

Sleep Disorder, Depression, and Suicidality in Female Sexual Assault Survivors

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The role of sleep in psychiatric illness in general, and depression and suicidality in particular, is poorly understood and has not been well researched despite the pervasiveness of sleep complaints in these conditions. As an exploratory, hypothesis-generating study, female sexual assault survivors with posttraumatic stress disorder (n = 153) who had enrolled in a nightmare-treatment program were assessed for subjectively determined sleep breathing and sleep movement disorders. Diagnoses of potential disorders were based on clinical practice parameters and research algorithms from the field of sleep disorders medicine. Potential sleep breathing and sleep movement disorders were present in 80% of the participants (n = 123) and included three subgroups: sleep-disordered breathing only (n = 23); sleep movement disorder only (n = 45); and both sleep disorders (n = 55). Based on the Hamilton Depression Rating Scale and Suicide subscale, participants with potential sleep disorders suffered greater depression (Cohen's $d = .73-.96$; $p < .01$) and greater suicidality (Cohen's $d = .57-.78$; $p < .05$) in comparison to participants without potential sleep disorders. The group with both sleep disorders suffered from the most severe depression and suicidality. A provisional hypothesis is formulated that describes how sleep disorders may exacerbate depression and suicidality through the effects of chronic sleep fragmentation.

Keywords: Depression, suicidality, nightmares, sleep disorders, posttraumatic stress disorder.

Insomnia and other sleep complaints are exceedingly prevalent in psychiatric illness [Benca et al., 1992; Benca, 1996; Nofzinger et al., 1993], particularly in suicidal patients [Agargun et al., 1997a; 1997b; 1998a; 1998b] in whom insomnia is a known risk factor that may be present: (a) within the year of a completed suicide [Fawcett et al., 1990]; (b) as a long-term behavioral characteristic in male suicides [Paffenbarger et al., 1994]; and (c) in severe suicide attempters in a managed care population [Hall et al., 1999]. Yet, de-

spite Fawcett et al.'s [1990] assertion that insomnia is a "modifiable risk factor" that should be viewed as one of four "target symptoms for vigorous treatment efforts" in suicidal patients, to our knowledge no study has ever assessed the underlying causes of poor sleep quality or nonrestorative sleep in suicidal patients, nor has any study thoroughly investigated self-reported insomnia complaints in suicidal patients to determine the specific type of insomnia, according to sleep medicine nosology [American Academy of

Sleep Medicine, 1997]. Moreover, the single sleep EEG study in suicidal patients [Sabo et al., 1991] did not investigate intrinsic sleep disorders such as sleep-disordered breathing and periodic limb movements, and it assessed insomnia solely with sleep stage data without integrating this objective information with clinical correlates. With respect to depression—one of the most important risk factors in suicidality [Jacobs, 1999; Lindeman et al., 1999]—only one prevalence study on sleep disorders in affective illness has been published [Reynolds et al., 1982]. Most importantly, no study has ever examined whether or not the direct treatment of sleep disorders in suicidal patients, using standard sleep medical therapies, would impact upon depression and suicidality.

Underestimating the role of sleep disturbance in suicidal patients was epitomized in a recent issue of the *Journal of the American Medical Association* in which a case was discussed regarding a 52-year-old man hospitalized for suicidal ideation. Although the patient was described as “sleeping poorly,” and experiencing “decreased energy and ability to concentrate,” this was presumed to be a function of his depression despite his current multitherapy with fluoxetine and trazodone. Due to an illness in a family member, the patient also suffered from acute sleep deprivation prior to his own hospitalization. The discussant’s only comment on these classic sleep disturbance symptoms was to note them as a potentiating risk factor because the patient had “not been sleeping regularly” [Jacobs, 2000]. There were no comments on (a) the differential diagnosis of sleep disorders that may have been causing this patient’s sleep complaints; (b) the chronicity and severity of the sleep complaints; (c) the potential therapeutic interventions for these sleep complaints; and (d) the possible beneficial effects on the patient’s depression and suicidal ideation and behavior if he were to receive sleep treatment.

