Behavioral Sleep Medicine, 15:39–52, 2017

ISSN: 1540-2002 print/1540-2010 online
DOI: 10.1080/15402002.2015.1065413

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Correlation of Disturbed Sleep and Cancer Stress

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To illuminate the course of insomnia in the presence of an acute comorbidity, we examined the association between insomnia severity and breast cancer symptom severity over time and determined if this association varies with insomnia history and presleep arousal. Twenty-nine newly diagnosed breast cancer patients, who also exhibited insomnia, completed sleep diary and cancer symptom severity questionnaires every other week (total of 28 days) over 7 weeks, as well as baseline and postobservation measures. Participants were defined as having insomnia prior to cancer (IPC) or insomnia secondary to cancer (ISC) based on precancer sleep status. Insomnia and cancer symptom severity were strongly correlated at baseline but significantly declined over the evaluation period. Among ISC individuals, there was an association between sleep severity and cancer severity at baseline but not 7 weeks later. IPC individuals showed a consistent pattern of no significant association between sleep severity and cancer symptom severity. IPC had higher levels of cognitive presleep arousal than ISC. The current study documented the evolution of the relation between insomnia and breast cancer symptom severity over time and identified factors (premorbid insomnia and presleep hyperarousal) that may influence this association.

The rate of insomnia in the presence of an intrusive comorbidity, such as cancer (Savard, Simard, Blanchet, Ivers, & Morin, 2001), chronic pain (Finan, Goodin, & Smith, 2013), or depression (Franzen & Buysse, 2008), is oftentimes fivefold or greater than the rate observed in the general population, implying some fashion of causal influence is at play. But seemingly insurmountable methodological problems impede drawing clear conclusions regarding the direction and magnitude of causality. One methodological strategy that could be used in the service of analyzing comorbid associations consists of correlating the course of abrupt onset of a

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compelling medical or psychiatric condition with that of sleep. The present study adopted such an approach in the hopes of illuminating the evolution of comorbid insomnia.

Sleep disturbance is a significant and commonly reported problem associated with the diagnosis and treatment of cancer (Ancoli-Israel et al., 2006; Savard et al., 2001; Sharma et al., 2012) and studies have linked poor sleep with numerous health, treatment, and psychosocial factors in cancer patients (Bardwell et al., 2008; Palesh et al., 2007; Sharma et al., 2012). Compared across cancer diagnoses, breast cancer may have the highest prevalence of sleep difficulties (Davidson, MacLean, Brundage, & Schulze, 2002; Savard, Simard, Ivers, & Morin, 2005), and research on distress surrounding the onset and early treatment of breast cancer indicates that insomnia is a highly prevalent (as high as 66% of patients studied) and persistent problem for newly diagnosed individuals (Boehmke, 2004; Cimprich, 1999; Liu et al., 2009).

For individuals with co-occurring cancer and insomnia, almost half report that the onset of their sleep difficulties occurred at or shortly after the diagnosis of cancer (Davidson et al., 2002; Savard, Simard, Hervouet, et al., 2005), with many more reporting that their symptoms began within one month post cancer diagnosis following radiation therapy (Savard et al., 2001). Either poor sleep or excessive sleepiness, often instigated by intrusive cancer treatment, can arise any time during the course of the disease (Davidson et al., 2002; Savard et al., 2001), but for most patients, cancer-related sleep difficulties emerge even before cancer treatment is begun (Ancoli-Israel et al., 2006; Liu et al., 2009). Roughly 50% of patients report that their cancer experience either caused or exacerbated their insomnia symptoms, and most commonly attribute their insomnia symptoms to stress, worry, pain or discomfort, concerns about their health and family, and the physical effects of cancer (Davidson et al., 2002; Savard et al., 2001).

For a significant number of individuals with cancer and insomnia, their insomnia symptoms persist following the end of cancer treatment (Alfano et al., 2011; Lindley, Vasa, Sawyer, & Winer, 1998; Sharma et al., 2012). However, the fact that not all individuals experience residual insomnia symptoms illustrates the complex nature of comorbid insomnia. If comorbid insomnia is purely a symptom or consequence of the breast cancer experience, then the onset and resolution of the insomnia symptoms should mirror the course of breast cancer treatment. Insomnia that persists beyond the resolution of the breast cancer is functioning independently of the cancer and may be perpetuated by other factors.

