Sleep Dynamic Therapy for Cerro Grande Fire Evacuees With Posttraumatic Stress Symptoms: A Preliminary Report

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Background: Sleep disturbance is common among disaster survivors with posttraumatic stress symptoms but is rarely addressed as a primary therapeutic target. Sleep Dynamic Therapy (SDT), an integrated program of primarily evidence-based, nonpharmacologic sleep medicine therapies coupled with standard clinical sleep medicine instructions, was administered to a large group of fire evacuees to treat posttraumatic insomnia and nightmares and determine effects on posttraumatic stress severity.

Method: The trial was an uncontrolled, prospective pilot study of SDT for 66 adult men and women, 10 months after exposure to the Cerro Grande Fire. SDT was provided to the entire group in 6, weekly, 2-hour sessions. Primary and secondary outcomes included validated scales for insomnia, nightmares, posttraumatic stress, anxiety, and depression, assessed at 2 pretreatment baselines on average 8 weeks apart, weekly during treatment, posttreatment, and 12-week follow-up.

Results: Sixty-nine participants completed both pretreatment assessments, demonstrating small improvement in symptoms prior to starting SDT. Treatment and posttreatment assessments were completed by 66 participants, and 12-week follow-up was completed by 59 participants. From immediate pretreatment (second baseline) to posttreatment, all primary and secondary scales decreased significantly (all p values < .0001) with consistent medium-sized effects (Cohen's d = 0.29 to 1.09), and improvements were maintained at follow-up. Posttraumatic stress disorder subscales demonstrated similar changes: intrusion (d = 0.56), avoidance (d = 0.45), and arousal (d = 0.69). Fifty-three patients improved, 10 worsened, and 3 reported no change in posttraumatic stress.

Conclusion: In an uncontrolled pilot study, chronic sleep symptoms in fire disaster evacuees were treated with SDT, which was associated with substantive and stable improvements in sleep disturbance, posttraumatic stress, anxiety, and depression 12 weeks after initiating treatment.

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Catastrophic events on September 11, 2001, sharply increased awareness of postdisaster psychological distress. Many Americans developed acute stress responses in the aftermath of the terrorist attacks on the World Trade Center and Pentagon.[1] Although research suggests that a majority of traumatized individuals would be expected to recover "spontaneously" or otherwise,[2–4] the magnitude of this disaster may cause a large increase in the incidence of chronic posttraumatic stress disorder (PTSD),[5] which may overwhelm mental health resources.[6] Crisis counseling and debriefing techniques are usually implemented early in the aftermath of a disaster,[7–12] but recent research on critical incident stress management indicates that debriefing may be ineffective or may even promote posttraumatic stress in susceptible individuals.[7–10] Few controlled studies aimed at disaster survivors have employed pharmacotherapy[13] or cognitive-behavioral therapies (CBT),[14] both considered first-line therapies for PTSD.[15] In sum, postdisaster treatment research is sparse, and therapeutic effects of postdisaster interventions have not been consistently demonstrated.[7]

Therapeutic objectives, appropriate treatment, and timing of intervention must be established for disaster-related

symptoms. Prevention or alleviation of chronic PTSD and other psychiatric distress is an obvious goal,[16] but if the majority of victims allegedly recover spontaneously from life-threatening events, how do we know whom to treat and when to treat them? Further, given the logistical problems associated with specific critical incidents, disaster survivors may have difficulty engaging in any treatment. We speculate that disaster survivors would be more likely to participate in brief programs that interfere minimally with their recently disrupted lifestyles and that offer immediate benefits for clear-cut symptoms. Moreover, the capacity to deliver an intervention to a large group might prove cost-effective in managing symptoms among disaster survivors.

Sleep disturbance might be a suitable target for post-disaster interventions because it is one of the most widely reported symptoms after traumatic exposure.[17] Following Hurricane Andrew, sleep disturbance was described as a complex symptom cluster[18,19]; and, in a recent study of Cerro Grande Fire evacuees in Los Alamos, New Mexico, posttraumatic sleep disturbance was extraordinarily complex and strongly associated with psychiatric distress.[20] On average, each of 78 treatment-seeking patients suffered 4 distinct behavioral or medical sleep disorders, most notably psychophysiologic insomnia, inadequate sleep hygiene, sleep-disordered breathing (SDB), and nightmares.[20] These findings align with recent studies demonstrating a similar pattern of complex sleep disorders among various trauma populations including sexual assault survivors, other crime victims, and possibly combat veterans.[21–25] Most importantly, sleep-oriented treatments have proven effective for trauma survivors.[21,26–30] Imagery rehearsal therapy (IRT) for nightmares in rape victims yielded large decreases in disturbing dreams and moderate-to-large improvements in sleep quality and posttraumatic stress severity compared with wait-list controls.[26,27] In crime victims, an open-label trial of IRT and evidence-based CBT for insomnia was associated with moderate-to-large improvements in nightmares, insomnia, and posttraumatic stress.[28] And, in 3 thought-provoking case series, continuous positive airway pressure (CPAP) treatment for SDB in trauma survivors was associated with elimination of insomnia[21,29,30] and large decreases in posttraumatic stress symptoms in patients receiving essentially no psychiatric intervention.[21,29]

In our clinical and research experience in sleep medicine, targeting the common stress symptoms of insomnia and nightmares generates more immediate interest among certain trauma survivors than interventions designed to help "relive the trauma and work through it."[26–28] We have recently developed a brief, symptom-specific, integrated sleep treatment program for trauma survivors—Sleep Dynamic Therapy (SDT)—consisting of primarily evidence-based sleep medicine therapies and standard instructions derived from clinical sleep medicine, delivered in 6, weekly, 2-hour sessions for the treatment of complex behavioral and physiologic sleep disturbance. SDT is offered in a psychoeducational format deliverable to a large group of trauma survivors. The current study piloted SDT in Cerro Grande Fire evacuees 10 months after the disaster. We hypothesized that SDT could be provided to a large group of adult men and women and would be associated with substantial improvement in insomnia and posttraumatic stress symptoms immediately posttreatment and 12 weeks from initiation of treatment.

METHOD

Recruitment and Eligibility

The study was approved by the University of New Mexico Health Sciences Center Human Research and Review Committee. In May 2000, the Cerro Grande Fire occurred in the Santa Fe National Forest surrounding Los Alamos and White Rock, New Mexico. The details of the fire, evacuation, and the sample's sleep disturbance and distress symptoms have been described.[20] A community-wide recruitment was initiated from October 2000 through January 2001 and included all forms of media as well as personal contact such as survivors' group meetings. Inclusion criteria comprised adults who, in the aftermath of the fire, reported the onset of insomnia or nightmares *allegedly caused* in part or wholly by the fire, evacuation, or related events. If these sleep problems were preexisting, then individuals were eligible if the fire *allegedly exacerbated* them. Exclusion criteria included age less than 18 years, acutely psychotic or suicidal, or acutely intoxicated or in withdrawal from alcohol or substances. Past history and current diagnosis of psychiatric disorders and usage of psychotropic medication were not exclusion criteria. After a complete description of the study was given to participants, informed written and oral consent was obtained.

