Insomnia as Predictor Versus Outcome of PTSD and Depression Among Iraq Combat Veterans

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Objectives: The study conducted a longitudinal assessment of insomnia as an antecedent versus consequence of posttraumatic stress disorder (PTSD) and depression symptoms among combat veterans. *Design*: Two postdeployment time points were used in combination with structural equation modeling to examine the relative strength of two possible directions of prediction: insomnia as a predictor of psychological symptoms, and psychological symptoms as a predictor of insomnia. Participants were active duty soldiers ($N = 659$) in a brigade combat team who were assessed 4 months after their return from a 12-month deployment to Iraq, and then again eight months later. *Results*: Although both insomnia and psychological symptoms were associated at both time periods and across time periods, insomnia at 4 months postdeployment was a significant predictor of change in depression and PTSD symptoms at 12 months postdeployment, whereas depression and PTSD symptoms at 4 months postdeployment were not significant predictors of change in insomnia at 12 months postdeployment. *Conclusions*: Results support the role of insomnia in the development of additional psychological problems and highlight the clinical implications for combat veterans, to include the importance of longitudinal assessment and monitoring of sleep disturbances, and the need for early intervention. © 2011 Wiley Periodicals, Inc. J Clin Psychol 67:1240–1258, 2011.

Keywords: insomnia; posttraumatic stress disorder; longitudinal; combat exposure; postdeployment mental health

Sleep disturbances are commonly reported by soldiers returning from combat (Bliese, Wright, Adler, Hoge, & Prayner, 2005; Hoge et al., 2008; Hoge, Terhakopian, Castro, Messer, & Engel, 2007; Mellman, Kulick-Bell, Ashlock, & Nolan, 1995; Raskind et. al., 2007). The prevalence of sleep problems is not surprising given that sleep disturbance is a diagnostic criterion for posttraumatic stress disorder (PTSD) and is also co-morbid with a range of psychological problems. However, there is increasing evidence that sleep disturbance plays a key role in mental health in that insomnia often precedes the development of conditions such as depression, anxiety, and alcohol abuse (see Breslau, Roth, Rosenthal, & Andreski, 1996; Picchioni et al., 2010; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005; Weissman, Greenwald, Niño-Murcia, & Dement, 1997).

For example, in a review of clinical trials of psychotherapy or pharmacotherapy for depression, insomnia was one of the most common residual symptoms in studies on partial remission, and the most common residual symptom in studies on full remission (Tranter, O'Donovan, Chandarana, & Kennedy, 2002), suggesting that it is an independent disorder that might have precipitated the depression rather than vice versa. In addition, there is

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 © 2011 Wiley Periodicals, Inc.
Published online in Wiley Online Library (wileyonlinelibrary.com/journal/jclp). DOI: 10.1002/jclp.20845

research indicating that insomnia can predict the relapse of depression. In one study, patients diagnosed with major depressive disorder were followed an average of 22 weeks after a 4-week remission. Those patients who relapsed exhibited a progressive increase in insomnia in the weeks immediately before the relapse in comparison to matched patients who did not relapse (Perlis, Giles, Buysse, Tu, & Kupfer, 1997).

It is now well recognized that the traditional perspective—that insomnia is secondary to a so-called primary disorder—is not accurate. Instead, insomnia is regarded as a comorbid disorder that is often an important maintainer of the other comorbid disorders (Harvey, 2001; NIH, 2005). Based on evidence from well-designed, large-scale, longitudinal studies, Harvey concluded that insomnia is a risk factor for the development of psychological disorders, thus making it a "primary" diagnosis. For example, Riemann and Voderholzer's (2003) review of longitudinal, epidemiological studies found that insomnia at baseline increased risk for depression at follow-up 1 to 3 years later. In addition, sleep disturbances have been found to mediate the relationship between combat stressors and psychological symptoms in Iraq War veterans. That is, when insomnia was added to a multiple regression model, the correlation between combat stressors and other depression symptoms significantly decreased and when nightmares were added to a multiple regression model, the correlation between combat stressors and other PTSD symptoms significantly decreased (Picchioni et al., 2010). This diverse set of studies and the associated findings have led several researchers to conclude that sleep disturbances are a predisposing factor that confers risk for the development of additional psychological disorders rather than vice versa (Germain, Buysse, & Nofzinger, 2008; Harvey, 2001; Lavie, 2001; Peterson & Benca, 2006; Pigeon & Perlis, 2007).

To understand how insomnia might trigger other psychological symptoms, it should first be viewed as an immediate reaction to stress and an important mediator in the Diathesis-Stress Model (Levi, 1974). In one seminal study by Haynes and colleagues (1981), a manipulation of presleep stressors was used to test the effects of laboratory stress on sleep onset latency as measured by polysomnography. The results in the insomnia patients were not significant; there was no change in sleep onset latency on nights with a presleep stressor compared with nights without a presleep stressor. The authors concluded that the sleep onset latency of these patients was already very high and the negative finding represented a ceiling effect in this population.

The question then becomes: How does the lack of restorative sleep associated with insomnia trigger other psychological symptoms? An answer comes from the theory that one of the functions of sleep is emotional regulation (Stickgold, 2002; Walker, 2009). Although the results of several preliminary studies support such a function of sleep, only recently have researchers begun to quantify such functions as sleep-dependent changes in emotional memories. For example, the presence of sleep leads to better memory for the emotionally salient objects in complex visual scenes (Payne, Stickgold, Swanberg, & Kensinger, 2008). The study employed a paradigm where participants were randomized to a sleep or wake condition. Participants in the sleep condition were given the test in the evening and again 12 hours later in the morning. Participants in the wake condition were given the test in the morning and again 12 hours later in the evening. This allowed the investigators to demonstrate increased recall of emotionally charged over emotionally neutral memories during a 12-hour delay with sleep versus a 12-hour delay with continuous wakefulness. Therefore, the lack of sleep in insomnia patients could prevent normal, sleep-dependent emotional memory processing and any associated emotional regulation benefits. Without such benefits, it could be hypothesized that insomnia patients would be at greater risk for the development of mood and anxiety disorders.

Even with increasing attention to alternative perspectives concerning sleep disturbance, a recent review of the literature concluded that insomnia is under-recognized and inadequately treated despite high prevalence rates and adverse personal consequences (Benca, 2005). In addition, the National Institutes of Health (NIH) Consensus Report on Insomnia (2005) stated that little is known about both the causes and consequences of insomnia and the clinical course and comorbidities. The report noted that there are few longitudinal studies to address these questions or to recommend effective treatment for the disorder. Therefore, although

most cases of insomnia are comorbid with other conditions, no firm conclusions can be drawn about the direction of causality. Thus, the report recommends longitudinal studies as a research priority to document the circumstances under which insomnia first appears in order to begin to identify the causal pathways.