One factor that has been shown to predict chronic insomnia following the treatment and resolution of breast cancer is a history of insomnia prior to the onset of cancer (Klink, Quan, Kaltenborn, & Lebowitz, 1992). One study found that previous insomnia was the only significant predictor of chronic insomnia symptoms following hospitalization for cancer-related procedures (Griffiths & Peerson, 2005). Hyperarousal also has been shown to contribute to the persistence of insomnia symptoms in women with breast cancer. Women who develop persistent sleep problems following breast cancer treatment show higher levels of sleep-related cognitive arousal (Griffiths & Peerson, 2005; Taylor, Espie, & White, 2003). Individuals with a previous insomnia history are more likely to experience sleep disturbance that is influenced by factors other than the co-occurring cancer, such as hyperarousal, than individuals without a prior history of insomnia.

The current study aimed to explore the association between sleep and breast cancer symptom severity during the first three months following cancer diagnosis, a time period when sleep problems originate for a majority of women with breast cancer. It was hypothesized that initially there is a significant association between insomnia severity and breast cancer symptom severity, but that this

association will abate over time as self-perpetuating influences accumulate. This study also sought to examine the impact of premorbid insomnia history and presleep hyperarousal, two factors associated with chronic insomnia, on the association between insomnia severity and breast cancer symptom severity. This is accomplished through two avenues: (a) comparing the association between insomnia severity and breast cancer symptoms severity separately for individuals with a history of insomnia prior to the cancer diagnosis, labeled insomnia prior to cancer (IPC), and individuals whose insomnia started after their diagnosis, labeled insomnia secondary to cancer (ISC), and (b) comparing levels of presleep physical and cognitive hyperarousal for both IPC and ISC groups. We hypothesized that individuals with a history of insomnia will exhibit a weaker association between insomnia severity and breast cancer symptom severity, and that they will also have higher levels of presleep hyperarousal that had presumably been well-established in maintaining their insomnia.

MATERIAL AND METHODS

Participants

Participants were recruited from a pool of newly diagnosed breast cancer patients ages 40–70. This age range captures the preponderance of breast cancer patients (Jemal et al., 2008), and also blunts insomnia confound from the force of aging effects (Foley, Monjan, Simonsick, Wallace, & Blazer, 1999; Morgan, 2000). Additional inclusion criteria were as follows: (a) within 6 weeks of their initial diagnosis of breast cancer; (b) no current diagnosis of another active sleep intrusive or unstable medical condition or sleep disorder (i.e., restless legs syndrome, sleep apnea); (c) satisfy International Classification of Sleep Disorders (ICSD) insomnia criteria at the time of the initial assessment (American Academy of Sleep Medicine, 2005), supplemented by empirically derived quantitative criteria requiring sleep onset latency or wake time after sleep onset to be greater than 30 min at least 3 times a week (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). To meet insomnia criteria, participants must have reported current sleep onset or maintenance disturbance (specified above) occurring at least three times a week, impaired daytime functioning attributed to poor sleep, and adequate opportunity for sleep. We did not use the diagnostic criterion that insomnia be present for one month. Because one of the goals of the study was to establish the nature of the association between insomnia symptoms and cancer symptoms as close to the cancer diagnosis as possible, it precludes the use of this diagnostic feature as a criterion for inclusion in the study. The presence of insomnia, daytime impairment, and other sleep disorders was determined through a semistructured interview.

We approached 55 cancer patients about the study, of whom 38 met criteria and were consented into the study. Of those 38 participants, 29 completed the entirety of the data collection procedure. Eight of the 9 dropouts outs gave no reason. They simply stopped responding to our attempts to contact them. One person expressed loss of interest in data collection. Participants derived no clinical benefits from participation and were given no compensation. It is not surprising that some decided to discontinue.

The course of cancer treatment for participants varied considerably. Most participants underwent surgery, either lumpectomy (n = 18) or mastectomy (n = 6). Most participants also underwent chemotherapy (n = 14) or radiation (n = 9) during the course of data collection, with six undergoing both. Unfortunately, we did not carefully track the correspondence between

timing or type of treatment and data collection for this study, but all participants commenced treatment prior to or during this study.