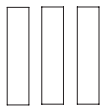
The most likely explanation for the above scenario is that sleep complaints are viewed as symptomatic phenomena of depression, and that treatment of depression ought to adequately address them. DSM-IV [American Psychiatric Association, 1994] emphasizes that sleep complaints are a symptom rather than a primary disorder, although it states that circumstances exist in which insomnia can be “sufficiently severe to warrant independent clinical attention.” Nonetheless, in clinical practice, therapeutic interventions are aimed

at the psychiatric illness with the expectation that sleep problems will resolve with successful treatment [Benca et al., 1992; Benca, 1996; Nofzinger et al., 1993], such as with sedating antidepressants [Benca, 1996; Kupfer, 1999]. However, although an enormous amount of anecdotal evidence and clinical experience strongly supports and entrenches this symptomatic approach [APA, 1994], no rigorous scientific studies using advanced polysomnographic and respiratory technology have ever confirmed the basis of this treatment perspective.

In contrast, research from various fields are now converging toward the view that sleep problems in depression and other psychiatric illnesses are actually comorbid, primary disorders in some proportion of patients [Ancoli-Israel et al., 1999; Aldrich et al., 1999; Krakow et al., 2000a; Kupfer, 1999; Reite, 1998]. Insomnia and other intrinsic sleep disorders may more than occasionally masquerade as a psychiatric illness or occur co-morbidly [Kalina et al., 1999; Krakow et al., 2000a; Millman et al., 1989; Reite, 1998]; and successful treatment usually has some beneficial impact on psychiatric symptoms [Derderian et al., 1988; Kupfer, 1999; Millman et al., 1989; Reite, 1998; Yu et al., 1999]. Depression has been observed to decrease or resolve with successful sleep apnea treatment, utilizing continuous positive airway pressure (CPAP) breathing masks [Derderian et al., 1988; Kalina et al., 1999; Millman et al., 1989; Mosko et al., 1989; Ramos Platon et al., 1992; Yu et al., 1999]. In PTSD research, 85% of a consecutive series of crime victims with PTSD, suffered from sleep-disordered breathing [Melendrez et al., 2000a], including obstructive sleep apnea and its subtle variant, upper airway resistance syndrome [Guilleminault et al., 1993, 1995]. Successful treatment of sleep disorders in PTSD have also been associated with decreases in nightmares, insomnia and posttraumatic stress [Youakim et al., 1998; Krakow et al., 2000b].

This emerging body of research indicates the need for greater scrutiny of intrinsic sleep disorders in appropriate psychiatric populations. Because polysomnographic studies are relatively expensive, we have taken an interim step by *subjectively* assessing intrinsic sleep disorders in PTSD patients. In a recent study, 77% of female sexual assault survivors with posttraumatic stress or PTSD endorsed classic symptoms for sleep breathing and sleep movement disorders [Krakow et

al., 2000a]. Those who reported symptoms of sleep-disordered breathing (SDB) and sleep movement disorders (SMD) suffered from greater PTSD severity than those without SDB or SMD. In the current study, potential sleep disorders were assessed in a similar sample of sexual assault survivors because they often report depressive symptoms and suicidal ideation or behaviors [Bagley et al., 1995; Chu, 1999; Davidson et al., 1996; Stepakoff, 1998]. We hypothesized that underlying intrinsic sleep disorders such as SDB and SMD would be highly prevalent and that more severe depression and suicidality would correlate with these potential sleep disorders.



Methods

Participants

The study was approved by the IRB of the University of New Mexico Health Sciences Center. All participants provided oral and written informed consent after complete description of the study. Women who complained of chronic nightmares, insomnia, and a history of sexual or physical assault were recruited ($n = 153$) from the general population, primarily through rape crisis centers, mental health centers, private therapists, and self-referral through newspaper and advertising sources. These patients were recruited for a nightmare-treatment research protocol; the results have been reported [Krakow et al., 2000c]. All participants were victims of sexual (95%) or physical (5%) violence, and 94% suffered from posttraumatic stress symptoms or PTSD in the moderate to severe range based on Foa's PTSD Symptom Scale [Foa et al., 1993]. Their mean (SD) age was 36.4 (11.1) years, and their average household income ranged between \$10,000 and \$30,000. Their average level of education ranged between some college, technical-vocational training or a bachelor's degree. Marital status included 27% married, 28% divorced, separated or widowed, 33% single, and 12% living with a partner; ethnicity included 58% Non-Hispanic White, 18% Hispanic, and 24% Mixed/Other.