Sample and Procedures

The study was originally designed as a controlled intervention, but one third of participants were blood relatives, mostly husbands and wives as well as adult sons and daughters or grandparents of other adults in the study, and an overlapping third of participants were living in closely situated temporary shelters. Because of our concern that these factors might have fostered a large bias due to patient interaction between groups, the study was changed near the end of recruitment to an open-label pilot protocol without a control group. Ninety-four participants screened eligible for the study, and 78 completed an initial intake. Once the study design was revised, an effort was made to assess for spontaneous recovery by conducting a second intake (second baseline) on average 7.5 weeks after the initial intake assessment and within 1 week of commencing treatment. Sixty-nine participants

Table 1. Sample Characteristics for Participants Who Completed Both Pretreatment and Posttreatment Assessments

Characteristic	Women (N = 40)	Men (N = 26)	Total (N = 66)
Age, mean (SD), y	50.10 (13.97)	54.50 (14.25)	51.83 (14.14)
Body mass index, mean (SD), kg/m^2	24.88 (3.87)	26.73 (4.50)	25.61 (4.20)
Race, N (%) white	27 (68)	23 (89)	50 (76)
Marital status, N (%) married	28 (70)	18 (69)	46 (70)
Education, N (%) with Bachelor's degree or higher	24 (60)	19 (73)	43 (65)
Annual income, N (%) earning ≥ $50,000	29 (73)	21 (81)	50 (76)

completed this second baseline and attended the first treatment session; and 66 of 69 completed the treatment program and posttreatment assessment. These 66 patients are the focus of this preliminary report (Table 1).

Treatment Protocol

Treatment was formatted for a large group to determine whether such an approach could be feasibly administered. All treatment sessions were conducted in the main auditorium of the Los Alamos Church of Christ. Due to the size of the group, missed sessions were anticipated, and the second (D.C.M.) and third authors (L.G.J.) conducted makeup sessions within a few days of each of the original large group sessions. Thirty-five people attended from 1 to 3 makeup sessions in groups typically ranging from 4 to 10 people. On average, 58 participants attended any given session, and 31 people attended all 6 regular sessions. Participants were encouraged to actively participate in the program by offering very brief examples of their experience with sleep disturbance and by answering questions raised in the session. There was no payment for participation in any portion of the program.

Therapeutic principles. Treatment incorporated 6 educational/therapeutic principles (Appendix 1) delivered in 6, weekly, 90-minute sessions, each followed by a 30-minute question and answer period. The overriding precept is that treatment-seeking trauma survivors are likely to suffer complex behavioral and medical sleep disorders that must be addressed to provide sustainable improvement in sleep.[22–25] The foremost principle involves sleep quality and its self-assessment. Many sleep disorders patients equate sleep quality with "sleeping through the night,"[31] which may or may not correlate with actual physiologic disruption of their sleep. Moreover, sleep disorders patients tend to deny or report limited awareness of sleep problems, for example, snoring.[32] In our experience, most sleep disorders patients "normalize" their perception of their own sleep problems regardless of severity. Admittedly, it is impossible to monitor one's sleep while sleeping. By focusing attention on sleep quality, patients are encouraged to adopt and develop a "personal scientist model"[33] in an attempt to explore and manage their own sleep problems. Additional principles include CBT to promote deconditioning of maladaptive learned behaviors and habits that promote sleep disturbance,[34] advanced CBT procedures such as stimulus control and sleep restriction[35] coupled with sleep hygiene principles,[33] emotional processing to examine links between anxiety and depressive symptoms and ruminations that foster insomnia,[36] IRT for chronic nightmares,[26,27] and sleep physiologic self-assessment to identify signs and symptoms of potential SDB or sleep movement disorders (SMD).[37]

Session 1. *Sleep quality assessment* teaches the patient to develop a capacity for assessing personal sleep quality, which is likely to reveal crucial insights into the real causes of one's sleep problems. Patients compare their sleep experiences with that of normal sleepers to facilitate awareness of poor sleep quality markers: restless sleep, feeling unrested in the morning, low energy reserves, fatigue or sleepiness, and caffeine use to promote wakefulness.[37,38] Yet, apparently, many sleep disorders patients as well as primary physicians do not readily connect these daytime sequelae to poor sleep.[39] *Sleep fragmentation* is a term that provides a broad paradigm to explain how persistent sleep disruption robs any sleeper of consolidated, deep or restorative slumber.[40] We use this term to explain how physiologic and behavioral factors may lighten or disrupt normal sleep. Because most insomniacs believe that psychological factors are of primary importance, they are instructed on how behavioral *and* medical sleep disorders fragment sleep and are encouraged to closely scrutinize signs and symptoms relevant to their sleep problems. Sleep quality assessment also helps to disengage insomniacs from their natural tendency to obsess and catastrophize about sleep quantity, which often produces self-perpetuating cycles of sleeplessness, that is, "losing sleep over losing sleep."

Behavioral deconditioning is needed for most troubled sleepers.[34] For example, patients must learn that clock-watching, while seemingly a logical endeavor to engage in during a sleepless night, actually promotes insomnia by creating unrealistic expectations about sleep quantity and triggering a conditioned response to monitor time in bed instead of sleeping.[41] Lying in bed not sleeping leads to further sleep fragmentation. Insomniacs learn that *sleep quantity* obsessions are time obsessions that hamper efforts to improve one's sleep quality. Patients are encouraged to remove clocks from the bedroom or face them away from the bed.

Session 2. Behavioral deconditioning is taught using a "day is done" metaphor in which patients learn that mental and physical factors thwart efforts to relax before bedtime and instead produce sleeplessness by triggering a state of greater alertness.[42] If the mind/body appear "motivated" to remain awake, then insomnia ensues. This point resonates

well with trauma survivors who realize that hypervigilance consistently signals that the day is *not* done[17,24,29]; that is, if recurrence of a traumatic episode seems remotely possible, then survival dictates remaining awake at all times to guard against renewed assaults or disasters.