This research was approved by the University of Alabama and the DCH Regional Medical Center Institutional Review Boards.

Measures

Pittsburgh Sleep Quality Index (PSQI)

The PSQI was used to gain a measure of precancer sleep status. It is an effective one-time self-report measure of sleep patterns and quality of sleep over a one-month period (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Participants were instructed to complete this measure retrospectively to cover the month before they received the cancer diagnosis. A total score of 0–21 is possible when domain scores are summed, with a cutoff score of > 5 achieving specificity for a "poor" sleeper (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). The cutoff for primary insomnia was used because we were trying to establish the presence of insomnia prior to the cancer diagnosis. The PSQI has shown good reliability and validity as a measure of sleep quality and disturbance in a clinical sample of breast cancer patients (Carpenter & Andrykowski, 1998), though we cannot rule out memory bias contaminating these data (Harvey & Tang, 2012).

Insomnia Severity Index (ISI)

The ISI is a 7-item questionnaire designed to retrospectively measure perceived severity of insomnia symptoms for a period of two weeks (Morin, 1993). Items relate to difficulty falling asleep, difficulty staying asleep, early morning awakenings, level of dissatisfaction with current sleep, level of interference of disturbed sleep on daytime functioning, level of distress caused by poor sleep, and degree to which other people notice their poor functioning. Specifically within breast cancer populations, the ISI shows high internal consistency (.91) and correlates highly with sleep diary data (Savard, Savard, Simard, & Ivers, 2005).

Pre-Sleep Arousal Scale (PSAS)

The PSAS is a measure of physical and cognitive arousal symptoms that people may experience during the time just prior to sleep onset (Nicassio, Mendlowitz, Fussell, & Petras, 1985). It contains 16 items, 8 pertaining to each physical and cognitive arousal component, rated on a 5-point Likert scale. The PSAS yields two separate measures of arousal, with scores ranging from 8 to 40. It shows good internal consistency for both the physical (.84) and cognitive (.67) components in individuals with insomnia and correlates well with other measures of sleep and arousal (Nicassio et al., 1985). Cancer patients have been shown to score higher on the cognitive arousal subscale than the physical arousal subscale both at 2 months and 14 months post diagnosis (Taylor et al., 2003).

Functional Assessment of Cancer Therapy—Breast Symptom Index (FBSI)

Participants assessed their breast cancer symptom severity using the FBSI breast cancer symptoms survey (Yost et al., 2005). The FBSI is composed of eight Likert scales, which

measure daily symptoms associated with breast cancer diagnosis and treatment over a seven-day period. The FBSI items tap into physical symptoms, such as pain, nausea, lack of energy, and fatigue, as well as social, emotional, and functional well-being. Each item is specifically tailored to symptoms most frequently reported by individuals with breast cancer (Yost et al., 2005). The FBSI short form has been shown to have adequate reliability (.90 internal consistency; .85 test–retest), validity, and sensitivity to change (Brady et al., 1997; Yost et al., 2005). This measure was used both as a seven-day and a 24-hour assessment tool in the current study.

Sleep Diary

A sleep diary is a subjective measure of daily sleep (given in Lichstein, Durrence, Riedel, Taylor, & Bush, 2004). It yields estimates of a number of sleep parameters including sleep onset latency (SOL), number of awakenings during the night (NWAK), and the amount of wake time after sleep onset (WASO). Other sleep measures are derived from the diary: total sleep time (TST) and sleep efficiency (SE, percentage of time in bed spent sleeping). The sleep diary measures and their definitions conform to recommended standards (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006).

Procedure

Participants were recruited by their first or second appointment at the cancer center, as gaining contact with them close to their diagnosis was crucial. Participants either received their cancer diagnosis at a previous cancer center appointment or from a surgeon. As part of their cancer center visit, potential participants were approached by a cancer center employee, most frequently the physician, to determine their initial interest. If they assented, the study was described in detail. Written consent was obtained from interested participants, and they were then briefly screened to determine if they met study criteria. If they met criteria, they completed the baseline battery of questionnaires, which included the PSQI, the ISI, the FBSI seven-day version, and the PSAS.