The current study adds to the research in this area in two ways. First, the study conducts a longitudinal assessment of insomnia as an antecedent versus consequence of two different mental health outcomes: PTSD and depression; second, the study specifically examines insomnia as an antecedent versus consequences of psychological health among combat veterans. Typically, longitudinal studies that identify insomnia as a predictor of psychological disorders have been conducted with community-based samples and clinical populations of patients with mood disorders. We are not aware of research that has examined whether insomnia is prospectively related to psychological problems frequently reported by combat veterans returning from Iraq and Afghanistan (Bliese, Wright, Adler, Thomas, & Hoge, 2007; Hoge et al. 2004). Combat veteran populations are particularly relevant to studies of the prospective relationships between insomnia and mental health problems given consistent findings that sleep disturbances can occur as a result of trauma-related nightmares (Dow, Kelsoe, & Gillin, 1996; Inman, Silver, & Doghramji, 1990; Lavie, Katz, Pillar, & Zinger, 1998; Neylan et al., 1998). Under high levels of combat intensity, numerous veterans would be expected to experience insomnia, emphasizing the importance of understanding the role of sleep disturbance in the development of mental health problems for this population.

Thus the present research examines the relationship between insomnia and psychological symptoms among combat veterans of the Iraq war using a cross-lagged longitudinal design to test a model of whether insomnia is a stronger predictor of insomnia than the reverse causal direction. Two postdeployment time points were used in combination with structural equation modeling to assess the relative strength of two possible directions of prediction: insomnia as a predictor of psychological symptoms and psychological symptoms as a predictor of insomnia. Based on the existing research, it was hypothesized that there would be stronger evidence for a link between insomnia at the first time point and psychological problems at the second time point than the reverse.

METHOD

Participants and Procedure

Participants were active duty soldiers ($N = 659$) in a brigade combat team who had completed a 12-month deployment to Iraq in 2005. Inclusion criteria were that the soldier had deployed with the brigade and was at least 18 years old. There were no exclusion criteria. Participants were 96% male and 4% female, with an average age of 25 years. In terms of rank, the sample was 64% Junior Enlisted (Private to Specialist), 31% Non-Commissioned Officers (Sergeant to Sergeant Major), and 5% Officers. In terms of ethnicity, the sample was 67% White, 10% African American, 13% Hispanic American, 7% Asian/Pacific Islander, and 3% "Other." Similarly to Kim, Thomas, Wilk, Castro, and Hoge (2010), the present samples had a slightly larger percentage of junior enlisted and females, and a slightly lower percentage of officers, than the population of deployed personnel.

Although the study did not include a comprehensive assessment of combat exposure, items were included to assess the number of times soldiers had been in danger of being killed or injured, handled dead bodies, and engaged in actions that led to the death of another. The responses to these items indicate that our sample was frequently exposed to traumatic events. Using "on occasion (2–5 times)" as a minimum response reflecting exposure, 89% of the sample was in danger of being killed, 90% of the sample was in danger of being injured, 61% saw or handled dead bodies, and 30% were involved in an action or inaction that resulted in someone being seriously injured or killed.

For the present research, soldiers were assessed approximately 4 months after their return from a 12-month deployment to Iraq, and then again approximately 8 months later. The survey scales reported here were sections of a larger survey on postdeployment transition

training (Adler, Castro, Bliese, McGurk, & Milliken, 2007). Surveys were administered to units and small groups of participants who were present for duty on that day and available. Participants were surveyed only if they stayed with their unit and there was no follow-up procedure if they were not available during the group survey administration (for example, they might have moved, been at a military school, or not been available for some other reason). Soldiers were linked by personal identifier and then assigned a random identification number to connect their responses to the two assessments. Confidentiality was ensured using individual envelopes. Participants were provided a survey in an unmarked envelope, and they then returned their survey in an unmarked envelope. Standard data collection procedures approved by the institutional review board ensured that individual identifying information was kept separate from the data and an arbitrary code was used instead. Participants were told that their data would be used for research purposes and would not be reported individually. The consent process included a discussion of the standard limit to confidentiality if an individual stated the intent to hurt themselves or someone else. Participants were not compensated for their participation. The study was conducted under a human use protocol approved by the Institutional Review Board of the Walter Reed Army Institute of Research. Study participants gave written informed consent prior to enrollment.

A total of 1,955 soldiers completed the assessment at 4 months postdeployment, and 659 of these soldiers completed both assessments for a response rate of 34%. This response rate is reasonable given the time lag of 8 months between the two assessments. During this time period, many of the soldiers in the original assessment would have moved on to a different duty station, transitioned out of the Army, or have been away on various training exercises. Demographic comparisons for those soldiers who completed both assessments versus those who completed only the Time 1 assessment revealed no rank or gender differences. The sample completing both assessments had slightly fewer African American and Hispanic American veterans than the sample who completed only the Time 1 assessment. An unexpected difference was that those soldiers who completed both assessments scored lower than those who failed to complete the second assessment on all of the Time 1 outcomes: insomnia, depression symptoms, aggressive behavior, and PTSD symptoms (p's $<.01$). This suggests that individuals with high rates of mental health problems at Time 1 are more likely to have left the unit by Time 2 (see also Stretch, 1986). Below we discuss analyses of the hypotheses with the intact longitudinal sample and a sample where missing data were imputed using the expectation maximization (EM) algorithm in EQS 6.1 for Windows.

Measures

Insomnia. Insomnia was measured using four items adapted from the seven-item Insomnia Severity Index (Morin, 1993; Morin & Espie, 2003; Ouellet, Beaulieu-Bonneau, & Morin, 2006). The measure was modified based on large-scale psychological screening validation studies conducted with soldiers returning from combat. This validation work is reported elsewhere (Bliese et al., 2005). The index is focused on quantitatively assessing insomnia and there is evidence indicating that the measure is reliable, valid, and sensitive to interventions designed to improve sleep (Ouellett et al., 2006). Previous research also has demonstrated the validity of this scale with sleep diary assessments of insomnia symptoms (Bastien, Vallières, & Morin, 2001). Participants indicated the severity of sleep problems during the past 2 weeks, such as "difficulty falling asleep" and "difficulty staying asleep," on a 5-point scale ranging from (*none*) to (*very severe*), and also reported on satisfaction with their current sleep pattern, on a 5-point scale ranging from (*very satisfied*) to (*very dissatisfied*), and the extent to which the sleep pattern interfered with daily functioning, on a 5-point scale ranging from (*not at all/no sleep problem*) to (*very much*). The total insomnia severity score is created by taking the mean of the five items. Internal consistency as assessed by Cronbach's alpha was .91 for the Time 1 assessment and .90 for the Time 2 assessment.