Sleep hygiene principles are explored to compare normal behaviors with sleep antagonistic behaviors.[33,34] Normal sleepers generally have regular bed and wake-up times, and mild insomniacs can be instructed to set such schedules. Posttraumatic insomniacs are likely to find such an approach futile, counterproductive, and somewhat offensive with respect to bedtimes because hypervigilance frequently prevents sleep onset.[28] Instead, patients are offered a menu of standard sleep hygiene behaviors that permit flexibility and induce minor changes. A regular wake-up time with the aid of a morning alarm (faced away from the bed) is easier to initiate than a regular bedtime. A brief instruction on an advanced deconditioning approach known as "stimulus control" is offered to eliminate stimuli that promote sleeplessness. Simply, if lying in bed awake promotes insomnia, then consider getting up out of bed until sleepiness returns.[35]

Session 3. *Stimulus control* may be used on intrusive thoughts (ruminations or self-talk) and feelings (ill-defined tension) that may linger despite following deconditioning and sleep hygiene procedures. One form of stimulus control is a specific, set-aside worry-period, which appears to be effective for some insomniacs.[43] However, all 3 approaches may be overly simplistic for sleep disorders patients with psychiatric distress[24,29] who instead might benefit from a traditional psychodynamic model in which ruminations are understood as a defense against sleep.[36] For example, if there is anxiety about the potential for unwanted or unpleasant feelings to emerge during sleep, bodily tension (somatization) and self-talk (intellectualization) at bedtime could produce the desired alternative—insomnia. Insomniacs may fear sleep because they fear loss of control. Through emotional processing, awareness of previously avoided feelings or expression of these feelings may lead to decreased tension, self-talk, and insomnia.[36]

Emotional processing is discussed in the context of anxiety and depression—2 common posttraumatic stress symptoms—that may be masking more powerful feelings, notably fear and anger. The intensity of any of these emotions is capable of inhibiting sleep. Thus, the assumption is conveyed that emotions are multilayered and that emotional processing may be most effective when layers are explored,[44] albeit emotional processing has not been formally studied in the treatment of insomnia. Pragmatically, the presence of tension and self-talk at bedtime can be discerned as "red flags" that require attention, perhaps through emotional processing or enhanced awareness of feelings. The objective is not to provide psychotherapy, although patients are encouraged to work with outside therapists as needed, albeit few used psychotherapists during this program. In this context, emotional processing educates the patient about the relationship between self-talk and inhibited emotional expression that may be fueling their own sleeplessness.

Session 4. IRT has been well-described[26,27] as a waking imagery technique for the treatment of nightmares in which the patient is instructed to note a disturbing dream and then mentally "change it any way you wish" per Neidhardt and colleagues' model.[45] The nightmare sufferer then rehearses the "new dream" in the waking state for a few minutes each day, but to limit overstimulation, the patient is instructed to work each week on changing only 1 or 2 disturbing dreams into new dreams. Anecdotal evidence suggests that some trauma survivors use IRT to deal with daytime distressing images and in facilitating emotional processing.[27]

Session 5. Participants are educated on the 3 defining sleep-related posttraumatic stress symptoms: intrusion (nightmares), avoidance (avoiding sleep to avoid dreams and unpleasant feelings in dreams), and arousal (insomnia).[17] Using techniques learned in the earlier sessions, patients are instructed to recognize and target their sleep-related symptoms to decrease their overall posttraumatic stress levels. Refresher information is provided on sleep quality, deconditioning, stimulus control, sleep restriction, sleep hygiene, emotional processing, and IRT.

Session 6. *Physiologic assessment* examines signs and symptoms of SDB and SMD. For example, insomniacs with SDB learn that abnormal sleep breathing produces cognitive impairments such as difficulties with memory and concentration[46] and physical signs and symptoms such as nocturia, awakening with a dry mouth, morning headaches, and problems controlling blood pressure.[47] Patients are advised that loud snoring and daytime sleepiness may not manifest as in classic SDB,[48] particularly in those with comorbid insomnia.[24,49] Participants are instructed to assess symptom clusters regarding their own health status to determine the need for additional consultation at a sleep disorders clinic.

Questionnaires and Follow-Up

Primary outcomes for sleep and distress included the Insomnia Severity Index (ISI)[50] and the Posttraumatic Stress Diagnostic Scale (PDS),[51] which assesses PTSD based on DSM-IV criteria.[52] The ISI and PDS were assessed 8 times (at both first and second baselines; during second, third, fourth, and fifth treatment weeks; posttreatment; and 12 weeks from the start of treatment) to demonstrate the trajectory of change in symptoms during the course of the protocol. This same 8-point trajectory of change was monitored for 3 standard sleep indices: an 8-item, ordinal Sleep Quality scale; total sleep time; and sleep efficiency (total sleep time/time in bed). Secondary measures were assessed 4 times, including both baselines, posttreatment, and 12 weeks after treatment initiation: the

Hopkins Symptom Checklist-25 (HSCL-25)[53] measures anxiety and depression as does the Symptom Questionnaire (SQ)[54]; the Impact of Event Scale-Revised (IES-R)[55] measures posttraumatic stress; and the Disturbing Dream and Nightmare Severity Index (DDNSI)[20] measures global nightmare severity. Psychometric properties of these scales have been well-described in the psychiatric literature except for ISI and DDNSI.

The ISI (formerly Sleep Impairment Index) is a reliable and valid instrument that quantifies perceived insomnia severity. It is useful both as a screening device and as an outcome measure in insomnia treatment research for both primary and psychiatric insomnia. The scale consists of 7 items, measuring severity of difficulties with sleep onset, sleep maintenance, early morning awakenings, sleep problem interference with daily functioning, indications to others of impairment due to sleep problems, degree of concern about current sleep problem, and satisfaction/dissatisfaction with current sleep pattern. Cronbach alpha averaged .76 in 2 previous studies.[50] Concurrent validity has been established with prospective sleep diary recording (r = 0.32 to 0.91). Each item is scored from 0 = essentially no problems to 4 = very severe problems, yielding a composite score ranging from 0 to 28. Scores >10 indicate clinical meaningful insomnia.[50]

The DDNSI (revised version of Nightmare Frequency Questionnaire)[56] uses 5 self-report items to measure nights of nightmares per week (0 to 7 nights), number of nightmares per week (0 to 14), awakenings due to nightmares (0 = never to 4 = always), severity of nightmare problem (0 = no problem to 6 = extremely severe), and nightmare intensity (0 = not intense to 6 = extremely severe intensity). The 5 items are summed to yield an index of nightmare severity (range = 0 to 37). Cronbach alpha is .91. Scores > 10 were most consistent with the diagnosis of a chronic nightmare disorder; however, because these disturbing dreams were in the context of posttraumatic stress, the nightmares may be "related to another mental disorder."[52,57]

Assessment procedures were not blinded, although participants received questionnaires primarily through the mail to be completed and returned. At second baseline and posttreatment, the large majority of participants completed questionnaires in person prior to the start of the first and final treatment sessions, respectively. Overall, posttreatment and 12-week follow-up focused on the effects of the initial 5 treatment sessions; whereas, the impact of the sixth session (sleep physiologic self-assessment of SDB or SMD) will be evaluated in the future on patients who seek treatment at sleep disorders centers.