Following the interview, participants underwent a seven-week data monitoring phase, during which data were collected every other week for a total of 28 days of sleep diary and breast cancer severity ratings. We expected that (a) seven weeks would be adequate to test our hypotheses, (b) asking these individuals to record data every day for seven weeks would be perceived as unduly burdensome, and (c) data collection in alternate weeks would be adequate for our purposes. Participants completed these ratings in four seven-day increments, which were interspersed with three seven-day periods where they were not completing the ratings. Each morning during the data collection phases, participants answered questions regarding their previous night's sleep and the previous day's cancer-related symptoms using the sleep diary and the FBSI 24-hour version.

After they completed this data collection phase, they completed a brief postassessment battery that included the ISI, FBSI seven-day version, and the PSAS. Participants were given the option of completing their daily records using paper questionnaires or online. About half chose the electronic data collection option (n = 14), and for those that did not choose this option, the primary reason cited was lack of computer or e-mail access. The timeline for administering all baseline, data monitoring, and postassessments is given in Table 1.

To identify IPC and ISC groups, participants were clustered based on the presence or absence of precancer sleep disturbance as determined by the PSQI. The baseline PSQI described sleep for

TABLE 1
Timeline for the Administration of Assessment Instruments

	Data collection time points							
Baseline assessment	*Week 1*	*Week 2*	*Week 3*	*Week 4*	*Week 5*	*Week 6*	*Week 7*	*Postassessment*
PSQI ISI FBSI PSAS	Daily sleep diary and FBSI		Daily sleep diary and FBSI		Daily sleep diary and FBSI		Daily sleep diary and FBSI	ISI FBSI PSAS

Note. PSQI (Pittsburg Sleep Quality Index), ISI (Insomnia Severity Index), FBSI (Functional Assessment of Cancer Therapy-Breast Symptom Index), PSAS (Pre-Sleep Arousal Scale).

the one-month time period directly prior to the participant's cancer diagnosis. Individuals with scores of 6 or higher were labeled IPC and those with 5 or lower were labeled ISC. We were interested in comparing the role of premorbid sleep disturbance versus contemporaneous onset of poor sleep with cancer, and though using PSQI for this purpose could not be considered a fine-grained insomnia diagnosis, it was the basis for making this distinction.

RESULTS

As this is an exploratory study, multiple statistical comparisons were conducted using a variety of techniques to best determine the associations present in the data. To balance Type I and Type II error, statistical results were reported without correction for multiple comparison. These guidelines conform to other exploratory studies in sleep and cancer (Ancoli-Israel et al., 2006).

Descriptive Statistics

A total of 29 participants were included in the study. Nine participants were African American and 20 participants were Caucasian American. All 29 participants met criteria for insomnia at the initial interview, excluding the duration criterion. One participant reported a history of restless legs symptoms, but stated that she had not experienced them in over two years, so she was included in the study. No other comorbid sleep diagnoses were reported by the other participants. Four participants reported using some sort of sleep medication to help their sleep at night, with one reporting the use of a prescription (alprazolam) and the others reporting use of over-the-counter medication. Out of the 110 nights of data for these four individuals, 40 (36%) of them were nights when medication was used. For the 29 participants, 14 (52%) reported having other medical problems, with the most common problem being high blood pressure. Of the women that reported other medical problems, 5 (36%) reported having two additional medical problems and 4 (29%) reported having three additional medical problems. Women with additional medical problems did not differ significantly from women without additional medical problems on (a) age, $t(27) = 0.75$, *ns*, (b) days between diagnosis and their initial assessment, $t(27) = 1.53$, *ns*, (c) baseline ISI scores, $t(27) = 1.32$, *ns*, or (d) baseline FBSI scores, $t(27) = 0.72$, *ns*. Means and standard deviations for baseline characteristics and measures are presented in Table 2.