Depression. Depression was assessed using an 8-item subscale from the Patient Health Questionnaire (PHQ; Spitzer, Kroenke, & Williams, 1999; the item assessing suicidal ideation

was not included). Participants responded to the items on a 4-point scale, indicating the number of days they had experienced various symptoms (e.g. "Feeling down, depressed, or hopeless," "Little interest or pleasure in doing things"). The item in the subscale referring to problems sleeping was omitted to minimize similarity in content with our primary insomnia measure, leaving seven items used in the initial measurement model. The depression subscale of the PHQ is a frequently used and previously validated instrument for assessing depression and has been included as an assessment measure in population-based studies of soldiers postcombat (Hoge et al. 2004). Cronbach's alpha for the depression subscale in the present study was .89 for the Time 1 and .90 for Time 2.

Posttraumatic Stress Disorder (PTSD). PTSD symptoms were assessed using the Posttraumatic Stress Disorder Checklist (PCL; Bliese et al., 2008; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Weathers, Litz, Herman, Huska, & Keane, 1993). This well-validated measure includes 17 items that correspond to the diagnostic criteria for PTSD in the *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revision* (DSM-IV-TR; American Psychiatric Association, 2000). The PCL items comprise three subscales that correspond to PTSD diagnostic criteria: intrusion (five items), avoidance (seven items), and arousal (four items). The items are responded to on a 5-point Likert-type scale ranging from (*not at all*) to (*extremely*). Sample items include "Suddenly acting or feeling as if the stressful experience on the deployment were happening again (as if you were reliving it)" and "Feeling irritable or having angry outbursts." Cronbach's alpha for the PCL in the present study was .94 for the Time 1 assessment and .95 for the Time 2 assessment.

Given our interest in examining the relationship between insomnia and PTSD symptoms, two items referring to problems sleeping and having disturbing dreams were omitted to minimize similarity in content with our primary insomnia measure, leaving 15 items in the PCL that were included in the initial measurement model.

RESULTS

Overall Descriptive Statistics for Variables at Time 1 and Time 2

Table 1 provides the means, standard deviations, and alphas for the measured variables at Time 1 and Time 2. The descriptive statistics reveal relatively low levels of insomnia and symptoms at each of the time periods, and a comparable magnitude of problems across the two time periods. The measures of PTSD and depression symptoms are not directly comparable to other military samples given that we eliminated the sleep items from the two scales. However, the PCL, which is more often examined as a continuous variable in research with combat soldiers, revealed comparable scores (Bliese et al., 2008). We are not aware of prior research using the insomnia severity measure with combat veterans.

Table 1
Means, Standard Deviations, and Internal Consistency Estimates for the Observed Variables at Time 1 and Time 2

Variable	Mean	SD	Alpha
Insomnia (Time 1)	2.245	1.01	.91
PTSD symptoms (Time 1)	27.95	11.70	.92
Depression symptoms (Time 1)	1.48	.55	.88
Insomnia (Time 2)	2.254	1.01	.90
PTSD symptoms (Time 2)	28.68	12.87	.94
Depression symptoms (Time 2)	1.48	.57	.89

Note: SD = standard deviation; PTSD = posttraumatic stress disorder. *N*'s range from 640 to 659.

Development of the Measurement Model

We first developed a measurement model using EQS 6.1 for Windows for insomnia, depression symptoms, and PTSD symptoms for the 659 veterans who responded at Time 1 and Time 2. A small number of missing data points (89% of the participants were not missing any data, and only 1.3% had 4 or more missing values) were imputed values using the EM algorithm. The measurement model that was initially examined is presented in Figure 1. The measurement model initially included 15 items representing the three subscales of PTSD (four items for intrusion, seven items for avoidance, and four items for arousal), seven items for depression, and four items for insomnia, at both Time 1 and Time 2. All factor loadings and error terms were freely estimated and factor variances were fixed to 1. Factor covariances were freely estimated. Disturbance terms for the items assessing the constructs at the two different points in time were allowed to covary.

Results from the initial measurement model suggested a number of changes to improve the fit of the model. First, the multivariate kurtosis of the model was relatively high. An inspection of the univariate statistics for the individual items indicated that two of the PTSD items ("Avoiding activities or situations because they reminded you of the stressful experience," "Trouble remembering important parts of the stressful experience") and three of the depression items ("Feeling bad about yourself, or that you are a failure and have let yourself or your family down," "Trouble concentrating on things, such as reading the newspaper or watching television," "Moving or speaking so slowly that other people could have noticed") had high levels of kurtosis (kurtosis values > 3.0) at both time periods. The mean for these items was near the lowest value of the scale, indicating relatively few people provided a value other than the lowest value. Given each factor still had four or more indicators remaining (with all indicators indexing a single latent variable), we eliminated the items with high kurtosis from the model.

In addition, the EQS program provided the five cases making the highest contribution to the multivariate kurtosis. These five cases each had a normalized Mardia's coefficient of over 2,765.01 and showed little variability in responses. Therefore, these cases were eliminated. This

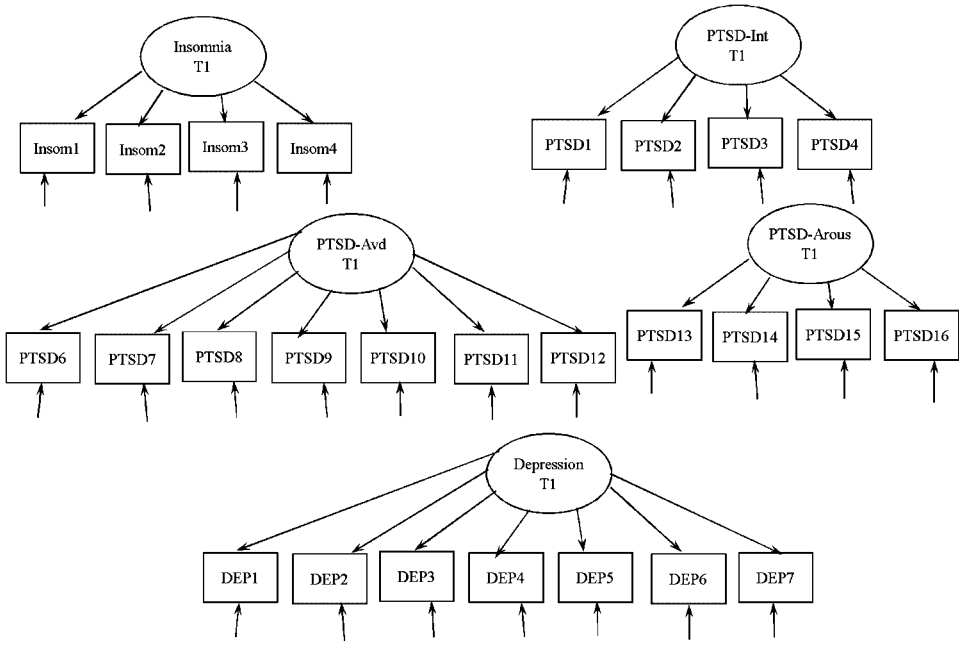

Figure 1. The measurement model for the present study is depicted. *Note*: The same latent variables at Time 2 are omitted for clarity. All latent variables were permitted to covary with one another at Time 1 and Time 2. Covariances between the same indicators at Time 1 and Time 2 were also allowed.