Measures

The *Wisconsin Cohort Sleep Survey* [Young et al., 1993] was used to ascertain sleep symptoms and behavioral information relevant to the subjective assessment of intrinsic sleep disorders based on an algorithm developed in the previously cited investigation [Krakow et al., 2000a]. The *Hamilton Depression Rating Scale* (HDRS) [Hamilton, 1967] measures severity of depression in those with a depressive disorder and is administered through a personal interview. The *Hamilton Suicide* subscale (contained within the HDRS) consists of a single, 0 to 4 scale, in which the score is based on responses to seven clinical questions about suicidality. It has been used with depressed attempters, nonattempters and healthy controls in a sleep EEG study on suicidal patients [Sabo et al., 1991]. The scale was coded: 0 = Absent (No suicidal ideation/behavior); 1 = Doubtful or trivial (Feels life is not worth living); 2 = Mild (Wishes she were dead or any thoughts of possible death to self); 3 = Moderate (Suicide ideas or gesture); 4 = Severe (Attempts at suicide). *PTSD Symptom Scale* (PSS) is reliable and valid for diagnosing PTSD and measuring posttraumatic stress severity [Foa et al., 1993], according to DSM-III-R criteria [APA, 1987]. *Nightmare Frequency Questionnaire* is a self-report instrument retrospectively assessing nightmare frequency as a continuous variable. Both "nights with nightmares" (nights/week) and actual "number of nightmares" (nightmares/week) are measured [Krakow et al., 2001]. *Pittsburgh Sleep Quality Index* (PSQI) is a standardized, self-report questionnaire to assess sleep quality [Buysse et al., 1989]. Higher scores reflect worse severity for all instruments. Current use of antidepressants was also elicited.

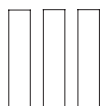
Potential Sleep Disorders

Potential sleep disorders were based on algorithms and clinical guidelines from current practice parameters in sleep medicine [AASM, 1997; ASDA, 1997] as had been applied in a previous study [Krakow et al., 2000a]. This method divided participants into four categories based on the presence or absence of sleep related movement disorders (SMD) and sleep-disordered breathing (SDB): No SMD or SDB; SMD only; SDB only; and, SMD and SDB. The categorizations do not in-

dicade objectively determined diagnoses but instead reflect a history of frequent symptom complaints consistent with these general categories. SMD was noted for patients who “often” or “always” kick people in bed or suffer from “restless legs” or who have had someone move from their bed because of kicking or other disruptive movements. For SDB, the criteria were based on the presence of snoring and frequent bouts of excessive daytime sleepiness or other respiratory irregularities (e. g., witnessed breathing cessation episodes or choking).

Statistical Analysis

Potential sleep disorders diagnostic subgroup means for depression and suicidality were subjected to a two-factor ANOVA based on the absence or presence of sleep-disordered breathing (SDB) and sleep-related movement disorders (SMD). Cohen’s *d* was used to calculate effect sizes for differences in mean depression and suicidality scores in the four sleep disorders diagnostic subgroups. χ^2 analysis was conducted on sleep disorders status versus antidepressant use. Statistical significance was set at .05.



Results

The mean (*SD*) suicidal scores were 1.33 (1.4). Frequency of suicidality was as follows: absent = 43%; trivial = 14%; mild = 18%; moderate = 16%; and severe = 9%. Thus, one-quarter of the sample suffered from clinical-

ly important levels of suicidality (moderate and severe), and overall 43% (mild, moderate, severe) were experiencing suicidal ideation at intake. Mean (*SD*) values for depression 25.2 (8.8), nights of nightmares/week 3.9 (2.0), nightmares/week 5.8 (4.3) and global sleep quality 12.2 (4.1) were all in the severe range, and the PTSD mean (*SD*) value of 28.8 (11.6) was in the moderately severe range. Anti-depressants were currently in use by 44% of the sample (*n* = 67).