Data Analysis

All analyses were conducted on the 66 participants who completed the study. During eligibility screening, ordinal-scaled (4, 5, or 6 levels) and dichotomous questions were used to gather data on self-reported effects of the evacuation, psychotherapy or medication use prior to the disaster and in the immediate postdisaster period, and past sleep and psychiatric disorders. Because we did not use a control group, spontaneous recovery was assessed with 2 separate analyses that compared the interval between first and second baseline (pretreatment phase) and the interval between second baseline and posttreatment (intervention phase). First, we used repeated-measures analysis of variance (ANOVA) to compare 2 clinically relevant groups—those whose ISI (N = 29) or PDS (N = 31) scores were unchanged or worsened pretreatment (PRE-WORSE) and those whose ISI (N = 37) or PDS (N = 35) scores improved pretreatment (PRE-IMPROVE). Within each group, ISI and PDS changes were compared between pretreatment and intervention phases. Second, we used a random-effects regression analysis to compare the slopes between pretreatment and intervention phases in the entire sample.

For the main analyses of the 66 participants, a last-observation-carried-forward, repeated-measures ANOVA was used for primary and secondary outcomes, comprising 59 participants who completed posttreatment and 12-week follow-up and 7 individuals who completed posttreatment follow-up only. For these 7 participants, the means of the posttreatment values carried forward were equal to or slightly worse than means of the 59 completers for all primary and secondary outcomes; thus, inclusion of these observations at the 12-week follow-up did not inflate effect sizes. To complement this analysis and to examine the problem of missing values more carefully, random-effects regression analysis in SAS PROC MIXED (SAS Institute, Cary, N.C.) was also performed on the 8-level repeated-measures design for ISI and PDS. This analytic method also modeled the cluster effect introduced by the presence of married couples among the subjects.

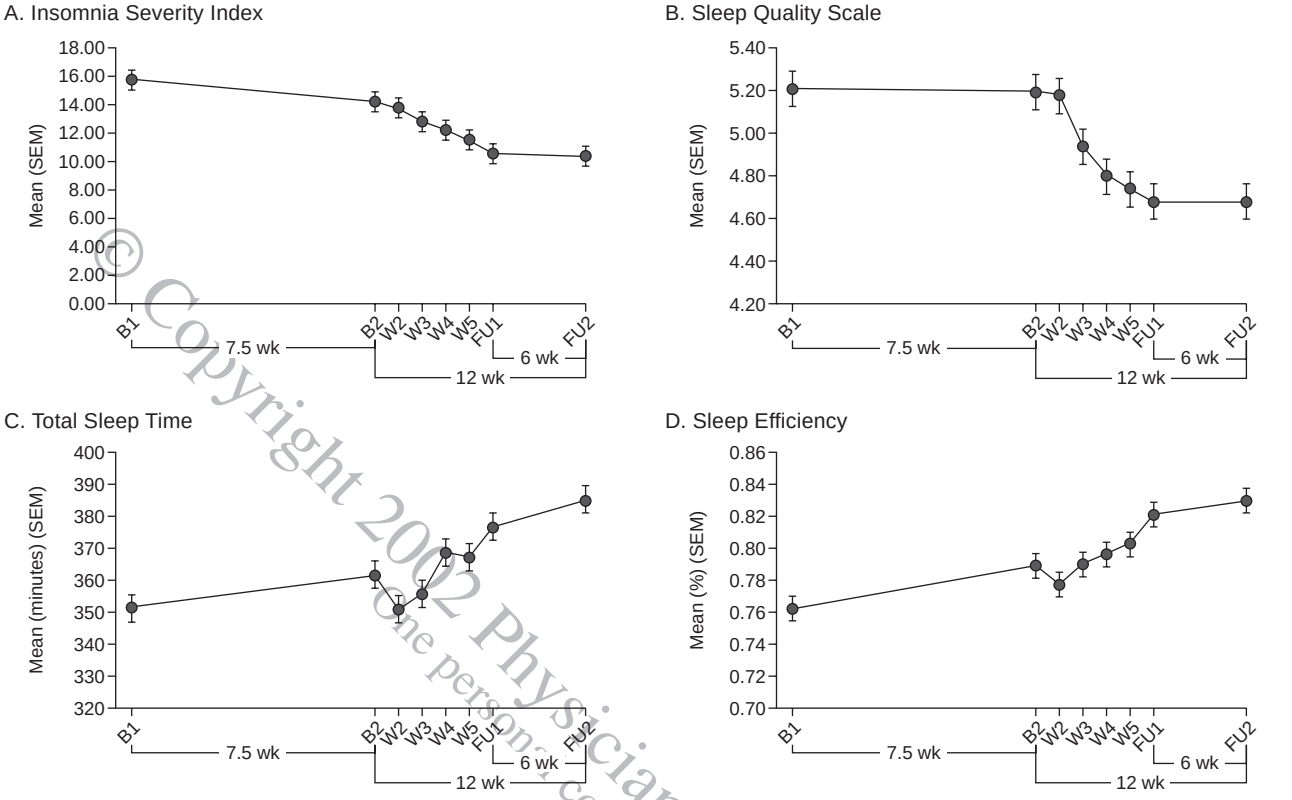
Posttreatment changes in ordinal clinical severity levels of insomnia were analyzed with McNemar test and changes in ordinal clinical severity levels of PTSD were analyzed with Wilcoxon signed rank test. Moderating effects of independent dichotomous variables (prior insomnia, nightmare severity, full session attendance, loss of home, prior psychiatric disorders, and gender) and for the number of sleep disorders on ISI and PDS were analyzed with the random-effects regression model. Alpha was set at .05. Effect sizes (Cohen's d, the mean standardized difference) were calculated for the pretreatment baseline period, for the treatment interval, and for the posttreatment follow-up period.