reduced the multivariate kurtosis, but it was still relatively high. Therefore, robust goodness of fit indexes and standard errors (Satorra & Bentler, 1994) were used when evaluating the fit of the model and when testing the significance of individual paths in the structural equations. These robust estimates have shown to be among the more reliable estimates for non-normal data under various sample sizes (Hu, Bentler, & Kano, 1992; Curran, West, & Finch, 1996).

Finally, the Lagrange Multiplier (LM) test for the initial measurement model and item-level statistics suggested the inclusion of four error covariances at Time 1 and four error covariances at Time 2, all among items already loading on the same underlying factor. Given the theoretical similarity in item content and wording between the items under consideration, the error covariances were included in the model (see MacCallum, Roznowski, & Necowitz, 1992). Although the χ^2 for the model was significant given the large sample size, Satorra-Bentler (SB) χ^2 (743) = 1433.73, $p < .05$, other fit indexes for the model ranged between acceptable and good, confirmatory factor analysis (CFI) = .95, non-normed fit index = .94, root mean square error of approximation (RMSEA) = .038 (90% confidence interval [CI], .035–.041; Kline, 2005). The factor loadings for the final measurement model are presented in Table 2 and were significant and moderate to large. A summary of the fit indices for the different models examined in the present study is provided in Table 3.

In addition, we conducted a test of the equality of factor loadings for the different latent variables across the two time periods to establish metric invariance. Constraining the loadings of items to be equal from Time 1 to Time 2 did not reduce the fit of the model, SB scaled χ^2 difference (21) = 29.51, $p = .10$, indicating the loadings on the five factors were invariant from Time 1 to Time 2. The means, standard deviations, and correlations among all the Time 1 and Time 2 indicators in the final measurement model are provided in Table 4.

Correlations Among the Latent Variables at Time 1 and 2

The correlations among the latent variables are provided in Table 5. Insomnia was correlated with depression symptoms and the PTSD subscales at both time periods. Insomnia at Time 1 was related to depression symptoms and the PTSD subscale symptoms at Time 2, and the mental health symptoms at Time 1 were also related to insomnia at Time 2. The relationships between insomnia and the different PTSD subscales were similar. Results revealed moderate correlations between insomnia at Time 1 and Time 2, as well as for the mental health outcomes at Time 1 and Time 2. Supporting the creation of a higher order PTSD symptoms factor for the structural model, the correlations among the three PTSD subscales were very high ($>.79$) at each time period.

Structural Model Including Insomnia, Depression Symptoms, and Higher Order PTSD Symptoms

We now examine our primary structural model, which involved the relationships between insomnia, depression symptoms, and PTSD symptoms over time. Equations were first constructed to create a higher order PTSD factor at Time 1 and Time 2. At each time period the higher order factor was indexed by the three factors representing the three PTSD subscales. As would be expected from the correlations among the latent variables, each of the three factors loaded highly on the higher order factor at each time period.

The structural model was then created through three equations with outcomes of insomnia at Time 2, higher order PTSD symptoms at Time 2, and depression symptoms at Time 2. In each equation, the predictors included insomnia at Time 1, higher order PTSD symptoms at Time 1, and depression symptoms at Time 1. This approach to creating the structural model insured that a given predictor's relationship with an outcome at Time 2 was tested controlling for the other predictors in the model. To model nonstructural relationships, covariances were estimated for the variables not included in the structural model, and error covariances were estimated for the dependent variables in the model. The χ^2 was again significant, SB χ^2 (764) = 1560.65, $p < .05$, but other fit indexes indicated acceptable to good fit of the model, CFI = .94, non-normed fit index = .93, RMSEA = .04 (90% CI, .037–.043).

Table 2
Confirmatory Factor Analysis for the Final Measurement Model of Insomnia, PTSD Symptoms, and Depression Symptoms at Time 1 and Time 2

Indicator	Insom	PTSD-Int	PTSD-Avd	PTSD-Ar	Depression
Time 1 Loadings					
Latent Factor					
Insom1 (T1)	.84*				
Insom2 (T1)	.84*				
Insom3 (T1)	.86*				
Insom4 (T1)	.83*				
PTSD1 (T1)		.77*			
PTSD3 (T1)		.76*			
PTSD4 (T1)		.83*			
PTSD5 (T1)		.79*			
PTSD6 (T1)			.57*		
PTSD9 (T1)			.75*		
PTSD10 (T1)			.78*		
PTSD11 (T1)			.71*		
PTSD12 (T1)			.71*		
PTSD14 (T1)				.79*	
PTSD15 (T1)				.82*	
PTSD16 (T1)				.65*	
PTSD17 (T1)				.70*	
Dep1 (T1)					.81*
Dep2 (T1)					.76*
Dep3 (T1)					.77*
Dep4 (T1)					.74*
Time 2 Loadings					
Insom1 (T2)	.83*				
Insom2 (T2)	.82*				
Insom3 (T2)	.82*				
Insom4 (T2)	.84*				
PTSD1 (T2)		.81*			
PTSD3 (T2)		.78*			
PTSD4 (T2)		.85*			
PTSD5 (T2)		.81*			
PTSD6 (T2)			.69*		
PTSD9 (T2)			.74*		
PTSD10 (T2)			.79*		
PTSD11 (T2)			.73*		
PTSD12 (T2)			.72*		
PTSD14 (T2)				.80*	
PTSD15 (T2)				.87*	
PTSD16 (T2)				.67*	
PTSD17 (T2)				.75*	
Dep1 (T2)					.81*
Dep2 (T2)					.80*
Dep3 (T2)					.80*
Dep4 (T2)					.76*

Note: PTSD = posttraumatic stress disorder; insom = insomnia; int = intrusion; avd = avoidance; ar = arousal.
*$p < .05$.