TABLE 2
Means (*SD*) of Baseline Characteristics and Measures for the Overall Sample and for IPC and ISC Groups

	Overall Sample	*Precancer diagnosis insomnia status*	
Baseline characteristics and measures	*(n = 29)*	*Insomnia secondary to cancer (ISC) (n = 12)*	*Insomnia prior to cancer (IPC) (n = 17)*
Age	54.7 (9.29)	53.9 (8.59)	55.2 (9.99)
Days since diagnosis	28.0 (12.69)	29.67 (14.24)	16.82 (11.79)
ISI	7.7 (6.51)	3.92 (4.06)	10.41[a] (6.66)
FBSI	9.1 (6.90)	5.0 (3.69)	12.0[a] (7.25)
Cognitive presleep arousal	19.0 (9.00)	13.3 (5.15)	23.1[a] (8.98)
Physical presleep arousal	12.1 (3.90)	10.9 (4.01)	12.9 (3.64)
PSQI	6.7 (4.55)	2.6 (1.5)	9.6[b] (3.57)

Note. PSQI: Pittsburg Sleep Quality Index. ISI: Insomnia Severity Index. FBSI: Functional Assessment of Cancer Therapy-Breast Symptom Index.
t-tests were performed to determine differences between ISC and IPC groups for all baseline characteristics. [a]$p < .01$, [b]*t*-test not performed as this was the measure used to define IPC and ISC groups.

Association Between Insomnia Severity and Breast Cancer Symptom Severity

The association between insomnia severity, as measured by the ISI, and breast cancer symptom severity, as measured by the FBSI seven-day version, was assessed at baseline as well as at the postdata collection period. Insomnia severity was significantly correlated with breast cancer symptom severity at baseline, $r(27) = .57$, $p < .01$. At the postevaluation assessment seven weeks later, this relation was no longer significant, $r(27) = .31$, *ns*.

To examine changes in the association between breast cancer symptom severity and insomnia severity, correlations between FBSI scores and different sleep parameters were computed daily for each of the 28 days of data collection. Given some missing data, a total of 26 correlations were calculated separately between the FBSI and each of five sleep parameters: SOL, NWAK, WASO, TST, and SE. These correlations represent the association between daytime cancer-related symptoms and sleep symptoms the following night across all 29 participants for each day of data collection. These five sets of correlations were defined as outcome variables and were then submitted to five separate regression analyses with time (26 days) as the predictor variable. Table 3 shows the daily correlations along with the parameters for the best-fit regression line for each of the five sets of correlations.

Results show there was a significant change in correlation between FBSI with WASO and NWAK over time. For both sets of correlations, there was a significant decrease in strength of a positive correlation over time. A scatter plot of data points for the correlation between FBSI and WASO with the regression line is presented in Figure 1 to more clearly document this unfolding.

If either sleep disturbance or stress (as measured by ISI and FBSI) diminished significantly from baseline to postassessment, this could affect the correlational analyses described above. To assess for the presence of this potential confound, we tested change in ISI and FBSI from baseline to post to inquire if reduced strength in either of these from baseline could account for the declining ISI-FBSI correlation over the approximate two months. For all participants combined, there was no significant change either in ISI [baseline mean (*SD*) = 7.72 (6.51),

TABLE 3
Regression Parameters for the Correlations of FBSI with SOL, NWAK, WASO, TST, and SE Regressed Over Time (n = 26)

Day[b]	*Correlation between FBSI and SOL*	*Correlation between FBSI and NWAK*	*Correlation between FBSI and WASO*	*Correlation between FBSI and TST*	*Correlation between FBSI and SE*
1	–0.13	0.02	0.33	0.17	0.01
2	0.05	0.36	0.47	0.03	–0.17
3	–0.16	0.29	0.16	0.21	–0.04
4	–0.11	0.32	0.48	0.28	–0.16
5	0.13	0.36	0.41	–0.28	–0.44
6	–0.19	0.23	0.57	0.00	–0.30
7	0.12	0.16	0.42	0.04	–0.36
8	0.24	0.31	0.33	–0.15	–0.41
9	0.08	0.32	0.30	–0.03	–0.16
10	–0.10	0.29	0.40	0.00	–0.19
11	–0.14	0.24	0.54	0.07	–0.31
12	–0.01	0.34	0.22	–0.01	–0.14
13	0.10	0.17	0.57	–0.14	–0.39
14	0.04	0.16	0.27	–0.04	–0.25
15	–0.15	–0.03	0.54	–0.17	–0.36
16	0.23	0.10	0.16	0.03	–0.36
17	0.13	0.30	0.59	0.08	–0.29
18	0.09	–0.04	0.07	–0.01	–0.29
19	–0.17	–0.11	0.10	0.21	–0.07
20	0.01	0.08	0.08	–0.07	–0.11
21	–0.20	0.08	0.26	0.26	0.03
22	–0.13	0.12	0.12	0.32	0.04
23	0.37	–0.24	0.33	–0.03	–0.53
24	0.11	0.26	0.08	0.34	–0.15
25	–0.22	–0.09	0.12	0.11	–0.03
26	0.12	0.05	0.36	0.00	–0.28
Regression parameters					
B	.003	–.013	–.012	.005	.002
SE(B)	.004	.003	.004	.004	.004
β	.144	–.621[a]	–.531[a]	.271	.090