RESULTS

Pretraumatic, Peritraumatic, and Posttraumatic Sleep and Psychiatric Complaints

From the screening data, which were indicative of either the predisaster or the immediate postdisaster period,

Figure 1. Mean (SEM) Changes From Baseline, 5 Weeks of Treatment, Posttreatment, and 12-Week Follow-Up for (A) Insomnia Severity Index, (B) Sleep Quality Scale, (C) Total Sleep Time, and (D) Sleep Efficiency (N = 66)[a]



[a]Data for the interval B1 to B2 describe the baseline period of change without intervention. Data for the interval of B2 to W5 reflect the period of treatment. Data at FU1 and FU2 reflect immediate posttreatment and 6 weeks after conclusion of treatment. Insomnia Severity Index and Sleep Quality Scale improvement is shown through lowering of scores, whereas in the scales of Total Sleep Time and Sleep Efficiency, improvement is shown through increases in time and percentage, respectively.

self-reported stress that was related to the evacuation averaged moderate to severe, yet concerns about safety averaged mild, and perceived threats to one's life were nearly absent. Only a handful of people reported incurring minor injuries related to the fire or evacuation activities. Average proximity to the fire was less than 1 mile, but participants reported being only mildly affected by smoke. Significant loss of personal property—"lost everything," "lost home," or "home was severely damaged" such that they had to live elsewhere—was reported by 50% of the sample. Disruption to home life averaged moderate to severe, disruption to social life averaged moderate, and disruption to work life averaged moderate.

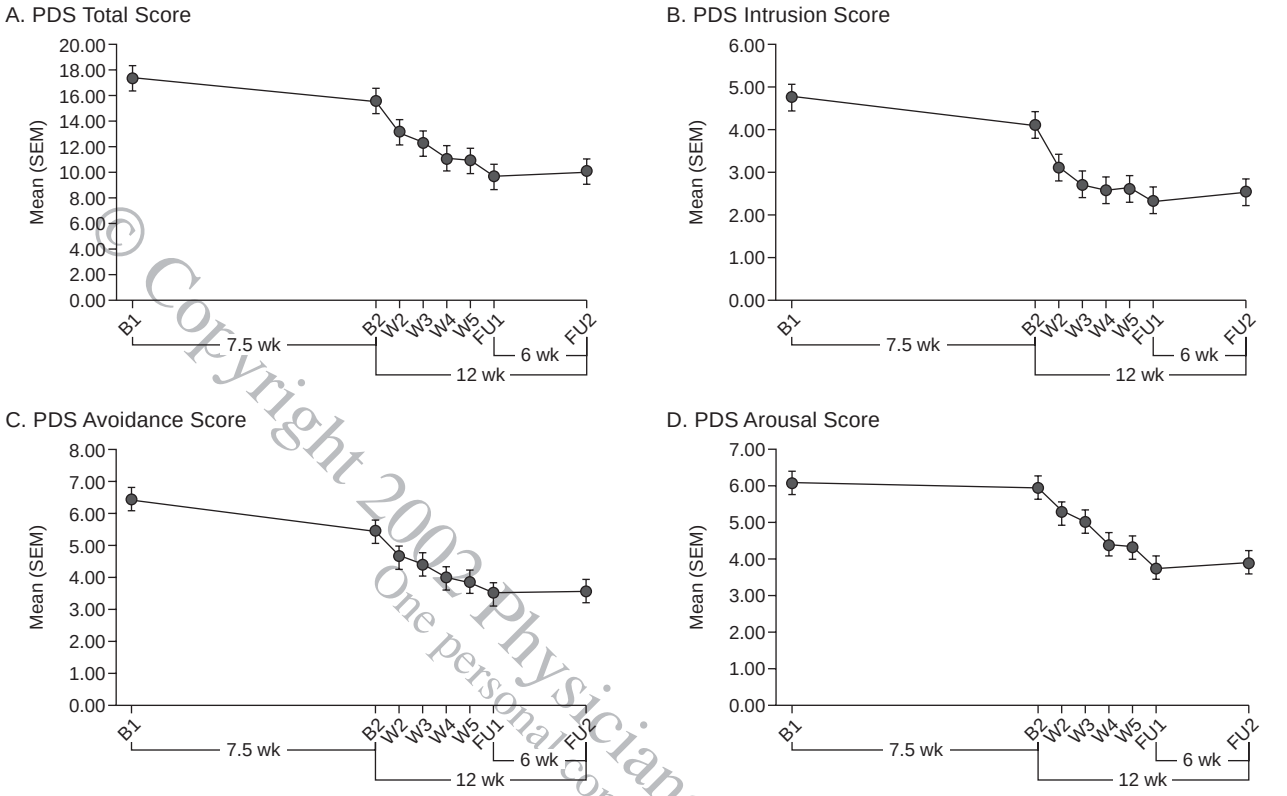
Additional screening information revealed that all patients complained of insomnia, of which 24 indicated that sleeplessness started after the fire, whereas 42 reported preexisting insomnia that worsened because of the fire. Nightmare complaints were registered by 47% (N = 31) of participants, of whom 68% (N = 21) indicated that disturbing dreams started after the fire and 32% (N = 10) reported preexisting nightmares that worsened because of the fire. For past problems with anxiety, depression, PTSD, insomnia, or nightmares, only 12% (N = 8) reported ever having used a psychotropic medication before the fire, whereas 46% (N = 30) reported taking medication for a short time after the fire (McNemar test, $\chi^2 = 18.75$, $p < .001$). For these same 5 conditions, only 17% (N = 11) reported ever having seen a therapist prior to the fire, whereas 33% (N = 22) reported seeing a therapist for a short time after the fire (McNemar test, $\chi^2 = 26.29$, $p < .01$).

Sample characteristics for the 66 participants are reported in Table 1. At first and second baseline, 56% (N = 37) met diagnostic criteria for current PTSD. Forty-seven percent (N = 31) of the sample reported a past history of a diagnosed psychiatric disorder or a major life stressor, primarily as childhood abuse, claustrophobia, anxiety, or depressive disorders.

Spontaneous Improvement

For insomnia, 29 PRE-WORSE patients had a medium increase in insomnia symptoms between baselines ($F = 14.58$, $df = 1,28$; $p = .001$; $d = –0.42$), yet showed a large decrease from second baseline to posttreatment

Figure 2. Mean (SEM) Changes From Baseline, 5 Weeks of Treatment, Posttreatment, and 12-Week Follow-Up for (A) Posttraumatic Stress Diagnostic Scale (PDS) Total Score, (B) PDS Intrusion Score, (C) PDS Avoidance Score, and (D) PDS Arousal Score (N=66)[a]



[a]Data for the interval B1 to B2 describe the baseline period of change without intervention. Data for the interval B2 to W5 reflect the period of treatment. Data at FU1 and FU2 reflect immediate posttreatment and 6 weeks after conclusion of treatment. In each scale, an improvement is shown through a lowering of scores.