The structural model is depicted in Figure 2 and provides support for insomnia at Time 1 being related to a change in PTSD and depression symptoms at Time 2. Insomnia at Time 1 was a predictor of PTSD symptoms and depression symptoms at Time 2 even when

Table 3
Summary of Fit Indexes for the Different Models Examined in the Present Study

Model	SB χ^2	CFI	NNFI	RMSEA (90% CI)
Measurement	1433.73*	.95	.94	.038 (.035 to.041)
Structural higher order PTSD	1560.65*	.94	.93	.04 (.037 to.043)
Structural higher order PTSD (Imputed)	2413.56*	.94	.93	.03 (.032 to.035)
Structural-3 PTSD subscales	1443.18*	.95	.95	.038 (.035 to.041)

Note: SB = Satorra-Bentler; CFI = confirmatory factor analysis; NNFI = non-normed fit index; RMSEA = root mean square error of approximation; CI = confidence interval. PTSD = posttraumatic stress disorder.
*$p < .05$.

controlling for the Time 1 mental health outcome. A one standard deviation increase in insomnia at Time 1 was associated with a .14 and .16 of a standard deviation increase in PTSD and depression, respectively, at Time 2. Although this amount was small, it is important to remember that the variance accounted for was calculated after controlling for the outcome measure at Time 1, and there were strong Time 1–Time 2 relationships for the outcome measures. In contrast, neither PTSD nor depression symptoms were related to insomnia at Time 2 after controlling for insomnia at Time 1.

In addition to running the above model with the sample of 659 veterans who completed surveys at both time points, we ran an additional model where the complete Time 1 sample was included ($N = 1{,}955$), and the EM method of imputation was used to impute the missing values of participants who failed to respond to the Time 2 assessment. Importantly, the fit of the model with the imputed data was comparable to the model based on those who provided data at both time points, SB χ^2 (764) = 2413.56, $p < .05$, CFI = .94, non-normed fit index = .93, RMSEA = .03 (90% CI, .032–.035). Furthermore, the significance of the coefficients identified in Figure 2 was replicated with the imputed data, with insomnia at Time 1 being significantly predictive of the higher order PTSD and depression symptoms at Time 2, whereas the higher order PTSD and depression symptoms at Time 1 were not significant predictors of insomnia at Time 2 (specific coefficients for the model are available from the second author).

However, the kurtosis values for the variables with large amounts of imputed data (i.e. the Time 2 variables) were very high (kurtosis values all greater than 3.0 and some as high as 11.0). Prior researchers have cautioned against imputing data when missing values are associated with non-normality (Shin, Davison, & Long, 2009). Therefore, given the large sample of respondents who completed both observations, the fact that missing data were expected given the study design, and the large percent of imputed values required based on the Time 1 survey, we highlight the results obtained with our completer-only sample.

Structural Model Including Insomnia, Depression Symptoms, and the Three PTSD Subscales

Recent research has emphasized the relationships among different PTSD subscales over time, with the arousal subscale being an important predictor of scores on other subscales (Schell, Marshall, & Jaycox, 2004). Even though the correlations between the latent variables of insomnia and the different PTSD subscales were similar to one another, we still wanted to examine a structural model including the separate subscales as predictors and outcomes. However, given the strong intercorrelations among the three subscales, we created structural equations where each subscale was an outcome and the corresponding Time 1 subscale was included as a predictor, along with insomnia and depression at Time 1. These three equations were added to the model with the equations where insomnia and depression symptoms at Time 2 were outcomes. The only significant path involving insomnia at Time 1 as a predictor of the three different PTSD subscales was insomnia predicting the intrusion subscale at Time 2 (standardized loading = .13, unstandardized loading = .11, corrected standard error = .053). In contrast, Time 1 intrusion did not predict Time 2 insomnia (insomnia at Time 1 continued

Table 4
Means, Standard Deviations, and Correlations Among the Indicator Variables at Time 1 and Time 2

Indicator	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1. STRS01T3	1.87	.98	–																					
2. STRS03T3	1.38	.72	.58	–																				
3. STRS04T3	1.77	.98	.63	.63	–																			
4. STRS05T3	1.55	.92	.60	.60	.65	–																		
5. STRS06T3	1.83	1.10	.33	.33	.37	.34	–																	
6. STRS09T3	1.74	1.08	.45	.44	.49	.46	.42	–																
7. STRS10T3	1.85	1.10	.46	.46	.50	.47	.37	.67	–															
8. STRS11T3	1.74	1.09	.42	.42	.46	.43	.40	.53	.65	–														
9. STRS12T3	1.67	1.09	.42	.42	.46	.43	.40	.53	.55	.50	–													
10. STRS14T3	2.13	1.20	.49	.49	.54	.51	.39	.52	.54	.50	.50	–												
11. STRS15T3	1.90	1.12	.51	.51	.56	.53	.41	.55	.57	.52	.52	.64	–											
12. STRS16T3	2.11	1.13	.40	.40	.44	.41	.32	.43	.45	.41	.41	.51	.53	–										
13. STRS17T3	1.91	1.09	.43	.43	.47	.45	.35	.46	.48	.44	.44	.54	.57	.60	–									
14. MOOD02T3	1.60	.78	.41	.41	.45	.42	.41	.55	.57	.52	.52	.54	.57	.44	.48	–								
15. MOOD03T3	1.56	.81	.39	.39	.43	.40	.39	.52	.54	.49	.50	.51	.54	.42	.45	.61	–							
16. MOOD05T3	1.93	.92	.39	.39	.43	.40	.39	.52	.54	.50	.50	.52	.54	.42	.45	.61	.58	–						
17. MOOD06T3	1.70	1.17	.38	.38	.42	.39	.38	.51	.53	.48	.48	.50	.52	.41	.44	.59	.56	.57	–					
18. SLEEP6T3	2.03	1.21	.43	.42	.46	.44	.32	.43	.44	.41	.41	.50	.52	.41	.44	.50	.47	.47	.46	–				
19. SLEEP7T3	2.06	1.11	.42	.42	.46	.43	.32	.42	.44	.40	.40	.50	.52	.41	.44	.49	.47	.47	.46	.70	–			
20. SLEEP4T3	2.91	1.04	.44	.43	.48	.45	.33	.44	.45	.42	.42	.51	.53	.42	.45	.51	.48	.49	.47	.72	.72	–		
21. SLEEP5T3	1.96	1.04	.42	.42	.46	.43	.32	.42	.44	.40	.40	.40	.49	.52	.41	.44	.49	.47	.45	.70	.69	.71	–	
22. STRS01T5	1.84	1.01	.49	.39	.42	.40	.23	.31	.32	.29	.29	.38	.39	.31	.33	.31	.30	.47	.45	.70	.69	.71	.34	–
23. STRS03T5	1.45	.83	.37	.44	.41	.38	.22	.30	.31	.28	.28	.36	.38	.30	.32	.30	.28	.38	.28	.33	.33	.34	.32	.63
24. STRS04T5	1.79	1.05	.40	.40	.50	.41	.24	.32	.33	.30	.30	.39	.41	.32	.35	.33	.31	.31	.30	.36	.35	.36	.34	.68
25. STRS05T5	1.63	.98	.39	.39	.42	.46	.23	.31	.32	.29	.29	.38	.40	.31	.33	.31	.30	.30	.29	.34	.34	.35	.34	.66
26. STRS06T5	1.81	1.13	.25	.25	.27	.25	.41	.33	.34	.32	.32	.31	.32	.25	.27	.33	.32	.32	.31	.28	.28	.29	.28	.44
27. STRS09T5	1.75	1.10	.26	.26	.29	.27	.27	.46	.37	.34	.34	.33	.35	.27	.29	.36	.34	.34	.33	.30	.30	.31	.30	.48
28. STRS10T5	1.84	1.12	.28	.28	.31	.29	.29	.38	.44	.36	.37	.36	.38	.29	.32	.39	.36	.37	.35	.33	.32	.33	.32	.51
29. STRS11T5	1.75	1.13	.26	.26	.29	.27	.27	.35	.37	.45	.34	.33	.34	.27	.29	.35	.33	.34	.33	.30	.30	.31	.30	.47
30. STRS12T5	1.87	1.22	.26	.26	.28	.26	.26	.35	.36	.33	.48	.32	.34	.26	.28	.35	.33	.33	.32	.29	.29	.30	.29	.46
31. STRS14T5	2.27	1.28	.32	.32	.35	.33	.24	.33	.34	.31	.31	.52	.41	.32	.35	.35	.33	.33	.32	.36	.35	.36	.35	.53