FBSI = Functional Assessment of Cancer Therapy–Breast Symptom Index, SOL = sleep onset latency, NWAK = number of awakenings, WASO = wake time after onset, TST = total sleep time, SE = sleep efficiency; [a]$p < .01$; [b]Day 27 and 28 were omitted due to missing data.

post = 8.21 (6.33), $t(28) = 0.56$, *ns*] or FBSI [baseline mean (*SD*) = 9.10 (6.90), post = 9.90 (7.59), $t(28) = 0.75$, *ns*)]. Similarly, no significant change for either measure occurred in either (a) the IPC [ISI baseline mean (*SD*) = 10.41 (6.66), post = 10.35 (6.88), $t(16) = 0.05$, *ns*, FBSI baseline mean (*SD*) = 12.00 (7.25), post = 11.53 (8.75), $t(16) = 0.34$, *ns*] or (b) the ISC [ISI baseline mean (*SD*) = 3.92 (4.06), post = 5.17 (4.00), $t(11) = 0.88$, *ns*; FBSI baseline mean (*SD*) = 5.00 (3.69), post = 7.58 (5.04), $t(11) = 1.65$, *ns*] subgroups.

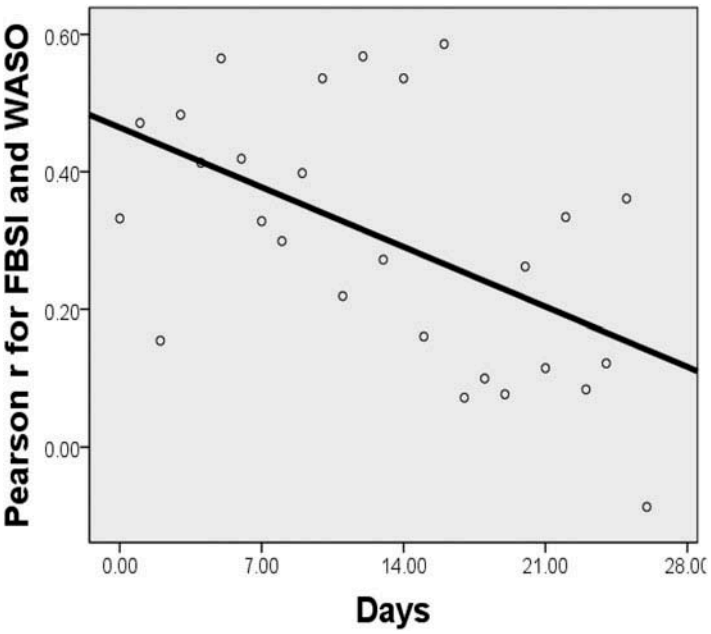

FIGURE 1 Regression line for daily correlations between Functional Assessment of Cancer Therapy–Breast Symptom Index (FBSI) and wake time after sleep onset (WASO) across 26 days distributed over seven weeks.

Differences Between IPC and ISC

To examine differences between IPC and ISC, *t*-tests were completed (see Table 2). Comparisons for age, physical presleep arousal, and the number of days between diagnosis and starting the study separately were nonsignificant. There were significant differences between ISI scores, $t(27) = 3.00$, $p < .01$, cognitive presleep arousal, $t(27) = 3.42$, $p < .01$, and FBSI scores, $t(27) = 3.07$, $p < .01$. IPC showed significantly higher levels of insomnia severity, cognitive presleep arousal, and breast cancer symptom severity than ISC at baseline.