Potential sleep disorders yielded four diagnostic subgroups: no SMD or SDB (*n* = 30); SMD only (*n* = 45); SDB only (*n* = 23); and SMD and SDB (*n* = 55). The ANOVA for depression yielded main effects for SMD ($F(1,149) = 9.047, p = .003$) and SDB ($F(1, 149) = 5.87, p < .02$) with no interaction ($p = .36$). The ANOVA for suicidality yielded a main effect for SMD ($F(1,149) = 7.373, p = .007$) and a marginal effect for SDB ($F(1,149) = 3.014, p < .09$) with no interaction ($p = .27$). This marginal effect was likely due to the small cell size of the SDB subgroup. Mean (*SD*) scores for depression and suicidality were significantly greater in the three sleep disorders diagnostic subgroups compared with the no disorders group; and, these mean differences were in the medium to large effect size range (depression, Cohen’s *d* = .73 – .96, $p < .01$; suicidality (Cohen’s *d* = .57 – .78; $p < .05$) (Table 1). The No SMD or SDB subgroup had the lowest proportion of antidepressant use (27% of group) with increasing use in the other three groups: SDB only, 39%; SMD only, 47%; SMD/SDB, 53%; average = 48%. The difference in antidepressant use between the no-disorders subgroup vs. the average for sleep disorders subgroups was 27% vs. 48% ($\chi^2 = 4.45$; Fisher’s exact test $< .05$).

Table 1
Univariate Comparisons and Effect Sizes for Mean (*SD*) Hamilton Depression and Suicidality Subscale Scores Between the No-Disorder Group and Three Sleep Disorders Groups

	<i>n</i>	Depression (Mean)	<i>t</i> **	<i>p</i>	<i>d</i> ***	Suicidality (Mean)	<i>t</i> **	<i>p</i>	<i>d</i> ***
No SMD or SDB*	30	20.03 (6.65)				.60 (1.13)			
SMD only	45	25.67 (8.61)	-2.86	.006	0.74	1.49 (1.36)	-2.9	.005	0.71
SDB only	23	24.83 (6.58)	-2.61	.01	0.73	1.26 (1.18)	-2.07	.04	0.57
SMD and SDB	55	27.84 (9.63)	-4.27	.0001	0.96	1.64 (1.53)	-3.53	.001	0.78

*SMD = Sleep movement disorders; SDB = Sleep-disordered breathing, **Comparison of no SMD or SDB group to each other group, ***Cohen’s *d*

Discussion

In this sample of female sexual assault survivors, enrolled in a nightmare-treatment research program, *potential* sleep breathing and movement disorders were very prevalent and were associated with worse depression and suicidality scores. The calculated effect sizes measuring the differences between the *potential* sleep disorders subgroups and the no-disorders subgroup were medium to large for both depression and suicidality, which suggests a clinically meaningful contrast between these groups. For example, the Hamilton Depression Rating Scale indicates that the sleep disorders diagnostic subgroups suffered from clinically severe depression (mean > 24) compared to the no disorders subgroup, which suffered from clinically moderate depression (mean = 20) [Hamilton, 1967].