TABLE 4. Continued

Indicator	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
32. STRS15T5	1.99	1.21	.35	.35	.38	.36	.27	.35	.37	.34	.34	.43	.57	.35	.38	.38	.36	.36	.35	.39	.38	.40	.38	.57
33. STRS16T5	2.05	1.18	.27	.27	.29	.28	.20	.27	.28	.26	.26	.33	.34	.45	.29	.29	.28	.28	.27	.30	.30	.30	.29	.44
34. STRS17T5	1.88	1.11	.30	.30	.33	.31	.23	.31	.32	.29	.29	.37	.39	.30	.50	.33	.31	.31	.30	.33	.33	.34	.33	.49
35. MOOD02T5	1.56	.82	.27	.27	.29	.27	.26	.34	.35	.32	.32	.35	.36	.28	.31	.42	.35	.35	.34	.35	.35	.36	.35	.42
36. MOOD03T5	1.56	.84	.26	.26	.29	.27	.25	.34	.35	.32	.32	.34	.36	.28	.30	.37	.44	.35	.34	.35	.34	.35	.34	.42
37. MOOD05T5	1.94	.95	.26	.26	.29	.27	.25	.34	.35	.32	.32	.34	.36	.28	.30	.37	.35	.44	.34	.35	.34	.36	.34	.42
38. MOOD06T5	1.65	.88	.25	.25	.27	.26	.24	.32	.33	.30	.30	.32	.34	.27	.29	.35	.33	.33	.42	.33	.33	.34	.32	.40
39. SLEEP06T5	2.03	1.17	.26	.26	.29	.27	.20	.27	.28	.26	.26	.32	.33	.26	.28	.31	.30	.30	.29	.54	.43	.44	.43	.42
40. SLEEP07T5	2.00	1.17	.26	.26	.28	.27	.20	.27	.27	.25	.25	.31	.33	.26	.28	.31	.29	.29	.29	.43	.47	.44	.42	.41
41. SLEEP04T5	2.97	1.15	.26	.26	.28	.27	.20	.26	.27	.25	.25	.31	.33	.26	.28	.31	.29	.29	.28	.43	.42	.54	.42	.41
42. SLEEP5T5	1.99	1.10	.27	.27	.29	.27	.20	.27	.28	.26	.26	.32	.34	.26	.28	.32	.30	.30	.29	.44	.43	.45	.50	.42

Interfactor corr.	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
23. STRS03T5	–																			
24. STRS04T5	.65	–																		
25. STRS05T5	.63	.68	–																	
26. STRS06T5	.42	.46	.44	–																
27. STRS09T5	.46	.49	.48	.50	–															
28. STRS10T5	.49	.53	.51	.53	.70	–														
29. STRS11T5	.45	.49	.47	.50	.63	.71	–													
30. STRS12T5	.44	.48	.46	.49	.52	.56	.52	–												
31. STRS14T5	.50	.55	.53	.47	.51	.55	.50	.49	–											
32. STRS15T5	.55	.59	.57	.52	.55	.60	.55	.54	.69	–										
33. STRS16T5	.42	.46	.44	.40	.43	.46	.42	.41	.53	.58	–									
34. STRS17T5	.47	.51	.50	.45	.48	.52	.47	.46	.60	.65	.67	–								
35. MOOD02T5	.41	.44	.43	.50	.66	.58	.53	.52	.53	.58	.44	.50	–							
36. MOOD03T5	.40	.44	.42	.49	.53	.57	.52	.51	.53	.57	.44	.49	.65	–						
37. MOOD05T5	.40	.44	.42	.50	.53	.57	.53	.52	.53	.57	.44	.49	.65	.64	–					
38. MOOD06T5	.38	.41	.40	.47	.50	.54	.50	.49	.50	.54	.42	.47	.61	.60	.61	–				
39. SLEEP06T5	.40	.44	.42	.35	.38	.41	.37	.37	.47	.51	.39	.44	.48	.48	.48	.45	–			
40. SLEEP07T5	.40	.43	.42	.35	.37	.40	.37	.36	.46	.50	.39	.44	.47	.47	.47	.44	.68	–		
41. SLEEP04T5	.40	.43	.41	.35	.37	.40	.37	.36	.46	.50	.39	.44	.47	.47	.47	.44	.68	.67	–	

Table 5
Correlations Among the Latent Variables of Insomnia, Depression Symptoms, and PTSD Subscale Symptoms at Time 1 and 2

Interfactor corr.	1	2	3	4	5	6	7	8	9	10
1. Insomnia (T1)		–								
2. PTSD-In (T1)	.67*	–								
3. PTSD-Av (T1)	.69*	.79*	–							
4. PTSD-Ar (T1)	.76*	.82*	.90*	–						
5. Depress (T1)	.68*	.68*	.92*	.86*	–					
6. Insomnia (T2)	.62*	.42*	.44*	.49*	.47*	–				
7. PTSD-In (T2)	.51*	.63*	.51*	.60*	.48*	.63*	–			
8. PTSD-Av (T2)	.50*	.48*	.66*	.58*	.61*	.62*	.80*	–		
9.PTSD-Ar (T2)	.53*	.53*	.55*	.63*	.55*	.71*	.82*	.87*	–	
10.Depress (T2)	.52*	.44*	.57*	.55*	.58*	.72*	.65*	.91*	.82*	–

Note: PTSD = posttraumatic stress disorder; insom = insomnia; int = intrusion; avd = avoidance; ar = arousal.
*$p < .05$.