To examine differences in the association between insomnia and breast cancer symptom severity for IPC and ISC, correlations between insomnia severity and breast cancer symptom severity were explored separately for both groups. There was a significant association between insomnia severity and breast cancer symptom severity at baseline for ISC, $r(10) = .60$, $p < .05$. This was not true for the IPC group, $r(15) = .39$, *ns*. At the postevaluation assessment, insomnia severity and breast cancer symptom severity were not significantly correlated for either ISC, $r(10) = .34$, *ns*, or IPC, $r(15) = .34$, *ns*.

DISCUSSION

There was a clear positive association between insomnia and breast cancer at the beginning of monitoring not long after establishment of the cancer diagnosis, but the strength of this association sharply dropped off after about a month of monitoring (see Figure 1). For the overall

sample, insomnia severity and breast cancer symptom severity were significantly correlated at the initial assessment point, but not at the two-month postassessment. Daily correlations between breast cancer symptom severity and both WASO and NWAK (but not other sleep parameters) exhibited a decrease in correlation strength over time. This evidence suggests that insomnia and breast cancer symptom severity may be linked during the initial stages of cancer detection and treatment, and that this connection weakens over a month or two.

There were also clear differences in the pattern of associations between insomnia severity and breast cancer symptom severity for IPC and ISC groups. ISC participants showed a significant connection between insomnia severity and breast cancer symptom severity at baseline, and IPC individuals did not. For ISC participants, this association was no longer significant at the two-month follow-up assessment, providing further evidence that the correspondence between insomnia and cancer symptom severity showed a decreasing trend over time.

In general, people with insomnia prior to their cancer tended to have higher levels of insomnia severity and breast cancer symptom severity. This may reflect the additive effects of stress on sleep disturbance. IPC individuals also had significantly higher levels of cognitive presleep arousal than ISC individuals, as we predicted. Individuals with previous insomnia symptoms displayed more cognitions and behaviors related to chronic insomnia concordant with previous research on premorbid insomnia, hyperarousal, and cancer (Griffiths & Peerson, 2005; Taylor et al., 2003). There were no differences between IPC and ISC individuals on physical presleep arousal. This may indicate that at the beginning of the cancer experience, individuals both with and without prior insomnia symptoms still experience a negative impact on their sleep related to the onset of this new cancer stressor. This may be further evidence for the additive effects of stress on poor sleep.

Overall, these findings fit well into existing theories on the changing nature of insomnia as it relates to the factors that drive it. Spielman's model illustrates the potential mechanism for the transformation of insomnia symptoms from reactive to self-sustaining (Spielman, 1986; Spielman, Caruso, & Glovinsky, 1987). The model suggests that predisposing, precipitating, and perpetuating factors play a coordinated role in determining if an individual experiences acute or chronic sleep disturbance. In this model, precipitating factors, such as health, social, and financial provocation, act as the event that triggers the onset of insomnia symptoms and are more influential in acute insomnia (Bastien, Vallieres, & Morin, 2004). As time progresses, the influence of the precipitating event subsides, and perpetuating factors, such as worry about sleep and misguided compensatory behaviors, become the more dominant influence maintaining insomnia symptoms (Roth & Drake, 2004). As Spielman predicted, this suggests an evolution in the relation between insomnia and the co-occurring condition. Specifically, it appears that a shift occurs in the category of factors that fuel insomnia: the influence of the precipitating co-occurring condition is replaced by perpetuating factors such as cognitive hyperarousal and poor sleep hygiene. If this is the case, it would be expected that the association between insomnia severity and the severity of the co-occurring condition (e.g., breast cancer) would decrease over time, which we observed, and that there would be a greater presence of perpetuating factors (i.e., hyperarousal) in those whose insomnia is not as strongly related to the co-occurring condition, which we also observed. Though Spielman's model has been widely accepted for many years, these are some of the first data to provide empirical support. In the present circumstance, however, because we are not studying a static condition, it should be emphasized that this theoretical model may not cleanly map onto

cancer. Cancer and its treatments unfold a series of dynamic challenges over time, crafting waves of precipitants. Over the course of two months of monitoring, we hesitate to specify exact processes, given the unstable nature of the comorbidity.