(F = 20.31, df = 1,28; p = .0001; d = 0.78). Although 37 PRE-IMPROVE patients had a large decrease in insomnia between baselines (F = 90.80, df = 1,36; p = .0001; d = 0.75), they demonstrated a further medium decrease during treatment (F = 22.48, df = 1,36; p = .0001; d = 0.52). For PTSD, 31 PRE-WORSE patients had a small increase in posttraumatic stress symptoms between baselines (F = 20.16, df = 1,30; p = .0001; d = –0.32), yet showed a medium decrease during treatment (F = 24.45, df = 1,30; p = .0001; d = 0.61). Although 35 PRE-IMPROVE patients had a large decrease in posttraumatic stress symptoms between baselines (F = 41.49, df = 1,34; p = .0001; d = 0.86), they demonstrated a further large decrease during treatment (F = 30.18, df = 1,34; p = .0001; d = 0.72).

Primary Sleep and PTSD Outcomes

Changes in all primary outcomes, based on the last-observation-carried-forward analysis, are depicted in Figure 1 and Figure 2. The random-effects regression model contrasts the slope (b values) during baseline to that during the treatment period, as well as to that during the posttreatment follow-up period. A significant improvement occurred during the baseline period (for insomnia: b = –1.58; t = –3.43; df = 440, p < .001; d = –0.34; for PTSD: b = –1.50; t = –2.17, df = 435, p = .03; d = –0.20) and during the treatment period (for insomnia: b = –3.61; t = –5.70, df = 440, p < .0001; d = –0.77; for PTSD: b = –5.69; t = –7.28, df = 435, p = .0001; d = –0.76), but no significant change occurred during the follow-up period. Improvement during the treatment period was significantly greater than during the baseline period (for insomnia: b = –2.03; t = –2.25, df = 440, p < .03; d = –0.43; for PTSD: b = –4.19; t = –3.34, df = 435, p < .001; d = –0.56), indicating that a large treatment effect occurred beyond the small (but significant) spontaneous improvement that was observed during the baseline period. The statistically significant cluster effect for married couples was included in all models, indicating that the spontaneous recovery and treatment effects occurred independent of the cluster effect.

Overall, for insomnia, 49 patients improved, 11 worsened, and 6 reported no change in symptoms. For PTSD, 53 patients improved, 10 worsened, and 3 reported no

Table 2. Pretreatment vs. Follow-Up Scores for Impact of Events, Anxiety, Depression, and Nightmare Severity

	Pretreatment (N = 66)				Follow-Up (N = 66)[a]								
	Baseline 1		Baseline 2		Posttreatment		12-Week Follow-Up		Change[b]		Statistic		
Measure	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	F	(df)	Cohen d[c]
Impact of Event Scale													
Intrusion	1.42	0.86	1.09	0.77	0.68	0.64	0.76	0.65	0.33	0.67	25.69	(3,63)*	0.46
Avoidance	1.06	0.88	0.82	0.85	0.53	0.61	0.53	0.55	0.29	0.68	12.39	(3,63)*	0.41
Arousal	1.08	0.85	0.86	0.78	0.47	0.63	0.58	0.71	0.28	0.63	20.09	(3,63)*	0.38
Hopkins Symptom Checklist													
Anxiety	1.51	0.47	1.49	0.54	1.34	0.44	1.33	0.52	0.16	0.28	11.89	(3,63)*	0.30
Depression	1.84	0.54	1.75	0.53	1.59	0.47	1.59	0.57	0.16	0.38	11.28	(3,63)*	0.29
Symptom Questionnaire													
Anxiety	10.05	5.07	9.88	5.43	7.74	5.75	6.36	5.74	3.52	3.80	20.44	(3,63)*	0.63
Depression	8.56	5.24	7.76	5.29	5.95	4.83	5.98	5.78	1.78	4.61	8.71	(3,63)*	0.32
Nightmare Severity Index[d]	13.23	5.76	16.13	6.00	9.81	8.74	7.56	9.40	8.57	8.16	9.62	(3,21)*	1.09

[a]Fifty-nine participants completed "12-Week Follow-Up"; scores of the other 7 were carried forward.
[b]Difference between "Baseline 2" and "12-Week Follow-Up."
[c]Effect size = standardized mean difference.
[d]N = 24 for Nightmare Severity Index, which reflects only the participants who met diagnostic criteria for nightmare disorder at pretreatment.
*p < .0001.

Table 3. Clinical Severity Changes for Insomnia and PTSD at Pretreatment vs. Follow-Up (N = 66)[a]

	Pretreatment				Follow-Up			
	Baseline 1		Baseline 2		Posttreatment		12-Week Follow-Up	
Measure (score range)	N	%	N	%	N	%	N	%
Insomnia Severity Index[b]								
Nonclinical insomnia (≤ 10)	9	14	15	23	39	59	40	61
Clinical insomnia (> 10)	57	86	51	77	27	41	26	39
Posttraumatic Stress Diagnostic Scale[c]								
None (0)	1	2	0	0	3	4	2	3
Mild (1–10)	19	29	23	35	38	58	38	58
Moderate (11–20)	22	33	25	38	21	32	20	30
Moderate-severe (21–35)	21	32	16	24	3	4	5	7
Severe (36–51)	3	4	2	3	1	2	1	2

[a]Data are expressed in column percentage for pretreatment and follow-up. Insomnia severity is based on scoring for the Insomnia Severity Index.[50] PTSD symptom severity is based on scoring for the Posttraumatic Stress Diagnostic Scale.[51] Fifty-nine participants completed "12-Week Follow-Up"; scores of the other 7 were carried forward.
[b]Using McNemar test, there is no significant difference in clinical severity between baselines, and highly significant difference (p < .0001) from "Baseline 2" to "12-Week Follow-Up."
[c]Using Wilcoxon signed rank test, there is no significant difference in clinical severity between baselines, and highly significant difference (p < .0001) from "Baseline 2" to "12-Week Follow-Up."

change in symptoms. Decreases in PTSD subscales showed at least medium effect sizes for intrusion (d = 0.56), avoidance (d = 0.45), and arousal (d = 0.69) (all F values > 6, all p values < .0001). Correlation coefficients between ISI and PDS scores were large (mean r = 0.54; range, 0.46 to 0.59) at each of the 8 assessment points.

For 3 sleep indices, small or no changes were noted between baselines (mean d = 0.10), but statistically reliable small effects for sleep quality (F = 10.69, df = 1,65; p = .002; d = 0.35), total sleep time (F = 4.61, df = 1.65; p = .04; d = 0.19), and sleep efficiency (F = 4.72, df = 1,65; p = .03; d = 0.24) were reported following treatment (Figure 1).