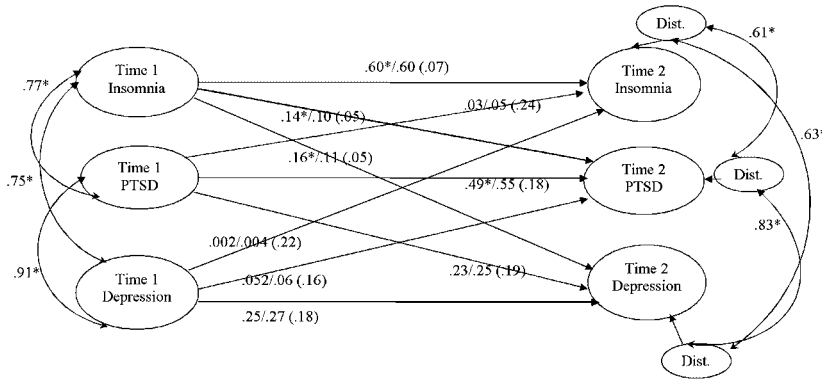

Figure 2. The structural equation model features insomnia and PTSD and depression symptoms at Time 1 as predictors of insomnia and PTSD and depression symptoms at Time 2. *Note*: Standardized loadings are presented on the left of the /, with unstandardized coefficients and robust standard errors appearing on the right of the /. Observed scales and their loadings on reflective factors as well as covariances and disturbances involving predictors were omitted for clarity. *$p < .05$.

to be a significant predictor of Time 2 depression in the model, whereas depression at Time 1 again failed to predict insomnia at Time 2).

DISCUSSION

The results supported the hypothesis that insomnia at an earlier time point is a stronger predictor of psychological symptoms at a later time point than the reverse. Insomnia at 4 months postdeployment was a significant predictor of depression and PTSD symptoms at 12 months postdeployment. In contrast, depression and PTSD symptoms at 4 months postdeployment were not significant predictors of insomnia at 12 months postdeployment in the cross-lagged analyses. We also found that this same pattern of results was obtained even when using the complete Time 1 sample and imputing missing values for those who were not followed up at Time 2. The ability to specifically address alternative predictive relationships represents a major advantage of the cross-lagged panel longitudinal design utilized in this study.

When the PTSD subscales were treated as separate constructs, as opposed to a higher order latent variable, insomnia severity at Time 1 predicted only a change in the intrusion subscale from Time 1 to Time 2, whereas the intrusion subscale did not predict a change in insomnia severity from Time 1 to Time 2. These results are inconsistent with the argument that insomnia is an indicator of hyperarousal, and for that reason, insomnia at Time 1 would be related to PTSD symptoms (and possibly depression symptoms) at Time 2 (Schell et al., 2004). For this sample of combat veterans, insomnia severity was more predictive of intrusive memories of traumatic events encountered during the operation. Note this relationship was obtained even after omitting the item on the PTSD scale assessing the prevalence of nightmares, suggesting that insomnia is related to intrusive memories in general, rather than only during sleep.

Theoretical Implications

The long-term effects of insomnia might provide a mechanism that explains the results of the present study. Presumably, the lack of sleep in those with insomnia prevents normal, sleep-dependent emotional memory processing and puts individuals at risk for the development of mood and anxiety disorders. This theoretical perspective has generally taken two forms, which are not mutually exclusive. In the first form, Stickgold argues that sleep performs a function similar to cognitive restructuring therapy (Stickgold, 2002). That is, sleep represents a unique brain state that facilitates memory integration or association. During sleep, emotional memories in confined neuronal networks become associated with other memories in larger neuronal networks, thus providing the cognitive perspective necessary for successful emotional adaptation. In the second form of the theory, Walker argues that sleep performs a function similar to systematic desensitization therapy (Walker, 2009). That is, sleep normally serves to strip the emotional component from memories. A failure or an absence of this process could then trigger psychological symptoms such as the reexperiencing symptoms of PTSD. Evidence that supports these theories comes from a study demonstrating that sleep is necessary for the generalization of extinction after aversive conditioning. Pace-Schott et al. (2009) used a mild electric stimulus as the unconditioned stimulus and skin conductance as the response, with participants randomized to a sleep or wake condition. Generalization of fear extinction from an extinguished conditioned stimulus to a nonextinguished conditioned stimulus depended on the presence of intervening sleep.

In addition, short-term diurnal effects of the sleep deprivation associated with insomnia might also trigger other psychological symptoms. Sleep deprivation often produces perceptual psychological symptoms such as hallucinations and dissociations. For example, hallucinations have been measured using self-reported and experimenter-observed phenomena during 72 hours of sleep deprivation (Babkoff, Sing, Thorne, Genser, & Hegge, 1989). In this study, there was a linear increase in hallucinations across the sleep deprivation period and, after removing the linear component from the data, there was a clear circadian rhythmicity in the percentage of individuals experiencing hallucinations at each time point. Moreover, dissociations have been measured during 24 hours of sleep deprivation (Giesbrecht, Smeets, Leppink, Jelicic, & Merckelbach, 2007) and increased during the early morning of the sleep deprivation period compared to baseline assessments during the normal waking portion of the day. These studies suggest a link to explain the relationship between insomnia and perceptual psychological symptoms that occur in disorders such as PTSD.

The experimental observation of hallucinations and dissociations during sleep deprivation as well as the phenomenological (Hobson, 1997) and neurochemical (Jacobs, 1978) similarity between dreaming and psychosis have led numerous researchers to propose that various psychological symptoms result from an intrusion of dreaming into wakefulness (Dement et al., 1969; Gillin & Wyatt, 1975; Howland, 1997; Mahowald, Woods, & Schenck, 1998; Saucerman, 1997; Watson, 2001). It is therefore possible that the association between insomnia and other psychological symptoms in the present study can be explained by the intrusion of dreaming, or other phenomena usually restricted to sleep, into sleep-deprived wakefulness.

In addition, there are substantial short-term changes in emotional expression that accompany sleep deprivation. Some evidence for this comes from a meta-analysis that compared the effect sizes associated with changes in vigilance (i.e., reaction time) to changes in self-reported measures of emotion (Pilcher & Huffcutt, 1996). Although changes in vigilance have become the gold standard for differentiating sleep-deprived and rested individuals (Lim & Dinges, 2008), the coefficients associated with emotional changes were greater than those associated with vigilance changes. Specific examples of these emotional changes demonstrated that sleep-deprived individuals do not necessarily express more or less of a particular emotion, but are instead extremely labile in their emotions. Insomnia patients undergo weeks or months of chronic, partial sleep deprivation, which quickly produces effects equivalent to the effects associated with acute, total sleep deprivation (Van Dongen, Maislin, Mullington, & Dinges, 2003). Thus, the short-term diurnal effects of sleep deprivation might also explain symptoms of PTSD such as emotional reactivity and irritability.

