46–31.94)	0.0008	9.30 (1.75–49.40)	0.009	4.89 (1.28–18.67)	0.02
In participants with anxiety (STAI-T ≥75th)						
Depression (Zung ≥50) versus no depression	1.14 (0.34–3.82)	0.82	0.80 (0.14–4.68)	0.80	0.86 (0.25–3.00)	0.82

CI = confidence intervals.

*Also adjusted for age, sex, and body mass index (BMI) in kg/height (m^2).

†There were 25 participants with self-reported cataplexy.

‡*P*-trend = 0.007 for relationship of depression severity to self-reported cataplexy.

OR, odds ratios; CI, confidence intervals; STAI-T, Trait-Anxiety Scale of Spielberger State-Trait Anxiety Inventory. In models B and D, there were 25 participants with self-reported cataplexy; in model C there were 15 participants with self-portrait cataplexy.

[*] Odds ratios estimated from logistic regression models and also adjusted for age, sex, and body mass index in kg/height (m^2).

[†] Depression severity categories used in other models were collapsed into Zung ≥50 given the small sample size and similar relationships of lesser and greater severity depression to this outcome.

[‡] *P*-values for interaction terms for depression × anxiety (trait) were 0.02 for model B, 0.05 for model C, and 0.06 for model D.

[§] Sleepiness (daytime) defined as an Epworth Sleepiness Score > 11 (versus ≤11); Insomnia (self-reported) defined as waking up repeatedly during the night ≥5/month (versus ≤4/month).

[¶] No depression refers to no depressive symptoms (Zung < 50). Its odds ratio is 1.00.