Secondary Distress Outcomes

All secondary measures of distress decreased significantly (all F values > 8, all p values < .0001) from second baseline to posttreatment, demonstrating small to medium effects (d = 0.29 to 0.63) for anxiety and depression and medium to large effects (d = 0.38 to 1.09) for IES-R and DDNSI; and these improvements in distress were maintained at 12-week follow-up (Table 2). Correlation coefficients between ISI and 2 depression scales (mean r = 0.50; range, 0.32 to 0.62) and between ISI and 2 anxiety scales (mean r = 0.51; range, 0.35 to 0.62) were large at each of 4 assessment points.

Supplemental Analyses

Statistically significant changes in insomnia and PTSD clinical severity levels are reported in Table 3. The only significant moderating effect on treatment effectiveness was for the presence or absence of a baseline PTSD diagnosis. For those with PTSD (N = 37), posttraumatic stress changes with intervention were larger compared with those without the diagnosis (N = 29) (F = 5.85, df = 1,64; p = .02; d= 0.81 vs. 0.52). The number of men and women using medication or psychotherapy throughout the program declined or stayed the same, with the largest drop occurring between baseline assessments in women for medication use. Thereafter, use was too small to warrant meaningful statistical analysis (Table 4). For newly initiated, supplemental insomnia treatment outside the scope of the program, 4 patients reported using prescription or over-the-counter sedating drugs at posttreatment, 2 reported medication use at 12-week follow-up, and 2 patients reported under-

Table 4. Number (Percentage) of Patients Using Psychotherapy and/or Psychotropic Medication During the Treatment Protocol

	Pretreatment				Follow-Up			
	Baseline 1		Baseline 2		Posttreatment		12-Week Follow-Up[a]	
Treatment	N	%	N	%	N	%	N	%
Psychotherapy								
Female (N = 40)	5	13	6	15	6	15	1	3
Male (N = 26)	1	4	2	8	2	8	1	5
Total (N = 66)	6	9	8	12	8	12	2	3
Medication								
Female (N = 40)	17	43	11	28	5	13	5	14
Male (N = 26)	4	15	3	12	1	4	1	5
Total (N = 66)	21	32	14	21	6	9	6	10
Either medication or therapy								
Female (N = 40)	17	43	14	35	8	20	5	14
Male (N = 26)	4	15	5	19	2	8	2	9
Total (N = 66)	21	32	19	29	10	15	7	12

[a]Female (N = 37), male (N = 22), total (N = 59).

going nasal surgery at 12-week follow-up. For newly initiated, supplemental distress treatment outside the scope of the program, 2 patients reported using prescription medication, 1 reported seeing a therapist at posttreatment, and 1 patient reported seeing a therapist at 12-week follow-up.

COMMENT

In our uncontrolled pilot study of Sleep Dynamic Therapy, the majority of patients reported improvements in behavioral sleep disorders, notably insomnia and nightmares, as well as in distress. At every point of assessment, insomnia severity scores consistently correlated with posttraumatic stress, anxiety, and depressive symptoms, indicating a strong association between sleep and distress measures. However, in the absence of a control group, no definitive comments can be made about the therapeutic effects of SDT. Nonetheless, this study points to the extreme complexity of posttraumatic sleep disturbance that may occur in patients such as these disaster survivors, and this complexity does not appear to be appropriately or accurately explained by the paradigms of "psychiatric insomnia"[58] or "insomnia related to another mental disorder"[59] that are frequently used to describe the sleep complaints of psychiatric patients.[52] Rather, sleep disorders' complexity consisted of stress-related sleep disturbance as well as behavioral and physiologic sleep disorders; and, notwithstanding the uncontrolled design, SDT appeared well-suited for various aspects of these patients' posttraumatic sleep complaints.

A primary objective of SDT aims to help trauma patients recognize that sleep problems, regardless of their psychiatric or stress components, often function as distinct disorders that respond to standard sleep medicine therapies. Standard, evidence-based sleep-oriented techniques, however, are not routinely offered to trauma survivors with sleep complaints.[28] Sleep treatments not only seem to be relatively inaccessible to trauma victims including disaster survivors, but also, sleep disturbance is rarely a primary therapeutic focus in PTSD treatment studies.[60] As an example, in our postdisaster interaction with local recovery personnel in Los Alamos, we observed that a recent U.S. Federal Emergency Management Agency (FEMA)–funded debriefing protocol (62-page training manual for mental health providers) provided limited information about or treatment recommendations for insomnia and nightmares even though these were listed as 2 common and disabling posttraumatic stress symptoms.[61] In light of controversies surrounding the treatment of acute stress disorder in general and the use of debriefing in particular, we believe that the current study highlights the potential for sleep measures and sleep therapies to serve as important assessment and intervention components in future postdisaster treatment studies of acute or chronic PTSD patients. Clinically, it is well established that standard sleep therapies, like some of those integrated into SDT, are very helpful to sleep disorders patients.[62] We predict that sleep medicine services will prove beneficial for disaster survivors, especially among those whose sleep problems appear refractory beyond the acute stress phase as occurred with this sample.

Notwithstanding the apparent, moderate success of the program, it is equally important to recognize that, on average, impaired sleep and distress symptoms persisted in many patients albeit at milder severity. This preliminary communication described the early posttreatment phase of the program. It will be informative to measure participants' subsequent follow-ups, especially among those who initiate treatment at sleep disorders centers for physiologic sleep disorders such as SDB. In another report assessing the sleep problems in these patients, 90% were presumptively diagnosed with SDB, and just over half of these potential cases were tested and screened positive for SDB on a portable sleep-breathing device.[20,63] In the current study, we were not surprised that sleep quality scores showed only modest improvement at 12-week follow-up, given the potential for persistent physiologic sleep disruption likely due to SDB. In subsequent follow-ups, we would expect greater improvement in sleep quality among those successfully treated for SDB, and we will be curious to monitor changes in distress levels that might be associated with SDB treatment.[21,29] Last, we speculate that future investigations might reap unexpected rewards by examining the combination of SDT or other sleep-oriented interventions and conventional first-line PTSD treatments such as exposure therapy or medication.[15]