Clinical Implications

In clinical populations, treating sleep problems that are comorbid with depression can be successful even without treating the depression directly (Lichstein, 2006; Stepanski & Rybarczyk, 2006). The results of the present study have similar implications for an at-risk population of combat veterans who might not have yet developed other psychological symptoms. That is, there is a possibility that the early treatment of insomnia might in fact *prevent* the onset of other psychological symptoms. For example, Harvey (2001) recommended advising those patients with previous depression or anxiety episodes to treat insomnia as a warning signal to make an appointment with their health care provider. Moreover, a review of prospective epidemiological research on insomnia revealed a pattern of results implicating insomnia as a risk factor in the development of depression and other psychological disorders (Taylor, Lichstein, & Durrence, 2003). As a whole, these studies highlight the importance of identifying and treating insomnia as a way to prevent subsequent psychological disorders.

Nightmares are another sleep symptom that can occur immediately after combat exposure without the diagnosis of another psychological disorder (Hublin, Kaprio, Partinen, & Koskenvuo, 1999), can be a predictor of PTSD at a subsequent time point (Creamer, O'Donnell, & Pattison, 2004), and often persist after treatment programs improve other PTSD symptoms (Forbes, Creamer, & Biddle, 2001). Thus, cross-lagged panel longitudinal designs could assess whether nightmares are stronger predictors of other psychological symptoms than the reverse. Such designs could also assess complex structural equation models with more than two time points or time points other than 4 and 12 months postdeployment. The interpretation of findings from the present research is that insomnia is a stronger predictor of other psychological symptoms than the reverse. However, this interpretation could change when examining veterans with chronic symptoms. For example, isolated insomnia symptoms immediately after deployment might contribute to the onset of the full psychological disorder, but that disorder might then increase the severity of the insomnia, resulting in a positive feedback cycle. This could indicate that the best model fit might depend on the timing of assessment postdeployment. Correspondingly, it would also be worthwhile to conduct assessments during the deployment cycle and to assess other complex models that do not have psychological symptoms as the only outcome.

While emphasis in the literature has been on comorbidity between insomnia and mood or anxiety disorders, perhaps researchers and clinicians should expand the investigation to other psychological problems such as impulse control and aggressive behaviors. In addition, examples of potentially salient unmeasured variables in the present study include genetic risk factors, predeployment and postcombat stressors, predeployment psychopathology, coping styles, social support, access to mental health care, substance abuse, levels of chronic sleep deprivation, and/or the presence of other sleep disorders. The inclusion of such variables in more complex causal models would build on the current study.

Limitations

One potential limitation of the present research was that the relationship between insomnia and depression/PTSD symptoms at Time 2 when controlling for the Time 1 outcomes was relatively small. This was almost certainly because of, in large part, the strong stability coefficients between the outcomes from Time 1-Time 2, coupled with the relatively long interval between the two assessments (8 months). Importantly, researchers have noted that even modest relationships can have applied significance in field settings for processes that are cumulative (Abelson, 1985; Bliese, Adler, & Castro, 2011). Distal outcomes associated with insomnia are almost certainly cumulative in that fragmented or disruptive sleep impacts a range of mental health processes over time. A two-wave longitudinal study, however, is not capable of fully capturing cumulative processes and, as such, will underestimate practical effects. We contend that interventions targeted to facilitate restorative sleep are likely to have a range of positive cumulative effects despite the relatively weak relationship based on two assessment periods.

A second limitation is that we had to eliminate some of the items in previously established measurement instruments because of their high level of skewness. Importantly, one advantage of structural equation modeling is that each item on a measure is considered an indicator of an underlying latent variable, and it is the latent variables that are related to one another. Given that all of the items of a specific measure are seen as representing the same latent variable, there is less of a concern that eliminating a small number of items biases the construct being measured. In addition, to address the problem with kurtosis we used robust statistical methods that have been found to perform well with data possessing this characteristic (Satorra & Bentler, 2001).

As with any type of survey-based research, a limitation is reliance on self-reported symptoms. Although mental-health related stigma is a concern in the military in general, the survey data collected in this study were not part of a screening or formal assessment process and responses are not documented in medical records and had no bearing on their military status. The confidential nature of the research data was described during the consenting process. Thus, we have no evidence that the participants systematically under-estimated or over-estimated their mental health symptoms. In addition, our goal was to examine the relationship between variables, not establish prevalence rates, so absolute rates of symptom reporting that might be influenced by stigma-related concerns are not the focus here. However, future research could include more in-depth measures of insomnia that assess severity as well as duration and frequency of insomnia episodes. Diary measures of insomnia and objective measures of insomnia such as actigraphy and structured clinical interview assessment of psychological symptoms would decrease the uncertainty of retrospective, self-report measures and improve the generalizability of the findings.

CONCLUSIONS

Finally, and particularly relevant for service members deployed to combat, a study assessing sleep disturbances in a deployed environment (Peterson, Goodie, Satterfield, & Brim, 2008) found approximately 75% of study participants rated their quality of sleep significantly worse than before deployment. In this study, sleep efficiency and sleep onset latency were in the range indicating insomnia. Moreover, there is evidence that good and poor sleepers (primary insomniacs without comorbid disorders) report experiencing similar numbers and types of stressful life events, but poor sleepers appraised the events as more stressful and perceived themselves as lacking control and unable to cope (Morin, Rodrigue, & Ivers, 2003). The NIH Consensus Report (2005) also emphasized the significant effects of insomnia on daily functioning and quality of life, to include performance and attendance at work. For service members in a deployed environment, the consequences of insomnia might not affect only performance and quality of life, but also lead to the development of additional psychological problems and to impaired stress appraisal and coping at a time when these skills are most needed.

Acknowledgments

The findings described in this article were collected under a Walter Reed Army Institute of Research Protocol.

The views expressed in this article are those of the authors and do not necessarily represent the official policy or position of the U.S. Army Medical Command or the Department of Defense.

We thank Matthew Baker, Oscar Cabrera, Carl Castro (co-principal investigator), Deena Carr, Wanda Cook, Dave Cotting, Anthony Cox, Nadia Kendall-Diaz, Rachel Eckford, Steven Franco, Nikolas Hamilton, Dennis McGurk, Julie Merrill, Lyndon Riviere, Angela Salvi, Kyle Shaul, Christina Terra O'Neil, Steven Terry, and Allison Thompson for their work on this study.

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