These findings suggest that the relationship between sleep complaints and psychiatric illness are more complex than previously appreciated [Kalina et al., 1999; Krakow et al., 2000a; Kupfer, 1999; Millman et al., 1989; Reite, 1998] and further support the hypothesis that additional sleep disorders, beyond insomnia, may be a primary factor in this complexity, at least for this sample. Sleep disorders would likely have their greatest impact on psychiatric illness by compromising sleep quality through a final common pathway of repetitive arousals caused by problematic breathing (SDB), leg jerking (SMD) or psycho-physiological conditioning (insomnia). Most patients with these sleep disorders suffer hundreds of arousals throughout the night or they never enter into deeper, restorative stages of sleep [AASM, 1997]. This creates a state of chronic sleep fragmentation, which may not be easily recognizable, and which can take an insidiously chronic toll on both mental and physical energy reserves [Decary et al., 2000; Bardwell et al., 1999; Martin et al., 1997]. Thus, depressed and suicidal patients, burdened with concurrent sleep disorders, may further strain their already fragile coping capacity. As chronic sleep fragmentation is one of the most important causes of poor sleep quality and subsequent exhaustion and low energy [Dement, 1993], it would be interesting to learn whether or not sleep disorders might be associated with suicidal patients' frequent reports of "emotional exhaustion" or having "run out of energy" or "being too tired to go any longer" [Motto, 1999]. It would also

be of interest to learn whether or not suicidal ideation and behavior can be decreased by the direct treatment of sleep disorders in patients suffering from depression and suicidality.

Currently, sleep medicine has not been well integrated into most mental health practices [Benca et al., 1992; Dement, 1993; Ford et al., 1989; Reite, 1998], in part, because insomnia in psychiatric illness is construed as a secondary process. Yet, this study suggests that the insomnia of our patients was also associated with additional sleep disorders that would require treatments distinct from psychiatric interventions, for example, CPAP breathing masks or oral airway devices for SDB [Sullivan et al., 1984; Wright et al., 2000]. For those with insomnia *and* SDB, we have suggested a provisional term "complex insomnia" to facilitate the recognition of such patients [Melendrez et al., 2000b; Krakow et al., in press] because they may be suffering a doubling effect on their distress levels (as in "double depression") due to two intrinsic sleep disorders. Last, on a clinical note, we would like to offer a speculation regarding the nearly twice as frequent use of antidepressants in the sleep disorders subgroups compared to those without sleep disorders. From a sleep medicine perspective, we observe that the use of psychotropic medications may mask or divert awareness away from the diagnosis of an underlying sleep disorder [Yantis, 1999; Banno et al., 2000; Berkowitz, 1984], which, per our main hypothesis, may exacerbate the severity of the psychiatric illness for which the patient was prescribed the medication. Therefore, although highly speculative, we imagine that antidepressant use may be a marker for some patients who suffer greater psychiatric distress associated with undiagnosed and untreated sleep disturbances.

This research is limited by the study design, which focused on the recruitment of participants who sought treatment for the specific problem of nightmares—a known sleep disorder that may ultimately prove to be a marker for other sleep disorders. If so, the results would have exaggerated the prevalence of sleep disorders in our sample of sexual assault survivors with depression and suicidality. The study utilized a vast network of resources for recruitment and also used self-reported insomnia as another inclusion criteria. Nevertheless, while these attempts may have broadened the basis for recruitment, it cannot eliminate the problems inherent in having a target symptom such as

nightmares as the most important and most widely advertised inclusion criteria in the research protocol. Thus, it is unknown whether or not these findings would be valid for sexual assault survivors or other PTSD patients who report minimal problems with nightmares and insomnia. Most importantly, the findings regarding *potential* sleep disorders must be viewed as preliminary because there was no corroboration with objective sleep studies. Consideration must also be given to the direction of causality; perhaps, depression and suicidality symptoms cause or worsen these intrinsic sleep disorders. These questions must be addressed in future investigations.

In conclusion, some proportion of female sexual assault survivors with nightmares, PTSD, depression and suicidality appear to suffer from distinctly diagnosable and treatable sleep disorders in addition to insomnia. Objective prevalence studies are needed to determine how frequently these disorders occur in suicidal populations and to what extent they influence psychiatric distress. Further, as sleep medicine treatments are widely available, treatment studies are needed to examine the direct effects of sleep therapies on depression, suicidality, and PTSD in patients who suffer co-morbid sleep disorders. These intervention studies may help us learn more about the fundamental role of sleep dynamics in depression and suicidality.

Acknowledgment

Research supported by a grant from the National Institute of Mental Health (MH 53239).

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