The absence of a control group and several unique sample factors warrant cautious interpretation of the

findings. Foremost, without a control group, it is impossible to determine a valid therapeutic effect size because nonspecific treatment effects may have influenced outcomes. Moreover, our self-selected sample comprising many individuals who appeared to have preexisting insomnia may have compounded this problem. In contrast, it is worth reiterating that the entire sample showed relative stability of symptoms during an average 7.5-week period before treatment, and patients who either improved or worsened *before* treatment subsequently improved *during* treatment. Nonetheless, spontaneous recovery or other nonspecific influences may have confounded posttreatment and follow-up results. The steady decrease in both insomnia and posttraumatic stress symptoms during the course of therapy was encouraging as was the maintenance of change during a 6-week period after the conclusion of treatment (Figures 1 and 2); nonetheless, the specific effects of SDT may be overestimated. Moreover, prospective monitoring with diaries or actigraphy would have been a useful addition to the study, but in their absence, some improvement in sleep parameters may have occurred for patients vis-a-vis a change in attitude about sleep as opposed to an objective change. Notwithstanding, attitudinal change about sleep is a core component of insomnia treatment.[33,34] Finally, the sample comprised an atypical group of highly educated individuals, many of whom were married, working or retired, and earning more than $50,000 year. Many were researchers themselves. The average severity level of posttraumatic stress symptoms in the sample was in the high moderate range, that is, one full standard deviation *below* the level observed in studies of rape and other crime victims.[28,51] In a recent study of PTSD crime victims, sleep and distress scores were markedly worse at treatment outset and therapeutic effects were larger than in the current study.[28] Participants in the current study may have been well-suited to this program, and they suffered from less severe distress levels at pretreatment; therefore, effects or outcomes may have been amplified by the former condition and suppressed by the latter.

In summary, hypotheses generated from this study, which to our knowledge is the first to examine sleep treatments for disaster survivors, suggest that sleep medicine may have a well-defined role in the management of chronic postdisaster sleep disturbance, although our enthusiasm for this perspective must be tempered by the limitations described above. Sleep medicine is a rapidly evolving field that offers new insights and therapies for many types of psychiatric patients with sleep problems.[21,26–30,64–66] This pilot study hints at a potentially important, new paradigm to explain the relationship between sleep disturbance and posttraumatic stress. At a minimum, this new perspective merits further research exploration as well as clinical consideration for those with complex sleep disorders in the aftermath of a disaster. Speculatively, it would be informative to determine whether or not this model of sleep disorders complexity fits some proportion of survivors reporting refractory posttraumatic sleep disturbance in the aftermath of 9/11.

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Appendix 1 appears on page 684.

Appendix 1. Principles of Sleep Dynamic Therapy (SDT)

Overview
SDT is predicated on the idea that many treatment-seeking trauma survivors suffer from complex behavioral and medical sleep disorders that need to be addressed with an integrated program of standard sleep medicine therapies. Six techniques or standard educational clinical procedures form the basis of SDT, the core of which is sleep quality self-assessment.

Principle 1: Sleep Quality Self-Assessment
Sleep disorders patients tend to be unaware or uneducated about basic facts of normal human sleep or, in more severe cases, are cognitively impaired by their sleep disorder and thus are unable to connect daytime symptoms with abnormal sleep.

Sleep Quality Education
1. Shift awareness away from sleep quantity and toward sleep quality.
2. Assume that a sleep quality problem reflects both a psychological and physiologic problem with sleep (rejection of mind-body dualism).
3. Describe and define the nature of normal sleep quality and contrast it with abnormal sleep quality.
4. Connect abnormal sleep quality with its indirect or peripheral markers, including:
   a. Restless sleep/bed linens in disarray/recurrent awakenings.
   b. Morning inertia/need for alarm clocks/caffeine to start the day.
   c. Low energy/daytime fatigue and sleepiness/poor concentration.
   d. Impaired psychosocial functioning throughout waking hours.
5. Illustrate how the correction of a sleep quality problem will positively impact upon every aspect of sleep disturbance, e.g., correction of a psychological sleep problem will also benefit a physiologic sleep problem and vice versa.

Principle 2: Cognitive-Behavioral Deconditioning Theory
Sleep disorders are accompanied by variable cognitive distortions and maladaptive conditioned behaviors that sustain the sleep disorder and limit recognition of it.

Cognitive-Behavioral Education
1. Illustrate how obsessive worry about sleep quantity, time spent awake in bed, and excessive monitoring of clocks evolves from cognitive distortions about sleep.
2. Show how ruminations about time monitoring during the night create a conditioned maladaptive behavior antagonistic to normal sleep.
3. Describe how these mental or psychological cognitions and behaviors produce physiologic sleep fragmentation.

Principle 3: Cognitive-Behavioral Therapy
Sleep hygiene and associated advanced cognitive-behavioral treatments (CBT) provide a framework through which sleep disorders patients can build a new series of healthy sleep habits and behaviors.

Cognitive-Behavioral Instructions
1. Sleep hygiene consists of many straightforward habits or behaviors that can be used to promote good quality sleep and can be summarized by the dictum to "use the bed and bedroom only for sleeping and sex."
2. Anything brought into the bedroom that thwarts sleep should be removed, including any mental or physical activity, behavior, or object (e.g., worrying, talking on phone, paying bills, television).
3. Advanced CBT techniques such as stimulus control or sleep restriction therapy can be used in the context of sleep hygiene and deconditioning practices.

Principle 4: Sleep-Related Emotional Processing
Sleep-related emotional processing focuses on how various emotional states, particularly anxiety and depression, tend to worsen insomnia by promoting physical tension and ruminations.

Emotional Processing Education
1. Connect the cycle of tension and ruminations induced by anxiety and depressive symptoms with a pattern of insomnia.
2. Clarify that anxiety and depression often serve as gatekeepers preventing stronger emotions from surfacing.
3. Learn to recognize and experience the stronger feelings that fuel anxiety and depression and monitor what impact this processing has on tension and ruminations.

Principle 5: Imagery Rehearsal Therapy (IRT)
IRT is a cognitive-imagery technique that attempts to cognitively shift the nightmare sufferer toward a recognition of the possibility that disturbing dreams are learned and controllable behaviors.

IRT Education
1. Illustrate how nightmares can be both trauma-induced and habit-sustained.
2. Use IRT by selecting a nightmare and "changing it any way you wish."
3. Rehearse the "new dream" several minutes per day, working on no more than 2 new dreams per week.

Principle 6: Sleep Physiologic Self-Assessment
Many complex sleep disorders patients also appear to suffer from medical sleep disorders, such as sleep-disordered breathing, but these are not easily identifiable among individuals whose primary sleep complaint—insomnia—appears to be induced by trauma or psychological stress.

Sleep Physiology Education
1. Identify obvious and subtle markers of physiologic sleep disturbance, focusing on sleep-disordered breathing (SDB) and sleep movement disorders (SMD):
   a. SDB: monitor for snoring, hypertension, obesity, nocturia, morning headache, problems with concentration and memory, and craniofacial features of airway resistance: small jaw, crowded airway, and narrow face.
   b. SMD: monitor for uncomfortable leg sensations that prevent sleep and signs of leg movements during sleep.