This paper has resurrected, however briefly, the outdated term *secondary insomnia*, to emphasize the point that psychologically or physically challenging agents take their toll on sleep in the short run consistent with the writing of Spielman and others, and consistent with the findings we report. Theories of secondary insomnia, claiming that an intrusive disorder asserted causal control of the sleep disturbance, ruled insomnia thought for many years (e.g., Tan et al., 1987; Walsh & Sugerman, 1989; Zorick, Roth, Hartze, Piccione, & Stepanski, 1981), although there were some early voices that questioned this position (Bootzin & Nicassio, 1978). More recently, many (Lichstein, 2000; Lichstein, McCrae, & Wilson, 2003; McCrae & Lichstein, 2001; State-of-the-Science Panel, 2005; Stepanski & Rybarczyk, 2006) have strongly criticized the concept of secondary insomnia. We know from philosophy of science that the mere co-occurrence of two disorders does not license assignment of causal attribution to either. Determining causal inference is a solemn process in science, and requires the presence of three conditions before declaring A causes B: A must precede B, A and B must covary, and alternative explanations must be ruled out (usually by random assignment to groups, including compelling control groups; Shadish & Sullivan, 2012). In the present study, the first two criteria hold for about a month, but the third is unaccounted for. Therefore, we cannot conclude without question that poor sleep was caused by cancer stress even in the first month of monitoring, although these data are consistent with the diagnosis of secondary insomnia to a greater extent than is usually present when this diagnosis is rendered. In any case, the strength of the association weakens presumably as the impact of the cancer stress wanes. If there is a transition from acute to chronic insomnia, it is likely due to emergent, potent perpetuating factors. If the insomnia was precipitated by the cancer, we assume it emerged about a month before we began monitoring. Therefore, we estimate it took about two months for the insomnia to disengage from its precipitant.

Our goal to catch the cancer diagnosis early to study the emergence of insomnia could not be met to our satisfaction. Characteristics of the health care system and respect for patient sensibilities introduced delay, but perhaps more importantly, the onset of cancer stress predates the formal diagnosis as patient apprehension stirs anxiety. In combination, if cancer stress did assert its influence on sleep, the sleep disturbance may have onset one or more months prior to our data collection. Furthermore, our method of identifying premorbid insomnia with a retrospective PSQI stretches the limits of accurate memory. Nevertheless, our approach to dichotomizing pre- and postcancer sleep disturbance did prove to be statistically useful.

The current study should be interpreted with caution given its exploratory nature and reliance on self-report data. In future studies, an objective measure of sleep, such as actigraphy, would add increased reliability to the measurement of sleep parameters. Breast cancer stage, treatment type, and treatment dose were not considered as possible exclusionary criteria, nor was there an attempt to use these as covariables. Surgery, radiation, and chemotherapy have all been shown to disrupt sleep throughout the course of treatment (Liu et al., 2009; Savard et al., 2001; Wang, Lee, Chang, & Lin, 2005). Though we recognize the weighty influence of cancer characteristics and treatments on sleep, their evaluation was beyond the scope of the current study.

Research in this area would be advanced by considering the following recommendations. The association between insomnia severity and breast cancer symptom severity should be studied throughout the entire breast cancer experience, from diagnosis, through treatment, and following treatment. This would allow for a fuller examination of the changes in this relation over time. It would also help determine if individuals who show a decrease in association between these factors are more likely to develop persistent insomnia symptoms beyond the resolution of the breast cancer treatment than individuals who maintain a strong association between insomnia severity and breast cancer symptom severity. Effort should be made to commence sleep-stress monitoring as soon after diagnosis as possible. Lastly, the stress experience of cancer patients varies by stage of cancer, treatment, and side effects, among many other psychosocial factors. Within the limits of patient tolerance of study procedures, assessing a wider range of psychological and physical variables may yield a more robust understanding of sleep experience. At the least, replication of the current findings is needed to bolster confidence in the applicability of the Spielman model to this population.

Despite the limitations, the primary goals of the study were met, which were to describe the evolution of correlated insomnia severity and breast cancer symptom severity and to examine differences in this association for individuals with and without factors associated with chronic insomnia. This study provided evidence of the changing nature of comorbid insomnia as it occurs, and also identified characteristics, namely premorbid insomnia and cognitive presleep hyperarousal, that may be related to the strength of the association between insomnia severity and breast cancer. This study may have important implications for how women with sleep disturbance and breast cancer are approached for sleep treatment and may lead to research that helps practitioners better understand when to intervene and where to focus their intervention.

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